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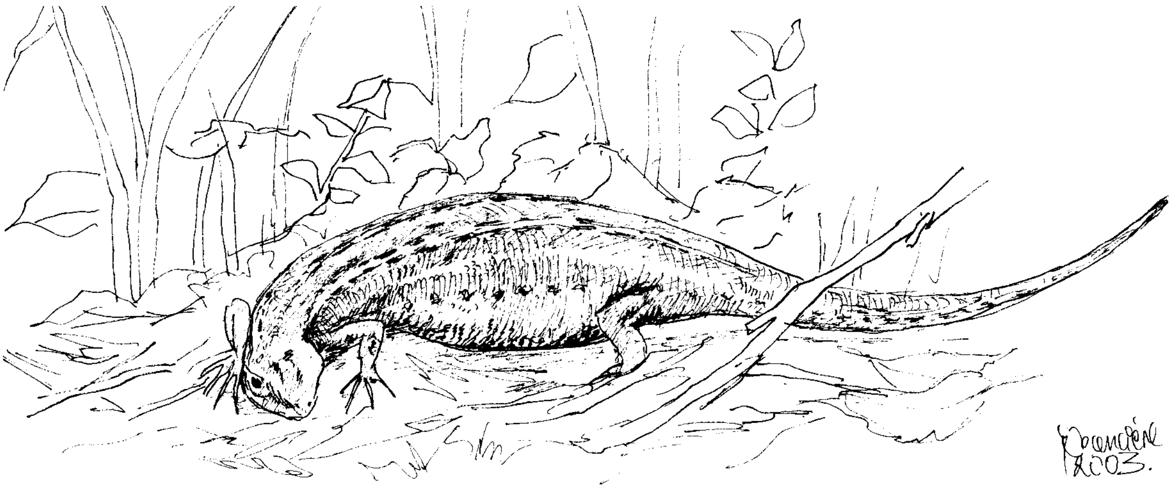
INTERACTIONS SOCIALES ET DISPERSION DANS DES POPULATIONS STRUCTUREES DANS L'ESPACE



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Pr Robert Barbault	**Université Pierre et Marie Curie**	**Président du jury**
Pr. Régis Ferrière	**Université Pierre et Marie Curie**	**Directeur de thèse**
Dr. Jean Clobert	**Université Pierre et Marie Curie**	**Co-directeur de thèse**
Dr. Xavier Lambin	**Université de Aberdeen, Ecosse**	**Rapporteur**
Pr. Nicolas Perrin	**Université de Lausanne, Suisse**	**Rapporteur**
Dr. Ophélie Ronce	**Université de Montpellier II**	**Examinatrice**
Dr Tom van Dooren	**Université de Leiden, Pays-Bas**	**Examinateur**

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RÉSUMÉ

L'hétérogénéité spatiale d'une population est engendrée à courte échelle par la portée limitée des interactions sociales et de la mobilité, à laquelle se superpose à plus longue échelle la fragmentation de l'habitat. Des facteurs intrinsèques (stochasticité démographique) et extrinsèques (fluctuations environnementales) génèrent alors de la variabilité spatio-temporelle à ces deux échelles. Cette thèse illustre certaines conséquences proximales et ultimes de cette hétérogénéité sur la dispersion et les interactions sociales.

A l'échelle du voisinage social, la stochasticité démographique permet d'ouvrir l'espace nécessaire à l'expansion d'une population altruiste favorisée par la sélection de parentèle. La variation génétique et démographique qui en résulte rend aussi possible l'évolution de certaines stratégies de dispersion : évitement des interactions avec la mère, de la consanguinité, ou de la compétition pour les ressources, et préférence pour les habitats denses. Elle accroît la valeur sélective de la dispersion par la colonisation de nouveaux habitats. Interactions sociales et dispersion se retrouvent alors associées dans un processus éco-évolutif qui résume les tensions entre coopération et compétition locale.

Mots-clés : dispersion, compétition, coopération, dynamiques adaptatives, hétérogénéité spatiale, valeur sélective.

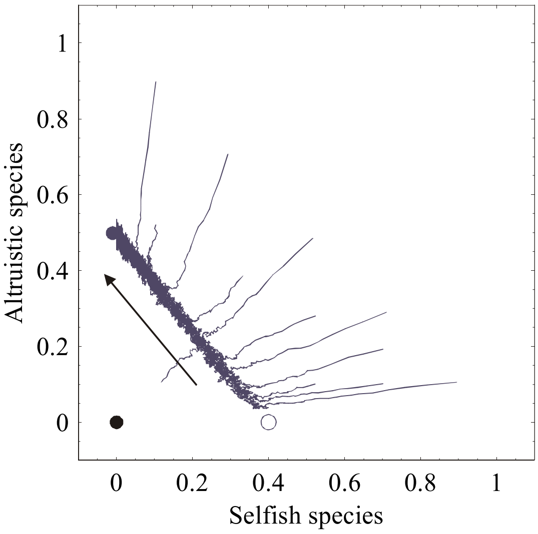
SUMMARY

Population spatial heterogeneity arises at small scales from limited interaction and dispersal ranges and additionally at larger scales from habitat fragmentation. Intrinsic (demographic stochasticity) and extrinsic (environmental fluctuations) factors then combine to generate spatio-temporal variability at these two scales. In this thesis, I illustrate some proximal and ultimate consequences of this heterogeneity on dispersal and social interactions.

At the scale of a social neighbourhood, demographic stochasticity is sufficient to provide the empty space necessary for the spread of an altruistic population favoured by kin selection. Stochasticity also gives rise to a genetical and demographic variation which allows the evolution of some dispersal strategies: avoiding competitive interactions with the mother, inbreeding, or resource competition, and cueing on population density. Fluctuations increase the fitness of dispersal through the colonization of new habitats. Social interactions and dispersal are then entangled in an eco-evolutionary process which summarises the tensions between cooperation and competition.

Keywords : dispersal, competition, cooperation, adaptive dynamics, spatial heterogeneity, fitness.

INTRODUCTION GENERALE



INTERACTIONS

INTERACTIONS SOCIALES

La vie, c’est l’interaction

Un jeune oisillon piaille comme un fou à l’arrivée de son parent et s’accapare toute la nourriture. Une mangouste suricate allaite les petits de sa sœur, puis surveille l’entrée du terrier communal. Elle a faim et attend avec impatience un relais qui tarde tant à venir. Suite à un conflit entre la vieille lionne et l’une de ses filles, la jeune fille doit quitter la troupe de sa mère pour fonder son propre groupe. Dans une prairie d’apparence si paisible, des plantes se combattent en utilisant à foison des armes chimiques libérées par leurs racines et en étouffant leurs voisines de leur ombrage ramifié. Pour le biologiste de l’évolution André Adoutte, la vie pouvait se résumer à trois concepts fondamentaux : un concept d’unité, le support génétique de l’information ; un concept de diversité, la capacité de réplication du support génétique ; et un concept de fonctionnalité, le métabolisme cellulaire. Mais que seraient l’oisillon, la mangouste, la lionne ou cette plante sans toutes ces capacités d’interactions sociales ? Pas grand chose à vrai dire. La vie, c’est donc aussi l’interaction sociale.

Interactions sociales, démographie et évolution

Les interactions sociales siègent au cœur du fonctionnement de toute population et de l’évolution adaptative de tout système biologique. La dynamique de la population est le résultat de l’intégration des effets des interactions sociales individuelles entre congénères et entre espèces sur les paramètres démographiques fondamentaux que sont la mortalité, la natalité et la migration (Caswell, 2001). L’évolution résulte de l’impact de ces paramètres démographiques sur le succès d’une nouveauté génétique, mesurée par sa capacité à envahir la population de son ancêtre (Fisher, 1930; Frank, 1998; Metz et al., 1992; Michod, 1999).

Pour Darwin, la démographie et l’évolution sont modelées par la *compétition* (une interaction sociale négative d’un individu focal envers ses congénères) : compétition entre membres des deux sexes pour la survie, la *sélection naturelle* (Darwin, 1859), ou compétition entre membres du même sexe pour l’accès au partenaire ou l’attraction du partenaire, la *sélection sexuelle* (Darwin, 1871). L’importance de la compétition continue de fasciner les écologistes qui construisent des pans entiers de théories et d’expériences autour de ce concept, depuis l’effet de la compétition sur la dynamique de la population jusqu’au rôle de la compétition dans la diversification des espèces (e.g., Bjørnstad et Grenfell, 2001 ; Dieckmann et Doebeli, 1999).

A l’inverse, pour les écologistes Clements (1916) ou Allee (1949), populations et écosystèmes fonctionnent comme une unité mutualiste, les espèces et les individus étant assimilés à des organes qui se développent, coopèrent et meurent. Plus récemment, l'intérêt porté au rôle de la *coopération* (une interaction sociale positive d'un individu envers ses congénères) a été stimulé par l'étude de *l'altruisme social* (un comportement de coopération impliquant un coût d'investissement pour l'individu) de certains insectes, vertébrés et micro-organismes (voir Annexe 1), ainsi que par l'étude de « grandes transitions majeures » impliquant la coopération entre des entités indépendantes au sein du génome, de la cellule, de l'individu ou de la colonie sociale (Maynard Smith et Szathmary, 1995).

La séparation entre compétition et coopération n'est cependant pas si claire. D'une part, de nombreuses interactions sociales impliquent à la fois des éléments de coopération, par exemple pour la production de descendants, et de compétition, par exemple entre les jeunes produits de manière coopérative (West et al., 2002). D'autre part, certains contextes sociaux impliquent à la fois une tentation à la compétition et une tentation à la coopération. En effet, l'intérêt privé de l'individu coïncide rarement avec l'intérêt commun des partenaires impliqués dans une interaction sociale (van Baalen et Jansen, 2001). Par exemple, l'intérêt d'un poussin quémandant la becquée auprès de ses parents est d'obtenir une quantité de ressources maximale au détriment de ses frères et sœurs, alors que l'intérêt de ses parents est d'assurer un nourrissage homogène de la descendance.

Voisinage social

Toutes ces interactions sociales ont lieu dans un espace limité autour de chaque individu, que j'appelle le *voisinage social* de l'individu. Par exemple, chez les végétaux terrestres, la sphère racinaire détermine les limites d'interaction dans le sol entre plantes voisines, alors que la surface foliaire et la structure aérienne déterminent les limites d'interaction au-dessus du sol entre plantes voisines (Begon et al., 1996). Chez les animaux, les interactions vont aussi être limitées à un voisinage social qui dépend des capacités de mobilité et de perception des congénères (Wilson, 1975). **De fait, toute population possède un degré plus ou moins prononcé de structuration sociale, qui se reflète dans la distribution des voisinages sociaux autour de chaque individu au sein de la population**.

Prenons l'exemple du modèle biologique de cette thèse, le lézard vivipare *Lacerta vivipara* (voir Annexe 2). A la sortie de l'œuf, le jeune est autonome. Il va explorer et trouver ses ressources dans son habitat de naissance. Pour cet animal, le voisinage social va se construire en fonction (i) des décisions d'interactions sociales prises au cours de sa vie par l'individu ; (ii) des décisions d'interactions sociales prises au cours de leurs vies par ses proches voisins, comme ses frères et sœurs ; ainsi que (iii) d'inévitables variations démographiques, provoquées par la mort de proches congénères ou l'arrivée d'individus non familiers. Le voisinage social est ainsi caractérisé par une *variabilité temporelle*.

L'environnement social du jeune lézard n'est pas comme tout les autres. D'une part, il coïncide avec le milieu de vie de sa mère et le site de ponte qu'elle a choisie. Ce site contient sûrement quelques éléments particuliers du paysage, car la variabilité spatiale des propriétés physiques et démographiques de l'habitat est détectable à une échelle de deux ou trois domaines vitaux de distance (Massot com. pers.). D'autre part, ce site est celui de la famille du jeune lézard, en particulier de ses jeunes frères et sœurs, de sa mère et peut-être aussi de son père biologique. Un site sur lequel pourrait déjà résider une lignée familiale impliquant d'autres proches généalogiques (voir O'Connor et Shine, 2003). Le voisinage social de notre jeune lézard focal est donc caractérisé par des *éléments de ressemblance* qui le distinguent d'un voisinage aléatoirement choisi dans la population : il implique à la fois des proches généalogiques et le milieu de vie de ses proches généalogiques.

Tout voisinage social possède ainsi trois propriétés fondamentales : ses **propriétés généalogiques** (par exemple, le degré moyen d'apparentement entre individus : Frank, 1998) ; ses **propriétés écologiques**, comme la structure d'âge chez Ronce et al. (2000) ou la qualité de l'habitat chez McPeek et Holt (1992) ; et ses **propriétés ontogéniques**, c'est-à-dire sa dépendance à l'histoire de l'individu et de ses congénères.

LA DISPERSION

Bouger

Le jeune lézard a pris sa décision quelques jours après sa naissance. Vraiment, cet habitat ne lui convient pas, trop humide à son goût et vraiment surpeuplé. En plus, ses frères et sœurs lui marchent sur les pattes en permanence, et sa mère est omniprésente. Le goût de l'indépendance a pris le dessus, et notre jeune lézard se lance dans une longue marche de plusieurs dizaines de mètres. Quand il a enfin quitté le territoire maternel, il pose ses pattes à l'ombre d'une callune. Il ne lui restera plus que de vagues souvenirs du petit coin de la tourbière où sa mère a mis bas. Contrairement à tous ses frères et sœurs, il a préféré changer d'espace.

Processus

La dispersion est le comportement de mouvement d'un individu de son site de résidence vers un autre site d'établissement. Ce mouvement est caractérisé par trois étapes principales sur lesquelles vont opérer des mécanismes proximaux et ultimes différents : la décision de départ (ou *émigration*), la phase de *transfert* ou d'exploration, et la décision de fixation (ou *immigration*) dans un site d'arrivée (Ims et Yoccoz, 1997). La dispersion est aussi un trait de l'histoire de vie de l'individu, c'est-à-dire une décision fondamentale impliquant des allocations d'énergie et pouvant influencer le succès reproducteur de l'individu, au même titre que l'âge à la première reproduction ou la vitesse de croissance (Stearns, 1992).

La dispersion affecte la répartition spatiale des individus, et influence donc la structure généalogique, les propriétés écologiques et l'histoire du voisinage social d'un individu dispersant

et de ses proches congénères. La dispersion est ainsi une composante fondamentale de la structuration sociale de la population. **En retour, les règles et les pressions de sélection qui régissent les décisions de dispersion vont dépendre de la structure du voisinage social de départ, de transfert et d'arrivée de l'individu**. Dispersion et interactions sociales sont donc impliquées dans une boucle de rétroaction éco-évolutive dont l'élément de contrôle fondamental se situe dans la structure sociale et spatiale de la population (chapitre 1). Il devient donc naturel de considérer conjointement les interactions sociales et la dispersion. La dispersion et les interactions sociales vont affecter mutuellement la dynamique de la population (voir chapitres 1-3, et chapitre 5). L'évolution adaptative de la dispersion va dépendre de l'évolution adaptative des interactions sociales, justifiant une approche conjointe des deux problèmes (voir chapitres 2 et 3).

Capacités locomotrices et phase de transfert

La phase de transfert de la dispersion nécessite des capacités d'exploration et d'interaction relativement élevées. **Les capacités locomotrices apparaissent dans ce contexte comme de bons candidats pour mesurer la compétence d'un individu à explorer son voisinage social car elles traduisent des différences physiologiques et morphologiques se reflétant dans des activités quotidiennes** (voir Annexes 4 et 5). Les capacités de locomotion fournissent une mesure totale de la compétence d'un organisme à se déplacer lors de tâches écologiquement pertinentes (Bennett et Huey, 1990). Par exemple, le régime d'endurance maximale en laboratoire, une mesure de la capacité d'un animal à résister à un effort prolongé, est corrélé à l'activité comportementale *in situ* chez les lézards (Clobert et al., 2000; Garland, 1999).

HETEROGENEITE SPATIALE

Hétérogénéité spatiale à l'échelle de l'habitat et de l'individu

Les paysages écologiques sont structurés à de multiples niveaux, et ceci de manière extrinsèque au fonctionnement démographique de la population. En particulier, il existe dans le paysage des éléments favorables à l'espèce, les *fragments* de la population, et des éléments externes à ces fragments, la *matrice* de la population constituée de corridors ou d'habitats défavorables utilisés lors des mouvements entre fragments (Wiens et al., 1993). Cette structure spatiale peut prendre diverses formes selon les espèces (Hanski, 1998; Harrison et Taylor, 1997), ce qui se reflète dans la diversité des approches théoriques de la structure spatiale en écologie (Kareiva et Wennergren, 1995). Certaines populations fonctionnent comme une collection indépendante de petits fragments isolés par la distance, susceptibles de s'éteindre du fait de leur petite taille, et connectés par des flux d'individus. On parle alors d'une *métapopulation*, et un tel système peut être maintenu dans un équilibre d'extinction et de colonisation (Hanski, 1999; Levins, 1969). Certaines populations fonctionnent au contraire sous la forme de sites stables, produisant de grandes quantités d'individus (les sources), et de sites instables, qui perdent une grande quantité d'individus (les puits ; Pulliam, 1988). A plus large échelle, on peut aussi considérer que la population est constituée de fragments de tailles très dissemblables, une formulation traduite par la notion de systèmes *continents-îles* (Harrison et al., 1988 ; MacArthur et Wilson, 1967). Globalement, la différenciation entre ces structures spatiales dépend de l'espèce considérée, de la structure de son environnement et de l'échelle spatiale, mais aussi des exigences de précision de l'étude. Il n'existe donc pas une « bonne » approche de la structure spatiale : tout au plus l'approche la plus informative dans le contexte de l'espèce et de l'étude. Par exemple, peu d'espèces de petits mammifères semblent se conformer à un système de métapopulation *sensu stricto* (Harrison et Taylor, 1997; Lambin et al., 2003). Par contre, la théorie des métapopulations fournit des concepts utiles à la description et à la compréhension de ces populations, même si ce n'est qu'en première approximation.

Indépendamment de l'habitat, la distribution des individus au sein des fragments d'habitat est hétérogène si les capacités d'interactions sociales et de dispersion sont limitées par rapport à la taille des fragments. Par exemple, la distribution des plantes adultes dans un habitat homogène dépend de processus internes à la population, en particulier du voisinage de compétition et du voisinage de dispersion. La structure spatiale qui se développe dans une population régulée par des processus compétitifs est définie par la taille des voisinages de compétition et de dispersion (Dieckmann et Law, 2000; Law et Dieckmann, 2000).

Quand la taille des voisinages sociaux est très large, la distribution des individus au sein des fragments converge vers un *champ moyen*. Si les individus perçoivent leur environnement dans les limites de la taille des fragments de la population, alors les processus démographiques individuels dépendront directement de la moyenne globale de la composition démographique de la population (van Baalen, 2000). Cette approximation est vraisemblablement vérifiée dans les systèmes très fragmentés où la taille des fragments correspond à quelques territoires, comme ceux du pika américain (*Ochotona princeps*) à Bodie en Californie (Smith, 1980). Par contre, chez le papillon modèle de la théorie des métapopulations, le fritillaire de Glanville *Melitea cinxia*, les œufs de différentes femelles sont pondus sur une même plante hôte qui constitue ainsi une unité spatiale fondamentale pour les interactions entre frères et sœurs, et pour la compétition entre familles (Hanski, 1999). Cette unité spatiale a une taille bien inférieure à celle des plus petits fragments du réseau d'habitat de l'espèce, ce qui pose la question de la pertinence d'une approche reposant exclusivement sur l'étude et la comparaison de fragments d'habitat. Cette thèse illustrera un contexte dans lequel s'écarter de l'hypothèse en champ moyen apporte une solution à un problème d'évolution des interactions sociales (voir chapitres 1-3).

On conçoit donc que **toute population possède un degré d'hétérogénéité spatiale, qui se reflète dans la distribution des habitats de l'espèce et dans la distribution des individus au sein**

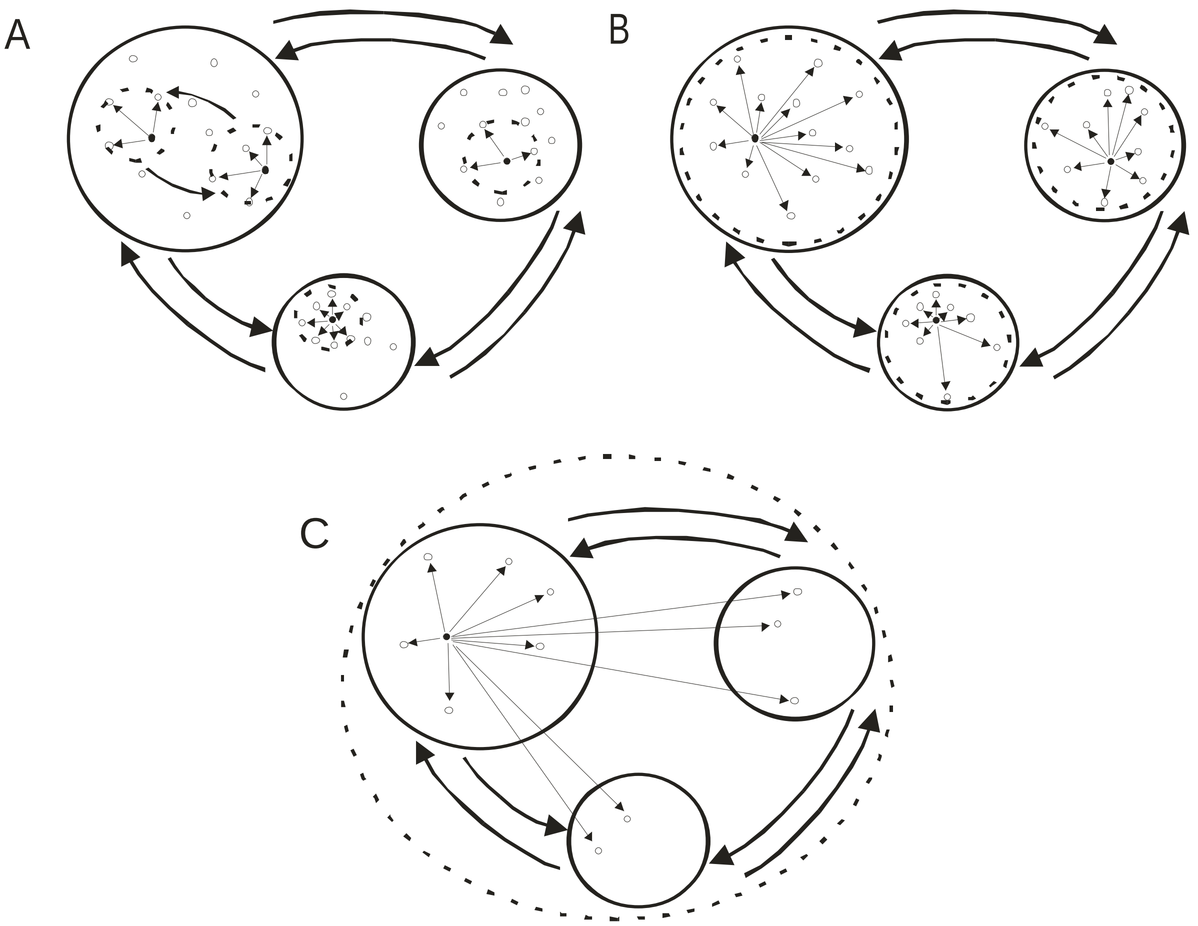
de ces habitats (Fig. 1). On parlera de *populations fragmentées* pour désigner une population structurée en fragments (chapitres 1, 4-6) et de *populations hétérogènes dans l'espace* pour désigner une population à distribution individuelle hétérogène au sein d'un espace continu (chapitres 1-3). Le terme *métapopulation* sera réservé pour désigner des populations fragmentées instables et typiquement asynchrones, tel que Levins (1969) l'a originellement conçu. Ces désignations ne présument pas de l'échelle spatiale de la fragmentation et de l'hétérogénéité ou de l'intensité de la variabilité spatiale.

Cependant, l'organisation réelle de toute population peut être organisée le long d'un continuum : depuis des systèmes très stables (e.g., les populations naturelles du lézard vivipare, Clobert et al., 1994) jusqu'à des systèmes très dynamiques (e.g., le fritillaire de Glanville, Hanski, 1998), depuis des systèmes synchrones vers des systèmes asynchrones (Bjørnstad et al., 1999), ou bien encore depuis des systèmes très structurés à des éléments continus du paysage (Wiens et al., 1993). Dès lors toute classification devient artificielle, et reflète avant tout le pragmatisme de l'approche et la sensibilité du problème au degré de structuration spatiale. Dans le cadre d'une étude de l'écologie et de l'évolution des interactions sociales et de la dispersion, la structuration de la population en voisinages sociaux peut difficilement être ignorée, ainsi que les travaux présentés dans cette thèse le démontrent. On peut alors considérer que la fragmentation de l'habitat vient se superposer à cette structuration sociale pour en affecter la dépendance à la démographie et à la génétique de la population (voir sections suivantes). Par contre, il me semble que la structuration du paysage n'est pas nécessaire à la description des phénomènes envisagés dans cette thèse, d'autant plus qu'elle nécessite de prendre en compte des éléments spécifiques à l'organisation spatiale d'une espèce (Wiens, 1989).

Structuration socio-spatiale

L'interaction entre la structuration sociale et spatiale génère trois organisations socio-spatiales fondamentales de la population (voir la Figure 1). Dans le premier cas, la taille du voisinage social est inférieure à la taille des fragments (Fig. 1A). Les fragments sont alors constitués d'unités sociales au sein desquelles sont déterminés les taux de mortalité et de natalité. La population est une collection de fragments qui échangent des individus. Cette double structuration permet la séparation de deux types d'évènements de dispersion : la *dispersion sociale*, qui a lieu entre les voisinages d'interaction, et la *dispersion d'habitat*, qui a lieu entre les fragments de la population. Dans le troisième cas, la taille du voisinage social est supérieure à la taille des fragments : le voisinage social implique alors des individus appartenant à plusieurs fragments et la dispersion d'habitat a lieu au sein du même voisinage social (Fig. 1C). Dans le cas intermédiaire, on retrouve l'organisation classique de la population fragmentée : la population est composée d'unités sociales correspondant aux fragments (Fig. 1B). Les interactions ont lieu de façon homogène entre tous les individus du même fragment, et les fragments échangent des individus par la dispersion.

Figure 1. Trois types de structuration socio-spatiale. Les individus sont formalisés par des petits ronds, les individus focaux étant indiqués en noir. Le voisinage social d'un individu focal est indiqué par un cercle pointillé. Les flèches droites connectent les congénères qui interagissent socialement avec l'individu focal. Les fragments d'habitat sont indiqués par des cercles pleins. Les flèches incurvées indiquent les mouvements individuels. A. Le voisinage social est structuré à une échelle inférieure à la taille du fragment : la population possède deux niveaux d'organisation. B. Le voisinage social coïncide avec la taille des fragments : la population possède un seul niveau d'organisation. C. Le voisinage social inclut tous les fragments de la population.



Considérons le cas de la Figure 1A que l'on pourrait appeler une *population fragmentée et structurée socialement*. Tout d'abord, natalité et mortalité ne dépendent pas ici directement de la structure démographique du fragment, mais de la structure des voisinages sociaux et de la répartition spatiale des individus au sein de ces voisinages (voir aussi chapitre 1). **Cette dépendance des évènements individuels à la structure du voisinage génère une variabilité spatiale, qui vient se superposer à la variabilité existant entre les fragments d'habitat**. Premièrement, la structure sociale impose des variations liées à la *fluctuation* aléatoire entre voisinages, même si les individus sont répartis de façon homogène dans l'espace (Durrett et Levin, 1994). En effet, on peut considérer chaque voisinage comme un échantillon aléatoire d'un nombre fini d'individus de la population

(Dieckmann et Law, 2000). Deuxièmement, si les individus sont répartis de façon hétérogène, la structure spatiale locale impose des variations liées aux *corrélations spatiales*. Le voisinage moyen autour d'un individu est en effet différent de l'environnement moyen en présence de corrélations spatiales. Ces fluctuations aléatoires et ces corrélations spatiales peuvent profondément affecter la démographie de la population (e.g., Harada et Iwasa, 1994; Law et Dieckmann, 2000; Morris, 1997; Rand, 1998; van Baalen, 2000). Par exemple, certaines stratégies démographiques utilisées par des plantes, comme l'exploitation, la colonisation, ou la tolérance, peuvent coexister parce qu'elles exploitent l'espace différemment dans une population hétérogène, mais pas dans un habitat strictement homogène. Une stratégie de colonisation utilise l'agrégation des autres stratégies pour exploiter les sites vides à l'aide d'une dispersion globale. Une stratégie d'exploitation utilise sa dispersion réduite et son avantage de croissance pour exploiter des sites occupés avant d'être remplacée. Une stratégie de tolérance utilise son agrégation et sa résistance à la compétition intraspécifique pour exploiter les sites occupés et les rendre imperméables aux autres stratégies (Bolker et Pacala, 1999).

De plus, la structure en voisinages sociaux engendre une redistribution de la variabilité généalogique, qui dépend du nombre de groupes sociaux impliqués, du mode de dispersion ou du régime de reproduction (e.g., Sugg et al., 1996). Prenons l'exemple de la musaraigne *Crocidura russula*, un mammifère insectivore à reproduction socialement monogame et à dispersion femelle (Favre et al., 1997). Cette espèce territoriale forme différents groupes de reproduction (en général, un jardin) au sein d'un même fragment d'habitat (en général, un village). La variabilité génétique d'une population suisse autour de Lausanne a été échantillonnée à ces deux échelles (Balloux et al., 1998). Les données génétiques mettent en évidence une structuration entre les villages, au sein des villages entre jardins pour les mâles et au sein d'un individu dans un jardin pour les mâles. Donc, la structuration génétique est présente à l'échelle du paysage et de la structure sociale au sein du paysage. La structure sociale produit un apparentement significatif entre voisins au sein d'un même fragment d'habitat pour le sexe philopatrique, et s'accompagne d'une consanguinité significative. Enfin, la taille efficace de la population (taille correspondant à une population idéale qui perdrait la variabilité génétique au même taux que la population d'étude) vaut environ 680 individus pour 140 reproducteurs, ce qui témoigne du rôle de la structuration sociale dans le maintien de la variabilité génétique.

Finalement, les voisinages sociaux sont une source de pressions de sélection qui peuvent conduire à l'évolution adaptative de combinaisons innovantes de traits d'histoire de vie, par exemple en favorisant l'émergence de comportements coopératifs familiaux (Emlen, 1997). Les conséquences évolutives des voisinages sociaux sont liées tant à leurs *propriétés écologiques* (chapitre 1), comme la variabilité spatiale, qu'à leurs *propriétés génétiques*, notamment la préservation d'unités d'individus apparentés (van Baalen et Rand (1998), chapitre 2).

POINTS DE VUE DE LA THESE

Ma thèse n’a pas l’ambition de répondre de manière générale aux problèmes posés par l’étude des interactions sociales et de la dispersion dans les populations structurées dans l’espace, de présenter un cadre théorique unifié des structures sociales et spatiales, ou d’en proposer une investigation approfondie et complète dans le cadre du modèle biologique d’étude. Au cours de ce travail, j’ai plutôt développé quatre points de vues complémentaires afin de décrire les effets de l’hétérogénéité et de la variabilité spatiale sur les interactions sociales et la dispersion. Les parties suivantes font une brève présentation des questions abordées dans cette thèse. Ces questions seront traitées en détail dans les chapitres 1 à 6 qui forment le corps du travail de la thèse. Des travaux complémentaires et des éléments de synthèse sont rassemblés dans les Annexes 1 à 5.

Hétérogénéité locale

J’envisage dans cette partie l’évolution de l’altruisme et de la dispersion dans une population hétérogène où les interactions sociales et la dispersion sont limitées aux territoires voisins. Je modélise une structure spatiale hébergeant des individus d’un organisme haploïde caractérisé par un phénotype d’altruisme et de mobilité. Je considère que les valeurs de ces phénotypes sont déterminés génétiquement et peuvent varier par mutation. Je décris le processus de sélection et le résultat de la dynamique adaptative de l’évolution conjointe de ces deux traits.

Les objectifs de la modélisation sont justifiés dans le chapitre 1 et dans l’introduction des chapitres 2 et 3. Le cadre théorique est fixé dans le chapitre 1, ainsi que dans les annexes des chapitres 2 et 3. Un point de vue général et un état des connaissances sur les comportements altruistes est donné dans l’Annexe 1. Il fournit le pesant biologique au déroulement du modèle théorique, et le place dans la perspective plus générale de l’étude de la coopération. Seuls les principes généraux du modèle et de sa construction sont présentés dans les paragraphes qui suivent.

La plupart des modèles d’évolution de l’altruisme et de la dispersion font deux hypothèses majeures : la structuration spatiale est envisagée globalement, c’est-à-dire que le paysage est composé de nombreux fragments connectés à longue distance par la dispersion (voir Fig. 1B), et la composition démographique des fragments d’habitats est donnée une fois pour toutes. Nous modéliserons au contraire une population structurée à une échelle locale et une démographie explicite, rendant compte du tirage aléatoire des évènements individuels de naissance, de mort et de mouvement. Par ailleurs, les problèmes de l’évolution de l’altruisme et de la dispersion partagent de nombreux points communs (chapitres 1, 3 et Annexe 1), mais sont classiquement étudiés séparément. Au contraire, nous envisagerons ici l’évolution conjointe de la dispersion et de la coopération. De plus, il est d’usage commun de considérer seulement deux stratégies d’interaction altruiste, comme Coopération et

Egoïste dans la théorie des jeux (Axelrod et Hamilton, 1981). Cette dichotomie recoupe mal la variabilité interindividuelle et interspécifique des comportements de coopération (voir Annexe 1). Au contraire, notre modèle suppose que l'altruisme peut se mesurer à l'échelle individuelle par l'investissement réalisé dans les interactions altruistes, et envisage de décrire les déterminants de la variation adaptative de cet investissement. Finalement, on tiendra compte de la diversité des coûts de la coopération (Heinsohn et Legge, 1999) en modélisant un coût accélérant (plus on investit dans l'altruisme, plus il sera coûteux d'investir plus), un coût linéaire (le coût d'un investissement supplémentaire est constant), et un coût décélérant (plus on investit dans l'altruisme, moins il sera coûteux d'investir plus).

En reprenant les prédictions verbales de Hamilton (1964) et les résultats théoriques de van Baalen et Rand (1998), on prédit une évolution continue de l'altruisme dans la population par un processus de mutation et de sélection de parentèle impliquant l'apparentement entre l'individu et ses proches voisins (Frank, 1998). Dans la mesure où le niveau adaptatif d'altruisme atteint dans la population dépend (i) d'interactions entre les bénéfices indirects de la coopération entre apparentés, les coûts indirects de la compétition entre apparentés et les coûts physiologiques directs de l'altruisme (West et al., 2002), et (ii) de l'effet rétroactif du niveau d'investissement dans l'altruisme sur ces pressions de sélection (Metz et al., 1996), on va s'attacher à décrire formellement ces processus. Cependant, on peut prédire qu'une mobilité réduite renforce l'apparentement, donc favorise probablement l'émergence de phénotypes plus altruistes (Hamilton, 1964). Par ailleurs, on va rechercher des processus permettant de franchir la barrière initiale d'invasion de l'altruisme liée à la disproportion des coûts physiologiques dans la situation où les coûts de l'altruisme décélèrent. Dans un deuxième temps, on va étudier les pressions de sélection contribuant à l'évolution de la mobilité individuelle dans ce type de population, et l'impact de l'évolution de la mobilité sur la persistance évolutive des interactions altruistes.

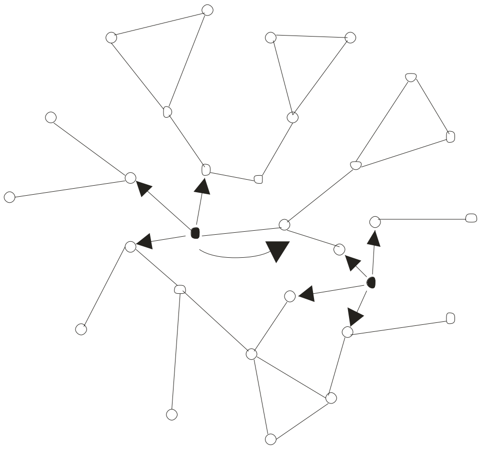


Figure 2. Structuration socio-spatiale du réseau envisagé dans le modèle théorique de la thèse (voir chapitres 1-3). Les liens entre sites voisins sont figurés par les traits fins et les sites par les ronds vides. Autour d'un individu focal (cercle plein), les interactions sociales (flèches droites) et la dispersion (flèche incurvée) sont limitées aux sites voisins.

Plus précisément, on envisage une structuration sociale qui prend la forme d'un voisinage de sites individuels connectés formant une structure globale en *réseau* (Matsuda et al. (1992), Fig. 2). Les interactions sociales et la dispersion ont lieu strictement entre sites voisins. Les liens de voisinage sont tirés aléatoirement entre les sites ou selon des règles qui dépendent de la proximité géographique (voir chapitres 1 et 2). Dans tous les cas, on supposera que le nombre de voisins est fixé *a priori*, dans la mesure où nos résultats ne sont pas fortement sensibles à cette hypothèse (Morris, 1997). On peut considérer ce réseau comme une métapopulation de sites discrets individuels, donc une structure spatiale où les interactions sociales et la dispersion sont limitées par la distance (comparer la Figure 2 à la Figure 1C). On peut aussi considérer ce réseau comme le résultat d'une hétérogénéité interne à un fragment d'habitat, donc une structure purement sociale (comparer la Figure 2 à la Figure 1A).

Par ailleurs, on suppose que l'état du réseau dépend des évènements démographiques qui ont lieu dans la population. Les sites sont vidés par la mortalité individuelle qui est supposée constante. Les sites sont remplis par la natalité et la mobilité individuelle à partir des sites voisins. La natalité comprend une part constante et une part influencée par *l'investissement dans l'altruisme* des voisins, qui augmente la natalité de ses voisins de manière additive et linéaire. La natalité résultant de ces deux parts est réduite par la *compétition locale* proportionnellement à la fréquence des sites occupés dans le voisinage. La *mobilité* entre sites voisins est constante, et est aussi réduite par la *compétition locale* proportionnellement à la fréquence des sites occupés dans le voisinage. L'investissement dans l'altruisme et la mobilité sont des traits coûteux, et leurs coûts augmentent en rapport à la valeur de ces deux traits. On envisage des coûts linéaires pour la mobilité, et j'envisage des coûts linéaires, décélérant et accélérant pour l'altruisme. Ces deux coûts affectent la natalité de l'individu.

L'analyse du modèle utilise une description écologique de la population, basée sur une méthode prenant en compte une correction des corrélations spatiales jusqu'au niveau des paires de sites (chapitre 1). La dynamique des sites et des paires de site étant explicitée, on utilise le formalisme des dynamiques adaptatives pour décrire l'évolution adaptative de l'investissement altruiste (chapitres 2 et 3) et de la dispersion (chapitre 3). Une mesure approximative de la valeur sélective d'invasion est dérivée et interprétée à la lumière des pressions de sélection affectant les deux traits. L'analyse complète de l'influence des paramètres du modèle sur cette dynamique adaptative est conduite numériquement. Elle est complétée par une simulation du processus individu centré et par une analyse de l'effet de mutations à effets importants.

Densité et structure généalogique du voisinage social

On étudie ici l'effet de la structure généalogique et de la densité du voisinage de naissance d'un jeune lézard sur son comportement de dispersion natale et son histoire de vie. On manipule la présence de la mère dans l'environnement de naissance du jeune pour tester l'effet des interactions entre la mère et ses enfants sur la dispersion natale du jeune. On manipule le nombre d'individus, toutes classes d'âge et de sexe confondues, pour mettre en évidence un effet de la densité en congénères sur la dispersion natale du jeune. On croise ces deux manipulations pour tester si l'interaction mère à enfant a un effet sur la dispersion du jeune indépendant ou non de la densité du voisinage. Les principes et résultats de l'expériences sont présentés dans le chapitre 4, et dans l'Annexe 3. Certains éléments propres à la compétition entre proches génétiques sont aussi discutés dans l'Annexe 1. Les caractéristiques du modèle d'étude sont rassemblées dans l'Annexe 2. Les objectifs de l'expérience et les données récoltées sont présentés dans les paragraphes qui suivent.

Après la naissance, la décision de dispersion du jeune lézard vivipare est rapide dans les populations naturelles (voir l'Annexe 2 pour la biologie de l'espèce). Certains résultats nous invitent à penser que cette décision dépend en partie d'une interaction entre la mère et ses enfants par des mécanismes agissant avant la naissance (de Fraipont et al., 2000; Massot et Clobert, 1995, 1998; Meylan et al., 2002; Sorci et al., 1994) ou après la naissance (Léna et al., 1998; Léna et de Fraipont, 1998). D'une part, certaines caractéristiques maternelles, comme l'âge de la mère ou la condition maternelle, affectent la dispersion natale. Les jeunes provenant de mères sénescentes ou en mauvaise condition ont tendance à disperser moins fréquemment (Meylan et al., 2002; Ronce et al., 1998). D'autre part, des manipulations simulant une mauvaise gestation maternelle, comme un faible nourrissage (Massot et Clobert, 1995, 2000), une application hormonale de corticostérone (de Fraipont et al., 2000), ou un parasitisme externe (Sorci et al., 1994) peuvent stimuler la philopatrie des jeunes. Enfin, les jeunes sont capables de discriminer leur mère biologique des autres femelles adultes et utilisent cette information pour choisir leur habitat (Léna et al., 2000; Léna et de Fraipont, 1998). Les réponses comportementales observées dépendent du sexe des jeunes, la réponse étant en général plus prononcée pour les jeunes femelles (Ronce et al., 1998 ; Sorci et al., 1994) ; de l'année, la relation pouvant s'inverser d'une année à l'autre (Massot et Clobert, 2000) ; et des caractéristiques maternelles (Meylan et al., 2002). Ces effets traduisent la dépendance au contexte des effets maternels et l'ambiguïté des messages véhiculés par ces effets : une bonne condition maternelle indique à la fois un risque élevé d'interactions futures entre la mère et ses enfants, mais aussi un bon environnement de naissance (Ims et Hjermann, 2001). Cependant, les manipulations pré-natales peuvent être confondues par les effets organisationnels de l'environnement maternel sur le développement du jeune et par des contraintes développementales (Dufty et al., 2002). Dans ce contexte, il nous est apparu utile de manipuler directement la présence de la mère afin de contraster des contextes sociaux sans possibilités d'interactions mère à enfants et des contextes sociaux où ces interactions sont possibles. On peut prédire l'effet de cette manipulation sur la dispersion natale en utilisant différentes explications, sous l'hypothèse que la mère n'est pas affectée par l'expérience :

- si la dispersion natale résulte uniquement d'un évitement de la compétition entre la mère et les enfants (Ronce et al., 1998), alors on devrait observer une augmentation de la dispersion natale en présence de la mère ;

- si la dispersion natale résulte uniquement d'un évitement de la consanguinité entre la mère et les enfants (Motro, 1991), alors on devrait observer une augmentation de la dispersion natale des fils en présence de leur mère, la dispersion des filles n'étant pas affectée ;
- si la dispersion natale résulte uniquement d'une facilitation entre la mère et ses enfants (Lambin et al., 2001), alors on devrait observer une diminution de la dispersion natale en présence de leur mère.

La dispersion natale dépend aussi de la présence d'individus non apparentés (ou plutôt, lointainement apparentés) dans le voisinage de l'individu. En particulier, le nombre de voisins traduit chez cette espèce le risque d'une compétition forte pour les ressources et l'espace (Massot et al., 1992). La dispersion devrait donc être augmentée à forte densité du fait des interactions compétitives entre congénères (Metz et Gyllenberg, 2001; Travis et al., 1999). Pour cette raison, on a manipulé conjointement la densité du voisinage social de l'individu en créant deux niveaux de densité, en dessous et au-dessus de la capacité de charge supposée du dispositif expérimental.

L'effet de la présence de la mère sur la dispersion des jeunes pourrait être dilué par des relations avec des individus non apparentés si un mélange entre les territoires familiaux voisins a lieu, si la dispersion adulte rend peu probable la présence d'un parent sur le lieu de naissance du jeune, ou si de nombreux individus partagent le même domaine vital. Chez notre espèce, les domaines vitaux individuels se chevauchent et les variations locales de la densité sont suffisantes pour permettre à la dispersion d'évoluer pour des causes indépendantes des interactions sociales avec la mère. De fait, on a croisé la manipulation de densité du voisinage avec la manipulation de la présence de la mère. On prédit une réponse plus forte à la présence de la mère pour un faible nombre d'individus dans le voisinage social.

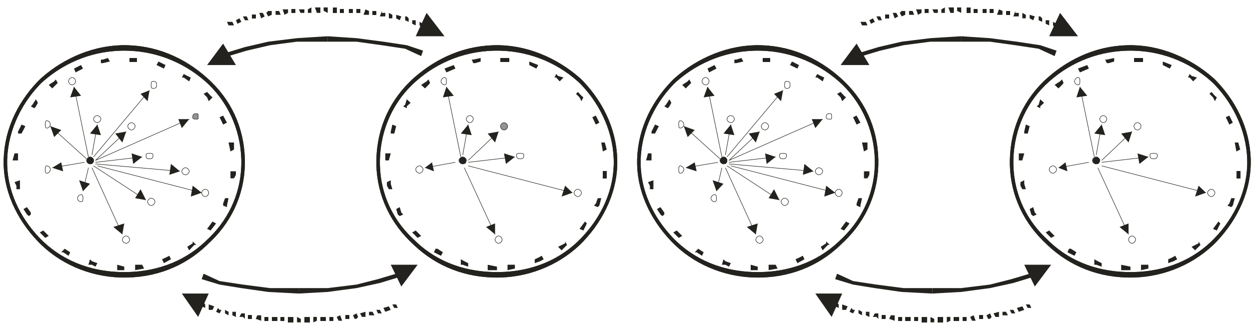


Figure 3. Structuration socio-spatiale de la première expérience de la thèse (voir chapitre 4 et Annexe 3). Autour d'un individu focal (rond noir), les interactions sociales (flèches droites) ont lieu au sein du fragment. La dispersion (flèche incurvée) est limitée à un fragment voisin (flèches pleines : mouvements aller, flèches hachurées : mouvements retour). On manipule la densité du voisinage au sein de la même population, et la présence de la mère (rond gris) dans le voisinage du jeune entre populations.

On réalise cette expérience à l'échelle d'un dispositif expérimental faisant coïncider le voisinage social et la structure spatiale de la population (Fig. 3). Chaque population est composée de deux fragments d'habitat fermés et connectés à l'autre fragment par des corridors. On met en rapport un fragment d'habitat à faible densité et un fragment d'habitat à haute densité au début de l'été 1999. Deux types de populations fragmentées sont établis : dans la moitié des systèmes, les jeunes sont lâchés en présence de la mère, alors que les jeunes sont lâchés en présence d'une femelle adulte non apparentée dans l'autre moitié du système. On laisse le système fonctionner de manière autonome jusqu'à l'été 2001. Chaque année, on identifie les individus dispersant d'un fragment d'habitat à l'autre, on capture à plusieurs reprises (printemps, été, automne) les individus résidant dans les fragments, et on garde les femelles gestantes au laboratoire en juin-juillet. Ce suivi permet d'étudier l'effet des traitements expérimentaux sur le comportement de dispersion, les histoires de vie individuelles (croissance, survie et reproduction), et la démographie de la population. L'analyse des données a été conduite entièrement pour le comportement de dispersion de toutes les classes d'âge (chapitre 4 et son appendice). L'analyse des caractéristiques de l'histoire de vie et de la démographie étant en cours de synthèse, elle sera présentée sous la forme de résultats bruts (Annexe 3).

Variabilité entre fragments d'habitats : occupation du fragment

On analyse ici l'effet de la structure d'occupation des fragments d'habitat sur le comportement d'immigration et l'histoire de vie d'un lézard dispersant. Je manipule la présence des congénères toutes classes d'âge et de sexe dans le fragment d'arrivée d'un dispersant afin de tester l'effet des interactions avec des résidents sur le comportement d'immigration d'un lézard et le succès de l'immigration. Les résultats de l'expérimentation sont présentés dans le chapitre 9.

Après la dispersion, tout individu doit s'établir dans un territoire et faire un choix d'habitat (Danchin et al., 2001). Le succès de l'individu au cours de sa vie va dépendre de ce choix si l'habitat choisi coïncide avec celui occupé en tant qu'adulte. En effet, le succès reproducteur individuel est une fonction de la qualité intrinsèque de l'habitat et des caractéristiques des congénères. Comment l'individu doit-il choisir son milieu de vie ? Quelles sont les conséquences de ce choix sur le succès reproducteur de l'individu au cours de sa vie ?

Une théorie traditionnelle du choix de l'habitat a été formulée par Fretwell et Lucas (1970), et est appelée distribution libre et idéale (Fretwell, 1972). Dans cette théorie, on suppose qu'il existe des habitats stables de qualités différentes, que le succès reproducteur individuel dans un habitat est influencé négativement par les congénères, que les individus ont une connaissance complète de leur environnement et que le choix d'un habitat s'effectue sans aucun coût. La théorie de la distribution libre et idéale prédit une répartition individuelle à l'équilibre évolutif proportionnelle aux différences intrinsèques de qualité de l'habitat (Fretwell, 1972).

On peut cependant supposer que des interactions sociales peuvent aussi avoir des effets positifs sur la démographie, notamment à faible densité (effets de Allee et interactions coopératives, Courchamp et al., 1999). De plus, on sait que de nombreux organismes ont une capacité limitée à accéder à l'information et procèdent par un processus d'échantillonnage pendant la prospection (Stamps, 2001). Dans le cas d'un effet de Allee, on prédit l'évolution d'une stratégie de choix de l'habitat basée sur la présence de congénères. A faible densité, les interactions positives génèrent une attraction envers les congénères, et le contraire est observé à forte densité (Greene et Stamps, 2001). Une telle attraction envers les congénères a été démontrée chez de nombreuses espèces à reproduction coloniale, mais aussi chez quelques espèces territoriales (Stamps, 2001). Par ailleurs, on doit s'attendre à voir évoluer des mécanismes de choix de l'habitat qui conduisent à une réduction des coûts de la prospection et à une augmentation de la qualité de l'information récoltée. Les coûts de prospection sont liés à l'échantillonnage, à des compensations avec d'autres activités et à la compétition entre prospecteurs. La qualité de l'information dépend de sa relation avec le succès reproducteur de l'individu et de sa prédictibilité, liée à l'autocorrélation temporelle de l'environnement (Doligez et al., 2003). Trois mécanismes de sélection de l'habitat ont donc été proposés : l'estimation directe d'un paramètre environnemental, l'attraction pour les congénères (attraction sociale), et l'utilisation du succès reproducteur des congénères (information publique, (Danchin et al., 2001)).

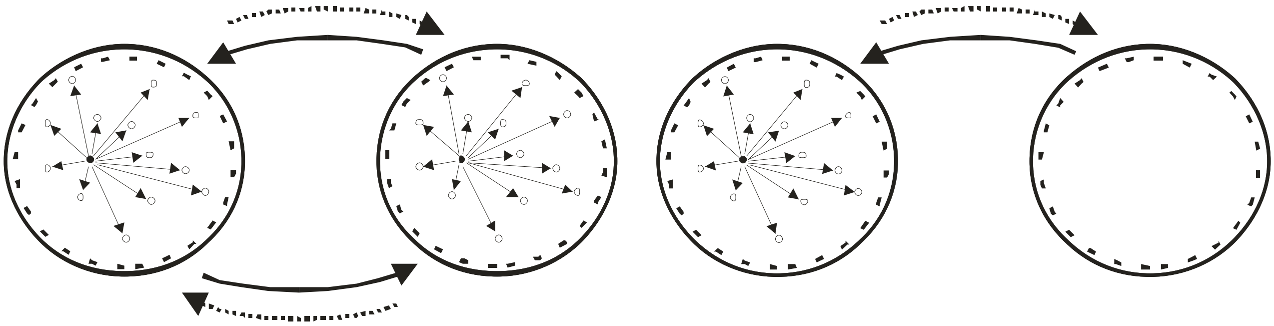


Figure 4. Structuration socio-spatiale de la deuxième expérience de la thèse (voir chapitre 5). Autour d'un individu focal, les interactions sociales ont lieu au sein du fragment. La dispersion est limitée à un fragment voisin. On manipule la présence de congénères au sein du fragment d'arrivée du lézard (gauche : les deux fragments sont occupés, mimant un contexte d'augmentation ; droite : un fragment est vide, mimant un contexte de colonisation depuis une source vers un habitat vide).

Si plusieurs études récentes chez les oiseaux suggèrent qu'un dispersant peut utiliser l'information dérivée du succès reproducteur de ses congénères pour choisir son milieu de vie (Boulinier et al., 2002; Danchin et Wagner, 1997; Doligez et al., 1999, 2002; Frederiksen et Bregnballe, 2001), relativement peu de travaux ont testé l'effet de la présence de congénères sur l'immigration à l'échelle d'une population et en comparant des individus dispersant (chez les invertébrés marins, voir par exemple Meadows et Campbell (1972) ; pour des expériences en milieu ouvert comparant la fixation d'oiseaux dans le cadre de programmes de réintroduction, voir par exemple Reed et Dobson, 1993). On a donc comparé le comportement d'établissement de lézards dispersant dans des unités expérimentales occupées par des congénères, à une densité proche de l'équilibre démographique du système, et dans des unités expérimentales initialement vides. On s'attendait à une préférence pour les sites vides si cet habitat était associé à un risque plus faible de compétition, surtout pour les individus jeunes. On s'attendait à une préférence pour les sites occupés si cet habitat était associé à un risque plus faible de coûts à l'établissement et la recherche du partenaire sexuel, surtout pour les individus âgés. On a donc aussi mesuré le succès démographique des immigrants dans des habitats occupées ou initialement vides.

On a réalisé cette expérience à l'aide du même dispositif que l'expérience précédente (Fig. 3). Au début de l'été 2001, on a mis en rapport deux fragments d'habitat occupés dans des unités dites d'augmentation, et un fragment d'habitat occupé à un fragment d'habitat vide dans les unités dites de colonisation. Le terme d'augmentation est utilisé en référence à son utilisation en biologie de la conservation et des métapopulations où il fait référence au rôle renforçant de l'immigration sur l'effectif des populations. Trois types de fragments sont donc établis : des fragments « augmentés » connectés deux à deux, des fragments « sources » connectés à un habitat vide, et des fragments initialement vides connectés à des fragments sources. On a laissé le système fonctionner de manière autonome jusqu'à l'été 2002. Un suivi a permis d'étudier l'effet des traitements expérimentaux sur le comportement de dispersion, les histoires de vie individuelles (croissance, survie et reproduction), et la démographie de la population. L'analyse des caractéristiques de l'histoire de vie, de la démographie et de la dispersion est résumée dans le chapitre 5.

Variabilité entre fragments d'habitats : structure de sexe

On envisage maintenant l'effet de la sexe ratio du fragment d'habitat sur le comportement de dispersion, l'histoire de vie et le mode d'appariement d'un lézard. On manipule la structure de sexe de la population adulte, afin de tester l'effet des interactions entre mâles ou femelles et un individu sur (i) son comportement de dispersion, (ii) son histoire de vie, et (iii) la dynamique des appariements. Certains résultats préliminaires sont présentés dans le chapitre 6. L'Annexe 2 donne les éléments fondamentaux de la biologie de la reproduction de notre espèce d'étude.

La dispersion est susceptible de dépendre d'interactions locales impliquant la structure généalogique du voisinage de l'individu (Le Galliard et al., 2003), mais aussi de la structure démographique du voisinage de départ et d'arrivée, comme la densité en congénères (voir par exemple Ozaki (1995) et Crespi et Taylor, 1990). Dans la dernière situation, on a supposé que les interactions étaient symétriques entre individus, en considérant la densité totale du voisinage toutes classes d'âge et de sexe confondues comme un bon indice du niveau de compétition. On peut cependant penser que la densité dépendance ne se résume pas à un paramètre aussi simple. Pour un vertébré, une stratification de la population s'opère par l'âge et le sexe : l'âge détermine entre autres le rang social de l'individu, les individus adultes étant en général dominants sur les individus jeunes ; et le sexe détermine les paramètres écologiques critiques du succès reproducteur, en dépendance du régime d'appariement. Chez les espèces à reproduction polygyne, les mâles et les femelles ont des stratégies démographiques différentes. Les mâles investissent peu dans les soins parentaux et la reproduction, hormis via la *compétition pour l'accès au partenaire*. Les femelles investissent beaucoup dans la production des jeunes et les soins parentaux (d'autant plus que la fécondation est interne et la reproduction vivipare), et sont en *compétition pour les ressources* (Andersson, 1994; Greenwood, 1980). De fait, les mâles sont plus sensibles que les femelles à des paramètres de la population reflétant la compétition locale pour le partenaire, alors que les femelles sont plus sensibles que les mâles à des paramètres de la population reflétant la compétition locale pour les ressources (e.g., Post et al., 1999). Ces différences entre mâles et femelles dans le régime de compétition ont été utilisées pour prédire une dispersion mâle biaisée chez les espèces polygynes (essentiellement des mammifères, voir Dobson, 1982; Perrin et Goudet, 2001; Perrin et Mazalov, 2000).

Les populations du lézard vivipare n'échappent pas à cette structuration par l'âge et par le sexe. Le régime de reproduction de l'espèce est polygynandrique, avec une variance du succès reproducteur plus forte chez les mâles que chez les femelles (Laloi et al. soumis). D'un point de vue comportemental, les mâles adultes sont dominants sur les femelles adultes, et les adultes sont dominants sur les sub-adultes et les juvéniles (Lecomte, 1993; Lecomte et al., 1994). Les données démographiques suggèrent aussi que les mâles adultes sont en compétition pour les femelles adultes (Massot, 1992; Massot et al., 1992; Pilorge, 1987; Pilorge et al., 1987). Par ailleurs, la dispersion natale est faiblement biaisée en faveur des mâles, et la dispersion de reproduction est plus fortement biaisée en faveur des mâles (Clobert et al., 1994; Massot, 1992), suggérant une asymétrie compétitive entre mâles et femelles dans la population (Perrin et Goudet, 2001). On a donc tenté de tester l'hypothèse selon laquelle la dispersion dépendrait du risque de compétition locale pour les ressources et pour les partenaires en manipulant la sexe ratio adulte de la population :

- si la dispersion de reproduction des femelles résulte d'un évitement de la compétition pour les ressources, on prédit une augmentation de la dispersion des femelles quand la sexe ratio de la population est biaisée en faveur des femelles (resp. mâles). Cette dispersion devrait être synchronisée sur la période estivale pendant laquelle les individus restaurent les réserves dépensées lors de la reproduction, surtout pour les femelles.
- si la dispersion de reproduction des mâles résulte d'un évitement de la compétition pour l'accès au partenaire, on prédit une augmentation de la dispersion des mâles quand la sexe ratio de la population est biaisée en faveur des mâles (resp. femelles). Cette dispersion devrait être synchronisée sur la période printanière pendant laquelle les appariements ont lieu.

Au début de l'été 2002, nous avons établi deux types de fragments d'habitat : la moitié des populations est constituée d'une sexe ratio adulte biaisée en faveur des mâles, et l'autre moitié d'une sexe ratio adulte biaisée en faveur des femelles (Fig. 5). La densité en adultes de la population est maintenue constante, et la structure de la population immature et juvénile est semblable entre les fragments. Nous avons laissé le système fonctionner de manière autonome jusqu'à l'été 2003. Nous avons identifié les individus dispersant d'un fragment d'habitat à l'autre, capturé à plusieurs reprises les individus résidant dans les fragments (été, automne, fin du printemps) et ramené les femelles gestantes au laboratoire à la fin de l'expérience. Contrairement aux expériences précédentes, on ne connecte pas les fragments deux à deux : la dispersion a lieu entre tous les fragments du même traitement. On simule ainsi une dispersion dans un paysage linéaire dont la sexe ratio adulte est déséquilibrée. Nous avons aussi établi la carte allélique des individus introduits et des juvéniles issus de la première génération de cette expérience sur plusieurs loci microsatellitaires (Boudjemadi et al., 1999). Une analyse préliminaire des comportements de dispersion est résumée dans le chapitre 6. En effet, l'expérience ayant débuté dans la dernière année de la thèse, elle est toujours en cours au moment de l'écriture de ce document.

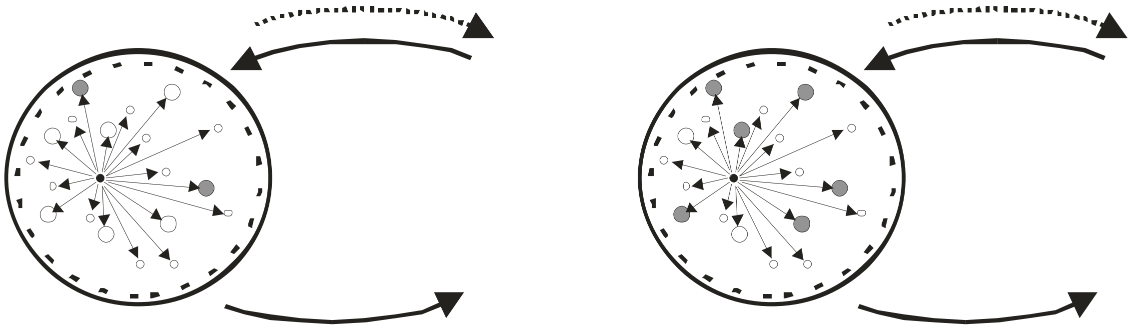


Figure 5. Structuration socio-spatiale de la troisième expérience de la thèse (voir chapitre 6). Autour d'un individu focal, les interactions sociales ont lieu au sein du fragment. La dispersion est limitée à plusieurs fragments du même traitement. On manipule la sexe ratio des congénères adultes (ronds de grande taille, rond gris : femelle, rond vide : mâle) au sein du fragment de vie du lézard (gauche : la sexe ratio est biaisée en faveur des mâles ; droite : la sexe ratio est biaisée symétriquement en faveur des femelles). La structure de la population immature et juvénile (petits cercles) n'est pas perturbée.

Capacités locomotrices

Dans cette dernière partie, on envisage certaines propriétés des capacités locomotrices, une mesure intégrative de la compétence physiologique et morphologique d'un individu à explorer son voisinage social. On a étudié ces variations dans deux contextes particuliers :

- l'effet de l'investissement dans la reproduction des femelles sur les performances locomotrices (Annexe 4) ;

- la relation entre la capacité locomotrice d'un jeune et sa survie pendant la première année de sa vie (Annexe 5).

On a utilisé une démarche corrélative pour répondre à ces questions. Cette démarche consiste à mesurer les performances locomotrices individuelles pour les mettre en relation avec des covariables individuelles. Cette approche est justifiée par le fait qu'il était difficile de manipuler directement les capacités locomotrices des lézards, en tout cas dans le cadre de ces travaux préliminaires (mais voir Miles et al. (2000) pour une étude expérimentale).

REFERENCES

Allee WC, Emerson AE, Park O, Park T, Schmidt KP, 1949. Principles of animal ecology. Philadelphia: Saunders.

Andersson M, 1994. Sexual selection. Princeton: Princeton University Press.

Axelrod R, Hamilton WD, 1981. The evolution of cooperation. Science 211:1390-1396.

Balloux F, Goudet J, Perrin N, 1998. Breeding system and genetic variance in the monogamous, semi-social shrew, *Crocidura russula*. Evolution 52:1230-1235.

Begon M, Harper JL, Towsend CR, 1996. Ecology. Individuals, populations and community, 3 ed: Blackwell Science.

Bennett AF, Huey RB, 1990. Studying the evolution of physiological performance. In: Oxford Surveys in Evolutionary Biology (Futuyma DJ, Antonovics J, eds); 251-284.

Bjørnstad ON, Grenfell BT, 2001. Noisy clockwork: time series analysis of population fluctuations in animals. Science 293:638-643.

Bjørnstad ON, Ims RA, Lambin X, 1999. Spatial population dynamics: analyzing patterns and processes of population synchrony. Trends in Ecology and Evolution 14:427-432.

Bolker BM, Pacala SW, 1999. Spatial moment equations for plant competition : understanding spatial strategies and the advantage of short dispersal. The American Naturalist 153:575-602.

Boudjemadi K, Martin O, Simon JC, Estoup A, 1999. Development and cross-species comparison of microsatellite markers in two lizard species, *Lacerta vivipara* and *Podarcis muralis*. Molecular Ecology 8:518-520.

Boulinier T, Yoccoz NG, McCoy KD, Eriskstad KE, Tveraa T, 2002. Testing the effect of conspecific reproductive success on dispersal and recruitment decisions in a colonial bird: design issues. Journal of Applied Statistics 29:509-520.

Caswell H, 2001. Matrix population models, 2 ed. Sunderland: Sinauer Associates.

Clements FE, 1916. Plant succession: analysis of the development of vegetation. Washington: Carnegie Institute of Washington Publication.

Clobert J, Massot M, Lecomte J, Sorci G, de Fraipont M, Barbault R, 1994. Determinants of dispersal behavior: the common lizard as a case study. In: Lizard ecology. Historical and experimental perspectives. (Vitt LJ, Pianka ER, eds). Princeton: Princeton University Press; 183-206.

Clobert J, Oppliger A, Sorci G, Ernande B, Swallow JG, Garland TJ, 2000. Trade-offs in phenotypic traits: endurance at birth, growth, survival, predation and susceptibility to parasitism in a lizard, *Lacerta vivipara*. Functional Ecology 14:675-684.

Courchamp F, Clutton-Brock T, Grenfell B, 1999. Inverse density dependence and the Allee effect. Trends in Ecology and Evolution 14:405-410.

Crespi BJ, Taylor PD, 1990. Dispersal rates under variable patch density. The American Naturalist 135:48-62.

Danchin E, Heg D, Doligez B, 2001. Public information and breeding habitat selection. In: Dispersal (Clobert J, Danchin E, Dhondt AA, Nichols JD, eds). Cambridge: Cambridge University Press; 243-258.

Danchin E, Wagner RH, 1997. The evolution of coloniality: the emergence of new perspectives. Trends in Ecology and Evolution 12:342-347.

Darwin C, 1859. The origin of species. London: John Murray.

Darwin C, 1871. The descent of man, and selection in relation to sex. London: John Murray.

de Fraipont M, Clobert J, John AH, Meylan S, 2000. Increased pre-natal maternal corticosterone promotes philopatry of offspring in common lizards *Lacerta vivipara*. Journal of Animal Ecology 69:404-413.

Dieckmann U, Doebeli M, 1999. On the origin of species by sympatric speciation. Nature 400:354-357.

Dieckmann U, Law R, 2000. Relaxation projections and the method of moments. In: The geometry of ecological interactions: simplifying spatial complexities (Dieckmann U, Law R, Metz JAJ, eds).

Dobson FS, 1982. Competition for mates and predominant juvenile male dispersal in mammals. Animal Behaviour 30:1183-1192.

Doligez B, Cadet C, Danchin E, Boulinier T, 2003. When to use public information for breeding habitat selection? The role of environmental predictability and density dependence. Animal Behaviour.

Doligez B, Danchin E, Clobert J, 2002. Public information and breeding habitat selection in a wild bird population. Science 297:1168-1170.

Doligez B, Danchin E, Clobert J, Gustafsson L, 1999. The use of conspecific reproductive success for breeding habitat selection in a non-colonial, hole-nesting species, the collared flycatcher. Journal of Animal Ecology 68:1193-1206.

Dufty AM, Clobert J, Moller AP, 2002. Hormones, developmental plasticity and adaptation. Trends in Ecology and Evolution 17:190-196.

Durrett R, Levin S, 1994. The importance of being discrete (and spatial). Theoretical Population Biology 46:363-394.

Emlen ST, 1997. Predicting family dynamics in social vertebrates. In: Behavioural ecology: an evolutionary approach (Krebs JR, Davies NB, eds). Oxford: Blackwell Science; 228-253.

Favre L, Balloux F, Goudet J, Perrin N, 1997. Female-biased dispersal in the monogamous mammal *Crocidura russula*: evidence from field data and microsatellite patterns. Proceedings of the Royal Society London B 264:127-132.

Fisher RA, 1930. The genetical theory of natural selection. Oxford: Oxford University Press.

Frank SA, 1998. Foundations of social evolution. Princeton: Princeton University Press.

Frederiksen M, Bregnballe T, 2001. Conspecific reproductive success affects age of recruitment in a great cormorant, *Phalacrocorax carbo sinensis*, colony. Proceedings of the Royal Society London B 268:1519-1526.

Fretwell SD, 1972. Theory of habitat distribution. In: Populations in seasonal environments (Fretwell SD, ed). Princeton: Princeton University Press; 79-114.

Fretwell SD, Lucas HL, 1970. On territorial behaviour and other factors influencing habitat distribution in birds. I. Theoretical development. Acta Biotheoretica 19:16-36.

Garland TJ, 1999. Laboratory endurance capacity predicts variation in field locomotor behaviour aming lizard species. Animal Behaviour 58:77-83.

Greene CM, Stamps JA, 2001. Habitat selection at low population densities. Ecology 82:2091-2100.

Greenwood PJ, 1980. Mating systems, philopatry and dispersal in birds and mammals. Animal Behaviour 28:1140-1162.

Hamilton WD, 1964. The genetical evolution of social behaviour, I. Journal of Theoretical Biology 7:1-16.

Hanski I, 1998. Metapopulation dynamics. Nature 396:41-49.

Hanski I, 1999. Metapopulation ecology: Oxford University Press.

Harada Y, Iwasa Y, 1994. Lattice population dynamics for plants with dispersing seeds and vegetative propagation. Research on Population Ecology 36:237-249.

Harrison S, Murphy DD, Ehrlich PR, 1988. Distribution of the bay checkerspot butterfly, *Euphydrias editha bayensis*: evidence for a metapopulation model. The American Naturalist 132:360-382.

Harrison S, Taylor AD, 1997. Empirical evidence for metapopulation dynamics. In: Metapopulation biology: ecology, genetics, and evolution (Hanski IA, Gilpin ME, eds). San Diego: Academic Press; 247-265.

Heinsohn RG, Legge S, 1999. The cost of helping. Trends in Ecology and Evolution 14:53-57.

Ims RA, Hjermann DO, 2001. Condition dependent dispersal. In: Dispersal (Clobert J, Danchin E, Dhondt AA, Nichols JD, eds). Oxford: Oxford University Press; 203-216.

Ims RA, Yoccoz NG, 1997. Studying transfer processes in metapopulations: emigration, migration, and colonization. In: Metapopulation biology: ecology, genetics, and evolution (Hanski IA, Gilpin ME, eds). San Diego: Academic Press; 247-265.

Kareiva P, Wennergren U, 1995. Connecting landscape patterns to ecosystem and population processes. Nature 373:299-302.

Lambin X, Aars J, Piertney SB, 2001. Dispersal, intraspecific competition, kin competition and kin facilitation: a review of the empirical evidence. In: Dispersal (Clobert J, Danchin E, Dhondt A, Nichols JD, eds). Oxford: Oxford University Press; 110-122.

Lambin X, Aars J, Piertney SB, Telfer S, 2003. Persistence in the face of correlated extinction in small mammals metapopulations. In:.

Law R, Dieckmann U, 2000. A dynamical system for neighborhoods in plant communities. Ecology 81:2137-2148.

Le Galliard J-F, Ferrière R, Clobert J, 2003. Mother-offspring interactions affect natal dispersal in a lizard. Proceedings of the Royal Society London B 270:1163-1169.

Lecomte J, 1993. Rôle du comportement dans l'organisation et la régulation des populations de lézards vivipares. Paris: Paris XI.

Lecomte J, Clobert J, Massot M, Barbault R, 1994. Spatial and behavioural consequences of a density manipulation in the common lizard. Ecoscience 1:300-310.

Léna JP, Clobert J, de Fraipont M, Lecomte J, Guyot G, 1998. The relative influence of density and kinship on dispersal in the common lizard. Behavioral Ecology 9:500-507.

Léna JP, de Fraipont M, Clobert J, 2000. Affinity towards maternal odour and offspring dispersal in the common lizard. Ecology Letters 3:300-308.

Léna JP, de Fraipont M, 1998. Kin recognition in the common lizard. Behavioral Ecology and Sociobiology 42:341-347.

Levins R, 1969. Some demographic and genetic consequences of environmental heterogeneity for biological control. Bulletin of the Entomological Society of America 15:237-240.

MacArthur RH, Wilson EO, 1967. The theory of island biogeography. Princeton: Princeton University Press.

Massot M, 1992. Movement patterns of the common lizard (*Lacerta vivipara*) in relation to sex and age. In: Proceedings of the Sixth Ordinary General Meeting of the Societas Europea Herpetologica (Korsos Z, Kiss I, eds). Budapest: Hungary Natural History Museum; 315-319.

Massot M, Clobert J, 1995. Influence of maternal food availability on offspring dispersal. Behavioural Ecology and Sociobiology 37:413-418.

Massot M, Clobert J, 1998. Similarities in dispersal behaviour among siblings in the common lizard (*Lacerta vivipara*). In: Proceedings of the Ordinary General Meeting of the Societas Europea Herpetologica (Miaud C, Guyétant R, eds). Le Bourget du Lac / France; 315-323.

Massot M, Clobert J, 2000. Processes at the origin of similarities in dispersal behaviour among siblings. Journal of Evolutionary Biology 13:707-719.

Massot M, Clobert J, Pilorge T, Lecomte J, Barbault R, 1992. Density dependence in the common lizard: demographic consequences of a density manipulation. Ecology 73:1742-1756.

Matsuda H, Ogita N, Sasaki A, Sato K, 1992. Statistical mechanics of population - The lattice Lotka-Volterra model. Progress of Theoretical Physics 88:1035-1049.

Maynard Smith J, Szathmary E, 1995. The major transitions in evolution.

McPeek MA, Holt RD, 1992. The evolution of dispersal in spatially and temporally varying environments. The American Naturalist 140:1010-1027.

Meadows PS, Campbell JI, 1972. Habitat selection by aquatic invertebrates? In: Advances in marine biology (Russell SS, Chazdon RL, Smith AP, eds). London: Academic Press; 271-382.

Metz JAJ, Geritz SAH, Meszéna G, Jacobs FJA, van Heerwaarden JS, 1996. Adaptive dynamics, a geometrical study of the consequences of nearly faithful reproduction. In: Stochastic and spatial structures of dynamical systems (van Strien S.J. VLSM, ed). Amsterdam: North Holland; 183-231.

Metz JAJ, Gyllenberg M, 2001. How should we define fitness in structured metapopulation models? Including an application to the calculation of evolutionarily stable dispersal strategies. Proceedings of the Royal Society London B 268:499-508.

Metz JAJ, Nisbet RM, H. GSA, 1992. How should we define fitness for general ecological scenarios ? Trends in Ecology and Evolution 7:198-202.

Meylan S, Belliure J, Clobert J, de Fraipont M, 2002. Stress and body condition as prenatal and postnatal determinants of dispersal in the common lizard (*Lacerta vivipara*). Hormones and Behavior 42: 319-326.

Michod RE, 1999. Darwinian dynamics - Evolutionary transitions in fitness and individuality. Princeton: Princeton University Press.

Miles DB, Sinervo B, Frankino WA, 2000. Reproductive burden, locomotor performance, and the cost of reproduction in free ranging lizards. Evolution 54:1386-1395.

Morris A, 1997. Representing spatial interactions in simple ecological models. Coventry: University of Warwick.

Motro U, 1991. Avoiding inbreeding and sibling competition: the evolution of sexual dimorphism for dispersal. The American Naturalist 137:108-115.

O'Connor D, Shine R, 2003. Lizards in 'nuclear families': a novel reptilian system in *Egernia saxatilis* (Scincidae). Molecular Ecology 12:743-752.

Ozaki K, 1995. Intergall migration in aphids: a model and a test of ESS dispersal rate. Evolutionary Ecology 9:542-549.

Perrin N, Goudet J, 2001. Inbreeding, kinship, and the evolution of natal dispersal. In: Dispersal (Clobert J, Nichols JD, Danchin E, Dhondt AA, eds). Oxford: Oxford University Press; 123-142.

Perrin N, Mazalov V, 2000. Local competition, inbreeding, and the evolution of sex-biased dispersal. The American Naturalist 155:116-127.

Pilorge T, 1987. Density, size structure, and reproductive characteristics of three populations of *Lacerta vivipara* (Sauria: Lacertidae). Herpetologica 43:345-356.

Pilorge T, Clobert J, Massot M, 1987. Life history variations according to sex and age in *Lacerta vivipara*. In: 4th Ordinary General Meeting of the Societas Europaea Herpetologica (van Gelder JJ, Strijbosch H, Bergers PJM, eds). Nijmegen: Faculty of Science; 311-315.

Post E, Forchhammer MC, Stenseth NC, 1999. Environmental variation shapes sexual dimorphism in red deer. Proceedings of the National Academy of Sciences USA 96:4467-4471.

Pulliam HR, 1988. Sources, sinks, and population regulation. The American Naturalist 132:652-661.

Rand D, 1998. Correlation equations and pair approximation for spatial ecologies. In: Advanced ecological theory (Mc Glade J, ed): Blackwell Science; 100-143.

Reed JM, Dobson AP, 1993. Behavioural constraints and conservation biology: conspecific attraction and recruitment. Trends in Ecology and Evolution 8:253-256.

Ronce O, Clobert J, Massot M, 1998. Natal dispersal and senescence. Proceedings of the National Academy of Sciences USA 95:600-605.

Ronce O, Gandon S, Rousset F, 2000. Kin selection and natal dispersal in an age-structured population. Theoretical Population Biology 58:143-159.

Smith AT, 1980. Temporal changes in insular populations of the pika (Ochotona princeps). Ecology 61:8-13.

Sorci G, Massot M, Clobert J, 1994. Maternal parasite load increases sprint speed and philopatry in female offspring of the common lizard. The American Naturalist 144:153-164.

Stamps JA, 2001. Habitat selection by dispersers: integrating proximate and ultimate approaches. In: Dispersal (Clobert J, Danchin E, Dhondt A, Nichols J, eds). Cambridge: Cambridge University Press; 230-242.

Stearns SC, 1992. The evolution of life histories. Oxford: Oxford University Press.

Sugg DW, Chesser RK, Fobson FS, Hoogland JL, 1996. Population genetics meets behavioral ecology. Trends in Ecology and Evolution 11:338-342.

Travis JM, Murrell DJ, Dytham C, 1999. The evolution of density-dependent dispersal. Proceedings of the Royal Society London B 266:1837-1842.

van Baalen M, 2000. Pair approximation for ecological interactions on different geometries. In: The geometry of ecological interactions: simplifying spatial complexities (Dieckmann U, Law R, Metz JAJ, eds): Cambridge University Press.

van Baalen M, Jansen VAA, 2001. Dangerous liaisons: the ecology of private interest and common good. Oikos 95:211-224.

van Baalen M, Rand D, 1998. The unit of selection in viscous populations and the evolution of altruism. Journal of Theoretical Biology 193:631-648.

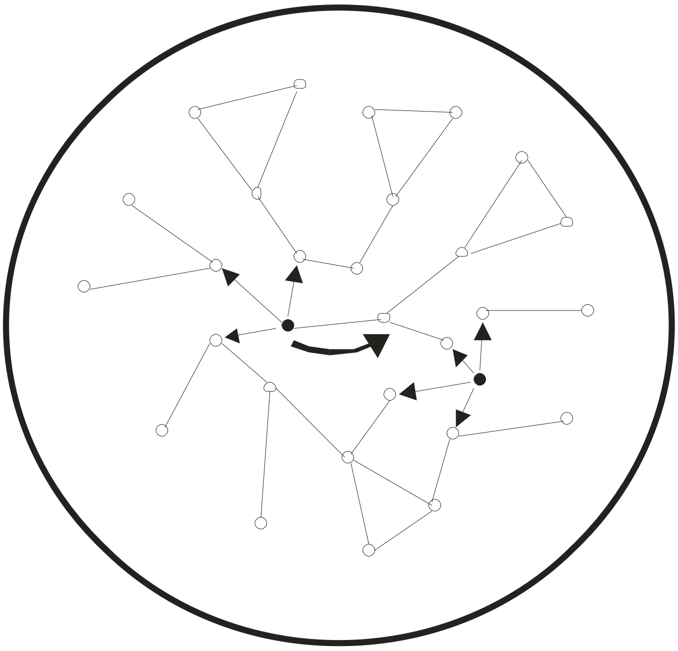
West SA, Pen I, Griffin AS, 2002. Cooperation and competition between relatives. Science 296:72-75.

Wiens JA, 1989. Spatial scaling in ecology. Functional Ecology 3:385-397.

Wiens JA, Stenseth NC, Van Horne B, Ims RA, 1993. Ecological mechanisms and landscape ecology. Oikos 66:1-12.

Wilson EO, 1975. Sociobiology: the new synthesis. Cambridge: Harvard University Press.

CHAPITRE 1 – DYNAMIQUES ADAPTATIVES SPATIALISEES



> « Explicit spatial models are instructive for they show that fitness (as measured by the likelihood of invasion) depends not on individual property alone but also upon aspects of the spatial environment » R. E. Michod dans *Darwinian dynamics. Evolutionary transitions in fitness and individuality*. 1999. p. 73.

Attention : les appels de chapitre dans cette partie font référence aux chapitres de l'ouvrage dans lequel cette partie a été publiée, pas aux chapitres de la thèse !

VALEUR SELECTIVE D'INVASION ET DYNAMIQUES ADAPTATIVES DANS DES MODELES ECOLOGIQUES SPATIAUX

Régis Ferrière
Jean-François Le Galliard

RESUME

Une mesure appropriée de la valeur sélective est nécessaire pour décortiquer les processus proximaux et ultimes agissant sur la dispersion et évaluer leurs effets respectifs. Cependant, il y a eu relativement peu de tentatives théoriques pour définir avec cohérence la valeur sélective à partir des principes de base de la démographie, quand l'adaptation de traits à dimension spatiale, comme la dispersion, est envisagée. Dans ce chapitre, nous présentons le système des dynamiques adaptatives et nous proposons que la valeur sélective d'invasion fournit un concept robuste pour prendre en compte les processus écologiques agissant à l'échelle individuelle. La construction de la valeur sélective d'invasion pour un scénario écologique à dimension spatiale est présentée. La valeur sélective d'invasion inclut les effets des voisins sur un individu focal, médiés par des coefficients analogues aux coefficients d'apparentement de la génétique des populations. La valeur sélective d'invasion peut être utilisée pour analyser l'évolution conjointe de la dispersion et de l'altruisme, deux traits qui ont une influence directe sur la distribution spatiale des individus, et dont l'évolution dépend de la distribution spatiale des individus. Nos prédictions déterministes de l'évolution de la dispersion et de l'altruisme basées sur la valeur sélective d'invasion sont en accord avec des simulations stochastiques du processus de mutation-sélection agissant sur ces traits.

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Mots-clés : dynamiques adaptatives, altruisme, dispersion, valeur sélective d'invasion, modèles de réseau.

INVASION FITNESS AND ADAPTIVE DYNAMICS IN SPATIAL POPULATION MODELS

Régis Ferrière
Jean-François Le Galliard

ABSTRACT

Disentangling proximate and ultimate factors of dispersal, and assessing their relative effects require an appropriate measure of fitness. Yet there have been few theoretical attempts to coherently define fitness from demographic 'first principles', when space-related traits like dispersal are adaptive. In this chapter, we present the framework of adaptive dynamics and argue that invasion fitness is a robust concept accounting for ecological processes that operate at the individual level. The derivation of invasion fitness for spatial ecological scenarios is presented. Spatial invasion fitness involves the effect of neighbors on a focal individual, mediated by coefficients analogous to relatedness coefficients of population genetics. Spatial invasion fitness can be used to investigate the joint evolution of dispersal and altruism, two traits that both have a direct influence on, and whose evolution is strongly responsive to, the spatial distribution of individuals. Our deterministic predictions of dispersal and altruism evolution based on spatial invasion fitness are in good agreement with stochastic individual-based simulations of the mutation-selection process acting on these traits.

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Key-words : Adaptive dynamics, altruism, dispersal, invasion fitness, lattice models.

INTRODUCTION

Even in homogeneous habitats, *spatial fluctuations* of population size arise inevitably as a result of demographic stochasticity, and *spatial correlations* build up from the imperfect mixing of individuals induced by the limited range of dispersal (Tilman and Kareiva, 1998 ; Dieckmann *et al.*, 2000). As a consequence, selective forces acting on the life-history traits of individuals are neither uniform nor independent across space. Dispersal propensity (in the broad sense of natal dispersal and breeding dispersal) is therefore a pivotal component of the individuals' phenotype, for it is both a target of selection and a primary factor of spatial fluctuations and correlations in the selective regime (Ferrière *et al.*, 2000).

Since the seminal work of Hamilton and May (1977) we know that the avoidance of competition with related individuals is an important factor in explaining the evolution of dispersal. It has been recently argued that dispersal probabilities evolving under the sole effect of kin competition provide a null model against which to assess the relative importance of alternative selective forces, as predicted by more elaborated kin selection models (Ronce, 1999). In kin selection theory based on diallelic, haploid genetics, the commonly used measure of fitness is *invasion fitness*, that is, the *per capita* growth rate of a mutant when rare. For pairwise interactions involving an 'actor' and a 'recipient', the definition of invasion fitness involves the *relatedness* of the recipient to the actor (Grafen 1979), for which the correct definition is the probability that the recipient is a mutant (Day and Taylor, 1998). However, this assumes that the altered phenotype of a mutant has no effect on that probability and therefore does not change relatedness. Obviously this does not hold true when phenotypic traits under consideration, like dispersal, modify the distribution of individuals across space. Furthermore, how to model mutants' initial rarity require some care in spatial models (Rousset and Billiard, 2000) : for the population size is everywhere locally finite, the initial number of mutants may not be regarded as locally infinitesimal.

The purpose of this chapter is to provide a modeling framework that allows to investigate the evolutionary dynamics of adaptive, continuous traits, while accounting explicitly for both the reciprocal effects of these traits on the spatial distribution of individuals, and for the effects of the spatial heterogeneity of selective pressures on the traits' evolutionary dynamics. In section 2, we provide a general argument that the notion of invasion fitness is appropriate to capture 'first' demographic principles operating at the level of individuals, and to describe the long-term evolutionary dynamics of adaptive life-history traits (Metz *et al.*, 1992, 1996 ; Dieckmann and Law, 1996 ; Geritz *et al.*, 1997, 1998). We then present in section 3 van Baalen and Rand's (1998) extension of the notion of invasion fitness to spatially heterogeneous populations. *Spatial invasion fitness* is derived from first demographic and behavioral principles operating at the levels of individuals and their nearby neighbors. In non-spatial populations where individuals are assumed to be constantly well-mixed and interactions occur at random between them, invasion fitness can be obtained as the Malthusian growth rate of a simple birth-and-death process (Ferrière and Clobert, 1992 ; Metz *et al.*, 1992 ; Ferrière and Gatto, 1995). In contrast, when interactions develop locally and dispersal is limited to neighborhoods, the process of mutant growth should be modeled by keeping track of spatial statistics that describe local population structures beyond global densities. The theory of *correlation equations* (Matsuda *et al.*, 1992 ; Morris, 1997 ; Rand, 1999) provides the appropriate mathematical tools. Under certain assumptions about habitat structure and the model's mathematical properties, invasion fitness can then be obtained as the dominant eigenvalue of a matrix (van Baalen and Rand, 1998), just as one would recover the population growth rate of a simple Leslie model (Caswell, 1989). In the spatial setting, the matrix involved contains demographic parameters that depend upon the local, spatial structure of the population.

In section 4 we operate this framework to investigate the evolution of dispersal jointly with altruistic behavior. The evolution of dispersal and the evolution of altruism have been the focus of two rather independent lines of research that trace back to the seminal work of Hamilton (1964). Yet there are serious reasons for trying to merge these lines. With limited dispersal, individuals are likely to interact with relatives, and kin selection models would then predict altruism to evolve. Yet neighbors not only interact socially : they compete with each other as well. Thus, clustering of relatives may not be sufficient for sociality to evolve. A dose of dispersal is needed, so that a locally successful strategy can be exported throughout the resident population. Co-adaptive changes of dispersal and social behavior may thus be expected (Le Galliard *et al.*, *in prep.*).

ADAPTIVE DYNAMICS AND THE CONCEPT OF INVASION FITNESS

We will first introduce the basics of a general and coherent mathematical theory of Darwinian evolution which aims at describing the evolutionary dynamics of adaptive, continuous traits. This *adaptive dynamics theory* (founding papers are Metz *et al.*, 1992, 1996 ; Dieckmann and Law, 1996 ; Geritz *et al.*, 1997) satisfies three important requirements :

- Adaptive dynamics are modeled as a macroscopic description derived from microscopic mechanisms. Selective pressures are set by ecological mechanisms operating at the 'microscopic' level of individuals.
- Adaptive dynamics incorporate the stochastic elements of evolutionary processes, arising from the random process of mutation, and from the extinction risk of initially small mutant populations in the process of selection.
- Adaptive dynamics describe evolution as a dynamical process, identifying potential evolutionary endpoints and among them those which indeed are attractors for the traits dynamics.

In this section we present a brief overview of the principles of adaptive dynamics modeling to show that a consistent measure of fitness arises naturally from the description of microscopic processes underlying ecological interactions. The reader should refer to Marrow *et al.* (1992), Metz *et al.* (1992) and Dieckmann and Law (1996) for a thorougher treatment. In the following sections, we shall see how to derive this fitness measure for a class of spatial population models where the individual probability of dispersal is one of the adaptive traits under consideration.

The canonical equation of adaptive dynamics

We consider a closed population of a single species. Individuals are characterized by a suite of adaptive, quantitative traits which define their *phenotype*. They reproduce and die at rates that depend upon their phenotype and their environment, including external factors as well as their own congeners population. Haploid inheritance is assumed, and there is a non-zero probability for a birth event to produce a mutant offspring, that is, an individual that differs from its parent in one of the traits. Individuals interact with each other, and the process of selection determines changes in the abundance of each phenotype through time. Direct individual-based models accounting for the stochasticity of birth, death and mutation events could be run to study how the distribution of phenotypes present in the population evolves through time. The theory of adaptive dynamics was developed as an alternative to intensive computer calculations, to provide a handy, deterministic description of the stochastic processes of mutation and selection.

Adaptive dynamics models rest on two basic principles (Metz *et al.*, 1996) : mutual exclusion, « in general two phenotypes x and x' differing only slightly cannot coexist indefinitely in the population » ; and timescale separation, « the timescale of selection is much faster than that of mutation ». Thus one may regard the adaptive dynamics as a trait substitution sequence. Each step occurs at a rate equal to the probability $w(x'|x)$ per unit time for a specific phenotype substitution, say x' substituted to x. The so-called canonical equation of adaptive dynamics then describes how the mean of the probability distribution of trait values in the evolving population changes through time. If we keep using x to denote this mean, the canonical equation reads (Dieckmann and Law, 1996) :

$$\frac{d}{dt}x = \int (x'-x) \cdot w(x'|x)\, dx' \tag{2.1}$$

where the integral sum is taken over the whole range of feasible phenotypes.

Following on the traditional view of the evolutionary process as a hill-climbing walk on an adaptive landscape (Wright, 1931), we seek to recast the canonical equation into the form

$$\frac{d}{dt}x = \eta(x) \cdot \frac{\partial}{\partial x'} W(x',x)\bigg|_{x'=x} \tag{2.2}$$

where the coefficient $\eta(x)$ would scale the rate of evolutionary change, and $W(x',x)$ would rigorously define the measure of fitness of individuals with trait value x' in the environment set by the bearers of trait value x.

Mutant invasion rate as a measure of fitness

To recast the canonical equation (2.1) in the form of equation (2.2), we first expand $w(x'|x)$ as the product of a mutation term and a selection term. To keep notations simple, we shall restrict ourselves to the case where phenotypes are characterized by a single trait. The mutation term is the probability per unit time that the mutant enters the population. It involves four multiplicative components : the *per capita* birth rate $b(x)$ of phenotype *x*, the fraction $\mu(x)$ of births affected by mutations, the equilibrium population size $\hat{n}_x$ of phenotype *x*, and the probability of a mutation step size $x'-x$ from phenotype *x*. The selection term is the probability that the initially rare mutant goes to fixation. Under the assumption that the population is *well mixed*, we can neglect the effects of the mutant density on the demographic rates of the mutant and resident populations. Let us denote the *per capita* birth and death rates of the rare mutant in a resident population of phenotype *x* by $b(x',x)$ and $d(x',x)$. Then the difference $b(x',x)-d(x',x)$ measures the mutant invasion rate, that is, the *per capita* growth rate of initially rare mutants, hereafter denoted by $s(x',x)$. The theory of stochastic birth-and-death processes (e.g. pp. 39-41 in Renshaw, 1991) shows that the probability that the mutant population escapes initial extinction starting from size 1 is zero if $s(x',x)<0$, and is approximately equal to $s(x',x)/b(x',x)$ otherwise.

Altogether we obtain

$$w(x'|x)=\mu(x)\cdot b(x)\cdot \hat{n}_x\cdot M(x,x'-x)\cdot\frac{[s(x',x)]_+}{b(x',x)} \tag{2.3}$$

The quantity $[s(x',x)]_+$ is equal to $s(x',x)$ if $s(x',x)>0$ and to zero otherwise ; this means that only advantageous mutants, with positive invasion rate, have a non-zero chance of getting established. Up to first order in the mutation step size $x'-x$ we further have

$$\frac{s(x',x)}{b(x',x)}\approx\frac{1}{b(x)}\cdot(x'-x)\cdot\left.\frac{\partial s}{\partial x'}\right|_{x'=x}, \tag{2.4}$$

where we have used $s(x,x)=0$ since the population of phenotype *x* is at demographic equilibrium. If we assume the mutation process to be symmetric, and denote the variance of the mutation distribution by $\sigma^2(x)$, we can insert equation (2.3) together with equation (2.4) into equation (2.1) and compute the integral to obtain (Dieckmann and Law, 1996)

$$\frac{d}{dt}x=\left[\mu(x)\cdot\frac{\sigma^2(x)}{2}\cdot\hat{n}_x\right]\cdot\left.\frac{\partial s}{\partial x'}\right|_{x'=x} \tag{2.5}$$

which precisely conforms to equation (2.2). According to this deterministic approximation of adaptive dynamics, the evolutionary rate $\eta(x)$ of equation (2.2) is given by the bracketed product which encapsulates the influence of mutation. Most importantly, this derivation identifies the mutant invasion rate $s(x',x)$ as the appropriate measure of fitness denoted by $W(x',x)$ in equation (2.2). Therefore we call $s(x',x)$ the mutant *invasion fitness*.

Invasion fitness, ESS, CSS, and evolutionary branching

The selection derivative (Marrow *et al.*, 1992), $\partial s/\partial x'|_{x'=x}$, determines the direction of adaptive change. When the selection derivative is positive (negative), an increase (a decrease) of the trait value *x* will be advantageous in the vicinity of the resident trait value. Phenotypes that nullify the selection derivative are called *evolutionary singularities* and represent potential end-points for the evolutionary process. Yet careful inspection of stability properties of evolutionary singularities is required before conclusions can be drawn about the adaptive dynamics in their vicinity (Geritz *et al.*, 1998) :

- If invasion fitness presents a local maximum at an evolutionary singularity, then this singularity is an *evolutionarily stable strategy* (ESS), in the classical terminology of evolutionary biology.
- An ESS needs not be attainable : if the selection derivative increases near the ESS, any evolutionary trajectory starting nearby will actually be repelled away from the ESS. In this case, the ESS also is an *evolutionary repellor*.
- Conversely, a singularity may attract evolutionary trajectories and yet correspond to a fitness minimum. In this perhaps most remarkable case, selection is initially stabilizing and drives the population to a point where ecological interactions turn the selective regime into a disruptive one, and dimorphism evolves. This phenomenon is known as *evolutionary branching*. The canonical equation for adaptive dynamics provides an approximate models for evolutionary trajectories heading to a branching phenotype, but obviously fails to capture the population's further evolutionary dynamics.

SPATIAL INVASION FITNESS IN HOMOGENEOUS HABITATS

One conclusion to be drawn from the previous section is that the derivation of invasion fitness must be underpinned on an ecological model for the population dynamics. The definition of a fitness measure as a function of space-related traits therefore requires that spatial structure and local interactions are incorporated in the underlying ecological model.

Spatial population models

Spatial models fall into two main categories, depending on the continuous versus discrete structure of the habitat. Traditional models for continuous space (*reaction-diffusion* models ; see Okubo, 1980) run into serious biological inconsistencies, like the assumption that infinitely many 'nano-individuals' may live on arbitrarily small areas. It is only recently that two new types of mathematically sound and biologically consistent models were derived. *Hydrodynamics limit* models are spatially explicit ; akin to reaction-diffusion equations, they involve correction terms that account for local interactions and dispersal (Durrett and Levin, 1994). *Moment equations* are spatially implicit ; they describe the dynamics of the statistical moments of the distribution of individuals in space (Bolker and Pacala, 1999 ; Dieckmann and Law, 2000). To model spatial population processes over discrete space, there is a long tradition of *metapopulation models* (Levins, 1969 ; Hanski and Gilpin, 1997 ; Hanski, 1999, and references therein). Classical models of metapopulations are not truly spatial in the sense that they do not involve the notion of neighborhood ; dispersal is global, and all dispersing individuals are mixed in a common pool before being redistributed in patches irrespective to their location1. *Stepping-stone* models (Kendall, 1948 ; Kingman, 1969 ; Renshaw, 1986) assume that a set of finite populations is distributed on a regular lattice of patches. Dispersal takes place between neighboring patches. In the field of population genetics, stepping-stone models usually assume that all patches are saturated at their carrying capacity (Malécot, 1948, 1975 ; Kimura, 1953). *Lattice models* (Matsuda *et al.*, 1992 ; Morris, 1997 ; Rand, 1999) have been developed recently as another tool to modeling population dynamics in discrete space. Lattice models prescribe the possible locations of individuals on a network of sites, each site hosting at most one individual. There is no saturation assumption : all sites need not be occupied. Local interactions and local dispersal occur between any site and its neighborhood of connected sites. Like moment equations, lattice models are spatially implicit, and they aim at describing neighbor-range spatial correlations.

When it comes to deriving a measure of invasion fitness from these ecological models, operational results are scant. So far no invasion criterion could be established rigorously for models of hydrodynamics limits or moments. Invasion fitness in metapopulations has been worked out by Olivieri *et al.* (1995) and in greater generality by Metz and Gyllenberg (2000). However, as we already pointed out, such models do not account for limited dispersal and therefore address spatial processes in a rather special way. The study of interacting populations on stepping-stone models remains very limited. Only lattice models have led to a rigorous mathematical definition of invasion fitness in space (van Baalen and Rand 1998), and it is this type of models that we shall consider further in the rest of this chapter.

Modeling the spatial dynamics of population lattices

The population is distributed over an infinite network, or lattice, of connected sites (Fig. 1). A site contains at most one individual. Connections have two meanings : interactions (social, competitive, parasitic, etc.) may occur only between individuals that inhabit connected sites, and movement may occur only from a given site to a connected site. This has the important consequence

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1 For the sake of completeness, we should mention the so-called two-patch or n-patch models frequently used (possibly overused) to describe local population regulation by means of simple nonlinear density-dependence (like the Ricker map). For examples and corresponding references, see chapter 3 in Hanski (1999). Unfortunately, as they treat the densities of local populations as continuous variables, they have to rely on the rather unsatisfactory premise that local population size is infinite.

that the spatial scale is the same for dispersal and interactions. For simplicity, we shall assume that each site is connected to the same number (n) of neighboring sites (e.g. a regular lattice). Each site is in one of several, finitely many possible states : empty, or occupied by an individual of one out of N possible types. The configuration of the whole lattice is given by the states of all sites. The lattice configuration changes as a result of two types of events potentially affecting any site during any short time interval : birth or immigration of an individual from a neighboring site, and death or emigration of the individual occupying a site. In general, dispersal (emigration-immigration) is not restricted to the newborn class.

We aim to describe the temporal dynamics of the frequencies of sites that are empty and sites that are occupied by any given phenotype (Matsuda *et al.*, 1992 ; Rand, 1999). The probability that the state of a site changes depends not only on its current state but also on the state of neighboring sites, for two different reasons. On the one hand, dispersal and birth are local events whose realization is conditional to the availability of empty sites in the neighborhood. The likelihood that an individual in a given site moves or exports its offspring is proportional to the frequency of empty sites in her neighborhood. On the other hand, local interactions with neighbors will affect the birth rate and death rate of any focal individual. For example, individuals might negatively affect each other's birth rate through local competition for food. In this case, the birth rate could be seen as a decreasing function of the number of neighbors.

Therefore the *frequency of sites in state i* among all sites of the lattice, p_i, must depend on the neighborhood structure as described by a second-order statistic for the distribution of the configurations of all pairs of nearest-neighbor sites. The dynamics of pair configurations depends in turn on the state of triplets including the pairs' neighbors, and so on. A full description of the lattice

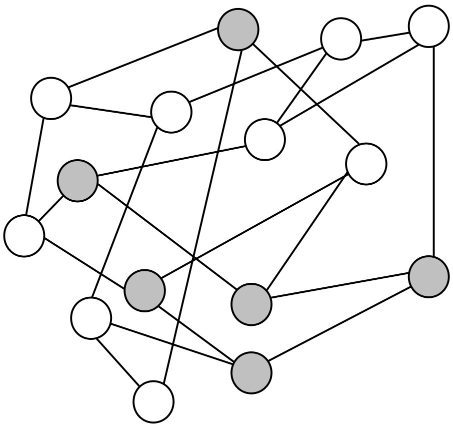

Figure 1. Example of a small random lattice. Each site is randomly linked to a fixed number n of other sites. Here $n = 3$. Dark circles are occupied sites, open circles are empty sites.

dynamics eventually requires an infinite hierarchy of statistics, each one describing the spatial structure on a particular scale (sites, pairs, triplets and so on) in relation to the immediately subsequent one (Morris, 1997). To make a model tractable, one has to choose a particular scale of description, and make appropriate approximations to close the exact, infinite system at that scale. This means that the frequencies of configurations beyond the chosen spatial scale are estimated from the frequencies of configurations up to that scale. No mathematical procedure is currently available to systematically identify the scale at which the system should be closed, and the closure that should be applied in order to obtain the best approximation of the dynamics of the infinite-dimensional model. This will depend on the particular model under consideration and of the biological motivation guiding the analysis (Morris, 1997 ; Dieckmann and Law, 2000).

Our aim is to describe the dynamics of lattices at the most local scale, that of pairs of nearest neighbors. Pair-dynamics models can account for the effect of spatial correlations which arise at a local scale and vanish quickly, although they are not concerned with the development of large-scale spatial structures. It should be noticed that, at least for regular lattices, one may straightforwardly recover the frequencies of sites in the various states (i.e., the p_i values) simply by adding the appropriate pair frequencies. Pair-dynamics models offer a handy compromise between the need to incorporate and describe some of the spatial complexity of the population dynamics, and the aim of deriving useful analytical results on population equilibrium and invasion conditions. The pair-dynamics approach has been used to construct appropriate correlation equations for plant dynamics models (Harada and Iwasa, 1994; Satō and Konno, 1995), spatial games (Morris, 1997 ; Nakamaru *et al.*, 1997), social interactions (Matsuda *et al.*, 1992 ; Harada *et al.*, 1995 ; van Baalen and Rand, 1998) and epidemic models (Keeling, 1996 ; Morris, 1997). In the case of a spatial game on a regular lattice, however, Morris (1997) showed that the pair-dynamics description could fail dramatically. Then moving up to the triplet dynamics is often sufficient to obtain a substantial improvement in the closure accuracy.

From individuals to pair dynamics and correlation equations

We define p_{ij} as the frequency of pairs of nearest-neighbor sites, one being in state *i*, and the other in state *j*. Such a pair is denoted by (i,j), and the frequency p_{ij} is calculated over all pairs[2] in the lattice. We shall take four heuristic steps in order to derive the so-called correlation equations ; that is, a set of nonlinear differential equations that describe the lattice dynamics at the spatial scale of pairs. The four steps are :

(1) Write the rates of local events for anchored pairs. We call *anchored pair* one that contains a given site *z* occupied by an individual in a specified state *i*. By definition, *local pair events* affect

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[2] Note that the pairs are symmetric, which implies $(i,j)=(j,i)$.

anchored pair, and are triggered by a site event at the anchored site *z* (see Fig. 2). Four local events have to be considered.

(2) Average the rates of local events for anchored pairs calculated at Step 1 over all sites *z* in state *i*.

(3) Calculate the rate of change of the frequency of all (i, j) pairs by bookkeeping all possible transitions of anchored pairs that may create or destroy an (i, j) pair.

(4) Apply an appropriate closure procedure designed to approximate all statistics involving triplets in terms of statistics for pairs.

Notations are introduced in Table 1. (See Morris, 1997, and Rand, 1999, for a rigorous account of all mathematical details involved).

Table 1. Variables and parameters of the lattice model

N	Set of all phenotypes present in the population
z	Generic notation for the location of a site in the graph
i, j, k	Generic notations for different site states
p_i	Frequency of sites in state *i* among all sites (*site frequency*)
p_{ij}	Frequency of (i, j) pairs among all pairs of sites (*pair frequency*)
$q_{i:j}$	Probability that next to a site in state *j*, there is a site in state *i* (*aggregation coefficient*)
$q_{i:jk}$	Probability that next to a site in state *j* in a (j, k) pair, there is a site in state *i*
n	Number of neighboring sites to any given site (constant)
$n_{k:ij}(z)$	Number of sites in state *k* in the neighborhood of a type *i* at site *z* in a (i, j) pair
ϕ	Probability to draw a connection at random uniformly among all connections to any given site ($\phi = 1/n$)
$b_i(z)$	Intrinsic *per capita* birth rate at location *z*
$d_i(z)$	Intrinsic *per capita* death rate at location *z*
$m_i(z)$	Intrinsic *per capita* dispersal rate of type *i* at location *z*
$E_{ij}^b(z)$	Additive effect (competition, cooperation) on the per capita birth rate of a type *i* individual located at *z* induced by interaction with a type j individual located in the neighborhood
$E_{ij}^d(z)$	Additive effect on the per capita death rate of a type *i* individual located at *z* induced by interaction with a type *j* individual located in the neighborhood
$C_i^b(z)$	Cost of type i strategy impacting the birth rate of a type i individual located at *z*
$C_i^d(z)$	Cost of type i strategy impacting the death rate of a type i individual located at *z*

Step 1. Transition rates for anchored pairs. We define the anchored pair $(i \in z; j \in z')$ to be the pair spanning the sites located at *z* and *z'*, and hosting a type *i* individual in site *z* while site *z'* is in

state *j*. We consider the four local events that can affect such a pair as a result of an individual event occuring at *z* (Fig. 2) : a birth event at *z* when *j* is the empty state; two mortality events affecting the *i* individual at *z*, differing in the presence or absence of an individual at *z'* ; a dispersal event from *z* to *z'*, assuming *z'* to be empty. The individual birth rate, death rate, and dispersal rate involve three additive components : an intrinsic, baseline rate that may depend on the individual's phenotype, an interaction term that measures the effect of neighbors, and a cost term that depends on the individual's phenotype. To calculate the rate of local events we must introduce the number $n_{k:ij}(z)$ of neighboring sites in state *k* next to the *z* site of an anchored pair $(i\in z; j\in z')$. We simply add the contributions to the event rate affecting the *i* individual at *z* resulting from all possible configurations of the neighborhood of site *z*. The per-capita rate of the birth and dispersal local events should be scaled by ϕ , the inverse neighborhood size. This reflects the fact that a birth or dispersal event affecting at a given rate a focal individual that belong to *n* pairs, will affect any of these pairs at a rate *n* times slower ; in contrast, a death event at *z* will concomitantly affect all *n* pairs containing *z*. Altogether this yields the following rates for each of the transitions depicted in Fig. 2 :

$$\phi\tilde{b}_i(z)=\phi\left(b_i(z)+\sum\nolimits_{k\in N}E^b_{ik}(z)n_{k:io}(z)-C^b_i(z)\right) \tag{3.1a}$$

$$\tilde{d}_{ij}(z)=d_i(z)+E^d_{ij}(z)+\sum\nolimits_{k\in N}E^d_{ik}(z)n_{k:ij}(z)+C^d_i(z) \tag{3.1b}$$

$$\tilde{d}_{io}(z)=d_i(z)+\sum\nolimits_{k\in N}E^d_{ik}(z)n_{k:io}(z)+C^d_i(z) \tag{3.1c}$$

$$\phi\,\tilde{m}_i(z)=\phi m_i \tag{3.1d}$$

Notice that for the sake of simplicity, we have assumed that the intrinsic dispersal rate $m_i(z)$ of any focal individual was merely equal to the intrinsic dispersal rate. There is no conceptual predicament, however, to extend the model and make dispersal conditional on the neighborhood composition (Rand, 1999).

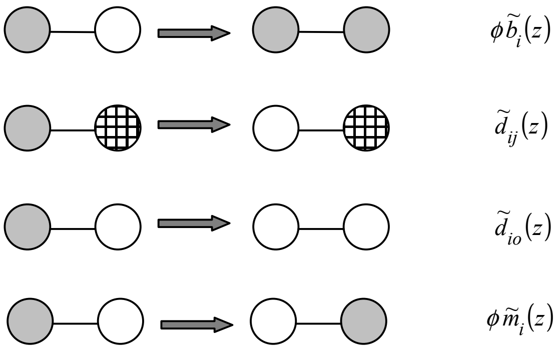


Figure 2. The four local pair events and their rate. Open circles are empty sites. Each dark circle is occupied by a type *i* individual. Hatched circles are in state *j*. See text for notations and explanations.

Step 2. Averaging transition rates for anchored pairs over the lattice. Assuming that the lattice is homogeneous, we can take the intrinsic rates, the interaction effects and the costs of interaction to be independent of the location *z* of any focal individual, and set $b(z) \equiv b$, $d(z) \equiv d$, $m(z) \equiv m$, $E_{ij}^{b}(z) \equiv E_{ij}^{b}$, $E_{ij}^{d}(z) \equiv E_{ij}^{d}$, $C_{i}^{b}(z) \equiv C_{i}^{b}$ and $C_{i}^{d}(z) \equiv C_{i}^{d}$. Transition rates for anchored pairs given by equations (3.1) are still influenced by the local configurations of the lattice, through the neighborhood-structure terms $n_{k:ij}(z)$ which depend on the location *z*. *Local fluctuations* caused by demographic stochasticity induce spatial variations in the neighborhood structure. If we would know at any time the state of every site *z*, then we could calculate each $n_{k:ij}(z)$ and obtain all transition probabilities for each anchored pair. However, the large number of sites makes this endeavor hopeless. Instead we aim at deriving average transition rates for anchored pairs across the lattice. We first compute an average measure of the neighborhood structure, $\overline{n}_{k:ij} = \sum n_{k:ij}(z) / |\, i \,|$, calculated as the total number $|\, i \,|$ of sites in state *i* is very large ; the sum is taken over all sites *z* that hosts a type *i* individual belonging to a (i, j) pair. Likewise we define $q_{k:ij}$ as the average proportion of sites in state *k* in the neighborhood of a site in state *i* within a (i, j) pair ; in other words, $q_{k:ij}$ is the conditional probability of having a site in state *k* in the vicinity of a site in state *i*, given that one of the latter's neighboring site is in state *j*. Since a focal site in an anchored pair is connected to *(n-1)* sites outside that pair, we have $\overline{n}_{k:ij} = (n-1) q_{k:ij}$. This averaging procedure applied to all local pair-events rates, equations (3.1), eventually yields the following average rates :

$$\phi \overline{b}_{i} = \phi \left(b_{i} + \sum\nolimits_{k \in N} E_{ik}^{b} (n-1) q_{k:io} - C_{i}^{b} \right) \tag{3.2a}$$

$$\overline{d}_{ij} = d_{i} + E_{ij}^{d} + \sum\nolimits_{k \in N} E_{ik}^{d} (n-1) q_{k:ij} + C_{i}^{d} \tag{3.2b}$$

$$\overline{d}_{io} = d_{i} + \sum\nolimits_{k \in N} E_{ik}^{d} (n-1) q_{k:io} + C_{i}^{d} \tag{3.2c}$$

$$\phi \overline{m}_{i} = \phi\, m_{i} \tag{3.2d}$$

Step 3. Pair transition rates and equations for pair dynamics. To compute the transition rates for all possible pairs, we have to complete the bookkeeping of all local pair events that may create or destroy any given pair, and use the average rates given by equations. (3.2). This is done in Box 1 for one particular type of pair, in the case of a lattice where there are three possible states for a site : empty, or occupied by one of two types. Once all pair transition rates are available, it is straightforward to assemble a system of differential equations that govern the temporal dynamics of pair frequencies. It turns out that the combinations of rates that enter these equations can be simplified by making use of the following composite rates (van Baalen and Rand, 1998) :

$\alpha_{ij} = (1-\phi)(\overline{b}_{i} + \overline{m}_{i}) q_{i:oj}$ is the rate at which type i enters a pair (o, j) with $j \neq i$,

Box 1 . Derivation of pair dynamics

We consider a dimorphic population with two types of individuals, x and y. We do the bookkeeping of all possible transitions and their rates that may create or destroy (x,o) pairs. The frequency of this pair is affected by six potential events which can be grasped easily by mere graphical depiction (Fig. B1 ; also see van Baalen and Rand, 1998). The rate of each transition is computed by summing the appropriate average rates of local pair events.

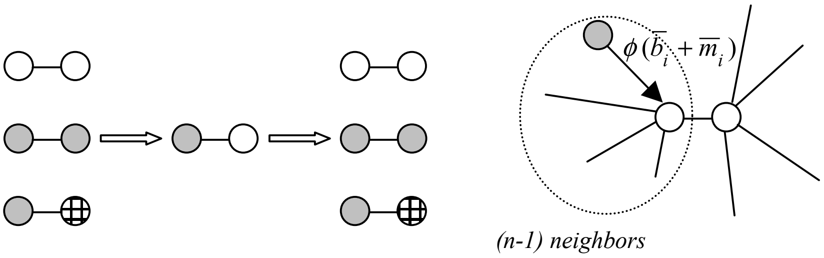


Figure B1. How local pair events affect the pair (x,o). (a) All possible transitions that may create and destroy the focal pair (in the middle). (b) An example of a local pair event showing how (x,o) can be created from a pair (o,o) : reproduction or dispersal occurs in an anchored pair that belongs to the neighborhood of one of the empty sites of the focal pair.

Pairs (x,o) are created by :

- the transition from (o,o), as illustrated in Fig. B1. There is on average $(n-1)q_{x:oo}$ anchored pairs (x,o) whose empty site belongs also to a pair (o,o) ; the empty pair (o,o) will be turned into an (x,o) pair by reproduction at the local pair-event rate $\phi\overline{b}_x$; and by dispersal at the rate $\phi\overline{m}_x$.
- the transition from (x,x) either due to death at rate $\overline{d}_{xx}$, or to movement towards a neighboring site. In the latter case there are $(n-1)q_{o:xx}$ anchored pairs that may undergo the corresponding transition, each at an average local pair-event rate $\phi\overline{m}_x$.
- and the transition from (x,y), which is calculated in a similar way.

Pairs (x,o) are destroyed by :

- the transition to (o,o) due to death at rate $\overline{d}_{xo}$, or to dispersal. Again we calculate the number of anchored pairs where this transition may take place to be $(n-1)q_{o:ox}$, and for each of them the transition occurs at the rate $\phi\overline{m}_x$.
- the transition to (x,x) due to reproduction within this pair at rate $\phi\overline{b}_x$, or due to a reproduction or dispersal event involving an *x* neighbor. The latter transition involves $(n-1)q_{x:ox}$ (x,o) anchored pairs which are affected by a local birth event at rate $\phi\overline{b}_x$ and by a local dispersal event at rate $\phi\overline{m}_x$.
- likewise, the transition to (x,y) involves $(n-1)q_{y:ox}$ anchored pairs (y,o), undergoing local birth at rate $\phi\overline{b}_y$ and local dispersal at rate $\phi\overline{m}_y$.

Collecting all these transition rates together, and using the notation $\overline{\phi}=(n-1)\phi$, we finally obtain the following rate of change for the pair frequency p_{xo} :

$$\begin{aligned}\frac{dp_{xo}}{dt}=&\left(\overline{b}_x+\overline{m}_x\right)\overline{\phi}\,q_{x:oo}p_{oo}+\left(\overline{d}_{xx}+\overline{\phi}\,\overline{m}_x\,q_{o:xx}\right)p_{xx}+\left(\overline{d}_{yx}+\overline{\phi}\,\overline{m}_y\,q_{o:yx}\right)p_{xy}\\&-\left(\phi\overline{b}_x+\overline{d}_{xo}+\overline{\phi}\,\overline{m}_x\,q_{o:xo}+\overline{\phi}\left(\overline{b}_x+\overline{m}_x\right)q_{x:ox}+\overline{\phi}\left(\overline{b}_y+\overline{m}_y\right)q_{y:ox}\right)\end{aligned}\qquad\text{(B1.1)}$$

$\beta_i = \phi \overline{b}_i + (1-\phi)(\overline{b}_i + \overline{m}_i) q_{i:oi}$ is the rate at which type *i* enters a pair (o,i),

$\delta_{ij} = \overline{d}_{ij} + (1-\phi)\overline{m}_i \, q_{o:ij}$ is the rate of loss of type *i* from (i,j) pairs.

We shall refer to these equalities as equations (3.3a), (3.3b), and (3.3c), respectively. It is also convenient to introduce the auxiliary parameter $\alpha'_{ij} = (1-\phi)(\overline{b}_i + \overline{m}_i)$.

Step 4. Closing the system. The equations for pair frequencies obtained at Step 3 involve the conditional probabilities $q_{k:ij}$. This implies that the system is not closed : the frequencies of pairs depend on the frequencies of triplets, and to avoid a cascade of dependency on even more complex configurations, the frequencies of configurations involved beyond pairs have to be approximated from the pairs. Finding an accurate approximation amounts to solving the « closure problem » posed by the dynamical system under concern.

The general form of such a pair approximation can be written as $q_{k:i}$, the probability that there is a site in state k next to a site in state *i*, plus an error term capturing an estimation bias due to local fluctuations (Morris, 1997). Different pair approximations have been developed, reflecting different ways of correcting for the neighborhood structure (Matsuda *et al.*, 1992; Van Baalen, 2000), the lattice regularity (Morris, 1997), and the distribution of local fluctuations (Morris, 1997). Ad hoc corrections accounting for the population clustering pattern have also been proposed (Satō *et al.*, 1994). In general, we can safely assume that an infinite random lattice, or a more regular lattice with weak aggregation, will produce a small bias. The standard pair-approximation (Matsuda *et al.*, 1992) precisely equals the bias to zero and therefore reads $q_{k:ij} \cong q_{k:i}$. It has been confronted to individual-based simulations in a number of models corresponding to various biological situations (Matsuda *et al.*, 1992 ; Harada and Iwasa, 1994 ; Satō and Konno, 1995 ; Kubo *et al.*, 1996 ; Nakamaru *et al.*, 1997 ; Iwasa, 2000). The match is often very good, but sometimes devastatingly bad. In such cases, moving up the description level to the spatial scale of triplets can suffice to improve matters substantially (Morris, 1997). Satō *et al.* (1994), Harada *et al.* (1995), Ellner *et al.* (1998), Morris (1997) and van Baalen (2000) have investigated the alternative path of deriving better pair approximations.

Here we shall content ourselves with the standard pair approximation and apply it to equations. (3.2) and (3.3). This yields

$$\phi \overline{b}_i = \phi \left(b_i + \sum\nolimits_{k \in N} E^b_{ik} (n-1) q_{k:i} - C^b_i \right) \tag{3.4a}$$

$$\overline{d}_{ij} = d_i + E^d_{ij} + \sum\nolimits_{k \in N} E^d_{ik} (n-1) q_{k:ij} + C^d_i \tag{3.4b}$$

$$\overline{d}_{io} = d_i + \sum\nolimits_{k \in N} E^d_{ik} (n-1) q_{k:i} + C^d_i , \tag{3.4c}$$

and

$$\alpha_{ij} = (1-\phi)\left(\overline{b}_i + \overline{m}_i\right)q_{i:o} = \alpha_i \tag{3.5a}$$

$$\beta_i = \phi\,\overline{b}_i + (1-\phi)\left(\overline{b}_i + \overline{m}_i\right)q_{i:o} \tag{3.5b}$$

$$\delta_{ij} = \overline{d}_{ij} + (1-\phi)\overline{m}_i\; q_{o:i} \tag{3.5c}$$

One can insert these approximate expressions into the system of differential equations for pair frequencies written with exact pair transition rates (see equation (B1.1)). If there is a single phenotype *x* in the population (resident phenotype), the dynamics of pairs obey the following system of so-called correlation equations (Rand 1999) :

$$\begin{pmatrix} dp_{ox}/dt \\ dp_{xx}/dt \end{pmatrix} = \begin{pmatrix} \alpha'_{xo}\, q_{o:o} - \left(\beta_x + \delta_{xo}\right) & \delta_{xx} \\ 2\beta_x & -2\delta_{xx} \end{pmatrix} \begin{pmatrix} p_{ox} \\ p_{xx} \end{pmatrix} \tag{3.6}$$

The equilibrium state of the population, fully characterized by $q_{o:x}$ and $q_{o:o}$, may then be found by solving the system $dp_{ox}/dt = 0$ and $dp_{xx}/dt = 0$.

Spatial invasion fitness

We now have the modeling machinery in place to tackle the calculation of invasion fitness, that is, a measure of the population growth rate of a mutant (phenotype *y*) introduced at low frequency in the resident population where only phenotype x is present. When two strategies *x* and *y* are represented in the population, there are six possible types of pairs. A simple bookkeeping procedure is applied to all possible transitions of these pairs, and the rates defined by equations. (3.3) are used to construct a system of correlation equations (3.7) :

$$\begin{pmatrix} dp_{ox}/dt \\ dp_{xx}/dt \\ dp_{oy}/dt \\ dp_{yx}/dt \\ dp_{yy}/dt \end{pmatrix} = \begin{pmatrix} \alpha'_{xo}\, q_{o:o} - \left(\beta_x + \alpha_y + \delta_{xo}\right) & \delta_{xx} & 0 & \delta_{yx} & 0 \\ 2\beta_x & -2\delta_{xx} & 0 & 0 & 0 \\ 0 & 0 & \alpha'_y\, q_{o:o} - \left(\beta_y + \alpha'_y\, q_{y:o} + \delta_{xo}\right) & \delta_{xy} & \delta_{yy} \\ 0 & 0 & \alpha_x + \alpha'_y\, q_{x:o} & -(\delta_{xy} + \delta_{yx}) & 0 \\ 0 & 0 & 2\beta_y & 0 & -2\delta_{yy} \end{pmatrix} \begin{pmatrix} p_{ox} \\ p_{xx} \\ p_{oy} \\ p_{xy} \\ p_{yy} \end{pmatrix}$$

The mutant rate of growth, denoted by $s(y,x)$, can be obtained by summing up the last three equations of system (3.5) :

$$\frac{dp_y}{dt} = \frac{dp_{oy}}{dt} + \frac{dp_{xy}}{dt} + \frac{dp_{yy}}{dt} = s(y,x)p_y \tag{3.8}$$

which after some algebra simplifies into :

$$s(y,x) = \overline{b}_y q_{o:y} - \overline{d}_y, \tag{3.9}$$

where

$$\overline{b}_y = b_y + (n-1)E^b_{yx} q_{x:y} + (n-1)E^b_{yy} q_{y:y} - C^b_y \tag{3.10}$$

$$\overline{d}_y = d_y + n\,E^d_{yx} q_{x:y} + n\,E^d_{yy} q_{y:y} + C^d_y. \tag{3.11}$$

Rearranging terms, we obtain the final expression :

$$\begin{aligned} s(y,x) = {} & \left[\left(b_y - C_y^b\right)q_{o:y} - d_y - C_y^d\right] \\ & + \left[(n-1)E_{yx}^b q_{o:y} - n\,E_{yx}^d\right]q_{x:y} + \left[(n-1)E_{yy}^b q_{o:y} - n\,E_{yy}^d\right]q_{y:y} \end{aligned} \tag{3.12}$$

This expression bears an interesting relationship to the notion of *direct* or *neighbor-modulated fitness* (Hamilton, 1970 ; Frank, 1998). Direct fitness is defined by summing the fitness effects on an individual caused by all the phenotypes of neighbors (including the individual itself). Likewise, spatial invasion fitness is obtained by adding the effects on a focal mutant of a resident or mutant neighbor weighted by the probability that the focal individual is neighbored by a resident or a mutant individual.

Further analysis based on spatial invasion fitness as defined by (3.12) requires that we solve equation (3.7) for $q_{y:y}$, $q_{x:y}$, and $q_{o:y}$. This can be done numerically by following the algorithmic recipe outlined in Box 2, or even analytically in the simplest cases (Matsuda *et al.*, 1992).

Box 2. A numerical recipe to compute spatial invasion fitness in lattice models

The expression of mutant population growth rate depends on the spatial statistics $q_{o:y}$, $q_{x:y}$ and $q_{y:y}$, which a priori vary over time. Yet the so-called *relaxation property* of the system entails that the statistics $q_{o:y}$, $q_{x:y}$ and $q_{y:y}$ converge very fast to equilibrium values, compared to the slow growth or decline of the system variables p_{oy}, p_{xy}, p_{yy} (Matsuda *et al.* 1992, our simulations). Therefore, to obtain a measure of spatial invasion fitness we may write an auxiliary system of differential equations for the variables $q_{o:y}$, $q_{x:y}$, $q_{y:y}$ only, solve it for equilibrium, and insert the result into equation (4.1).

The numerical derivation of this auxiliary system relies on the initial rarity of the mutant in the resident population. This, be definition, means : $q_{y:o} = 0$. This property allows us to write a closed model for the mutant pair dynamics, using the 3×3 lower-right block M of the transition matrix which appears in equation (3.5) :

$$\frac{d\vec{p}_y}{dt} = \mathbf{M}(\vec{q}_y)\vec{p}_y \text{ with } \vec{p}_y = (p_{oy}, p_{xy}, p_{yy}) \tag{B2-1}$$

Using the relations $dp_y \big/ dt = s(y,x)p_y$ and $\vec{p}_y = p_y\vec{q}_y$, we can further transform this system into

$$\frac{d\vec{q}_y}{dt} = \left[\mathbf{M}(\vec{q}_y) - s(y,x)\,\mathbf{I}\right]\vec{q}_y \tag{B2-2}$$

(I is the 3×3 identity matrix). At equilibrium, $d\vec{q}_y \big/ dt = 0$ and the spatial statistics $\vec{q}_y$ are obtained by solving (numerically or analytically in the simplest cases) the nonlinear system $M(\vec{q}_y)\vec{q}_y = \lambda\vec{q}_y$, which involves four unknowns ($q_{o:y}$, $q_{x:y}$, $q_{y:y}$, and the corresponding eigenvalue λ) and three equations, along with the constraint $q_{o:y} = 1 - q_{x:y} - q_{y:y}$. Solving for λ at the same time yields the numerical value of the spatial invasion fitness $s(y,x)$ (4.1).

APPLICATION : COADAPTATION OF DISPERSAL AND ALTRUISM

Empirical work has stressed the importance of spatial structure and spatial processes for the evolution of dispersal (Hanski, *this volume* ; Ims and Hjermann, *id.* ; Ronce *et al.*, *ibid.*). Coadaptation of other life history components is also expected to have a decisive influence on the evolution of dispersal, because of physiological and/or genetic correlations (Ronce *et al.*, *this volume* ; Roff and Fairbairn, *id.*) or behavioral alternatives (see Lambin *et al.*, *this volume*, for a discussion of the joint adaptation of dispersal, competition and cooperation). There is an urgent need for theory to incorporate these empirical facts.

The purpose of this section is to take a step forward in that direction. We make use of the framework of lattice population models to investigate the joint evolution of dispersal and social behavior while accounting explicitly for local interaction and dispersal processes. More specifically, our main objectives are (i) to identify selective pressures acting on these traits, (ii) to make predictions on their relative effects on the direction of evolution, and (iii) to relate them quantitatively to basic individual and interaction traits. The material presented here provides a short review of analyses expounded in Le Galliard (1999), Le Galliard *et al.* (*in prep.*), and Ferrière and Le Galliard (*in prep.*).

Model assumptions

We focus on two adaptive components of the individual's phenotype: dispersal and altruism (Table 2). The former trait is measured by the dispersal rate m. The altruistic trait is measured by the total investment in altruism u and the amount u/n of help an actor individual may distribute over its neighborhood. This amount affects any recipient's intrinsic birth rate additively. Note that this is a simplified description of altruism because individuals will have a total potential amount u to give and will always give the same amount of help per neighbor, whatever the number of receivers. In the biological realm, this would mean the absence of any kind of strategical distribution of altruism.

Both traits are costly to the bearer. A linear model for the cost of dispersal m is assumed, whereas the cost of altruism scales algebraically with the amount of total investment u (Table 2). The total cost is substracted from the intrinsic birth rate. The costs of dispersal and altruism are paid unconditionally, irrespective to the movement actually performed by the individual and the average amount of help actually given to the neighborhood. A representative biological instance would be an organism where both dispersal and altruism imply an initial ontogenetic shift towards a fixed physiological or morphological state that would determine the lifetime level of dispersal and altruism. This state would permanently impact the birth rate. This might be the case of a dispersal structure (O'Riain *et al.*, 1996).

Starting from the general model presented in section 3, we make two simplifying assumptions on our way to derive the measure of spatial invasion fitness : the intrinsic birth and death rates are independent of the phenotype, and costs and benefits impact the birth rate only. Referring to notations introduced in Table 1, this means : $b_i \equiv b$, $d_i \equiv d$, $E_{ij}^d \equiv 0$, $C_i^d \equiv 0$. We use the notation $C(u,m)$ to designate the total cost associated with altruism u and dispersal rate m, $C(u,m) = \kappa u^\gamma + \nu m$ (Table 2). Parameters κ and ν measure the sensitivity of the cost to altruism and dispersal. The parameter γ further indicates how the sensitivity of the cost to altruism varies with altruism. A high value of γ means that the cost of altruism increases slowly with altruism when altruism is low, and becomes more sensitive to altruism as altruism increases.

Table 2. Specific variables and parameters of the model

b	Intrinsic per capita birth rate (*b=2*)
d	Intrinsic per capita death rate (*d=1*)
m	Intrinsic per capita dispersal rate (*adaptive trait*)
u	Intrinsic per capita altruism rate (*adaptive trait*)
κu^γ	Cost of altruism impacting the birth rate
νm	Cost of dispersal impacting the birth rate

Adaptive dynamics of dispersal and altruism

Spatial invasion fitness *s* follows from the general model equation (3.10) and is given here by

$$s \equiv \left(b + u(1-\phi)q_{x:y} + u'(1-\phi)q_{y:y} - C(u',m')\right)q_{o:y} - d \qquad (4.1)$$

where $x = (u,m)$ denotes the resident phenotype, $y = (u',m')$, the mutant phenotype. The canonical equation (2.5) reads

$$\frac{d}{dt}\begin{pmatrix} u \\ m \end{pmatrix} = \begin{pmatrix} \eta \cdot \frac{\sigma^2}{2} \cdot p_x \cdot \left.\frac{\partial s}{\partial u'}\right|_{u'=u} \\ \eta \cdot \frac{\sigma^2}{2} \cdot p_x \cdot \left.\frac{\partial s}{\partial m'}\right|_{m'=m} \end{pmatrix} \qquad (4.2)$$

where η and σ^2 respectively denote the mutation rate and the mutation step variance, that we assume to be the same for both traits and independent of the current phenotypic mean. By making use of the facts that the resident population is at equilibrium and that the mutant is little different from the resident, a first-order approximation of spatial invasion fitness reads :

$$s/q_{o:y} \approx d\left(1/q_{o:x} - 1/q_{o:y}\right) + (1-\phi)q_{y:y}(u'-u) - \left[C(u',m') - C(u,m)\right] \qquad (4.3)$$

(see Le Galliard *et al.*, *in prep.*, for details). This expression clearly identifies three components of selection operating on dispersal and altruism. The first term in the right-hand side of equation (4.3) quantifies the pressure for reducing local competition for space. This pressure increases with the intrinsic death rate d : when mortality is low, there is little selective advantage to be gained from opening space by reducing altruism or increasing dispersal. The second term in equation (4.3) expresses the pressure for increased altruism under aggregated conditions ; the third term measures the pressure for reducing the direct costs of dispersal and altruism. By following the numerical recipe for the calculation of aggregation coefficients and spatial invasion fitness (Box 2), one can obtain explicit analytical expressions for $q_{y:y}$ and $q_{o:y}$. It is thus possible to write each component of selection as a function of individual parameters.

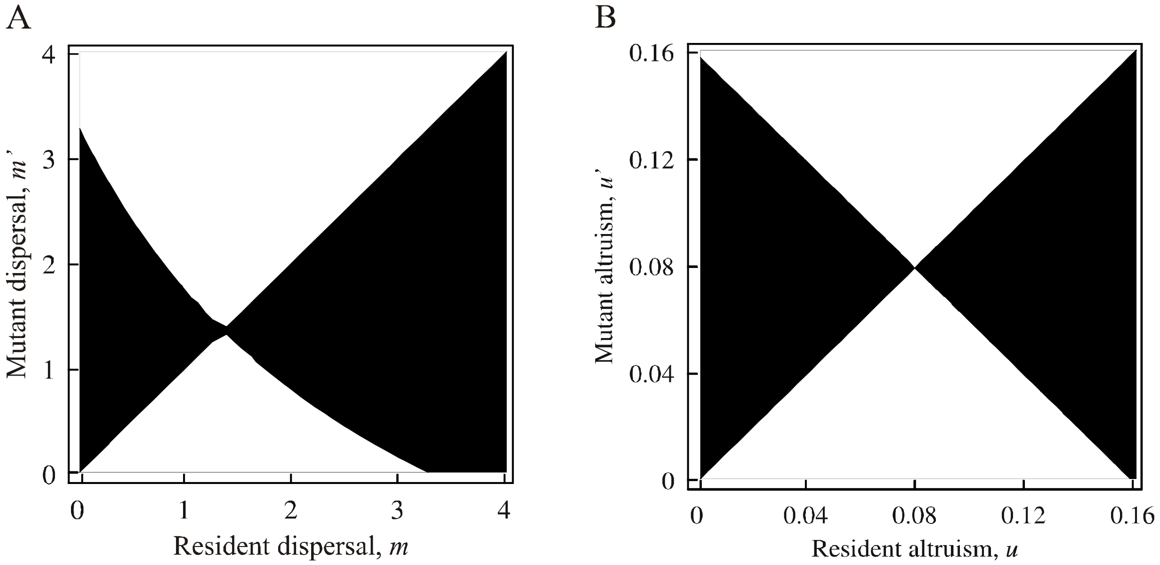


Figure 3. Pairwise invasibility plots when either the altruism trait or the dispersal trait is fixed. Spatial invasion fitness (see equation (4.1)) is positive in the dark region. A, Evolution of dispersal for fixed altruism ($u = 0.1$). B, Evolution of altruism for fixed dispersal ($m = 0.5$). In both cases, there is a single evolutionary singularity, which is attracting and evolutionarily stable. Parameter values : $n = 4$, $b = 2.0$, $d = 1.0$, $\gamma = 2.0$, $\kappa = 1.0$, $v = 0.1$.

In general, when the evolution of one trait alone is considered, the adaptive dynamics of the trait are monotonous and converge to a point attractor. This attractive point corresponds to a singularity of the adaptive dynamic, e.g. a point where the selection derivative vanishes. A mutant appearing around this phenotype value is actually counterselected and cannot invade (Fig. 3). The pattern of stabilizing selection is well explained by the relative effects of conflicting pressures. Focusing on the case of dispersal, we can see that at low dispersal, the predominant selective pressure is induced by local competition for space ; reduced aggregation is favored, and this selects for higher dispersal rates. As dispersal increases the intensity of the opposed selective pressure induced by the cost of dispersal also raises. An intermediate equilibrium value is reached at which both pressures exactly compensate each other. Numerical analysis of the dispersal rate at this attractor suggest that its value is mainly sensitive to the parameter v, which scales the cost of dispersal.

We now consider the coadaptive dynamics of dispersal and altruism. The selective gradient respective to either trait vanishes along the corresponding isocline (Fig. 4), which is the set of evolutionary singularities obtained for this trait, for each possible value of the other trait. Both isoclines cross at the singularity of the coadaptive dynamics, denoted by (m^*,u^*). When the cost of altruism is high and very sensitive to a change in the degree of altruism, the singularity is always a stable node (Le Galliard *et al.*, in prep.). The dispersal rate still converges monotonically to the singularity, but explaining the adaptive dynamics in the two dimensional trait space now requires that we consider how the three selective pressures interplay. This can be done by identifying the sign of each selection component locally in the direction of adaptation (Fig. 5). For example one can interpret the four trajectories (1-4) depicted in Fig. 4 in this way. (1) Starting from low dispersal and low altruism, mutants that invest more in altruism and dispersal are initially favored (selection components I and II are positive) ; being more altruistic is advantageous because the level of aggregation is high ; being slightly more mobile is also beneficial for it reduces local competition for space. In a second phase of the dynamics, mutants dispersing more are selected for (selective component I is positive) ; this reduces spatial aggregation and therefore promotes invasion by less altruistic phenotypes. (2) Initially dispersal is low and altruism is high. Only the first phase of the adaptive dynamics differs : here the adaptive dynamics begins with the reduction of the cost of altruism and the reduction of local competition for space (components III and I are positive). (3) Starting with a high dispersal-low altruism phenotype, selection favors an increase of altruism and a decrease of dispersal : at low altruism, mutants with lower dispersal rate pay a significantly reduced cost (component III is positive), and the benefit of more altruism in a population that develops more aggregation dominates the cost of increased local competition (component II is positive). (4) Finally, when ancestral dispersal and altruism are high, the selective pressure for reduced costs dominates (component III is positive) and drives the system all the way down to the singularity where both traits stabilize.

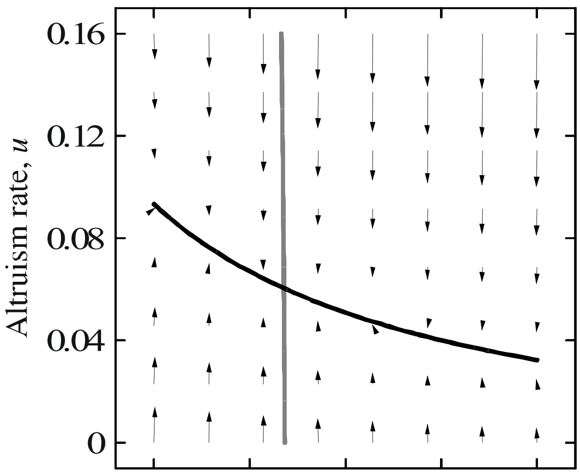


Figure 4. Co-adaptive dynamics of dispersal and altruism. Predictions from the canonical equation (4.2). Vector field : canonical equation of adaptative dynamics. Thin lines : evolutionary isoclines of altruism (black) and dispersal (gray). The crossing point of the isoclines gives the singularity, which is attracting and evolutionarily stable. Parameter values : same as in Fig. 3.

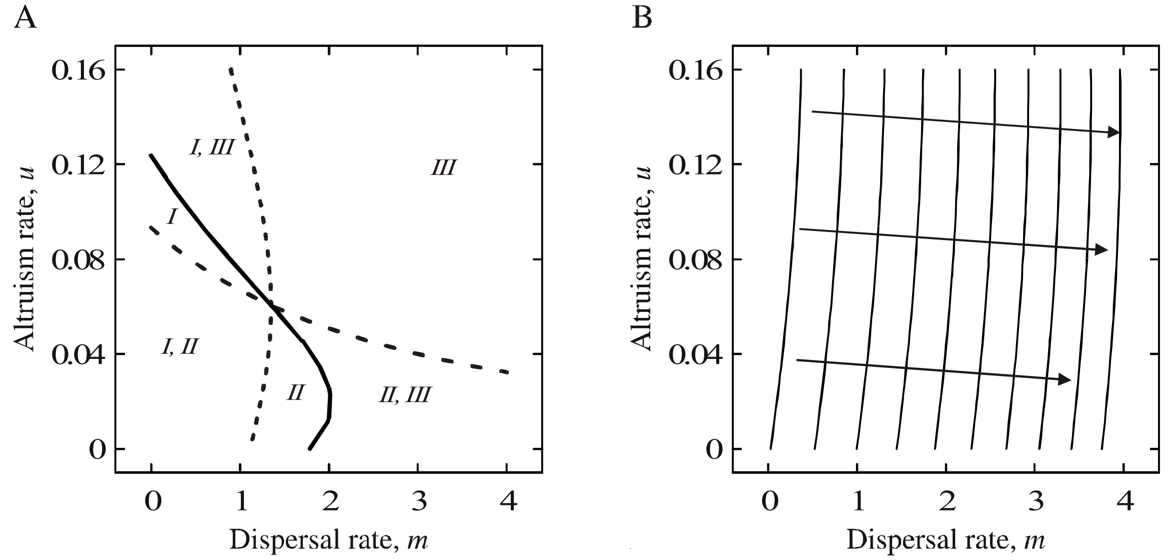


Figure 5. A, Zero-contour lines of the components of selection along adaptive trajectories. In each of the six delineated regions, positive pressures are indicated. Component I (dotted curve) : pressure for reducing local competition for space ; component II (dashed curve): pressure for increased altruism under aggregated conditions ; component III (continuous curve) : pressure for reducing the direct costs of dispersal and altruism. B, Spatial aggregation, shown as a contour plot of the aggregation coefficient $q_{x:x}$ for a pure population of phenotype *x*. Arrows indicate a decreasing aggregation coefficient. Parameter values : same as in Fig. 3.

Revisiting the Hamilton's rule

Hamilton (1964) formulated his famous rule according to which if an actor expresses a behavior that costs him C offspring and increases by B the number of individuals related to the actor, this behavior is selected for if $B\,r > C$. There has been much debate over the interpretation of the fitness costs C, benefits B, and of the *relatedness r* which make Hamilton's rule work, and by which this rule can be generalized for more complex ecological scenarios.

Defining and measuring relatedness in spatially structured populations is a longstanding problem of population genetics (Malécot, 1948 ; Rousset and Billiard, 2000). The spatial invasion condition provides a natural definition of relatedness as a measure of phenotypic correlation between neighbors (Frank, 1998 ; van Baalen and Rand, 1998). When altruistic and selfish individuals are identical in their basic demographic rates (*b*, *d*, and *m*), altruists with phenotype $y = (u', m)$ can invade non-altruists with phenotype $x = (0, m)$ if

$$u' \cdot (1-\phi) \cdot q_{y:y} > \kappa \cdot u'^{\gamma} \qquad (4.4)$$

that is, we have recovered a variant of Hamilton's rule in which $B \equiv u'$, $C = \kappa \cdot u'^{\gamma}$, and the coefficient of relatedness *r* is given by

$$r = (1-\phi) \cdot q_{y:y} . \qquad (4.5)$$

As already mentioned, $q_{y:y}$, and therefore *r*, can be computed from the invasion matrix (Box 2). This coefficient *r* estimates how much of an altruist's environment consists of other altruists, an interpretation that is consistent with Day and Taylor (1998). The precise interpretation of *B*, *C*, and *r* in Hamilton's rule, however, is dependent of the details of demographic processes operating in the population. For example, van Baalen and Rand (1998) note that if the cost of altruism is incurred as an increased mortality rate instead of a decreased birth rate, for zero dispersal the invasion condition of altruists in a selfish population becomes

$$u'\cdot(1-\phi)\cdot q_{y:y} > \left(b/d\right)\cdot \kappa \cdot u'^{\gamma}. \tag{4.6}$$

This provides another version of the Hamilton's rule where the cost *C* is recovered as the cost of altruism corrected for intrinsic birth and death rates. Other variants of the spatial Hamilton's rule, where relatedness similarly depends on local demographic processes, have been established by Ferrière and Michod (1995, 1996) for the invasion of cooperation in a spatial iterated Prisoner's Dilemma.

How kin selection models handle relatedness is usually problematic (Day and Taylor, 1998 ; Rousset and Billiard, *manuscript*). This is not to mean that kin selection is not the ultimate cause of the evolution of altruism in viscous populations, as Hamilton originally asserted (1964), but that measure of inclusive fitness may not correctly predict the evolutionary dynamics of social traits when selection is density dependent. Using spatial invasion fitness, Hamilton's principle is recovered as an emergent property of the model. This backs up Nunney's (1985) statement that kin selection is the only form of group selection that is able to maintain altruism.

Does spatial invasion fitness rightly predict evolutionary dynamics ?

Although our coevolutionary model of dispersal and altruism incorporates salient features of the ecological and evolutionary processes (including density-dependence, demographic stochasticity and evolutionary feedbacks), it remains underpinned on several critical simplifications. We assume an infinite lattice size, and describe the dynamics of local densities by making use of the pair-approximation (Morris, 1997). The derivation of the fitness measure relies on the small frequency of mutants as they originate and on the relaxation assumption that they instantaneously build up a characteristic cluster that may serve as a vehicle for the potential invasion process (Dieckmann and Law, 2000). Furthermore, the deterministic description of the adaptive dynamics gives an approximation for the mean path of the stochastic mutation selection-process (Dieckmann and Law, 1996), which itself already represents averaging over an infinite number of realizations.

Notwithstanding all this, the properties of stochastic simulations are remarkably well captured by the deterministic predictions (Fig. 6 ; see Le Galliard *et al.*, *in prep.*, for a thorougher comparison). The position of isoclines and the attracting singularity $\left(m^*,u^*\right)$ remain nearly unchanged. Overall trends of stochastic trajectories are correctly predicted by the deterministic model. Wilder fluctuations in trait values, involving the repeated rise and fall of altruism, are observed nearer to the singularity, as the selection gradient tends to weaken there. In our case, these complex regimes in the altruism level, which have received some attention elsewhere (Doebeli and Knowlton, 1999), are best explained by genetic drift in regions of low selection pressure across the phenotypic space.

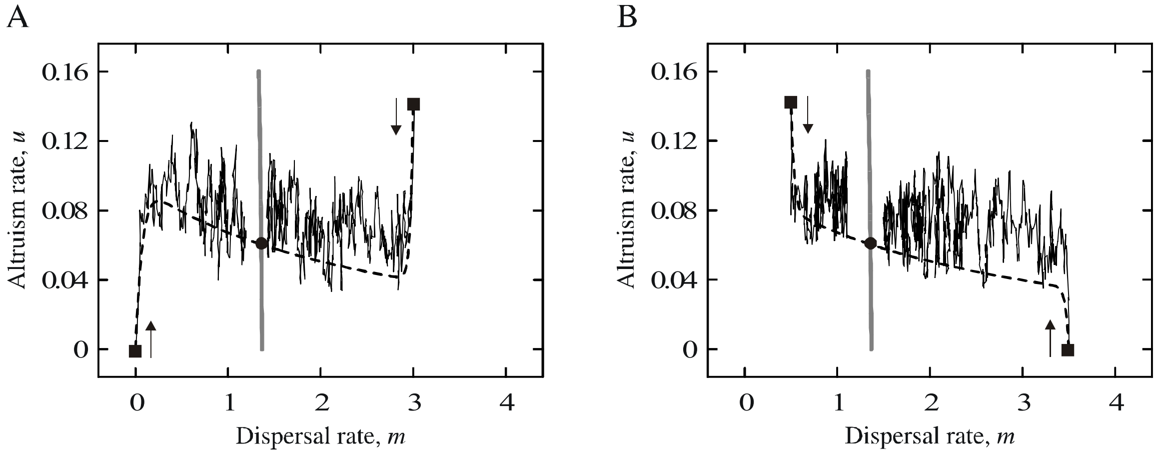


Figure 6. Mean trajectory of ten individual-based simulations of dispersal and altruism evolutionary dynamics. Grey line is the mobility isoclines predicted by the canonical equation (4.2) (see Fig. 4), black squares indicate initial states, dashed curves are predictions from the canonical equation and black dot is the singularity. A, Simulations of two trajectories starting at $(m,u)=(0,0)$ and $(m,u)=(3,0.15)$ respectively. B, Simulations of two trajectories starting at $(m,u)=(0.5,0.15)$ and $(m,u)=(3.5,\,0)$ respectively. The stochastic trajectories, although rather jerky near the convergence state at the isoclines intercept, hit rather close to it after following closely the deterministic path predicted by spatial invasion fitness. For both traits, the mutation rate is 10^{-2} and the mutation variance is 10^{-2}. Lattice size : 900 sites.

CONCLUDING REMARKS

Defining invasion fitness for spatial ecologies is no trivial matter. Starting from demographic and behavioral processes operating at the individual level and locally between close neighbors, the invasion exponent of a simple system of correlation equations for a mutant population dynamics provides a tractable solution to this problem. The notion of spatial invasion fitness allows one to *derive*, rather than postulate, an explicit relationship between distinct components of selection on the one hand, and the characteristics of the individuals and their interactions on the other. Numerical simulations of individual-based models confirm that the resulting spatial invasion fitness correctly predicts the dynamics of the stochastic mutation-selection process. On the empirical side, Rainey and Travisano's (1998), in their experiments on the evolution of polymorphism in bacteria, have shown that invasion fitness measured in spatially heterogeneous populations successfully predicts the maintenance of morphs diversity. In contrast, the destruction of local structures developed in the course of population growth alters the phenotypes' invasion fitnesses and modifies the eventual phenotypic composition of the population.

The mathematical derivation of spatial invasion fitness proceeds by averaging over space the transition rates of pairs. This amounts to look at the local structure of the mutant population as homogeneously replicated across the whole (infinite) lattice. The non-homogeneous distribution of the pairs containing mutants, induced by the finite size of the mutant population and the non-typical clustering pattern that may develop at the earliest stage on invasion, may also require to bring corrections to the measure of spatial invasion fitness. There may be an interesting parallel to be drawn with the theory of evolutionary games in continuous space. In this context the initial clustering of mutants entails that fitness should be defined not from space averages of individual traits, but as the speed at which the front of a mutant cluster moves forward and propagates mutants through space (Hutson and Vickers, 1992 ; Ferrière and Michod, 1995, 1996 ; Ellner *et al.* 1998).

We have used the notion of spatial invasion fitness to model the joint adaptive dynamics of dispersal and altruism. Even without further corrections for more subtle spatial effects, spatial invasion fitness appears to predict very well how these two behavioral traits coevolve. The analysis of this particular model underlines three important and general achievements of adaptive dynamics based on the notion of spatial invasion fitness. First, it unravels the interplay of the ecological (spatial) dynamics of a population and the evolutionary dynamics of the individual traits. The spatial structure shapes the selective pressures, which in return may alter the aggregation pattern. Here we have seen that a high degree of spatial aggregation is not a prerequisite for, but rather a consequence of the joint evolution of altruism and dispersal. Second, this analysis underlines important transient effects. A state of high dispersal or high altruism may be maintained transitorily, up to the point where the direction of selection changes or even reverts. In general, this means that variations, under the same environmental conditions and for the same species, of adaptive traits may be explained by different ancestral states and the observation of populations at different points in time in their evolutionary history. Finally, this approach allows to separate out distinct components of the selective regime and to express these components in terms of individual traits and characteristics of the population aggregation structure. In practice, there is potential here to predict how the selective pressures should equilibrate to produce patterns observed empirically, and how dispersal-related traits may respond to the experimental manipulation of each component of the selective regime.

Acknowledgements. Initially the chapter was planned to be co-authored with Denis Couvet, whose expertise in population genetics would have been critical to achieve a complete reinterpretation of traditional kin selection models in a spatially explicit context. Obviously the shot is longer than we thought, and we are still far away from the big integration of spatial ecology and population genetics. Yet discussions with Denis were most useful to delineate the critical issues and pitfalls in this enterprise. We ought to acknowledge him very gratefully. We also owe many thanks to Ulf Dieckmann who kindly wrote the C code which was used to produce the individual-based simulations presented in Section 4, and is contributing substantially to our ongoing research project on the coadaptative dynamics of altruism and dispersal. Many thanks also to Mats Gyllenberg, Laurent Lehman, Nicolas Perrin, François Rousset and Minus van Baalen for stimulating discussions and invaluable comments on a previous draft of this chapter, and to the Editors of this volume for their constant support and patience. Part of this work has been supported by the Adaptive Dynamic Network (IIASA, Laxenburg, Austria).

LIST OF REFERENCES

Bolker B.M., Pacala S.W. 1999. Spatial moment for plant competition: understanding spatial strategies and the advantages of short dispersal. Am. Nat. 153:575-602.

Caswell H. 1989. *Matrix Population Models*. Sinauer.

Day T., Taylor P.D. 1998. Unifying genetic and game theoretic models of kin selection for continuous traits. J. Theor. Biol. 194:391-407.

Dieckmann U., Law R. 1996. The dynamical theory of coevolution: a derivation form stochastic ecological processes. J. Math. Biol. 34:579-612.

Dieckmann U., Law R. 2000. Relaxation projections and the method of moments. In *The geometry of ecological interactions: simplifying spatial complexity* (Dieckmann U., Law R., Metz J.A.J. eds.). Cambridge Univ. Press.

Dieckmann U., Law R., Metz J.A.J. (eds.). 2000. *The geometry of ecological interactions: simplifying spatial complexity*. Cambridge Univ. Press.

Doebeli M., Knowlton N. 1998. The evolution of interspecific mutualisms. Proc. Natl. Acad. Sci. USA 95:8676-8680.

Durrett R., Levin S. 1994. The importance of being discrete (and spatial). Theor. Pop. Biol. 46:363-394.

Ellner S.P., Sasaki A., Haraguchi Y., Matsuda H. 1998. Speed of invasion in lattice population models: pair-edge approximation. J. Math. Biol. 36:469-484.

Ferrière R., Clobert J. 1992. Evolutionarily stable age at first reproduction in a density-dependent model. J. Theor. Biol. 157:253-267.

Ferrière R., Gatto M. 1995. Lyapunov exponents and the mathematics of invasion in oscillatory or chaotic populations. Theor. Pop. Biol. 48:126-171.

Ferrière R., Michod R.E. 1995. Invading wave of cooperation in a spatial iterated prisoner's dilemma. Proc. Roy. Soc. Lond. B 259:77-83.

Ferrière R., Michod R.E. 1996. The evolution of cooperation in spatially heterogeneous populations. Am. Nat. 147:692-717.

Ferrière R., Belthoff J.R., Olivieri I., Krackow S. 2000. Evolving dispersal: where to go next? TREE 15:5-7.

Ferrière R., Le Galliard J.F. Cooperation and mobility: adaptive reversals and population suicide. *Manuscript in preparation*.

Frank S.A. 1998. *Foundations of social evolution.* Princeton Univ. Press.

Geritz S.A.H., Metz J.A.J., Kisdi E., Meszéna G. 1997. The dynamics of adaptation and evolutionary branching. Phys. Rev. Lett. 78:2024-2027.

Geritz S.A.H., Kisdi E., Meszena G., Metz J.A.J. 1998. Evolutionary singular strategies and the adaptive growth and branching of the evolutionnary tree. Evol. Ecol. 12:35-37.

Grafen A. 1979. The hawk-dove game played between relatives. Anim. Behav. 27:905-907.

Hamilton W.D. 1964. The genetical evolution of social behaviour I, II. J. Theor. Biol. 7:1-52.

Hamilton W.D. 1970. Selfish and spiteful behaviour in an evolutionary model. Nature 228:1218-1220.

Hamilton W.D., May R.M. 1977. Dispersal in stable habitat. Nature 269:578-581.

Hanski I., Gilpin M.E. (eds.). 1997. *Metapopulation biology. Ecology, genetics and evolution.* Academic Press.

Hanski I. 1999. *Metapopulation ecology*. Oxford Univ. Press.

Hanski I. 2001. Population dynamic consequences of dispersal in local populations and in metapopulations. In *Dispersal* (J. Clobert, E. Danchin, A.A. Dhondt, and J.D. Nichols eds.). Oxford Univ. Press.

Harada Y., Iwasa Y. 1994. Lattice population-dynamics for plants with dispersing seeds and vegetative reproduction. Res. Pop. Ecol. 36:237-249.

Harada Y., Ezoe H., Iwasa Y., Matsuda H., Satō K. 1995. Population persistence and spatially limited social interaction. Theor. Pop. Biol. 48:65-91.

Hutson V., Vickers G.T. 1992. Travelling waves and dominance ESSs. J. Math. Biol. 30:457-471.

Ims R., Hjermann D.Ø. 2001. Condition-dependent dispersal. In *Dispersal* (J. Clobert, E. Danchin, A.A. Dhondt, and J.D. Nichols eds.). Oxford Univ. Press.

Iwasa Y. 2000. Lattice models and pair approximation in ecology. In *The geometry of ecological interactions: simplifying spatial complexity* (Dieckmann U., Law R., Metz J.A.J. eds.). Cambridge Univ. Press.

Keeling M.J. 1996. *The ecology and evolution of spatial host-parasite systems.* Unpublished PhD Thesis. Univ. of Warwick.

Kendall D.G. 1948. On the role of variable generation time in the development of a stochastic process. Biometrika 35:316-330.

Kimura M. 1953. 'Stepping stone' model of population. Ann. Rep. Natl. Inst. Gen. Jap. 3:62-63.

Kingman J.F.C. 1969. Markov population processes. J. Appl. Prob. 6:1-18.

Kubo T., Iwasa Y., Furumoto N. 1996. Forest spatial dynamics with gap expansion: total gap area and gap size distribution. J. Theor. Biol. 180:229-246.

Lambin X., Aars J., Piertney S.B. 2001. Dispersal, intraspecific competition, kin competition and kin facilitation : a review of the empirical evidence. In *Dispersal* (J. Clobert, E. Danchin, A.A. Dhondt, and J.D. Nichols eds.). Oxford Univ. Press.

Le Galliard J.F. 1999. Local competition, cooperation and mobility. A theoretical and experimental approach. Unpublished Masters thesis, Univ. of Paris VI.

Le Galliard J.F., Ferrière R., Dieckmann U. The coevolutionary dynamics of altruism and dispersal. *Manuscript in preparation.*

Levins R. 1969. Some demographic and genetic consequences of environmental heterogeneity for biological control. Bull. Entomol. Soc. Am. 15:237-240.

Malécot G. 1948. *Les mathématiques de l'hérédité*. Masson.

Malécot G. 1975. Heterozygosity and relation ship in regurlarly subdivided populations. Theor. Pop. Biol. 8:212-241.

Marrow P., Law R., Cannings C. 1992. The coevolution of predator-prey interactions : ESSs and Red Queen dynamics. Proc. Roy. Soc. Lond. B 250:133-141.

Matsuda H., Ogita N., Sasaki A., Satō K. 1992. Statistical mechanics of population: the lattice Lotka-Volterra model. Progress in Theoretical Physics 88:1035-1049.

Metz J.A.J., Nisbet R.M., Geritz S.A.H. 1992. How should we define fitness for general ecological scenarios? TREE 7:198-202.

Metz J.A.J., Geritz S.A.H., Meszéna G., Jacobs F.J.A., van Heerwaarden J.S. 1996. Adaptive dynamics, a geometrical study of the consequences of nearly faithful reproduction. In *Stochastic and spatial structures of dynamical systems* (van Strien S.J., Verduyn Lunel S.M. eds.). North Holland.

Metz J.A.J., Gyllenberg, M. 2000. How should we define fitness in structured metapopulation models? Including an application to the calculation of evolutionarily stable dispersal strategies. Proc. Roy. Soc. Lond. B 268:499-508.

Morris, A.J. 1997. Representing spatial interactions in simple ecological models. Unpublished PhD thesis. Univ. of Warwick.

Nakamaru M., Matsuda H., Iwasa Y. 1997. The evolution of cooperation in a lattice-structured population. J. Theor. Biol. 184:65-81.

Nunney L. 1985. Group selection, altruism and structured-deme models. Am. Nat. 126:212-230.

Okubo A. 1980. *Diffusion and ecological problems*: mathematical models. Springer.

Olivieri I., Michalakis Y., Gouyon P.H. 1995. Metapopulation genetics and the evolution of dispersal. Am. Nat. 146:202-228.

O'Riain J.M., Jarvis J.U.M., Faulkes C.G. 1996. A dispersive morph in the naked mole-rat. Nature 380:619-621.

Rainey P.B., Travisano M. 1998. Adaptative radiations in a heterogenous environment. Nature 394:69-72.

Rand D.A. 1999. Correlation equations and pair-approximations for spatial ecologies. In *Advanced Ecological Theory: advances in principles and appl*ications (Mc Clade J.M. ed.). Blackwell.

Renshaw E. 1986. A survey of stepping-stone models in population dynamics. Adv. Appl. Prob. 18:581-627.

Renshaw E. 1991. *Modelling biological populations in space and time.* Cambridge Univ. Press.

Roff D.A., Fairbairn D.J. 2001. The genetic basis of dispersal and migration, and its consequences for the evolution of correlated traits. In *Dispersal* (J. Clobert, E. Danchin, A.A. Dhondt, and J.D. Nichols eds.). Oxford Univ. Press.

Ronce O. 1999. Histoires de vie dans un habitat fragmenté: étude théorique de la dispersion et d'autres traits. Unpublished PhD thesis. Univ. of Montpellier.

Ronce O., Olivieri I., Clobert J., Danchin E. 2001. Perspectives on the study of dispersal evolution. In *Dispersal* (J. Clobert, E. Danchin, A.A. Dhondt, and J.D. Nichols eds.). Oxford Univ. Press.

Rousset F., Billiard S. 2000. A theoretical basis for measures of kin-selection in subdivided populations : finite populations and localized dispersal. J. Evol. Biol. 13:814-825.

Satō K., Matsuda H., Sasaki A. 1994. Pathogen invasion and host extinction in lattice structured population. Evol. Ecol. 6:352-356.

Satō K., Konno N. 1995. Successional dynamic models on the 2-Dimensional Lattice Space. J. Phys. Soc. Japan 64:1866-1869.

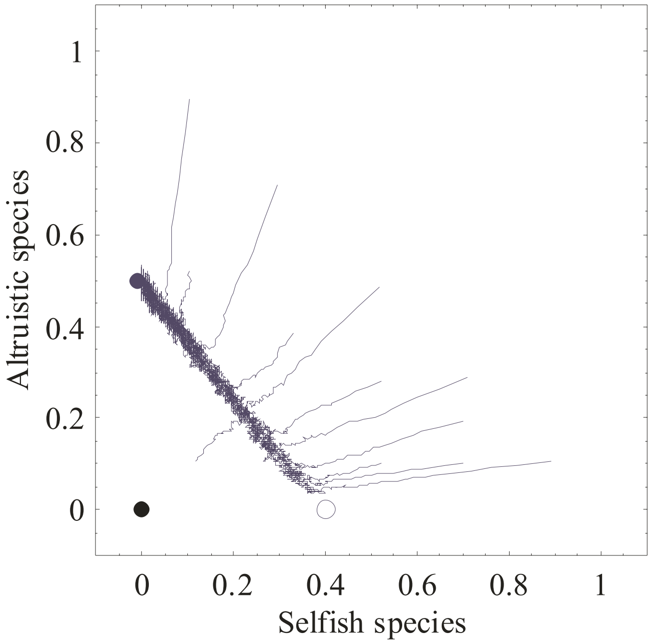
Tilman D., Kareiva P. 1998. *Spatial ecology: the role of space in population dynamics and interspecific interactions.* Princeton Univ. Press.

van Baalen M., Rand D.A. 1998. The unit of selection in viscous populations and the evolution of altruism. J. Theor. Biol. 193:631-648.

van Baalen M. 2000. Pair approximations for different spatial geometries. In *The geometry of ecological interactions: simplifying spatial complexity* (Dieckmann U., Law R., Metz J.A.J. eds.). Cambridge Univ. Press.

Wright S. 1931. Evolution in Mendelian populations. Genetics 16:96-159.

CHAPITRE 2 – EVOLUTION DE L’ALTRUISME



“With many natural populations it must happen that an individual form the center of an actual local concentration of his relatives which is due to a general inability or disinclination of the organisms to move far from their place of birth. In such a population, which we may provisionally term ‘viscous’, [kin selection] may apply fairly well to genes which affect vagrancy, [and we] would expect to find [cooperation] commonest and most highly developed in the species with the most viscous populations whereas uninhibited competition should characterize species with the most freely mixing populations.” W. D. Hamilton dans *The genetical evolution of social behaviour*. 1964.

LES DYNAMIQUES ADAPTIVES DE L’ALTRUISME DANS UNE POPULATION HETEROGENE DANS L’ESPACE

Jean-François Le Galliard, Régis Ferrière & Ulf Dieckmann

RESUME

Nous étudions les dynamiques adaptatives d’un trait continu qui mesure l’investissement dans l’altruisme. Notre étude repose sur un modèle écologique d’une population hétérogène dans l’espace à partir duquel nous dérivons une mesure appropriée de la valeur sélective. L’analyse de cette mesure de valeur sélective met en évidence trois processus sélectifs contrôlant l’évolution de l’altruisme: le coût physiologique direct, les bénéfices génétiques indirects de la coopération, et les coûts génétiques indirects de la compétition pour l’espace. En contraste avec les suggestions des études précédentes, nous trouvons que le coût génétique indirect de la compétition pour l’espace exerce une pression négligeable contre l’évolution de l’altruisme. Par ailleurs, notre étude fournit une classification des états adaptatifs de l’altruisme en fonction de la forme des coûts physiologiques de l’altruisme (avec une dépendance décélérante, linéaire ou accélérante à l’investissement altruiste). L’invasion de l’altruisme est aisée chez une espèce égoïste avec des coûts de type accélérant, mais des mutations à effets larges sont nécessaires chez des espèces à coûts de type décélérant. L’égoïsme stricte n’est maintenu que sous des conditions restreintes. Chez les espèces à coûts accélérant rapidement, l’adaptation conduit à un taux d’investissement altruiste évolutivement stable qui décroît continûment avec le niveau de mobilité de l’espèce. Un régime adaptatif différent émerge chez les espèces à coûts accélérant lentement : un altruisme fort évolue pour une mobilité faible de l’espèce, alors qu’un état quasi-égoïste est sélectionné pour une mobilité forte de l’espèce. Le niveau d’altruisme fort peut être prédit sur la base des paramètres caractérisant la connectivité de l’habitat et les coûts physiologiques de l’altruisme. Nous montrons aussi que des changements environnementaux qui favoriseraient une augmentation de la mobilité chez des espèces fortement altruistes peuvent provoquer l’auto-extinction de l’espèce par un processus de suicide adaptatif. Ceci pourrait contribuer à la rareté des espèces sociales.

Référence : Le Galliard, J.-F., Ferrière, R. et U. Dieckmann. 2003. « The adaptive dynamics of altruism in spatially heterogeneous populations ». *Evolution* 57(1): 1-17.

THE ADAPTIVE DYNAMICS OF ALTRUISM IN SPATIALLY HETEROGENEOUS POPULATIONS

Jean-François Le Galliard, Régis Ferrière & Ulf Dieckmann

ABSTRACT

We study the spatial adaptive dynamics of a continuous trait that measures individual investment in altruism. Our study is based on an ecological model of a spatially heterogeneous population from which we derive an appropriate measure of fitness. The analysis of this fitness measure uncovers three different selective processes controlling the evolution of altruism: the direct physiological cost, the indirect genetic benefits of cooperative interactions, and the indirect genetic costs of competition for space. In contrast with earliest suggestions, we find that the cost of competing for space with relatives exerts a negligible selective pressure against altruism. Our study yields a classification of adaptive patterns of altruism according to the shape the of costs of altruism (with decelerating, linear, or accelerating dependence on the investment in altruism). The invasion of altruism occurs readily in species with accelerating costs, but large mutations are critical for altruism to evolve in selfish species with decelerating costs. Strict selfishness is maintained by natural selection only under very restricted conditions. In species with rapidly accelerating costs, adaptation leads to an evolutionarily stable rate of investment in altruism that decreases smoothly with the level of mobility. A rather different adaptive pattern emerges in species with slowly accelerating costs: high altruism evolves at low mobility, whereas a quasi-selfish state is promoted in more mobile species. The high adaptive level of altruism can be predicted solely from habitat connectedness and physiological parameters that characterize the pattern of cost. We also show that environmental changes that cause increased mobility in those highly altruistic species can beget selection driven self-extinction, which may contribute to the rarity of social species.

Reference : Le Galliard, J.-F., Ferrière, R. and U. Dieckmann. 2003. « The adaptive dynamics of altruism in spatially heterogeneous populations ». *Evolution* 57(1): 1-17.

Key-words : Adaptive dynamics, altruism, mobility, spatial heterogeneity, relatedness, kin competition, kin selection.

Altruism is a cooperative behavior by which a donor individual increases a recipient's fitness at the cost of its own fitness. Major progress in the study of the evolution of altruism has been made over the last decade on both theoretical and empirical sides. On the theoretical side, models have gained a significant dose of realism from the explicit inclusion of spatial factors and the consideration of conditional behavior (Nowak and May 1992; Ferrière and Michod 1995; Roberts and Sherrat 1998). On the empirical side, ecological and genetic determinants of altruistic behavior have started to be identified, and physiological costs and benefits have been measured (Crespi 1996; Bourke 1997; Cockburn 1998; Heinsohn and Legge 1999). However, the merging between theory and facts has led to conflicting interpretations of processes and patterns in the evolution of altruism.

Two main processes have been put forward to explain the evolution and maintenance of altruism: kin selection (Hamilton 1964) and reciprocity (Trivers 1971; Axelrod and Hamilton 1981). Kin selection initially met with great success in explaining empirical observations (for example in social insects). However, recent theoretical developments, based on spatially implicit models, have pointed out a critical issue in this framework: the deleterious effects of kin competition should cancel out the indirect benefits of an altruistic behavior, thereby preventing the evolution of altruism (Taylor 1992a, 1992b; Wilson et al. 1992; Queller 1992, 1994). In contrast, game-theoretic spatial models involving conditional reciprocity have shown that reciprocal altruism evolves readily in spatially heterogeneous populations (Nakamaru et al. 1997, 1998). These theoretical findings are altogether in sharp contrast with empirical advances. On the one hand, unequivocal evidence is lacking for the expectedly widespread occurrence of reciprocal altruism, and reciprocity involves already fairly elaborate behavioral mechanisms (e.g., memory of past interactions) that are unlikely to be relevant to our understanding of the evolution of primitive forms of altruism (Pusey and Packer 1997). On the other hand, kin selection is still regarded as essential to explain the transition from selfish to cooperative units at all levels of biological organization (Maynard-Smith and Szathmary 1995), and many empirical examples of the specific transition from solitary to social life in animals seem indeed to fall under the scope of kin selection (Bourke 1997; Emlen 1997).

Widely different patterns of altruistic behavior have been described in a large array of taxa spread across bacteria, slime moulds, arachnids, insects and vertebrates. Levels of altruism, as described by the qualitative and quantitative natures of the investment by donors and benefit to recipients, have been found to vary between different species (Edwards and Naeem 1993; Crespi 1996) or within the same lineage across evolutionary time (Jarvis et al. 1994; Wcislo and Danforth 1997), between different populations within the same species (Spinks et al. 2000) and between groups of individuals within the same population (Cockburn 1998; Velicer et al. 2000; Strassmann et al. 2000). Most models have aimed at understanding how altruism can evolve in a selfish world, and how altruists can persist in the face of cheaters that reap the benefits of altruism while providing less or no help. Yet little theory is currently available to probe the adaptive significance of such variation in patterns of altruism and to identify physiological, ecological and genetic determinants.

This study offers a theoretical framework to address these tensions. We consider a population of asexual organisms that live in a spatially homogeneous, temporally constant habitat where competition and cooperation take place between kin and non-kin neighbors (van Baalen and Rand 1998). First, we address the robustness of previous investigations that questioned the role of kin selection for the evolution of altruism. To this end, we relax two of their critical assumptions. A dose of movement may help export the local benefits of altruism. Thus, we expect the conclusion that kin competition cancels kin cooperation to be sensitive to the inclusion and intensity of individual mobility (Queller 1992). We therefore relax the "pure viscosity" hypothesis according to which individuals (except offspring) are sessile (Hamilton 1964; Taylor 1992a,b). In our model, offspring are born locally, but in contrast with most viscous population models, individuals move during their lifetime (van Baalen and Rand 1998). Also, most kin selection models assume that the population is saturated and constantly maintained at carrying capacity. This lack of environmental "elasticity" might prevent the spread of altruism (Queller 1992, Mitteldorf and Wilson 2000). To overcome this restriction, we assume that population regulation arises locally from the limited empty space being available for offspring. In our model, the habitat is not saturated, because occupied sites coexist with empty sites generated by demographic stochasticity (Ferrière and Le Galliard 2001).

Second, we want to understand adaptive variation in altruism from basic physiological, ecological and genetic properties that could be documented in natural populations. This is achieved by assuming that altruism is not an all-or-nothing behavior and is better modeled as a quantitative trait that measures the amount of time, energy or resources invested in the altruistic function (Doebeli and Knowlton 1998; Roberts and Sherrat 1998; Koella 2000). At the physiological level, populations or species may differ according to the pattern of energy allocation to altruism versus other costly functions (Heinsohn and Legge 1999). We therefore assume that the physiological cost of altruism relates quantitatively to the actual altruistic investment. However, in contrast with previous models (Doebeli and Knowlton 1998; Roberts and Sherrat 1998), we envision three alternative costs patterns: a decelerating, a linear and an accelerating dependence of costs on investment in altruism. At the level of ecology, populations or species may also differ with respect to interaction structure. Two determinants of this structure are habitat connectedness and individual mobility (Ferrière and Michod 1995, 1996; Nakamaru et al. 1997; Frank 1998). Introducing specific parameters for these two factors allows us to investigate their effect on the adaptive evolution of altruism. At the level of the genetic processes, mutation rates and mutational effects determine the population phenotypic diversity upon which selection operates. Our study addresses to what extent variations in these basic genetic features can contribute to variations in adaptive patterns of altruism.

From a methodological point of view, we develop a model of population dynamics based on spatial correlation equations (Matsuda et al. 1992; van Baalen and Rand 1998; Rand 1999; Ferrière and Le Galliard 2001) to study the evolutionary dynamics of altruism in the framework of adaptive dynamics theory (Metz et al. 1996; Dieckmann and Law 1996). The central notion is that selective pressures acting on mutant phenotypes are generated by the background population dynamics of resident phenotypes. After identifying selective pressures and incorporating them in a deterministic model of adaptive dynamics, we provide a classification of adaptive patterns of altruism according to the shape of physiological costs, the levels of individual mobility and the degree of habitat connectivity. The stability of the evolutionary endpoints and the effect of large mutations are investigated to gain insight into how variation in the mutation process may determine the adaptive outcome. Finally, the robustness of the salient conclusions drawn from our analytical study is tested against stochastic, individual-based simulations.

MODEL ASSUMPTIONS

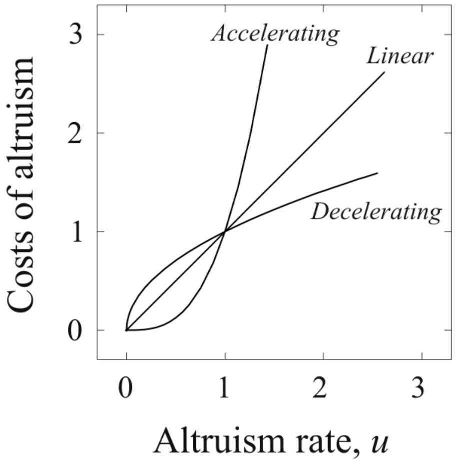
We consider a population of haploid individuals that inhabit a network of homogeneous sites, modeled as an infinite lattice (Appendix 1). Each site may be empty, or occupied by one individual. Each site is randomly connected to a set of sites that defines a neighborhood (Appendix 1). We assume that every site is connected to the same number of sites, denoted by n. Thus, the neighborhood size n provides a measure of the habitat's connectedness.

Mobility and interaction are defined locally, at the neighborhood scale. During any small time interval, an individual may move to an empty site within its neighborhood, reproduce by putting an offspring into an empty neighboring site, or die. The per capita mobility rate m and death rate d are unaffected by local interactions. Mobility is assumed to be costly to the individual, with a permanent negative effect on the individual's birth rate. This is expected in organisms where a stronger ability to move, resulting from specific structures (e.g., gliding flagella or muscles), imposes a developmental or maintenance cost. For example, the dispersive morph in the naked mole rat diverts more energy into growth and fat storage to compensate for the risks of moving in inhospitable habitats (O'Riain et al. 1996). The cost of mobility is assumed to impact linearly on the intrinsic birth rate such that the net per capita birth rate (in the absence of interaction) is given by $b(m) = b - \nu m$, where b measures the intrinsic per capita birth rate in sessile organisms that do not invest energy into mobility, and ν measures the sensitivity of the cost to mobility.

We assume that two types of local density-dependent factors affect movement and reproduction (Appendix 1). First, both movement and reproduction are conditional on the availability of empty sites within the neighborhood. Thus, local crowding negatively affects the rates of mobility and birth. Second, reproduction is enhanced by altruistic interactions with neighboring individuals, which induces a positive effect of local crowding. Here we assume that an altruistic interaction improves the quality of neighboring sites. This may involve storage of resources, habitat engineering, or signaling. The altruistic phenotype is defined by the per capita rate of investment u into the altruistic function. The altruistic behavior is directed evenly toward all neighboring sites, regardless of the presence or phenotypes of neighbors. In effect, every neighbor of a focal individual that invests at rate u into altruism receives a birth rate increment equal to u/n (Wilson et al. 1992). Therefore, altruism is only effective in practice provided some recipients are present in the neighborhood of the donor and some space is available in the neighborhood of the recipient for its offspring. We use the terms selfishness to describe a phenotype that does not invest in altruism ($u = 0$) and quasi-selfishness to refer to a phenotype that hardly invests in altruism ($u \approx 0$).

We further assume that altruism involves a physiological cost on the donor's reproduction, and we distinguish three patterns of dependence of costs on the investment in altruism: accelerating, linear, and decelerating (Fig. 1). With accelerating costs, the increase of the cost resulting from an increased altruistic investment becomes disproportionately larger as the initial investment increases. For example, in the cooperative bird Corcorax melanorhamphos physiological costs are detected only among individuals that invest strongly in altruism (Heinsohn and Cockburn 1994). Conversely, a decelerating pattern yields a disproportionate increase of costs at lower investment. This would apply to organisms in which the initiation of altruism from a selfish state would be very costly. In the limiting case of a linear pattern, the cost sensitivity is independent of the level of investment. The physiological cost of altruism $C(u)$ is modeled as a simple, allometric function that encapsulates the three patterns of decelerating, linear, and accelerating costs: $C(u) = \kappa u^{\gamma}$, where κ scales the sensitivity of the cost to the investment ($\kappa > 0$), and γ determines whether costs are accelerating ($\gamma > 1$), linear ($\gamma = 1$) or decelerating ($\gamma < 1$).

Figure 1. Costs of altruism as a function of the individual investment in altruism (*u*). This function is given by $C(u) = \kappa u^{\gamma}$ (with $\kappa = 1$ in this display). Decelerating costs, $\gamma < 1$: the cost increases with the rate of altruism first steeply and then more slowly. This pattern makes the origin of altruism from selfishness harder. Accelerating costs, $\gamma > 1$: the cost increases with the rate of altruism first slowly and then steeply. This pattern turns out to influence the long-term adaptive level of altruism. Linear costs, $\gamma = 1$: the rate of increase of the cost is independent of the level of altruism.



Mutations cause the altruistic phenotypes of offspring to differ from those of their parents. Mutations occur with a fixed probability per birth event, denoted by *k*. The mutant phenotype is obtained by adding the mutation effect to the progenitor phenotype. Mutation effects are drawn randomly from a normal probability distribution, with zero mean and a mutational variance σ^2. The resulting polymorphic, stochastic process can be simulated on a finite lattice (Appendix 1). Table 1 summarizes the used notation.

Table 1. Notation used in this paper.

	Model parameters
n	Neighborhood size
ϕ	Probability to draw a site at random in the neighborhood ($\phi = 1/n$)
b	Intrinsic birth rate
d	Intrinsic death rate
m	Intrinsic mobility rate
u_i	Intrinsic investment rate in altruism of a phenotype i (*adaptive trait*)
$C(u_i)$	Cost of altruism, impacting the birth rate of a phenotype i
κ	Cost sensitivity with respect to the level of investment in altruism
γ	Cost acceleration with respect to the level of investment in altruism
k	Mutation probability per birth event
σ^2	Mutational step variance
	Model variables
p_i	Global frequency of sites i
$q_{i\|j}$	Local frequency of sites i neighboring a j site
$\overline{p}_x$	Equilibrium global frequency of a resident x
$\overline{q}_{i\|x}$	Equilibrium local frequency of sites i neighboring a resident x
$\widetilde{q}_{i\|y}$	Pseudo-equilibrium frequency of sites i neighboring a rare mutant y

SPATIAL POPULATION DYNAMICS OF RESIDENTS

Selective pressures acting on a mutant phenotype result from the interaction between the initially scarce mutant population and the background population. The finite size of the interaction neighborhood and the finite range and rate of dispersal and fecundity cause spatial fluctuations and spatial correlations to develop in population density (Dieckmann and Law 2000). Thus, spatial population heterogeneity develops in a spatially homogeneous habitat. We use the framework of correlation equations to derive an analytical model describing the spatial dynamics of such a background population (Appendix 2). Assuming that mutation occurs rarely, the background population may be considered as monomorphic (Dieckmann and Law 1996; Metz et al. 1996). In this section we apply the polymorphic, ecological model to the specific case of a monomorphic population. This will provide the basic ingredients needed in the next section to model the dynamics of a mutant phenotype against this resident population.

Let us consider a single phenotype x which invests in altruism at rate u_x. The temporal dynamics of a population of x can be described by tracking over time t the frequency $p_x(t)$ of occupied sites. These dynamics depend on the neighborhood composition, described by the local frequencies $q_{i|x}$, i.e. the probabilities that an occupied site is neighbored by at least one site in the state i (Matsuda et al. 1992). The frequency p_x obeys the ordinary differential equation

$$\frac{dp_x(t)}{dt} = \left[\left(b(m) + (1-\phi)\cdot u_x \cdot q_{x|x}(t) - C(u_x)\right)\cdot q_{0|x}(t) - d\right]\cdot p_x(t), \tag{1}$$

involving the local frequencies $q_{x|x}(t)$ and $q_{0|x}(t)$ of occupied and empty sites next to an occupied site at time t, and $\phi = 1/n$. A closed system of correlation equations for the dynamics of local frequencies is constructed in Appendix 2 by making use of the standard pair approximation (Matsuda et al. 1992; Rand 1999; Iwasa 2000; van Baalen 2000).

At equilibrium, the spatial structure of a monomorphic population depends on the mobility rate and the altruistic investment (Appendix 3). The spatial structure is characterized by some degree of aggregation (Fig. 2). The spatial structure vanishes at high mobility rates, and for large birth rates, because birth is associated with offspring dispersal. More aggregation is found in organisms with low mobility, and also in organisms with very high mobility that consequently incur a severe reduction of their birth rate (due to the cost of mobility). The relationship between altruism and aggregation depends on the pattern of cost. In species with linear and decelerating costs, strongest aggregation is observed at low altruistic investment (Figs. 2B to D). In species with accelerating costs, strongest aggregation is observed in organisms with low altruism, or with high altruism, when the birth rate is drastically reduced by the cost of altruism (Fig. 2F).

For some parameter combinations, extinction is the only stable population equilibrium (Fig. 2). Extinction results from the total cost of mobility and altruism not being compensated. High mobility causes extinction because it implies a large direct cost that depresses the intrinsic birth rate $b(m)$, along with the reduction of the indirect benefits of altruism due to the loss of local aggregation. Species with accelerating costs can also undergo extinction at high investment in altruism (Fig. 2F). In all other cases, the altruistic population is viable and two types of population dynamics can be distinguished (Fig. 2). Borrowing terminology from the field of mutualism studies, we call the corresponding phenotypes facultative versus obligate. Thus, altruism is said to be facultative when the population growth rate in the limit of very low population density, $b(m) - d - C(u_x)$, is positive. The population is then characterized by a single, globally stable, and positive equilibrium $(\bar{p}_x, \bar{q}_{x|x})$ (Fig. 2A). Altruism is said to be obligate when the limit growth rate is negative and the population dynamics thus shows bistability. In this latter case, the positive equilibrium is locally stable and coexists with the extinction equilibrium, which is also locally stable. A population of obligate altruists may attain the viable equilibrium state only if its initial density lies above a critical threshold (Fig. 2A). This phenotypic state is therefore associated with low colonization ability and an elevated risk of extinction since a viable population can neither be established from an initially low density nor be maintained below a critical density threshold.

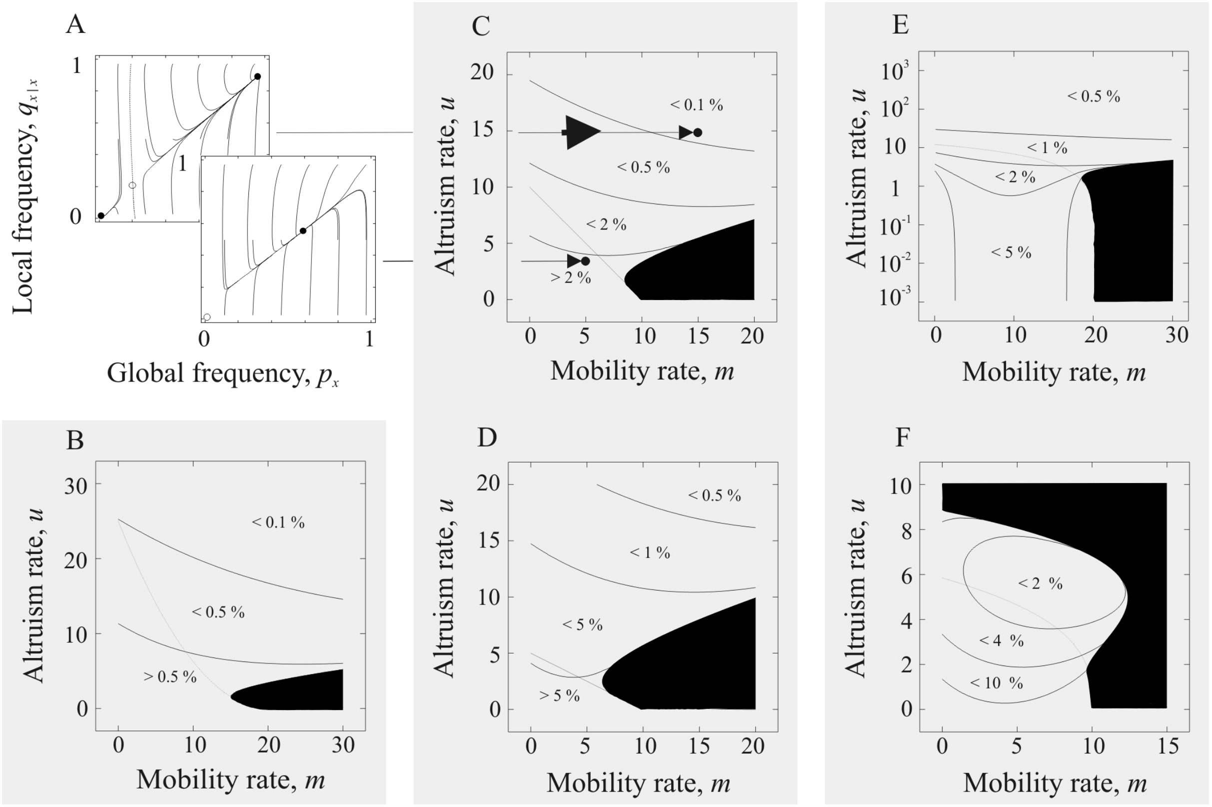


Figure 2. Monomorphic population dynamics. A, Population trajectories of global and local frequencies, as predicted by Equations (A4) and (A10). Starting from any initially rare state, the local frequency converges fast toward a one-dimension manifold along which most of the global frequency dynamics take place. The upper panel shows a case of obligate altruism ($m = 15$, $u_x = 20$): a positive equilibrium and the extinction equilibrium are both locally stable (filled circles) and coexist with a saddle point (open circle). The dotted curve separates the basins of attraction of the two stable equilibria. The lower panel shows a case of facultative altruism ($m = 5$, $u_x = 3$): there is a globally stable equilibrium (filled circle); the extinction equilibrium (open circle) is unstable. Parameters: $\gamma = 1$, $\kappa = 0.1$, and $\nu = 0.1$. B-F, Parameter regions of facultative altruism (below dashed curve), obligate altruism (above dashed curve) and population extinction (black area). Black curves are contours of the relative percent of deviation of spatial structure from mean-field equilibrium. B, Species with decelerating costs ($\gamma = 0.5$, $\kappa = 0.2$, and $\nu = 0.05$). C, Species with weak linear costs (parameter values as in A). D, Species with strong linear costs ($\gamma = 1$, $\kappa = 0.2$, and $\nu = 0.1$). E, Species with slowly accelerating costs ($\gamma = 1.2$, $\kappa = 0.05$, and $\nu = 0.05$); extinction also occurs at higher values of altruistic investment (not shown). F, Species with rapidly accelerating costs ($\gamma = 3$, $\kappa = 0.005$, and $\nu = 0.1$). Life-history and connectedness parameters are $b = 2$, $d = 1$ and $n = 4$, here as well as in all other figures.

SPATIAL INVASION FITNESS OF MUTANTS

The invasion fitness of a mutant is defined by its per capita growth rate while being rare in a resident population at ecological equilibrium (Metz et al. 1992). In the present section, we analyze the growth of such a small mutant population in the resident population described in the previous section (Appendix 4).

The invasion dynamics of a rare phenotype involves three phases (van Baalen 2000; Fig. 3). In a first, short phase, the small mutant population locally spreads from a single mutant individual up to the point where the mutant population attains a pseudo-equilibrium correlation structure. The build-up of this structure is highly stochastic but occurs with certainty on a finite time scale (Matsuda et al. 1992). Indeed, the cost of altruism dooms any single altruistic mutant in an established population of selfish individuals. Drift is first needed to drive the mutant population to its pseudo-equilibrium spatial structure. Also, the initial spread of the mutant depends on the local spatial structure of the resident population. For example, an altruistic mutant that arises in a neighborhood where selfish residents are more frequent than expected on average will face an increased risk of extinction. Denoting the mutant by *y*, we use the pseudo-equilibrium local frequencies $\tilde{q}_{0|y}$, $\tilde{q}_{x|y}$ and $\tilde{q}_{y|y}$ of (respectively) empty, resident and mutant sites around a focal mutant site to describe this transient structure. These statistics are calculated in Appendix 5. Conditional on non-extinction during this first phase, the mutant dynamics then enter the second phase during which the mutant population expands or contracts while its population keeps its pseudo-equilibrium structure and the resident population remains close to its own equilibrium (Fig. 3). Spatial invasion fitness can be defined as the mutant population growth rate during this second phase (van Baalen and Rand 1998). A positive fitness implies that the invasion process enters a third phase during which the mutant phenotype displaces the resident (Fig. 3), while a negative fitness implies mutant population extinction.

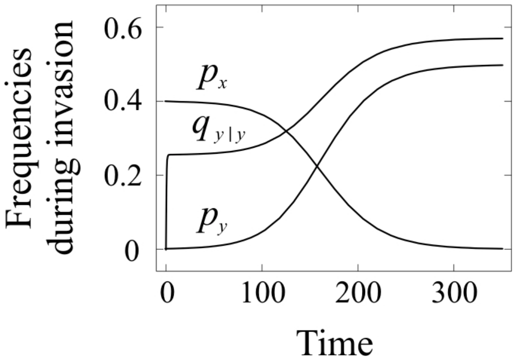


Figure 3. Successful invasion of an initially rare, altruistic mutant ($u_y = 1$) into a selfish resident population at equilibrium. Dynamics of the resident global frequency (p_x) and of the mutant global (p_y) and local frequency ($q_{y|y}$), as predicted by the deterministic system of correlation equations (A13). The three phases of invasion apparent in the dynamics of $q_{y|y}$ are discussed in the text. Parameter values: $\gamma = 2$ (accelerating costs), $\kappa = 0.1$, $v = 0.1$, and $m = 0$.

The spatial invasion fitness $s_x(y)$ can be expressed as a function of the pseudo-equilibrium statistics of the mutant population. Combining the population growth rate (1) with the expression of the mutant pseudo-equilibrium local frequencies, $s_x(y)$ is given by

$$s_x(y) = \left[\left(b(m) - C(u_y)\right)\tilde{q}_{0|y} - d\right] + (n-1)\phi\; u_x\,\tilde{q}_{0|y}\;\tilde{q}_{x|y} + (n-1)\phi\; u_y\,\tilde{q}_{0|y}\;\tilde{q}_{y|y}\,. \qquad (2)$$

Notice that the benefit of altruism (second and third terms) is measured conditionally on the presence of at least one empty site for breeding and depends upon the amount of help received from $n-1$ (not *n*) neighboring sites and the local frequency of empty sites $\tilde{q}_{0|y}$.

This expression bears an interesting relationship to the notion of direct or neighbor-modulated fitness of additive behavioral effects (Frank 1998). Direct fitness is derived by summing the effects on a focal individual's fitness of all phenotypes present in the neighborhood (including the focal individual itself). Likewise, the spatial invasion fitness of a focal mutant is obtained by adding to the mutant neighbor-independent fitness (first term) the effects of a resident neighbor (second term) and that of a mutant neighbor (third term), weighed by the probabilities of occurrence of such neighbors.

SELECTIVE PRESSURES

We now derive a simplified version of the spatial invasion fitness to analyze the selective pressures acting on the altruistic trait under small mutational steps. This will be the basis for studying the evolutionary dynamics of altruism. By using the fact that the resident's fitness in its own environment is always zero, $s_x(x) = 0$, the selection derivative can be derived from a first-order approximation of the spatial invasion fitness, and equals

$$\left.\frac{\partial s_x(y)}{\partial y}\right|_{y=x} = \bar{q}_{0|x}\left((1-\phi)\bar{q}_{y|y} - a\left((1-\phi)u_x - \frac{d}{\bar{q}_{0|x}^{\;2}}\right) - \left.\frac{\partial C(u_y)}{\partial u_y}\right|_{y=x}\right), \qquad (3)$$

where *a* measures the gain (or loss) of open space in a mutant's pseudo-equilibrium neighborhood relative to the resident's at equilibrium (see Appendix 5 for details). This expression exhibits three selective pressures driving the evolution of altruism. The first term on the right-hand side of Equation (3) quantifies the pressure for increased investment in altruism. The second term measures the pressure for opening free space in an individual's neighborhood. The last term measures the pressure for reducing the physiological cost of investing in altruism.

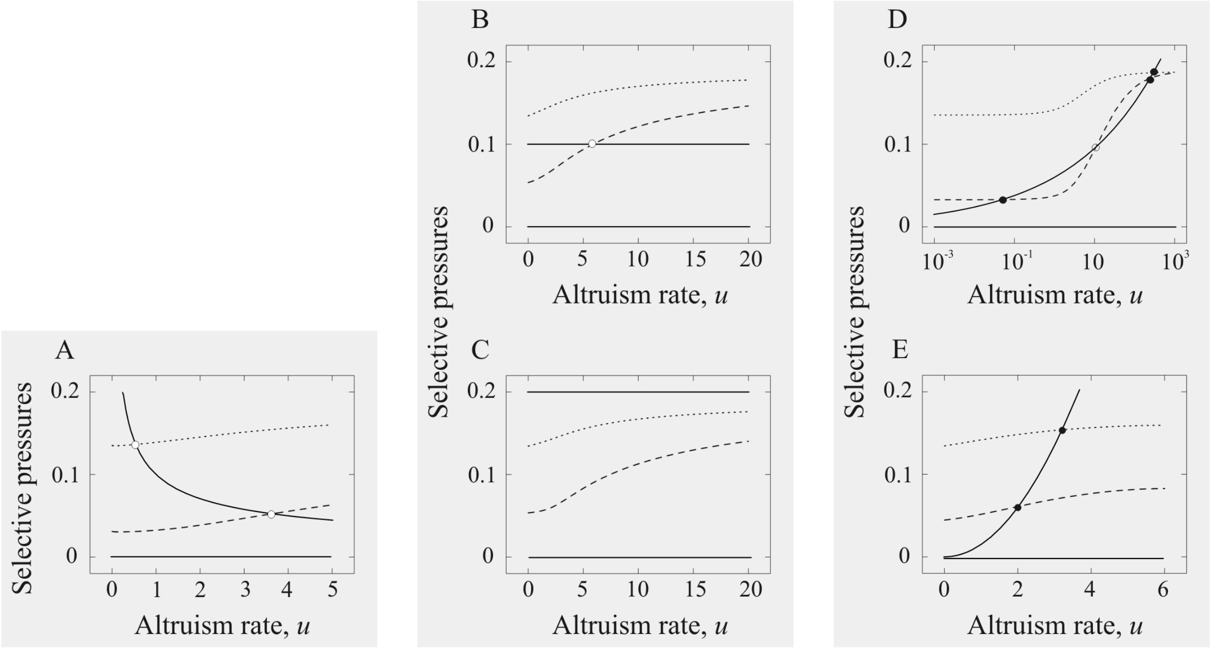


Figure 4. Selective pressures acting on altruism under small mutational steps calculated by following the recipe described in Appendix 5. Broken curves: positive pressure for increasing altruism, $(1-\phi)\,\bar{q}_{y|y}$, evaluated at low ($m = 1$, dotted curves) and high mobility ($m = 10$, dashed curves). Continuous curve: negative pressure for reducing the cost of altruism. Lower continuous line: negative, negligible selective pressure for decreasing local competition. Within the range of altruism where a broken curve is below (above) the continuous curve, selection favors the increase (decrease) of altruism. Circles indicate singular points where selective pressures exactly balance each other. Filled circles: attracting evolutionary singularities. Open circles: repelling evolutionary singularities. Parameter values in A to E are the same, respectively, as in Fig. 2B to F.

Extensive computations suggested that the pressure for opening space, albeit not vanishing, is negligible compared to the two other selective components (Fig. 4). This implies that $\bar{q}_{0|x} \approx \tilde{q}_{0|y}$, hence $a \approx 0$ in the Equation (3) (see Appendix 5). This also implies that as long as the mutant phenotype stays rare, the resident correlation structure is redistributed over the pairings of mutants with their own type and the resident type: $\bar{q}_{0|x} = \tilde{q}_{y|y} + \tilde{q}_{x|y}$. Thus, the mutant is less aggregated than expected when common, and therefore rare mutants are less likely to interact among themselves during the initial phases of invasion (Fig. 3). Globally, the evolution of altruism is not limited by competition for empty sites within the invasion structure, and the condition for an adaptive increase in altruism, $\partial s_x(y)/\partial y > 0$, is equivalent to

$$(1-\phi)\,\tilde{q}_{y|y} > \left.\frac{\partial C(u_y)}{\partial u_y}\right|_{y=x} . \tag{4}$$

This condition is a spatial form of Hamilton's rule (see also Ferrière and Michod 1995, 1996; Frank 1998; van Baalen and Rand 1998). The right-hand side is the marginal cost of altruism. The left-hand side measures the marginal benefit of altruism, weighed by the average frequency $\tilde{q}_{y|y}$ of recipient neighbors that are phenotypically identical to the focal mutant individual. For a haploid mutant population descended from a single mutation event, the identity in phenotype is equivalent to the identity by descent, and $\tilde{q}_{y|y}$ provides a measure of relatedness (Day and Taylor 1998).

The mutant relatedness can be expressed as a function of the resident population structure (Appendix 5), hence in terms of the basic demographic, mobility and lattice parameters, according to the following equality

$$\tilde{q}_{y|y} = \frac{d\phi}{d+(1-\phi)m\bar{q}_{0|x}}. \tag{5}$$

Thus relatedness is higher in a population with lower $\bar{q}_{0|x}$, which promotes the invasion of even more altruistic phenotypes. Insofar as the local frequency $\bar{q}_{0|x}$ correlates negatively with the investment u_x, this relation establishes a positive ecological feedback on the evolution of altruism: a negative effect of altruism investment on the local frequency $\bar{q}_{0|x}$ increases the relatedness of mutants, thereby enhancing the selective pressure for increased altruism. Also, Equation (5) shows that there is a direct negative effect of mobility on mutant relatedness, and an indirect effect through $\bar{q}_{0|x}$. Both effects add up to decrease mutant relatedness and weaken the selective pressure that favors altruism. Finally, increasing the neighborhood size n decreases $\tilde{q}_{y|y}$: a larger neighborhood size hampers the evolution of altruism.

CANONICAL EQUATION OF ADAPTIVE DYNAMICS

We use the results of the previous section to develop a deterministic model of adaptive dynamics under small mutations. This allows us to identify general patterns in the adaptive dynamics of altruism, to characterize the evolutionary endpoints and to study transient evolutionary dynamics. In a large population where mutations are rare and mutational steps are small, the stochastic mutation-selection process can be approximated by a deterministic process whose trajectories are the solution of the so-called canonical equation of adaptive dynamics (Dieckmann and Law 1996):

$$\frac{du_x}{dt} = \left[k \cdot \frac{\sigma^2}{2} \cdot \bar{p}_x\right] \cdot \left.\frac{\partial s_x(y)}{\partial y}\right|_{y=x}. \tag{6}$$

The bracketed term captures the effect of mutations, involving the mutation probability *k*, the mutational variance σ^2, and the equilibrium population frequency $\bar{p}_x$ of a monomorphic population of phenotype u_x. The local direction of phenotypic change is given by the selection derivative (Marrow et al. 1992) that we approximate according to Equation (4) by

$$\left.\frac{\partial s_x(y)}{\partial y}\right|_{y=x} = \overline{q}_{o:x}\left[(1-\phi)\overline{q}_{y|y} - \left.\frac{\partial C(u)}{\partial u}\right|_{u=u_x}\right]. \tag{7}$$

The resting points that satisfy $du_x/dt = 0$ are called evolutionary singularities and correspond to phenotypic states where the selection derivative vanishes (Marrow et al. 1992). Thus, at an evolutionary singularity, the marginal cost of altruism balances exactly the marginal benefit weighted by mutant relatedness. A singularity u^* can be locally evolutionarily attractive ("convergence stable"), or acts as an evolutionarily repellor.

Classification of Adaptive Dynamics

We develop a classification of the adaptive dynamics of altruism depending on the cost pattern specified by parameters κ and γ. To this end, we perform a numerical bifurcation analysis of the evolutionary singularities generated by (6) and (7) with respect to the mobility rate. We obtain five generic bifurcation diagrams as parameters κ and γ are varied (Fig. 5A).

For a decelerating cost of altruism, there is a single positive singularity that is unstable for any mobility rate (Fig. 5B). For linear costs, two cases can be distinguished. Either the selfish state undergoes a transcritical bifurcation as mobility increases, turning from unstable to stable and then coexisting with an unstable positive singularity. This is characteristic of "weak linear costs" (Fig. 5C). Alternatively the selfish singularity remains stable irrespective of the mobility rate, which characterizes "strong linear costs" (Fig. 5D). For accelerating costs, there are also two distinct patterns. For low values of κ and γ, there is a range of intermediate mobility rates over which the adaptive dynamics of altruism possess one unstable and two stable singularities. As mobility decreases, the lower stable equilibrium and the unstable one collide, leaving the upper stable singularity alone. Such combinations of γ and κ values define "slowly accelerating costs" (Fig. 5E). For higher values of κ and γ, characterizing "rapidly accelerating costs", there is a single stable positive equilibrium for every mobility rate (Fig. 5F).

Generically, natural selection favors altruism in species characterized by a large intrinsic birth rate *b* and a small death rate *d*. Also, altruism is selected against in species characterized by a large mobility rate. Selection against the altruistic trait may even lead to the "evolutionary suicide" of the population, if the adaptive dynamics start from an intermediate level of altruism and a high level of mobility (Figs. 5C to F). Although highly mobile organisms could persist on the ecological timescale provided that they behave sufficiently altruistically, the adaptive process would drive their altruistic investment down to the point where the population becomes non-viable. Increasing the size of the neighborhood selects strongly against altruism. The whole patterns are not sensitive to variations in the mobility cost parameter ν .

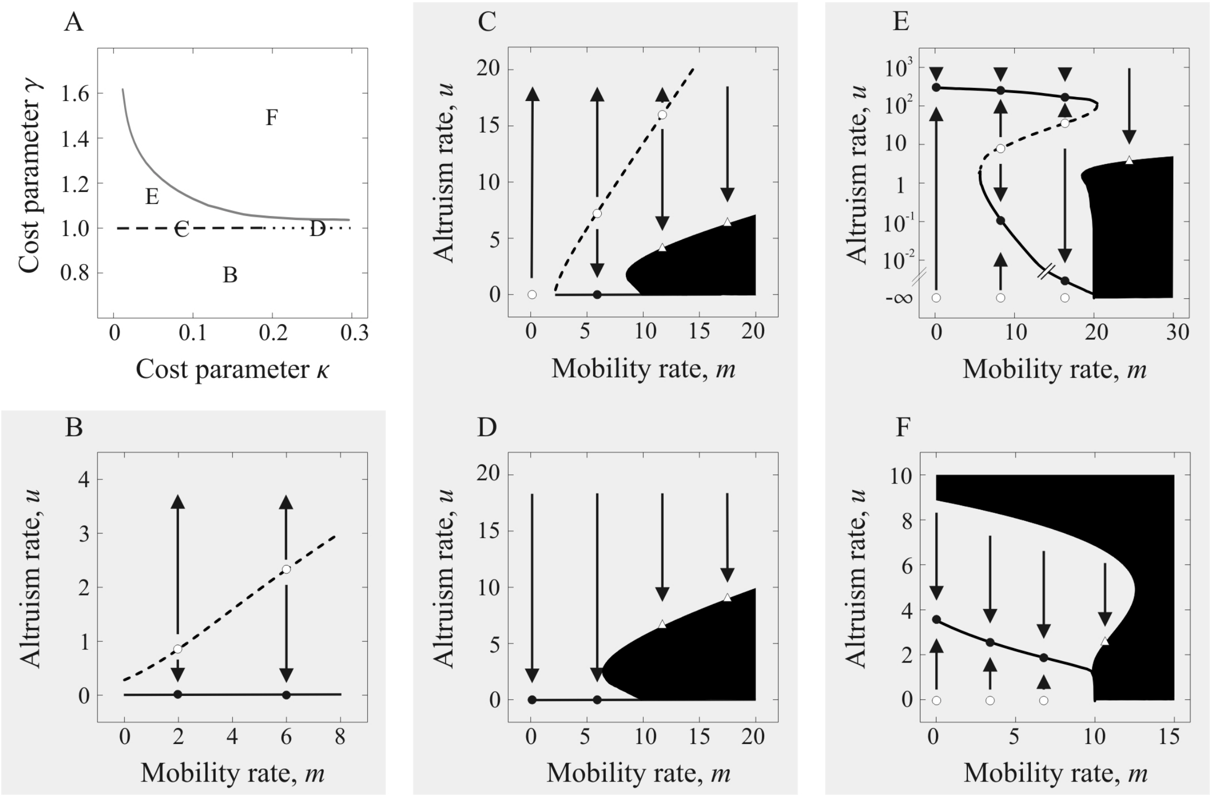


Figure 5. Adaptive dynamics of altruism. A, Classification of adaptive dynamics according to cost parameters γ and κ . Lettering refers to panels B to F showing bifurcation diagrams of the evolutionary singularities with respect to the mobility rate. In B-F, plain black curves are sets of convergence stable (attracting) singularities; dashed black curves are sets of convergence unstable (repelling) singularities. Population extinction occurs in black regions. Arrows indicate the direction of selective pressures at particular values of the mobility rate. Filled circles: attracting evolutionary singularity; open circles: repelling evolutionary singularity; triangles: evolutionary self-extinction. B, Species with decelerating costs. Inner singularities are repelling, resulting in bistable adaptive dynamics. C, Species with weak linear costs. Below a mobility threshold, altruism invades a purely selfish population and increases monotonically; above the threshold, the adaptive dynamics are bistable. D, Species with strong linear costs. Pure selfishness is globally attractive. E, Species with slowly accelerating costs. High altruistic investments are selected at low mobility. At higher mobility, an unstable singularity separates the basins of attraction of two locally attracting singularities that differ dramatically in their level of altruism (high altruistic investment versus quasiselfishness). F, Species with rapidly accelerating costs. The adaptive dynamics typically converge to a globally stable singularity. In all cases (not shown in B), the adaptive process can hit a region of extinction when the population originates from an ancestral state characterized by high mobility and intermediate or high altruism. Values of parameters κ and γ in B to F are the same, respectively, as in Fig. 2B to F. In all panels $\nu = 0.1$.

Decelerating Costs

With decelerating costs, there is a single, repelling evolutionary singularity for any mobility rate, and adaptive dynamics exhibit bistability (Fig. 5B). At the evolutionary singularity, the positive selective pressure on altruism resulting from mutant relatedness and the negative pressure exerted by the physiological cost balance exactly. The evolutionary singularity increases with the mobility rate, which is due to the effect of increased mobility on mutant relatedness as described by Equation (5).

Since the cost pattern is decelerating, a slight increase of altruism within the range below the singularity is counter-selected by a cost disproportionately larger than the gain. As a result, the adaptive dynamics ought to converge to selfishness (Fig. 4A). If the ancestral population state is sufficiently altruistic, the adaptive process will result in ever-increasing altruism. This is because, with decelerating costs, the cost of altruism increases more slowly than the benefits of altruism resulting from increased relatedness (Fig. 4A). In real systems, the adaptive increase of altruism should be limited by physiological or functional constraints, and the evolutionary process is expected to halt at such a limiting trait value.

Linear Costs

The adaptive dynamics of altruism in species with linear costs can be classified in two categories according to the cost parameter κ (Fig. 5A). For species with low κ, costs are said to be "weak linear" and the adaptive dynamics depend on the mobility rate (Fig. 5C). For low mobility, the selfish state is invadable by altruism and the adaptive process leads to the maximum physiologically feasible investment in altruism. Above a threshold on mobility, there exists a positive, repelling singularity and the adaptive process behaves as in the case of decelerating costs. If the initial investment in altruism lies below the singularity, the marginal benefit is too low to compensate for the marginal cost, and decreased altruism evolves. Above the singularity, the adaptive process causes the rise of altruism up to the physiological bound. For species with high κ ("strong linear" costs), the selfish state is evolutionarily attractive at any value of the mobility rate (Fig. 5D).

This pattern can be understood by comparing selective pressures (Figs. 4B, C). In the case of species with linear costs, the marginal benefit of altruism, $(1-\phi)\cdot\tilde{q}_{y|y}$, increases monotonously towards $\phi\cdot(1-\phi)$ as the altruistic investment becomes larger. If κ is larger than this value, the marginal costs of altruism always oppose the evolution of altruism (Fig. 4C). Otherwise, in species with low mobility marginal benefits are sufficiently high in the selfish state to select for altruism (Fig. 4B); in species with high mobility, marginal benefits exceed marginal costs only at high investment in altruism, and selfishness is locally attractive. The mobility threshold, where the stability of selfishness switches from global repulsion to local attraction, is given by the mobility rate m_l of a selfish population at which marginal benefits $(1-\phi)\cdot\tilde{q}_{y|y}$ and marginal costs κ equalize:

$$m_l = b\frac{\phi\cdot(1-\phi)-\kappa}{\nu(\phi\cdot(1-\phi)-\kappa)+\kappa\cdot(1-\phi)}. \tag{8}$$

This relation shows that among slowly reproducing organisms (small b), altruism may evolve only in species that exhibit little mobility. In species with very weak linear costs, the mobility threshold may not be observed, for it may exceed the critical value $(b-d)/\nu$ above which the population becomes non-viable (Appendix 3, $\kappa < 0.05$ in Fig. 5A). As a consequence, selfishness is invaded by altruism at any mobility rate smaller than this critical value, whereas at higher mobility, the evolution of decreasing altruism always drives the population to extinction (not shown).

Accelerating Costs

With accelerating costs, the cost of altruism is negligible compared to the benefits as long as the investment in altruism is not too high, and the selfish state is always invadable. This is in sharp contrast with predictions from well-mixed populations, in which selfishness is uninvadable even by only slightly altruistic mutants as soon as altruistic individuals incur a non-zero cost (Equation (A14) in Appendix 4). As altruistic phenotypes gain a foothold in the population, there are two possible outcomes depending on the combination of cost parameters.

Under a pattern of "slowly accelerating cost" (Fig. 5E), altruism rises toward a high evolutionary singularity in species with low mobility. With higher mobility the adaptive dynamics regime is bistable: the adaptive process converges to a high or a low singularity depending on the ancestral state. Extensive numerical explorations show that the altruism is always obligate (facultative) at the high (low) singularity. Under a pattern of "rapidly accelerating cost" (Fig. 5F), the adaptive dynamics converge monotonously to a low altruistic investment, whatever the ancestral state (including selfishness). The selected altruistic trait is found to correlate negatively with mobility. The evolved altruistic interactions shift from facultative to obligate as the cost parameters γ and/or κ increase.

The analysis of selective pressures helps us to understand these results (Figs. 4D, E). With slowly accelerating costs, when mobility is low, the marginal benefits start high and increase slowly (Fig. 4D). Then mutant relatedness easily opposes the initially low but faster-growing marginal cost. The selective force that favors altruism keeps dominating as the investment in altruism increases, until the marginal costs and benefits of altruism balance each other, which occurs at a high value of altruism. At higher mobility rates, the initial level of relatedness is lower, yet it remains sufficient for altruism to invade (Fig. 4D). The increase of relatedness with altruism is slower, which causes the selective pressures to balance at a low-altruism singularity. Beyond this point, the negative pressure exerted by the cost grows smoothly, while the positive pressure catches up rapidly across a range of intermediate investments (Fig. 4D). This generates a second unstable singularity, above which the net selective pressure turns positive again and favors the increase of altruism until a third, attractive singularity is reached at high altruistic investment. With rapidly accelerating costs, the selective pressures balance at a low-altruism singularity (Fig. 4E). Above the singularity, the net selective pressure against altruism increases, whereas the positive ecological feedback through relatedness remains weak.

Although in general there is no analytical expression for the singularities, the selected altruistic trait reaches a maximum value in the limit where mobility becomes very low, which is given explicitly by

$$u^*_{\max} = \left[\frac{\phi(1-\phi)}{\kappa\gamma}\right]^{\frac{1}{\gamma-1}} . \tag{9}$$

This maximum is independent of the organism's birth and death rates, and it decreases as the neighborhood size n increases. In species that experience rapidly accelerating costs, the singularity smoothly rises toward $u^*_{\max}$ as mobility decreases. In contrast, species with slowly accelerating costs fall into two main categories: quasi-selfish species, and obligatory altruistic species. In these obligatory altruistic species, the level of altruism is approximately equal to $u^*_{\max}$, and thus primarily depends on cost parameters and habitat connectedness.

EVOLUTIONARY STABILITY AND LARGE MUTATIONS

The previous analysis based on the canonical equation assumes small mutational steps and does not yet address the potential invasibility of attractive states. A locally attractive singularity would give rise to evolutionary branching if it is invadable (Metz et al. 1996). Evolutionary stability is probed by inspecting pairwise invasibility plots (PIPs; Geritz et al. 1998) that display the sign of $s_x(y)$ as u_x and u_y vary throughout the trait space (Fig. 6). Mutation effects are actually small but not infinitesimal, and even large mutations may occur, albeit rarely. The PIPs also describe the invasion potential of mutants that may differ substantially from their resident progenitors.

For species with decelerating costs, the PIPs show that selfishness is locally uninvadable: a slightly altruistic phenotype may not thrive in a primeval egoistic world. However, large mutations can move the population out of the basin of attraction of the selfish state, thus allowing for the adaptive increase of altruism (Fig. 6A). Even starting from a purely selfish population, rare mutations of large effect, together with random drift in a finite size population, makes this occur with certainty, although the waiting time can be long. The case of species with linear costs is radically different: even very large mutations may not move the population out of the basin of attraction of the selfish state, which is thus globally evolutionary stable (Figs. 6B, C).

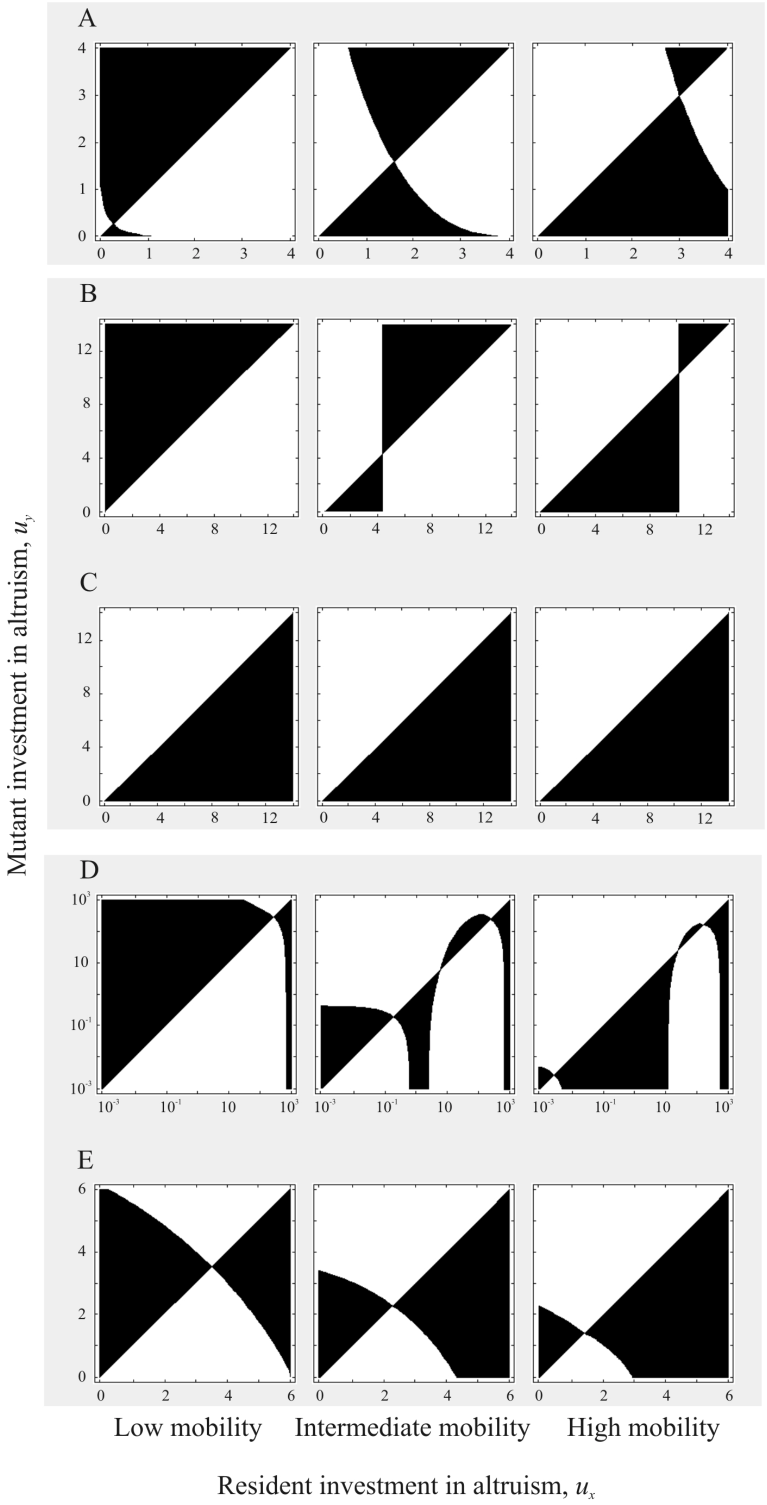


Figure 6. Invasibility of altruism phenotypes at different mobility rates. Each 'pairwise invasibility plot' represents the sign and zero contour of spatial invasion fitness as a function of the resident (horizontal axis) and mutant (vertical axis) trait values. Black (white) areas indicate combinations of resident altruism and mutant phenotypes for which spatial invasion fitness $s_x(y)$ is positive (negative). Singularities lie at the intersection of the diagonal line $s_x(x)=0$ and non-trivial zero contour $s_x(y)=0$ with $y \neq x$. A singularity x^* is locally uninvadable if for any y in the vicinity of x^* $s_{x^*}(y)$ is negative. A singularity x^* is attracting if the spatial invasion fitness is positive above the diagonal on the left of x^* and below the diagonal on the right of x^*. A, Decelerating costs, with $m=0$, $m=4$ and $m=8$ from left to right. The singularity is attracting. B, Weak linear costs with $m=0$, $m=4$ and $m=8$ from left to right. At low mobility, the increase of altruism is always favored. At high mobility, there is one repelling singularity. C, Strong linear costs with $m=0$, $m=3$ and $m=6$ from left to right. Selfishness is attracting, and also locally and globally evolutionarily stable. D, Slowly accelerating costs with $m=0$, $m=7.5$ and $m=15$ from left to right. At low mobility, altruism converges to a high singularity that is uninvadable. For higher mobility, two evolutionary attracting singularities are separated by a repelling singularity. E, Rapidly accelerating costs with $m=0$, $m=4.5$ and $m=9$ from left to right. The singularity is evolutionarily attracting and uninvadable by mutations of any size. All unspecified parameters in panels A to E are as in Fig. 2B to F, respectively.

In species with accelerating costs, the PIPs indicate that the attractive singularities are uninvadable (Figs. 6D, E). Starting from any viable trait value, altruism gradually evolves toward the singularity, which is robust against invasion by any alternative mutant. Large mutations may fail to invade even if they occur in the direction of adaptation predicted by the selection derivative (Figs. 6D, E). This will cause the adaptive process to slow down, all the more as it approaches the singularity. The non-trivial zero contour of fitness flattens in response to increased mobility, indicating that this "evolutionary slowing down" (Dieckmann and Law 1996) should be more pronounced in more mobile species. Overall, the inspection of many generic pairwise invasibility plots conclude that in our model the continuous evolution of altruism never undergoes evolutionary branching.

POLYMORPHIC SIMULATIONS

Although our analytical investigation of the evolution of altruism incorporates salient features of the ecological and evolutionary processes, it also involves several important simplifications. We assume an infinite lattice size, and describe the ecological dynamics with the standard pair approximation (Appendix 2). The derivation of the fitness measure relies on the small frequency of mutants as they originate and on the assumption that the build up of the mutant's pseudo-equilibrium correlation structure can be regarded as instantaneous (Fig. 3). Furthermore, the deterministic description of the adaptive dynamics is an approximation for the mean path of a stochastic mutation selection-process (Dieckmann and Law 1996). Individual-based simulations that track the fate of each individual in the population (Appendix 1) provides a natural way to circumvent these limitations and can be used to test the robustness of our main findings.

Patterns of invasion can be probed by running a large number of stochastic simulations in which a single individual mutant arises in a stable, resident population (Fig. 7A). We observe a sharp increase of the mutant local frequency $q_{y|y}$ at low values of p_y which corresponds to the rapid phase of convergence towards the pseudo-equilibrium of the mutant correlation structure (i.e., $\tilde{q}_{y|y}$) predicted by the pair approximation (Fig. 3).

The evolutionary patterns predicted by the canonical equation can be tested by running individual-based simulations of the mutation-selection process in which the assumption that mutants arise one at a time is relaxed, and averaging over a set of simulations (Figs. 7B to F). In species with decelerating costs, we predict that selfishness is invaded by altruistic phenotypes after a potentially long waiting time. Individual-based simulations confirm this prediction and show that the adaptive increase of altruism starts earlier when mutational effects are larger (Fig. 7B). Possibly, for very small mutational effects, the corresponding waiting time may be too long for being observed in simulations of feasible duration. Keeping mutational variance constant but increasing mobility or decreasing mutation rate causes a similar increase of the waiting time for altruism to take off. In species with weak linear costs, individual-based simulations confirm both the adaptive increase of altruism at low mobility and the evolutionary stability of selfishness at high mobility, even when very large mutations are feasible (Fig. 7C). In species with strong linear costs, convergence to selfishness or evolutionary suicide, depending on mobility, occurs as predicted by the canonical equation (Fig. 7D). Species with slowly accelerating costs are characterized by two clear-cut patterns, namely quasi-selfishness versus high altruism. At intermediate mobility, the adaptive dynamics bistability is confirmed by individual-based simulations run from different ancestral conditions (Fig. 7E). This implies that alternative adaptive investments can be reached for identical life-history profiles, due to different ancestral states or to contingent events during evolutionary history. Notice also that quasi-selfish states are practically indistinguishable from the stable selfish state of a stochastic mutation-selection process. In species characterized by rapidly accelerating costs the individual-based simulations closely match the predictions (Fig. 7F). The negative correlation between selected altruism and mobility expected in this case is confirmed. Overall, the agreement between stochastic simulations and the deterministic approximation is satisfactory. In general, the deterministic approximation converges slower than the stochastic process and underestimates the adaptive altruistic investment. This may be due to small systematic errors introduced by the standard pair approximation.

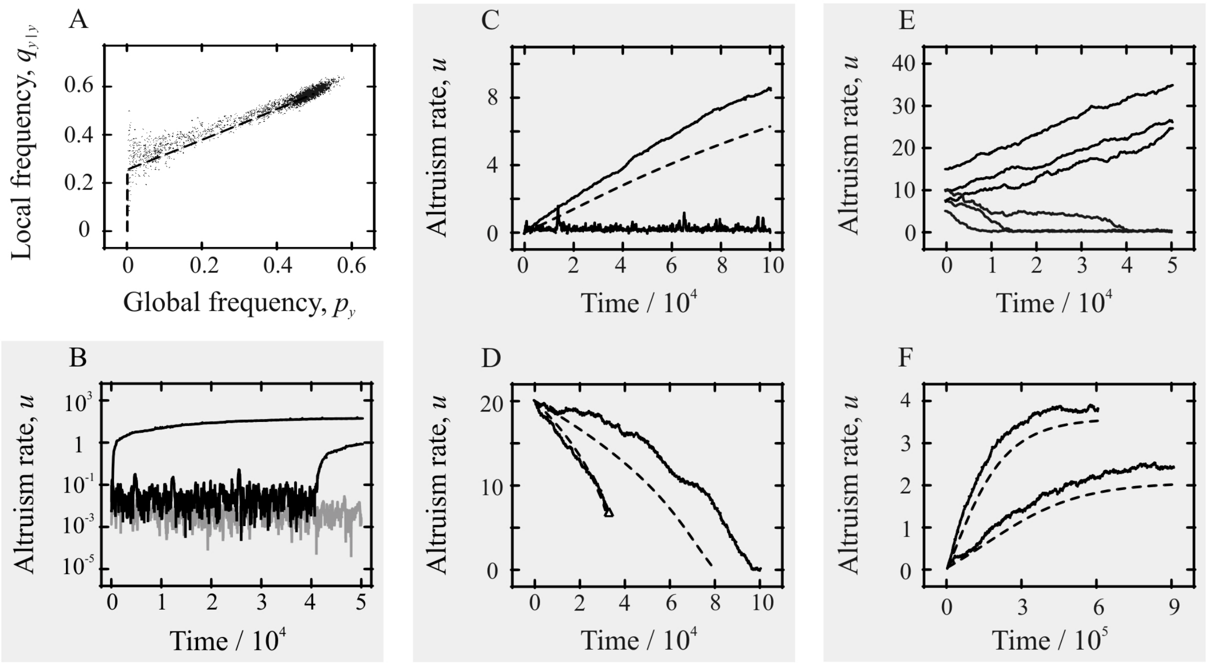


Figure 7. Individual-based simulations. A, Invasion dynamics. Deterministic dynamics (dashed curve) paralleled the stochastic simulations (continuous curve, mean of 1500 runs). Each dot gives the altruist local frequency $q_{y|y}$ and the altruist frequency p_y value at one point in time. Parameter values as in Fig. 2. B-F, Adaptive dynamics. B, Species with decelerating costs. Adaptive dynamics with large ($\sigma = 0.5$,first rising black trajectory), intermediate ($\sigma = 0.1$, second rising black trajectory) and low mutational variance ($\sigma = 0.01$, gray trajectory). Mobility: $m = 0$. Mutation rate: $k = 0.01$. C, Species with weak linear costs. Average of 10

independent stochastic trajectories (continuous upper curve) against the deterministic prediction computed from Equation (7) (dashed curve) at low mobility ($m = 0$, $k = 0.1$, $\sigma = 0.1$). Stochastic adaptive dynamics at high mobility with large mutation effects (lower trajectory, $m = 6$, $k = 0.05$, $\sigma = 0.1$). D, Species with strong linear costs. Average of 10 independent stochastic trajectories (continuous trajectories) compared to the deterministic prediction computed from Equation (7) (dashed curves) at low mobility (right-hand side of panel; $m = 0$, $k = 0.1$, $\sigma = 0.02$) and at high mobility (left-hand side of panel; $m = 10$, $k = 0.1$, $\sigma = 0.02$). Triangle indicates population extinction. E, Species with slowly accelerating costs and intermediate mobility. Adaptive dynamics starting from a low ancestral altruistic investment $u_x = 5$ or a high ancestral state at $u_x = 15$ diverge. Starting from the same ancestral state, $u_x = 8$ or $u_x = 10$, stochastic trajectories either rise to a high singularity or decline toward quasi-selfishness. Average from ten stochastic runs per starting condition. Parameter values: $m = 15$, $k = 0.05$, $\sigma = 0.1$. F, Rapidly accelerating cost. Average of 10 independent stochastic trajectories (continuous curves) compared to the deterministic prediction (dashed curves), at low mobility ($m = 0$, upper curves, simulations run over 600,000 time units) and high mobility ($m = 6$, lower curves, simulation run over 900,000 time units). Mutation parameters: $k = 0.01$, $\sigma = 0.01$. Unspecified parameters as in Fig. 2B to F, respectively.

Random lattices (featuring randomly assigned connections between sites) have been proposed for the purpose of modeling social networks (Rand 1999). The alternative of a regular habitat geometry, where interactions are limited to the geographically closest sites, compromises the use of the standard pair approximation to derive correlation equations for the population dynamics (van Baalen 2000). Individual-based simulations involving a regular square lattice indicate that our main findings are not altered qualitatively whereas selected trait values tend to be higher (results not shown).

DISCUSSION

We study the adaptive dynamics of a quantitative trait that measures the individual investment in altruism. The habitat is constant and homogeneous, but selective pressures arise from the phenotypic heterogeneity of the population. Altruism evolves from selfishness under a gradual kin selection process. This pattern is similar to the continuous evolution of cooperative investment observed in a spatial evolutionary game (Killingback et al. 1999). However, our model is not restricted to accelerating costs and pure viscosity, which would make up the most favorable case for the rise of altruism in a selfish population. In fact, we show that the qualitative and quantitative features of the adaptive evolution of altruism depend on the patterns of the cost and their interactions with mutation, individual mobility and habitat structure. Even when an accelerating cost pattern allows altruism to evolve easily from selfishness (Killingback et al. 1999), the adaptive increase of altruism appears to be often halted at very low levels. In contrast, when the cost pattern is decelerating, the selfish state will usually be displaced upon to the occurrence of rare mutations of large effects.

Kin Selection, Cooperation and Competition

The conceptual path followed here to define and measure fitness differs from the usual approach of kin selection theory (Frank 1998; Michod 1999 for reviews), although the results from both angles can be formulated in similar terms. Starting from demographic and behavioral processes operating at the level of individuals and their neighbors, we follow on from van Baalen and Rand (1998) to define fitness as the invasion exponent of a system of correlation equations for the spatial dynamics of a mutant population. This notion extends the concept of invasion fitness defined for well-mixed populations (Metz et al. 1992; Rand et al. 1994; Ferrière and Gatto 1995). In our model, invasion fitness is found to compound the per capita intrinsic growth rate and the per capita "neighbor-modulated" growth rate (Frank 1998) that accounts for the respective effects of competitive and cooperative interactions between a mutant focal individual and mutant *versus* resident neighbors.

In the terminology of kin selection, this invasion fitness is analogous to a "direct fitness function" from which one can derive a direct fitness gradient and decipher the selective pressures that operate on the trait under consideration (Frank 1998). We identify three selective pressures acting on altruism: the direct physiological cost to the individual, the indirect beneficial effect of altruistic interactions, and the indirect negative effect of competition for space. According to the analysis of Taylor (1992a, 1992b) and Queller (1994), spatial kin selection models raise a critical difficulty for the evolution of cooperation, because costs of competing for space with relatives exactly cancel out the benefits of altruism. Similarly, Wilson et al. (1992) concluded "local population regulation often, if not always, cancels the effects of relatedness". We arrive to the conclusion that the effect of competition with relatives can become negligible in organisms with a continuous life cycle, which reemphasizes that kin selection is effective at explaining the evolution of unconditional altruism (also see Taylor and Irwin 2000). This result holds independently of the level of individual mobility; and therefore cannot be attributed to relaxing the "pure viscosity" hypothesis of traditional kin selection models, according to which only offspring are dispersed (Hamilton 1964). Thus, why is the indirect cost of kin competition so low in our model ? Help is processed by any recipient to produce offspring in sites that are unlikely to be located in the donor's neighborhood because of the random structure of the habitat. Even with more regular habitat structure, the availability of free sites to host any donor's offspring is made likely by individual mortality that keeps reopening sites irrespective to social interactions between neighbors.

One key feature of our model therefore lies in the fact that the habitat offers empty sites for the local spread of altruistic mutants. This occurs because the stochastic death process keeps reopening space in the neighborhood of any helping individual. Consequently, the population does not reach a steady state of saturation, and the selective pressure generated by kin competition remains negligible. Such a dominant effect of the availability of free space on the evolution of altruism was anticipated by Taylor (1992b) and Queller (1992, 1994) who introduced the notion of "population elasticity" to refer to it. In a recent study of a contest of selfishness versus altruism, Mitteldorf and Wilson (2000) included a similar effect as generated by environmental stochasticity, and came to the same conclusion. However, these authors assumed an initial mixture of egoists and altruists instead of a single mutant in a resident background population, which does not address the crucial phase of the invasion process.

Whether it is demographic stochasticity or environmental stochasticity that underlies the site-opening process is unlikely to make much difference as long as there is no feedback of the adaptive trait dynamics on that stochastic process. This is the case both in our model and in the study by Mitteldorf and Wilson (2000) because the process of site opening amounts to a form of individual mortality whereas the altruistic trait affects fecundity. In a different setting where altruism would impact on the individual mortality rate, such a feedback could exist depending on whether sites are opened by demographic or environmental stochasticity. The adaptive increase of altruism would reduce the death rate, hence the rate of site opening by demographic stochasticity. As a consequence, the selective pressure of local competition against altruism would be enhanced, as can be demonstrated by an extension of our analysis (results not shown) and by the studies of Nakamaru et al. (1997, 1998) and Taylor and Irwin (2000). By contrast, the rate of site opening due to environmental stochasticity could remain independent of the adaptive change in altruism. Environmental stochasticity might then become critical for the evolution of altruism.

Because the selective pressure resulting from competition for open space turns out to be negligible, the criterion for mutant invasion amounts simply to comparing the marginal physiological cost of altruism to the marginal benefit withdrawn from interaction with the mutant's own kind. This is a variant of Hamilton's rule (Hamilton 1964) where the coefficient of relatedness is given by the local frequency of mutants neighboring a focal mutant during the invasion process. That the probability for the recipient of a focal mutant's act also to be a mutant is the appropriate definition of relatedness has already been shown in kin selection models involving pairwise interactions (Day and Taylor 1998, Frank 1998). However, such kin selection models assume that relatedness is constant and that the phenotype of a mutant has no effect on relatedness, which obviously cannot be true when invasion is a dynamical process and altruism impacts the distribution of individuals across space. The approach first advocated by van Baalen and Rand (1998) and followed up in this study shows that the same concept of relatedness holds nonetheless. In effect, relatedness is a dynamical variable for which the relevant equilibrium value can be expressed as a function of basic features of the organism's life cycle and behavior (also see Ferrière and Michod 1995, 1996; Hutson and Vickers 1995; van Baalen and Rand 1998).

Adaptive Patterns of Altruism

The pattern of physiological costs, individual mobility, the mutation process and habitat connectedness all interact to determine the adaptive dynamics of altruism (Table 2). The consideration of decelerating costs is more relevant to study the rise of altruism from selfishness. In species with decelerating costs, altruism is under the most stringent conditions to evolve from selfishness. However, once altruism is established by the mutation-selection process, the population invariably evolves toward the physiologically maximum investment. In contrast, the assumption of accelerating costs is more relevant to study the determinants of adaptive variation in altruism. In species with accelerating costs, altruism evolves right away from selfishness (Killingback et al. 1999); but contrary to the case of decelerating costs, the evolutionary endpoint is predicted to vary according to the physiological and ecological parameters.

Table 2. Overview of the results derived in this paper.

Physiological cost	*Adaptive dynamics*	*Evolutionary outcome from a selfish ancestral state*
Decelerating costs	Bistability	• Selfishness always displaced if finite mutations occur • Waiting time for the adaptive rise of altruism increases as mutation rate or mutational variance decreases, or mobility increases • Evolution toward physiologically maximum investment in altruism
Weak linear costs	Monotonic increase at low mobility Bistability at high mobility	• Evolution toward physiologically maximum investment in altruism if mobility is low • Persistent selfishness if mobility is high
Strong linear costs	Monotonic decrease	• Persistent selfishness
Slowly accelerating costs	Monotonic convergence at low mobility Bistability at high mobility	• Evolution toward high altruistic investment if mobility is low • Evolution toward quasi-selfishness if mobility is high
Rapidly accelerating costs	Monotonic convergence	• Evolution toward low altruistic investment, correlating negatively with mobility

Following on from this dichotomy, an important result is that generically any ancestral selfish population will evolve some degree of altruism. In the case of accelerating costs, the evolution of altruism is not influenced by the mutation process. On the contrary, assuming that in the primeval selfish state the cost of altruism is decelerating, the adaptive initiation of altruism depends primarily on the mutation process. We found that higher mutation frequency and larger mutational steps decrease the waiting time for altruism to evolve. For a given mutation process, the waiting time also increases with mobility, as higher mobility carries a larger direct cost and lowers the indirect benefit of altruism. The study of a linear cost function, which can be interpreted as a degenerate case in between decelerating and accelerating cost patterns, pinpoints the fallacious consequences that result from restricting attention to such a simple case (e.g., Roberts and Sherrat 1998). Incorporating a strong linear cost in the model completely hides the potential effect of the genetic process on the displacement of selfishness. The assumption of weak linear costs yields a type of bistable adaptive dynamics similar to the case of decelerating costs. However, large mutations have no effect in this case.

Mobility is expected to be an important factor of adaptive variation in altruism, as shown by the study of accelerating costs of altruism. First, mobility is found to impact on the speed of the adaptive process — the altruistic trait evolves at a slower pace among more mobile individuals. Second, mobility is a significant determinant of the evolutionary endpoint. In species with rapidly accelerating costs, the selected altruistic trait value correlates negatively with mobility. In species with slowly accelerating costs, mobility has a profound qualitative effect. High altruistic investments evolve in species with low mobility whereas quasi-selfishness evolves in species with high mobility. The finding of altruism being associated with low mobility is in line with previous insights into the evolution of cooperation in the Iterated Prisoner's Dilemma game (Dugatkin and Wilson 1991; Enquist and Leimar 1993; Hutson and Vickers 1995; Ferrière and Michod 1995, 1996), although the mechanisms involved are different. In these studies, mobility opposes the evolution of altruism by reducing the probability of repeated interactions between the same partners. On the empirical side, many independent studies have related the evolution of complex social systems with reduced mobility. For example, the emergence of cooperative breeding in birds may have been driven by delayed dispersal in a context of intense competition for space ("habitat saturation" hypothesis; Emlen 1982, 1997). Also, African mole-rat populations exhibit strong levels of philopatry, and the eusocial species of this mammal group occur in the harshest habitats, namely arid zones where benefits of group-living are high and dispersal is low (Jarvis et al. 1994; Spinks et al. 2000).

In species with slowly accelerating costs and low mobility, most variations of altruism between species are expected in response to differences in physiological costs and the degree of habitat connectedness. Our model outlines the importance of describing and measuring these parameters (Heinsohn and Legge 1999). Controlled experiments including the analysis of a broad range of levels of investment in altruism would enhance our knowledge of the shape and values of costs of altruism. In reality, the costs of cooperation may impact on different life history traits at different periods of the individual lifetime, and these experiments would require multivariate approaches and long-term studies.

In contrast, in species with slowly accelerating costs and high mobility, quasi-selfishness is expected to evolve. This state is eventually indistinguishable from a stable selfish population in which slightly altruistic phenotypes would chronically spread by mutation and drift. High altruistic investments could still be observed by the phylogenetic conservation of an ancestrally altruistic state while a slow environmental change favors an increase in mobility (Fig. 5E). High altruistic investment would have evolved under the early low mobility, and be preserved throughout the subsequent environmental increase of mobility. In support of the role of phylogenetic conservation, recent comparative analyses of sociality have demonstrated a phylogenetic component of sociality in birds (Edwards and Naeem 1993) and different groups of arthropods (Crespi 1996; Wcislo and Danforth 1997). High levels of investment in sociality seems to be maintained in more various ecological contexts once they evolve from cooperative ancestors. Possibly, a slow, gradual environmental change could even cause the loss of altruistic behavior at very high mobility through the catastrophic extinction of the population (Fig. 5E). An environmentally driven loss of sociality may provide another element of explanation for the uneven distribution of social species across taxa (Velicer et al. 1998).

Our analysis highlights the critical importance of mobility, which we modeled as a fixed parameter. This simplifying assumption may apply to species in which mobility is strongly constrained by the environment, the developmental program, or the genetic system. Otherwise, mobility and altruism should be entangled in a co-adaptive process, the dynamics of which will be investigated in a forthcoming paper.

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LITERATURE CITED

Axelrod, R., and W. D. Hamilton. 1981. The evolution of cooperation. Science 211:1390-1396.

Bourke, A. F. G. 1997. Sociality and kin selection in insects. Pp. 203-227 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. Blackwell, Oxford.

Cockburn, A. 1998. Evolution of helping behavior in cooperatively breeding birds. Ann. Rev. Ecol. Syst. 29:141-177.

Crespi, B. J. 1996. Comparative analysis of the origins and losses of eusociality: causal mosaics and historical uniqueness. Pp. 253-287 *in* E. Martins, ed. Phylogenies and the comparative method in animal behavior. Oxford Univ. Press, Oxford.

Day, T., and P. D. Taylor. 1998. Unifying genetics and game theoretic models of kin selection for continuous traits. J. Theor. Biol. 194:391-407.

Dieckmann, U., and R. Law. 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. J. Math. Biol. 34:579-612.

Dieckmann, U., and R. Law. 2000. Relaxation projections and the methods of moments. Pp. 412-455, *in* U. Dieckmann, R. Law, and J. A. J. Metz, eds. The geometry of ecological interactions: simplifying spatial complexity. Cambridge Univ. Press, Cambridge.

Doebeli, M., and N. Knowlton. 1998. The evolution of interspecific mutualisms. Proc. Natl. Acad. Sci. 95:8676-8680.

Dugatkin, L. A., and D. S. Wilson. 1991. ROVER: a strategy for exploiting cooperators in a patchy environment. Am. Nat. 138:687-701.

Edwards, S. V., and S. Naeem. 1993. The phylogenetic component of cooperative breeding in perching birds. Am. Nat. 141:754-789.

Emlen, S. T. 1982. The evolution of helping. I. An ecological constraints model. Am. Nat. 119:29-39.

Emlen, S. T. 1997. Predicting family dynamics in social vertebrates. Pp. 228-252 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. Blackwell, Oxford.

Enquist, M., and O. Leimar. 1993. The evolution of cooperation in mobile organisms. Anim. Behav. 45:747-757.

Ferrière, R., and M. Gatto. 1995. Lyapunov exponents and the mathematics of invasion in oscillatory or chaotic populations. Theor. Pop. Biol. 48:126-171.

Ferrière, R., and R. E. Michod. 1995. Invading wave of cooperation in a spatial iterated prisoner's dilemma. Proc. Roy. Soc. London B 259:77-83.

Ferrière, R., and R. E. Michod. 1996. The evolution of cooperation in spatially heterogeneous populations. Am. Nat. 147:692-717.

Ferrière, R., and J.-F. Le Galliard. 2001. Invasion fitness and adaptive dynamics in spatial population models. Pp. 57-79 *in* J. Clobert, E. Danchin, A. A. Dhondt and J. D. Nichols, eds. Dispersal. Cambridge Univ. Press, Cambridge.

Frank, S. A. 1998. Foundations of social evolution. Princeton Univ. Press, Princeton.

Geritz, S. A. H., E. Kisdi, G. Meszéna, and J. A. J. Metz. 1998. Evolutionary singular strategies and the adaptive growth and branching of the evolutionary tree. Evol. Ecol. 12:35-57.

Gillespie, T. D. 1976. A general method for numerically simulating the stochastic time evolution of coupled chemical reactions. J. Comp. Phys. 22:403-434.

Hamilton, W. D. 1964. The genetical evolution of social behaviour I, II. J. Theor. Biol. 7:1-52.

Heinsohn, R., and A. Cockburn. 1994. Helping is costly to young birds in cooperatively breeding white-winged coughs. Proc. Roy. Soc. London B 256:299-303.

Heinsohn, R., and S. Legge. 1999. The costs of helping. TREE 14:53-57.

Hutson, V., and G. T. Vickers. 1995. The spatial struggle of tit-for-tat and defect. Phil. Trans. Roy. Soc. Lond. B 348:393-404.

Iwasa, Y. 2000. Lattice models and pair approximation in ecology. Pp. 227-251 *in* U. Dieckmann, R. Law, and J. A. J. Metz, eds. The geometry of ecological interactions: simplifying spatial complexity. Cambridge Univ. Press, Cambridge.

Jarvis, J. U. M., M. J. O'Riain, N. C. Bennett and P. W. Sherman. 1994. Mammalian eusociality: a family affair. TREE 9:47-51.

Killingback, T., M. Doebeli, and N. Knowlton. 1999. Variable investment, the Continuous Prisoner's Dilemma, and the evolution of cooperation. Proc. Roy. Soc. London B 266:1723-1728.

Koella, J. 2000. The spatial spread of altruism versus the evolutionary response of egoists. Proc. Roy. Soc. London B 267:1979-1985.

Marrow, P., R. Law, and C. Cannings. 1992. The coevolution of predator-prey interactions: ESSs and Red Queen dynamics. Proc. Roy. Soc. London B 250:133-141.

Matsuda, H., N. Ogita, A. Sasaki, and K. Sato. 1992. Statistical mechanics of population: the lattice Lotka-Volterra model. Progress in Theoretical Physics 88:1035-1049.

Maynard-Smith, J., and E. Szathmary. 1995. The major transitions in evolution. Oxford Univ. Press, Oxford.

Metz J. A. J., R. M. Nisbet, and S. A. H. Geritz. 1992. How should we define fitness for general ecological scenarios? TREE 7:198-202.

Metz J. A. J., S. A. H. Geritz, G. Meszéna, F. J. A. Jacobs, and J. S. van Heerwaarden. 1996. Adaptive dynamics, a geometrical study of the consequences of nearly faithful reproduction. Pp. 183-231 *in* S. J. van Strien and S.M. Verduyn Lunel, eds. Stochastic and spatial structures of dynamical systems, North Holland, Amsterdam.

Michod, R. E. 1999. Darwinian dynamics, evolutionary transitions in fitness and individuality. Princeton Univ. Press, Princeton.

Mitteldorf, J., and D. S. Wilson. 2000. Population viscosity and the evolution of altruism. J. Theor. Biol. 204:481-496.

Nakamaru, M., H. Matsuda, and Y. Iwasa. 1997. The evolution of cooperation in a lattice-structured population. J. Theor. Biol. 184:65-81.

Nakamaru, M., H. Nogami, and Y. Iwasa. 1998. Score-dependent fertility model for the evolution of cooperation in a lattice. J. Theor. Biol. 194:101-124.

Nowak, M. A., and R. May. 1992. Evolutionary games and spatial chaos. Nature 359:826-829.

O'Riain, M. J., J. U. M. Jarvis, and C. G. Faulkes. 1996. A dispersive morph in the naked mole-rat. Nature 380:619-621.

Pusey, A. E., and C. Packer. 1997. The ecology of relationships. Pp. 254-283 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. Blackwell, Oxford.

Queller, D. C. 1992. Does population viscosity promote kin selection? TREE 10:322-324.

Queller, D. C. 1994. Genetic relatedness in viscous populations. Evol. Ecol. 8:70-73.

Rand, D. A., H. B. Wilson, and J. M. McGlade. 1994. Dynamics and evolution: evolutionarily stable attractors, invasion exponents and phenotype dynamics. Phil. Trans. Roy. Soc. Lond. B 343:261-283.

Rand D. A. 1999. Correlation equations and pair-approximations for spatial ecologies. Pp. 100-142 *in* J. M. McGlade, ed. Advanced ecological theory: advances in principles and applications. Blackwell, Oxford.

Roberts, G., and T. N. Sherrat. 1998. Development of cooperative relationships through increasing investment. Nature 394:175-179.

Spinks, A. C., J. U. M. Jarvis, and N. C. Bennett. 2000. Comparative patterns of philopatry and dispersal in two common mole-rats populations: implications for the evolution of mole-rat sociality. J. Anim. Ecol. 69:224-234.

Strassmann, J. E., Y. Zhu, and D. Queller. 2000. Altruism and social cheating in the social amoeba *Dictyostelium discoideum*. Nature 408:965-967.

Taylor, P. D. 1992a. Inclusive fitness in a homogeneous environment. Proc. Roy. Soc. London B 249:299-302.

Taylor, P. D. 1992b. Altruism in viscous populations – an inclusive fitness approach. Evol. Ecol. 6:352-356.

Taylor, P. D. and A. J. Irwin. 2000. Overlapping generations can promote altruistic behavior. Evolution 54:1135-1141.

Trivers, R. L. 1971. The evolution of reciprocal altruism. Quat. Rev. Biol. 46:35-57.

van Baalen, M., and R. A. Rand. 1998. The unit of selection in viscous populations and the evolution of altruism. J. Theor. Biol. 193:631-648.

van Baalen, M. 2000. Pair approximations for different spatial geometries. Pp. 359-387 *in* Dieckmann, U., R. Law, and J. A. J. Metz, eds. The geometry of ecological interactions: simplifying spatial complexity. Cambridge Univ. Press, Cambridge.

Velicer, G. J., L. Kroos, and R. E. Lenski. 1998. Loss of social behaviors by Myxococcus xanthus during evolution in an unstructured environment. Proc. Natl. Acad. Sci. USA 95:12376-12380.

Velicer, G. J., L. Kroos, and R. E. Lenski. 2000. Developmental cheating in the social bacterium *Myxococcus xanthus*. Nature 404:598-600.

Wcislo, W. T., and B. N. Danforth. 1997. Secondarily solitary: the evolutionary loss of social behavior. TREE 12:468-474.

Wilson, D. S., G. Pollock, and L. E. Dugatkin. 1992. Can altruism evolves in purely viscous populations? Evol. Ecol. 6:331-341.

APPENDIXES

APPENDIX 1 - POLYMORPHIC INDIVIDUAL-BASED MODEL

Here we describe the stochastic, individual-based model of polymorphic population dynamics that forms the basis of our study. The habitat structure is generated by randomizing the edges of a regular, square lattice of 30×30 sites with Moore neighborhoods (which, locally, gives a so-called Cayley or Bethe lattice). The state of any site is either empty or occupied by a type *i* individual with a certain phenotype u_i. The demographic parameters at time *t* of such an individual located at *z* are the birth rate

$$b_i(z,t)=\left[b(m)+\sum_{k=1}^{n}\frac{u_k}{n}-C(u_i)\right]\frac{n_{0|i}(z,t)}{n}, \tag{A1}$$

the intrinsic death rate *d* and the mobility rate

$$m_i(z,t)=m\frac{n_{0|i}(z,t)}{n}, \tag{A2}$$

where *k* (varying from 1 to *n*) labels each neighboring site, u_k is the altruistic investment of a neighbor or zero if that site is empty at that moment, and $n_{0|i}(z,t)$ denotes the number of empty sites neighboring a type *i* individual located at *z* at time *t.* The local birth rate and movement rate are multiplied by the proportion of empty sites within the neighborhood.

The simulation starts by distributing individuals of an ancestral phenotype randomly over half of the lattice. Mutations generate variability with a probability *k* per birth event. The mutant phenotype is obtained by adding a mutation effect drawn randomly from a normal probability distribution, with zero mean and mutational variance σ^2. When a negative investment in altruism is produced, the mutant phenotype is reset to selfishness. We use the minimal process method (Gillespie 1976) to simulate the time-continuous stochastic dynamics of the population. This means that the waiting time between two events is drawn form an exponential distribution with the inverse of its mean given by the total sum of event rates per unit time. Typically the completion of such a stochastic simulation requires around a day of computation on the fastest personal computer available at the time this work was conducted.

APPENDIX 2 - SPATIAL POPULATION DYNAMICS

Here, we derive an analytical model of the polymorphic population dynamics. The frequency of occupied sites varies through time along with the neighborhood configuration. The configuration of the neighborhood of a focal individual is given by the states of all pairs containing that individual, which themselves typically depend on the states of higher-order structures that contain those pairs (triplets, quadruplets, and so on). This kind of dependence cascades through all orders and spatial scales. Therefore, a full description of the lattice dynamics requires an infinite hierarchy of dynamical equations, also called correlation equations (Dieckmann and Law 2000). The system, however, can be closed at any order of description by making use of appropriate approximations. We close the system at the order of pairs with the standard pair approximation (Matsuda et al. 1992, Ferrière and Le Galliard 2001). This is the simplest approximation as it assumes that third-order correlations just vanish, and it has been used to construct correlation equations for spatial games (Nakamaru et al. 1997) and social interactions (Matsuda et al. 1992).

We first describe the temporal dynamics of the frequency of occupied sites. We assume that a fixed number of phenotypes are present in the population initially. We use the indices *i* and *j* to label phenotypes, and more generally the indices *k* or *l* to designate the state of a site, including the empty state 0. We proceed by averaging the local birth rate (A1) over the lattice, which after some algebra yields the average birth rate of a type *i* individual at time *t*:

$$\bar{b}_i(t) = \left[b(m) + \sum_j u_j (1-\phi) q_{j|i}(t) - C(u_i) \right] q_{0|i}(t), \tag{A3}$$

where $q_{j|i}(t)$ is the local frequency of phenotype *j* around a focal *i* individual at time *t*, the sum is taken over all phenotypes in the population and $\phi = 1/n$. The mean change in the frequency of the *i* phenotype at time *t* during an infinitesimal time step *dt* then is

$$\frac{dp_i(t)}{dt} = (\bar{b}_i(t) - d)\, p_i(t) = \lambda_i(t)\, p_i(t). \tag{A4}$$

We now describe the temporal dynamics of the local frequency of sites. As local frequencies are simply determined by pair frequencies according to

$$q_{k|i}(t) = p_{ki}(t) / p_i(t), \tag{A5}$$

where $p_{ki}(t)$ designates the frequency of *ki* pairs at time *t*, it is enough to describe the dynamics of the frequencies of pairs involved in (A5) to obtain equations for the local frequencies involved in (A4). An inventory of all different events that may affect any type of pairs at time *t* yields three elementary fluxes (van Baalen and Rand 1998, Rand 1999). The first is the average per capita input rate of a type *i* into a 0*j* pair with $j \neq i$

$$\alpha_{ij}(t) = (1-\phi)(\bar{b}_{i0}(t) + m)\, q_{i|0j}(t) = \alpha'_{ij}(t)\, q_{i|0j}(t). \tag{A6}$$

The second flux corresponds to the average per capita input rate of a type *i* into an 0*i* pair:

$$\beta_i(t) = \phi \bar{b}_{i0}(t) + (1-\phi)(\bar{b}_{i0}(t) + m) q_{i|0i}(t) \ . \tag{A7}$$

The third flux is the average per capita output rate of a type *i* from an *ij* pair:

$$\delta_{ij}(t) = d + (1-\phi) m q_{0|ij}(t) \ . \tag{A8}$$

The term $\bar{b}_{i0}(t)$ involved in these fluxes is the average birth rate of a type *i* individual inside a *i0* pair, given by

$$\bar{b}_{i0}(t) = (b(m) + \sum_j u_j (1-\phi) q_{j|i0}(t) - C(u_i)) \ . \tag{A9}$$

From equations (A7), (A8) and (A9), we can obtain the dynamics of the frequencies for the three general types of pairs, $0j$, *ij* and *jj*. Unfortunately, the dynamics of these pair frequencies depend on higher-order configurations because $q_{k|il} = p_{kil} / p_{il}$. Using the standard pair approximation, we assume that $q_{k|il} = q_{k|i}$, which gives

$$\begin{aligned}
\frac{dp_{0j}(t)}{dt} &= (\alpha'_j(t) q_{0|0}(t) - (\beta_j(t) + \delta_j(t))) p_{0j}(t) + \sum_{k \neq (0,j)} \delta_k(t) p_{kj}(t) + \delta_j(t) p_{jj}(t) \\
\frac{dp_{ij}(t)}{dt} &= (\alpha_i(t) + \alpha'_j(t) q_{0|0}(t)) p_{0j}(t) - (\delta_j(t) + \delta_i(t)) p_{ij}(t), \quad i \neq (0,j) \\
\frac{dp_{jj}(t)}{dt} &= 2\beta_j(t) p_{0j}(t) - 2\delta_j(t) p_{jj}(t)
\end{aligned} \tag{A10}$$

APPENDIX 3 - MONOMORPHIC POPULATION

A monomorphic version of the analytical model can be derived from (A4) and (A10). We assume there is only one phenotype and we denote by *x* the state of an occupied site. We are interested here in the feasible population equilibria. According to equation (A4), the non-trivial population equilibrium $\bar{q}_{0|x}$ must satisfy the quadratic equation

$$((b(m) + u_x (1-\phi)(1 - \bar{q}_{0|x}) - C(u_x)) \, \bar{q}_{0|x} - d = 0 \, . \tag{A11}$$

The spatial population is also characterized by a second, independent statistic, $\bar{q}_{0|0}$. From (A10) $\bar{q}_{0|0}$ satisfies

$$\bar{q}_{0|0} = \delta_x / \alpha'_x \ . \tag{A12}$$

We now analyze the viability and stability of the equilibria if *b* is sufficiently larger than *d*. The resident population is non-viable when no real solution exists for $\bar{q}_{0|x}$, which gives a first extinction boundary $\Delta = 0$ and the extinction domain $\Delta < 0$ (where Δ denotes the discriminant of the quadratic

Equation (A11)). In the area where $\Delta > 0$, there are two sub-domains. In the sub-domain defined by $b(m) - d - C(u_x) > 0$, the maximal root of (A11) corresponds to a viable equilibrium that is globally stable. In the disjunct sub-domain of obligate altruism, this solution is locally stable and coexists with the locally stable, trivial solution. A saddle point separates these two locally attractive solutions. The resident population goes extinct when the saddle point collides with the upper boundary, which leads to a second extinction domain adjacent to the first one.

APPENDIX 4 - DIMORPHIC POPULATION

Here we make use of the general polymorphic equations (A4) and (A10) to describe the population dynamics when there are only two phenotypes, indexed by *x* and *y*. Deriving a closed dynamical system requires equations for the two global frequencies p_x and p_y and for the three local frequencies $q_{x|x}$, $q_{x|y}$, and $q_{y|y}$. Using (A10) and (A5) for the dynamics of local frequencies and (A4) for the dynamics of global frequencies yields

$$\begin{aligned}
\frac{dp_x(t)}{dt} &= \lambda_x(t)\,p_x(t) \\
\frac{dp_y(t)}{dt} &= \lambda_y(t)\,p_y(t) \\
\frac{dq_{x|x}(t)}{dt} &= 2\beta_x(t)q_{0|x}(t) - (2\delta_x(t) + \lambda_x(t))q_{x|x}(t) \\
\frac{dq_{x|y}(t)}{dt} &= (\alpha_x(t) + \alpha'_y(t)q_{0|0}(t))q_{0|y}(t) - (\delta_y(t) + \delta_x(t) + \lambda_y(t))q_{x|y}(t) \\
\frac{dq_{y|y}(t)}{dt} &= 2\beta_y(t)q_{0|y}(t) - (2\delta_y(t) + \lambda_y(t))q_{y|y}(t)
\end{aligned} \quad . \qquad \text{(A13)}$$

As pointed out by van Baalen and Rand (1998), the mean-field version of system (A13) with selfishness (*x* type) and altruism (*y* type) interacting on the lattice is given by

$$\begin{aligned}
\frac{dp_x}{dt} &= \Big(\big(b(m) + u_y(1-\phi)p_y - C(0)\big)p_0 - d\Big)p_x \\
\frac{dp_y}{dt} &= \Big(\big(b(m) + u_y(1-\phi)p_y - C(u_y)\big)p_0 - d\Big)p_y
\end{aligned} \quad . \qquad \text{(A14)}$$

This system readily shows that selfishness is uninvadable because it benefits from the same amount of altruism without paying the costs. This serves to highlight that spatial population heterogeneity is a key prerequisite for the invasion of altruism in a selfish world.

APPENDIX 5 – INVASION STRUCTURE AND FITNESS

Let $\tilde{q}_{0|y}$, $\tilde{q}_{x|y}$, and $\tilde{q}_{y|y}$ denote the pseudo-equilibrium local frequencies characterizing the mutant correlation structure during invasion. These terms are the steady states of (A13) when *x* is a resident type at ecological equilibrium and *y* is a mutant type at low frequency, or

$$\begin{aligned} \frac{dq_{x|y}(t)}{dt} &= (\bar{\alpha}_x + \tilde{\alpha}'_y\,\bar{q}_{0|0})\tilde{q}_{0|y} - (\tilde{\delta}_y + \bar{\delta}_x + \tilde{\lambda}_y)\tilde{q}_{x|y} = 0 \\ \frac{dq_{y|y}(t)}{dt} &= 2\tilde{\beta}_y\tilde{q}_{0|y} - (2\tilde{\delta}_y + \tilde{\lambda}_y)\tilde{q}_{y|y} = 0 \end{aligned} \quad . \tag{A15}$$

Noting that $\tilde{q}_{y|0} = 0$ when the mutant is rare, the non-linear system involves three unknowns ($\tilde{q}_{0|y}$, $\tilde{q}_{x|y}$, and $\tilde{q}_{y|y}$) and two equations, along with the constraint $\tilde{q}_{0|y} = 1 - \tilde{q}_{x|y} - \tilde{q}_{y|y}$. The non-linear system (A15) can be used generically to evaluate numerically the spatial invasion fitness. However, one further analytical step can be taken by introducing the Taylor expansion of spatial invasion fitness up to the first order:

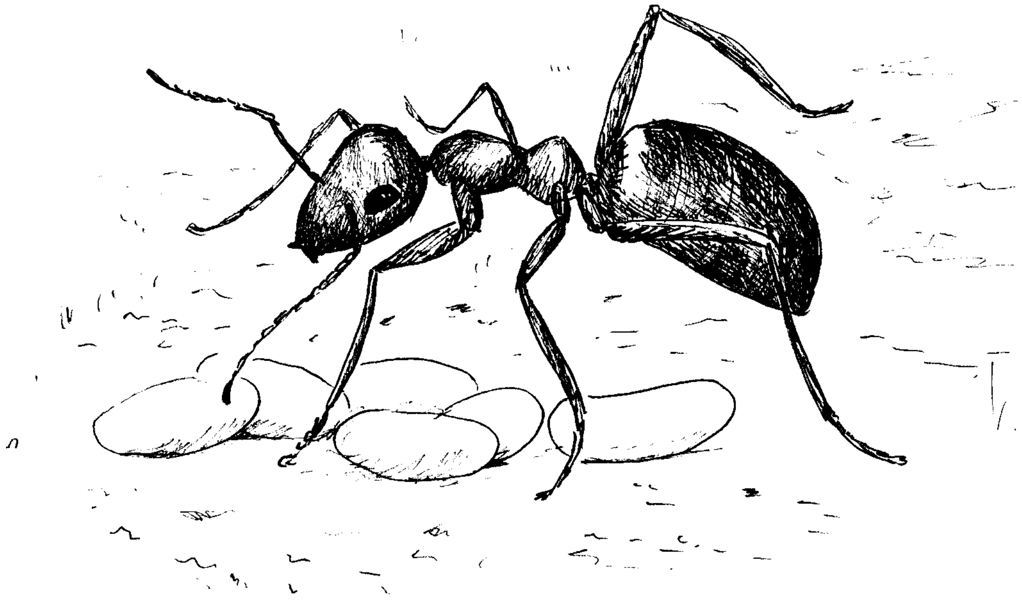
$$s_x(y) = s_x(x) + (y - x)\left.\frac{\partial s_x(y)}{\partial y}\right|_{y=x} + o(y - x)\,. \tag{A16}$$

For the degenerate mutant, $y = x$, we can solve analytically the non-linear system (A15) using symbolic resolution. This yields the solutions $\tilde{q}_{0|y} = \bar{q}_{0|x}$, $\tilde{q}_{y|y} = \bar{q}_{y|y}$ given as Equation (5) in the text, and $s_x(y) = s_x(x) = 0$. We now consider a slightly perturbed resident investment, i.e., $u_y = u_x + \varepsilon$, $\tilde{q}_{0|y} = \bar{q}_{0|x} + a\varepsilon$, and $\tilde{q}_{y|y} = \bar{q}_{y|y} + b\varepsilon$. The term *a* measures the marginal gain or loss of open space in a mutant's neighborhood relative to a resident. The term *b* measures the marginal gain or loss of relatives in a mutant's neighborhood relative to a resident. Plugging these approximations in the spatial invasion fitness defined by (1) yields after some algebra

$$s_x(y) = \varepsilon\,\bar{q}_{0|x}\left((1-\phi)\bar{q}_{y|y} - a\left((1-\phi)u_x - \frac{d}{\bar{q}_{0|x}^{\,2}}\right) - \frac{C(u_y) - C(u_x)}{\varepsilon}\right), \tag{A17}$$

from which we derive the selection derivative (3) at the limit where ε vanishes. Next, we use the non-linear system (A15) to solve for the *a* and *b* terms introduced before and to evaluate numerically the different components of (A17).

CHAPITRE 3 – ORIGINE ET EVOLUTION DES STRUCTURES SOCIALES



"What set species that form multigenerational families apart is the tendency for offspring to remain in association with their parent(s) beyond the age of sexual maturity and, commonly, throughout their lifetimes. The key to understanding the evolution of families is understanding delayed dispersal." S. T. Emlen dans *Behavioural Ecology*. 1997.

"Aspects of genetics, phenotype, ecology, and demography interact in their influences on social systems. Thus, to explain the phylogenetic distribution of social systems, the effects of these variables should be considered jointly." B. J. Crespi & J. C. Choe dans *Social behavior in insects and arachnids*. 1997.

EVOLUTION ADAPTATIVE DES TRAITS SOCIAUX: ORIGINE, HISTOIRE, ET PATRONS DE CORRELATION DE L'ALTRUISME ET DE LA MOBILITE

Jean-François Le Galliard, Régis Ferrière & Ulf Dieckmann

RESUME

Le comportement sociale implique un délai de la dispersion et une entraide locale — deux attributs individuels qui varient considérablement entre organismes. Cette étude analyse les facteurs ultimes de cette variation. L'évolution conjointe de l'altruisme et de la mobilité est influencée par les coûts physiologiques des deux traits, des rétroactions éco-évolutives, et une interaction sélective complexe entre les deux traits. La saturation de l'habitat, autour des individus (agrégation locale) ou autour des sites vides (concurrence locale), est un nœud critique du processus éco-évolutif. La promiscuité locale induit de la sélection de parentèle pour et est réciproquement augmentée par l'altruisme; la mobilité favorise l'invasion de mutants moins agrégés, et maximise la concurrence locale au cours de l'évolution unidimensionnelle. Quand les deux traits évoluent conjointement, l'agrégation locale est le filtre principal de l'interaction sélective et des effets indirects de la connectivité de l'habitat et des traits d'histoire de vie. Les dynamiques de la coévolution ont trois propriétés principales. (1) Le coût à la mobilité est la clef de l'origine de l'altruisme, en déterminant si l'égoïsme est envahissable, et l'échelle de temps de l'invasion. (2) Il existe deux voies typiques vers la socialité: l'évolution de la philopatrie, résultant en plus d'agrégation, peut avoir lieu avant ou après l'accroissement adaptatif d'altruisme. (3) A l'équilibre évolutif, une corrélation positive entre l'altruisme et la mobilité est attendue sous l'effet de changements dans les contraintes sur la mobilité, ou quand les traits d'histoire de vie varient conjointement.

Référence : Le Galliard, J.-F., Ferrière, R. et U. Dieckmann. 2003. « Adaptive evolution of social traits: origin, history, and correlation patterns of altruism and mobility ». *Manuscript en préparation pour soumission à The American Naturalist.*

Mots-clés : Dynamiques adaptatives, valeur sélective d'invasion, altruisme, mobilité, saturation de l'habitat, sélection de parentèle.

ADAPTIVE EVOLUTION OF SOCIAL TRAITS: ORIGIN, HISTORY, AND CORRELATION PATTERNS OF ALTRUISM AND MOBILITY

Jean-François Le Galliard, Régis Ferrière & Ulf Dieckmann

ABSTRACT

Social behavior involves "staying and helping"—two individual attributes that vary considerably among social organisms. This study investigates the ultimate factors of such variation. Altruism and mobility co-evolution is driven by physiological costs, eco-evolutionary feedbacks, and a complex selective interaction between the traits. Habitat saturation, around individuals (local aggregation) or around empty space (local contention), is a critical node of the co-evolutionary process. Local aggregation induces kin selection for, and is reciprocally enhanced by more altruism; mobility favors the spread of less aggregated mutants, and, as a single-evolving trait, maximizes local contention. When both traits co-evolve, local aggregation is the predominant mediator of the traits' selective interaction, and of the indirect effects of habitat structure and life-history traits. Co-evolutionary dynamics display three general properties. (1) The cost of mobility is key to the origin of altruism, determining whether selfishness is invadable, and the timescale of invasion. (2) There are two archetypal routes to sociality: the evolution of less mobility, resulting in stronger aggregation, can occur either before or after the adaptive rise of altruism. (3) At evolutionary equilibrium, a positive correlation between altruism and mobility is expected as constraints on mobility change, or as life-history traits covary.

Référence : Le Galliard, J.-F., Ferrière, R. et U. Dieckmann. 2003. « Adaptive evolution of social traits: origin, history, and correlation patterns of altruism and mobility ». *Manuscript in preparation for submission to the American Naturalist.*

Key-words : Adaptive dynamics, spatial invasion fitness, altruism, mobility, habitat saturation, kin selection

INTRODUCTION

Sociality is an essential characteristic of life. Sociality involves specific individual behaviors that lead to collective properties, new levels of natural selection, and the adaptive complexification of living systems (Maynard Smith and Szathmary 1995; Michod 1999). One of the most intriguing feature of sociality is that it induces at least a two-fold cost to the individual. Sociality often requires some form of altruistic behavior whereby individuals sacrifice their own fitness to help others (Hamilton 1964a, 1964b), and some reduction in individual mobility that raises the risks of competition for local resources (Frank 1995; Perrin and Goudet 2001). Genes involved in such deleterious effects are expected to be eliminated by natural selection. Yet a wide diversity of social behaviors are observed in the wild (Choe and Crespi 1997; Crespi 2001; Wilson 1975), and one of the challenge for evolutionary theory is to explain the role that genetic adaptation can play in molding individual altruism along with population structure, and more generally the diversity of social systems (Crespi and Choe 1997).

The two-fold cost of sociality reflects only some of the selective pressures acting on social traits. Low individual mobility may increase local genetic relatedness between interacting individuals, thus promoting the evolution of helping behavior through kin selection (Frank 1998; Hamilton 1964b). Yet the enhancement of neighbors reproduction ensuing from helping may also generate habitat saturation and exacerbate competition among kin (Grafen 1984; Queller 1992; West et al. 2002). Increased competition between relatives for local resources can in turn reduce or even totally negate the indirect genetic benefits of altruism (Taylor 1992; Wilson et al. 1992). The extent to which kin competition resulting from low mobility fundamentally alters our understanding of kin selection on social traits has been demonstrated by recent comparative work on fig wasps. In these insects, strict philopatry of males competing for mates results in extremely strong local competition and completely cancels out any indirect genetic benefits of decreasing aggressiveness toward relatives (West et al. 2001). Substantial overlap between social and competitive neighborhoods may also occur in other species. In cooperatively breeding vertebrates, local offspring recruitment can result in competition among relatives for dominance and breeding opportunities within a group (Cockburn 1998; O'Riain and Braude 2001). Limited dispersal in some social insects can lead to competition between colonies founded by relatives (Thorne 1997). In general, the importance of kin competition for the evolution of altruism should depend upon the species' life history profile, the spatial scale over which cooperation and competition occur, and the structure of the habitat (Kelly 1992, 1994; Queller 1992).

In a recent study, Le Galliard et al. (2003) showed that fluctuations in local population size caused by demographic stochasticity suffices to turn local competition into a weak selective force against reproduction altruism (see also Mittledorf and Wilson 2001). As a consequence, cooperation can almost always evolve from pure selfishness. However, the level of individual mobility has a paramount influence on the evolutionary outcome: high altruism can only evolve in species with low mobility, and the evolutionary trajectory of highly mobile species is halted in a state of 'quasi-selfishness'. The underlying assumption that mobility can be treated as a constant trait may apply if mobility is strongly constrained by the environment, the developmental program, or the genetic system. Otherwise, mobility and altruism will be entangled in a co-adaptive process. The costs and benefits of altruism should depend on local spatial structures, which are shaped by the current levels of individual mobility and helping in the population (Ferrière and Michod 1996; Le Galliard et al. 2003). The costs and benefits of mobility should also depend upon local levels of competition and cooperation (Ferrière and Le Galliard 2001; Lambin et al. 2001). Understanding the origin and evolutionary diversification of social behaviors then raises the need to conduct a joint analysis of the intertwined adaptive dynamics of altruism and mobility (Emlen 1997; Helms Cahan et al. 2002). The purpose of this paper is to develop such a unifying approach.

By expanding the framework laid out by Le Galliard et al. (2003), we study the joint evolution of altruism and mobility in a spatially explicit model. In this model, individuals move and interact locally on a lattice of suitable sites (Matsuda et al. 1992; van Baalen 2000b). The study requires to define an appropriate measure of fitness; the notion of invasion fitness, that is, the per capita growth rate of a mutant when rare in the environment set by the wild-type population (Metz et al. 1992), appropriately extends to kin selection processes involving diallelic, haploid genetics (Frank 1998; Michod 1999). Following on from van Baalen and Rand (1998), we derive invasion fitness from a set of correlation equations describing the population's spatial heterogeneity (Ferrière and Le Galliard 2001; Le Galliard et al. 2003). We then use this measure of spatial invasion fitness to (i) identify the selective pressures acting on altruism and mobility traits, and relate these pressures to the model underlying parameters; (ii) analyze the course of evolution of both traits, and their interplay with the population spatial structure; and (iii) make predictions about the correlation patterns between altruism and mobility at evolutionary equilibrium—patterns which are expected to emerge across populations or species in response to variation of life-history traits or ecological constraints.

MODEL OF SOCIAL NETWORKS

We assume that interactions and mobility occur locally between neighbors. Altruism and mobility are continuous characters affecting individuals' demographic parameters. Demographic parameters and individual interactions mold the local population structure from which selective pressures on the individual traits arise.

The model takes the form of a lattice of sites over which individuals are distributed. Each site may be empty, or occupied by one individual, and is connected to a set of sites that defines a neighborhood. Connections are assigned randomly between sites and every site is connected to the same number of sites, denoted by n which defines a degree of 'habitat connectivity'. This representation of a spatial habitat is appropriate to model social interactions, and may be typical of the social structure of some vertebrates which defend individual territories and disperse primarily among adjacent sites (e.g., Rand 1998).

Mobility and interaction are defined at the neighborhood scale. During any small time interval, an individual may move to an empty site within its neighborhood, reproduce by laying an offspring into an empty neighboring site, or die. The per capita mobility rate *m* and death rate *d* are unaffected by local interactions. Mobility is assumed to be costly to the individual, with a permanent negative effect on the individual's birth rate. This cost may result from differential allocation of resources between dispersal and fecundity (Cohen and Motro 1989). The cost of mobility is assumed to impact linearly the intrinsic birth rate such that the net per capita birth rate (in the absence of interaction) is given by $b(m) = b - \nu m$, where *b* measures the intrinsic per capita birth rate in sessile organisms that do not invest energy into mobility, and ν measures the sensitivity of the cost to mobility.

We assume that movement and reproduction are affected by two types of local density-dependent factors. First, movement and reproduction are conditional to the availability of a neighboring empty site, because of a prior-resident advantage. Thus, local crowding negatively affects the rates of mobility and birth. Second, reproduction is enhanced by altruistic interactions with neighbors, which implies a positive effect of local population size. Here we assume that an altruistic donor improves the quality of the neighboring sites at the expense of its own reproduction. For example, in cooperatively breeding vertebrates, helpers usually participate in alloparental care (Cockburn 1998). The altruistic phenotype is defined by the per capita rate of energetic investment *u* into the altruistic function. The altruistic behavior is directed evenly towards all neighboring sites, regardless of the presence or phenotypes of neighbors. In effect, every neighbor of a focal individual that invests at rate *u* into altruism sees her birth rate augmented by the amount u/n. We use the terms "selfishness" to describe a phenotype that does not invest in altruism ($u = 0$) and "quasi-selfishness" to refer to a phenotype that invests nearly zero in altruism ($u \approx 0$).

We further assume that altruism carries a physiological cost. This is known for example in the suricate *Suricata suricatta*: adults engaging in the altruistic activity of baby-sitting lose significant body weight (Clutton-Brock et al. 1998). We distinguish three patterns of dependence of costs on the amount of energy invested in altruism, that we call accelerating, linear, and decelerating. With accelerating costs, the increase of the cost resulting from an increased altruism rate becomes disproportionately larger as the initial investment increases. Conversely, a decelerating pattern yields a disproportionate increase of costs at lower investment. This would apply to organisms in which the initiation of altruism from a selfish state would be very costly. In the limiting case of a linear pattern, the cost sensitivity is independent of the level of investment. The physiological cost of altruism $C(u)$ encapsulates the three patterns of decelerating, linear, and accelerating costs: $C(u) = \kappa u^{\gamma}$, where κ

scales the sensitivity of the cost to altruism ($\kappa > 0$), and γ determines whether costs are accelerating ($\gamma > 1$), linear ($\gamma = 1$) or decelerating ($\gamma < 1$).

Mutations may cause the altruism or mobility phenotypes of offspring to differ from their parents’. Mutations occur with a fixed probability per birth event, denoted by *k*. The mutant phenotype is obtained by adding the mutation effect to the progenitor phenotype. Mutation effects are drawn randomly from a normal probability distribution, with zero mean and a mutation step variance σ. There are no genetic correlations between the altruism and mobility traits. The polymorphic, stochastic process can be simulated on a finite lattice (Appendix A). Symbols of the model are defined in Appendix E.

CANONICAL EQUATION OF ADAPTIVE EVOLUTION

In a large population where mutations are rare and mutational steps are small, the stochastic mutation-selection process can be approximated by a deterministic process whose trajectories are solution of the so-called canonical equation of adaptive dynamics (Dieckmann and Law 1996):

$$\frac{d}{dt}\begin{pmatrix} u_x \\ m_x \end{pmatrix} = k\,\frac{\sigma^2}{2}\,N_x \begin{pmatrix} \left.\dfrac{\partial s_x(y)}{\partial u_y}\right|_{u_y=u_x} \\ \left.\dfrac{\partial s_x(y)}{\partial m_y}\right|_{m_y=m_x} \end{pmatrix}, \qquad (1)$$

where $x=(u_x,m_x)$ denotes a resident phenotype, and $y=(u_y,m_y)$, a mutant phenotype. Parameters *k* and σ are the mutation rate and the mutation step variance, which we assume to be the same for both traits. N_x is the resident population size at equilibrium, and $s_x(y)$ denotes the invasion fitness of a mutant phenotype *y* in a resident phenotype *x*. The three first terms of the right hand-side quantify the effects of mutation and population size on the evolutionary rate. The bracketed term is the selection gradient which defines the local direction of the adaptive process.

The resting states of the canonical equation (1) correspond to resident phenotypes where the selection gradient vanishes. A stability analysis of these singular points requires examining independently the evolutionary attractivity, or convergent stability, and the non-invasibility or evolutionary stability (Eshel 1983; Geritz et al. 1998). The local evolutionary attractivity of a singular state x^* means that trajectories starting in the vicinity of x^* converge to the singularity. Under the assumption of canonical equation (1), the convergence stability of x^* is characterized by the Jacobian matrix

$$\boldsymbol{J} = \begin{bmatrix} \dfrac{\partial^2 s_x(y)}{\partial^2 u_y} + \dfrac{\partial^2 s_x(y)}{\partial u_x \partial u_y} & \dfrac{\partial s_x(y)}{\partial m_x \partial u_y} \\ \dfrac{\partial s_x(y)}{\partial m_y \partial u_x} & \dfrac{\partial^2 s_x(y)}{\partial^2 m_y} + \dfrac{\partial^2 s_x(y)}{\partial m_x \partial m_y} \end{bmatrix}_{y=x=x^*} . \qquad (2)$$

For a singular point to be locally attractive, it is sufficient that the two eigenvalues of the Jacobian matrix have negative real part. The local non-invasibility of a singular point x^* means that a mutant with a phenotype close to the singular phenotype is unable to invade. Under the assumption of the canonical equation (1), the Hessian matrix characterizes the evolutionary stability of the singular point

$$\mathbf{H} = \begin{bmatrix} \dfrac{\partial^2 s_x(y)}{\partial^2 u_y} & \dfrac{\partial^2 s_x(y)}{\partial m_y \partial u_y} \\ \dfrac{\partial^2 s_x(y)}{\partial m_y \partial u_y} & \dfrac{\partial^2 s_x(y)}{\partial^2 m_y} \end{bmatrix}_{y=x=x^*} \qquad (3)$$

(Leimar In press; Marrow et al. 1996). For the singular state x^* to be locally non-invadable, it is sufficient that the two eigenvalues of the Hessian matrix have negative real part (Leimar In press). Convergence and evolutionary stability can also be characterized globally by geometrical tools. Global evolutionary attractivity has been analyzed here by plotting adaptive trajectories in the trait plane, and global non-invasibility has been studied by the use of pairwise invasibility plots, that display the sign of the invasion fitness $s_x(y)$ with regard to x and y values (Geritz et al. 1998).

SPATIAL INVASION FITNESS

The spatial invasion fitness of a mutant (denoted by y) can be defined as the propensity of an initially rare mutant population to establish in a resident population at ecological equilibrium (Metz et al. 1992). The dynamics of a mutant phenotype y is derived by tracking its population size across time. Mutant dynamics depend on the resident population configuration and are affected by two types of density-dependent effects. Local competition affects the likelihood of movement and birth events. Birth and movement rates of mutant are proportional to the frequency of empty sites in the neighborhood of mutant sites. Altruistic interactions affect additively the birth rate of any focal individual. The benefits obtained from altruistic interactions are proportional to both the altruism rate and the frequency distribution of neighboring phenotypes.

The growth of the mutant population depend on the neighborhood structure, which involves the configuration of pairs of sites (Appendix B). This neighborhood structure can be described in terms of 'local frequencies', the average probabilities that a site is neighbored by a site in a given state (Matsuda et al. 1992). The dynamics for the population size of the mutant are then governed by

$$\frac{dN_y}{dt} = \left[\left(b + (1-\phi)\, u_x\, q_{x|y} + (1-\phi)\, u_y\, q_{y|y} - C(u_y, m_y) \right) q_{0|y} - d \right] N_y , \qquad (4)$$

where $q_{k|y}$ is the average local frequency of sites in state *k* neighboring a mutant individual. Obviously, the dynamics of the neighborhood depend also on the state of higher order terms including the pairs' neighbors, and so on. A complete description of the lattice dynamics therefore requires an infinite hierarchy of statistics governed by an infinite system of correlation equations, each one describing the spatial structure on a particular scale in relation to the subsequent one. To make a model tractable at the level of pair dynamics, we use a pair-approximation to close the exact system at that scale (Matsuda et al. 1992; Morris 1997, Appendix B).

The spatial invasion fitness $s_x(y)$ can then be defined as the per capita exponential growth rate of the mutant population governed by equation (4) (see Ferrière and Le Galliard 2001; Metz et al. 1992). The initial population dynamics of a rare mutant involve two distinct phases (van Baalen 2000b). In a first, short phase, a single mutant individual is born near its resident progenitor and either dies without living any descendants, or begins to invade locally until the spatial correlation structure stabilizes at a pseudo-equilibrium neighborhood structure denoted by $\tilde{q}_{0|y}$, $\tilde{q}_{x|y}$, $\tilde{q}_{y|y}$. Conditional on non-extinction during this first phase, the mutant population then expands or contracts while keeping its pseudo-equilibrium correlation structure. At the same time, the resident population remains close to its own equilibrium correlation structure. Spatial invasion fitness can therefore be defined as the mutant population growth rate during this phase (van Baalen and Rand 1998), and is given by

$$s_x(y)=\Big[\big(b(m)-C(u_y,m_y)\big)\tilde{q}_{0|y}-d\Big]+(n-1)\phi\ u_x\,\tilde{q}_{0|y}\,\tilde{q}_{x|y}+(n-1)\phi\ u_y\,\tilde{q}_{0|y}\,\tilde{q}_{y|y} \qquad (5)$$

A positive fitness implies that the invasion process enters a third phase during which the mutant phenotype displaces the resident. This expression relates to the notion of "direct" or "neighbor-modulated" fitness obtained by adding the effects on a focal individual's fitness of all phenotypes present in the neighborhood (see Frank 1998). Likewise, the spatial invasion fitness of a focal mutant sums the mutant neighbor-independent fitness (first term) and the effects of a resident (second term) and a mutant neighbor (third term), each weighed by the probabilities of such neighborhoods.

In practice, the spatial invasion fitness is determined by the equilibrium correlation structure of the resident population, as characterized by $\bar{q}_{x|x}$ and $\bar{q}_{x|0}$, and the pseudo-equilibrium correlation structure of the mutant population. The latter can be derived from the dynamics of a dimorphic population, when the mutant phenotype is rare and the resident phenotype is at equilibrium (Appendix D). The former is obtained from a model of the monomorphic resident population (Appendix C). The spatial statistics $\bar{q}_{x|x}$ and $\bar{q}_{x|0}$ relate to the empirical notion of habitat saturation, as originally introduced by (Brown 1978; Emlen 1982). The complementarity of $\bar{q}_{x|x}$ and $\bar{q}_{x|0}$ as descriptors of the population spatial structure pinpoints that, in fact, habitat saturation involves two distinct aspects of the population structure: "local aggregation", measured by $\bar{q}_{x|x}$, which is the level of crowding felt locally by any given individual; and "local contention", measured by $\overline{q}_{x|0}$, which is the level of competition between the neighbors of any vacant site, in which each of them could lay down an offspring.

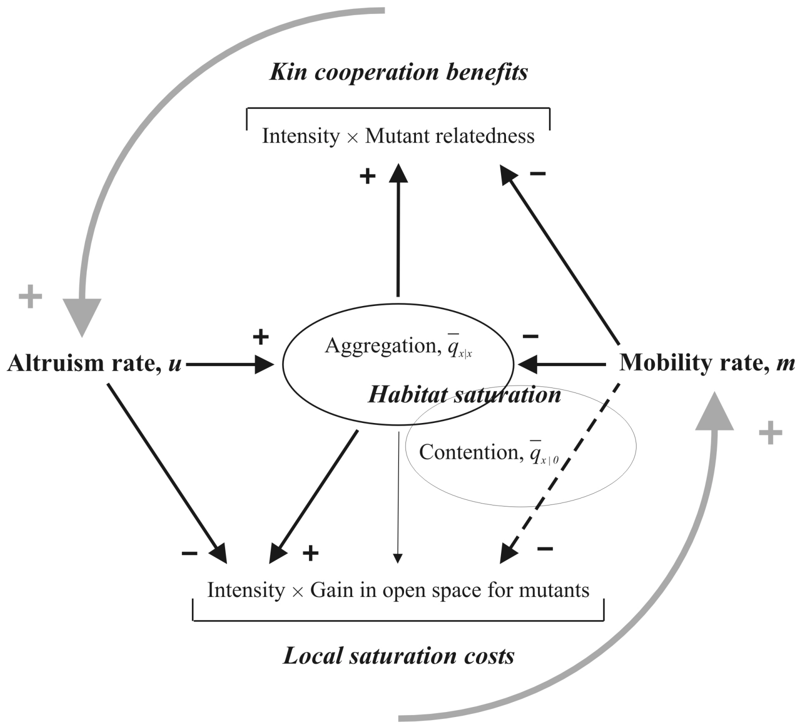


Figure 1. Selective pathways affecting altruism and mobility. The multiple links of both eco-evolutionary feedbacks (altruism→habitat saturation→kin cooperation benefits→altruism, and mobility→habitat saturation→local saturation costs→mobility) and of the selective interactions (altruism→habitat saturation→local saturation costs→mobility, and mobility→habitat saturation→kin cooperation benefits→altruism) can all be traced on this diagram. Curved gray arrows indicate selective pressures. Each pressure is evaluated as a marginal gain or loss in fitness, multiplied by an intensity factor. The pressure exerted by local saturation costs on altruism is negligible and thus not displayed here. Plain arrows refer to positive or negative effects established from the analysis of equations (6) to (8) (see also Appendix D). The strength of the effects is modulated by underlying parameters (cost parameters, habitat connectivity, life-history traits). The thinner arrow indicates a constantly weak effect. The dotted arrow indicates a complex combination of direct and indirect (via local contention) effects of mobility on the marginal gain in open space; the negative sign of this effect was established numerically. No arrow points to the intensity of the kin cooperation benefits, which depends solely on habitat connectivity (fixed parameter).

SELECTION GRADIENT AND SELECTIVE INTERACTION

The selection gradient (bracketed term in the right hand-side of the canonical eq. [1]) determines the direction of the adaptive process. Its first coordinate measures the selective pressures acting on mobility (see Appendix D)

$$\partial_m s_x(y) = \bar{q}_{0|x}\left[\left(\frac{d}{\bar{q}_{0|x}^{\ 2}} - (1-\phi)u_x\right)\partial_m \tilde{q}_{0|y} - \partial_m C\right], \tag{6}$$

where ∂_m denotes the derivative with respect to m_y evaluated at $m_y = m_x$. The derivative $\partial_m C$ is the marginal cost of mobility which measures the selective pressure for reducing the physiological cost of mobility ($\partial_m C = \nu$, the cost sensitivity). A second selective pressure is represented by $\partial_m \tilde{q}_{0|y}$, which is the marginal gain (or loss) of open space in the neighborhood of a mutant individual. This shows that the evolution of mobility is driven primarily by the advantage conferred during invasion to mutants who are surrounded by more open space than residents are. The intensity of this pressure, i.e. $\left(\frac{d}{\bar{q}_{0|x}^{\ 2}} - (1-\phi)u_x\right)$, is determined by the death rate, habitat connectivity, altruism rate, and local aggregation. The marginal gain of open space for mutants, $\partial_m \tilde{q}_{0|y}$, translates into a positive pressure on the mobility rate, whose strength appears to be a complex combination of the current mobility rate, local aggregation, and local contention (app. D).

The second coordinate of the selection gradient is given by

$$\partial_u s_x(y) = \bar{q}_{0|x}\left[(1-\phi)\bar{q}_{y|y} - \partial_u C\right] \tag{7}$$

(Appendix D). This expression describes two selective pressures acting on altruism (Le Galliard et al. 2003). The derivative $\partial_u C$ indicates the pressure for reducing the physiological cost of altruism. The local frequency $\bar{q}_{y|y}$ reflects the benefit of increasing altruism among mutants; the intensity of this selective pressure, i.e. $(1-\phi)$, only depends on habitat connectivity. The statistic $\bar{q}_{y|y}$ measures the probability that the recipient of an action performed by a mutant individual is a mutant itself, and so provides an appropriate measure of relatedness (Day and Taylor 1998; Ferrière and Le Galliard 2001). Equation (7) (from which a marginal Hamilton's rule can be derived, see Le Galliard et al. 2003) identifies the role that kin selection plays in the evolution of altruism. Relatedness can be expressed as a function of the basic environmental and life-history parameters

$$\bar{q}_{y|y} = \frac{d\ \phi}{d + (1-\phi)\ m\ \bar{q}_{0|x}} \tag{8}$$

which shows that the marginal gain resulting from interactions between relatives is higher in more aggregated populations (lower $\bar{q}_{0|x}$).

These results show altogether that the adaptive dynamics are mediated by eco-evolutionary feedback loops involving both altruism and mobility, a selective interaction between altruism and mobility (fig. 1), and a physiological feedback loop on altruism. Eco-evolutionary feedback loops involve the effects of one trait on the individuals' environment which in return modifies the selective pressures acting on adaptive variation in that trait. Insofar as the selection component $\partial_m \tilde{q}_{0|y}$ decreases with mobility (see Appendix D), equation (6) demonstrates a negative feedback of mobility on its own adaptive dynamics: the marginal gain in open space for mutants diminishes as mobility increases. This feedback involves both local aggregation (through the coefficient of $\partial_m \tilde{q}_{0|y}$ and the expression of $\partial_m \tilde{q}_{0|y}$ itself) and local contention (upon which $\partial_m \tilde{q}_{0|y}$ depends). In the degenerate though instructive case of a zero mobility cost, local aggregation becomes independent of mobility, whereas local contention increases monotonically with the mobility rate (Appendix C); thus, the corresponding eco-evolutionary feedback on mobility is mediated by local contention—not by local aggregation. From extensive numerical analysis it appears to be general that local contention plays the predominant role over local aggregation in shaping the eco-evolutionary feedback on mobility.

Likewise, given that in general local aggregation ($\bar{q}_{x|x}$) correlates positively with the level of altruism, equations (7) and (8) establish a positive feedback on altruism mediated by the local aggregation-component of habitat saturation and its effect on relatedness: local aggregation, hence relatedness, are usually higher in more altruistic populations, which favors the evolution of even more altruistic phenotypes. The evolution of altruism is also controlled by a physiological feedback when the cost pattern is non-linear. Then the pressure to reduce the physiological cost of altruism depends on the altruism rate: when costs are decelerating (accelerating) the marginal cost of altruism decreases (increases) with the level of altruism. Thus, the effect of this physiological feedback is positive for decelerating costs of altruism and negative for accelerating costs.

The selective interaction of altruism and mobility has five components (fig. 1)—two involving an effect of mobility on the selection gradient of altruism; three involving an effect of altruism on the selection gradient of mobility. As for the effect of mobility, equation (8) shows that mutant relatedness is negatively affected by mobility both directly (dependence on *m* of the right-hand side of eq. [8]) and indirectly via local aggregation ($\bar{q}_{x|x}$). Thus, increasing mobility tends to weaken the selective pressure acting on altruism. As for the effect of altruism, the intensity of the selective pressure for opening space, measured by $\left(\frac{d}{\bar{q}_{0|x}^{\,2}} - (1-\phi)u_x \right)$, indicates that there is, first, a direct, negative effect (term $-(1-\phi)u_x$) which arises because it pays off to interact with more neighbors in a strongly altruistic population. Notice that the corresponding cost of opening space due to the loss of help received from neighbors has been highlighted in the empirical literature as a 'benefit of philopatry' (Stacey and Ligon 1991). Second, there is an indirect, positive effect (term $d/\bar{q}_{0|x}^{\,2}$) which stems from the fact that there is more of a selective advantage to be gained from opening space by moving when local aggregation is high. The net effect depends on the level of altruism in the population. Numerical simulations show the selective pressure for mobility is weakened at low altruism, whereas it is enhanced at intermediate rates of altruism. Moreover, in species with accelerating costs, a further rise of altruism results in reduced local aggregation (for the large cost of altruism then severely depletes natality), hence a reversal to a negative net effect on the intensity of selection for opening space. Finally, there is an additional effect of altruism on the selection derivative $\partial_m \tilde{q}_{0|y}$ through local aggregation (see Appendix D), but this effect turns out to be negligible.

ADAPTIVE EVOLUTION OF SINGLE TRAITS

In general, the evolutionary dynamics of each single trait are monotonous and converge to a point attractor (which, under certain circumstances, depends on the population ancestral state). Each attractive point corresponds to a singularity of the adaptive dynamics where the selection derivative vanishes. Any small mutation arising around these singularities is selected against and fails to invade.

Altruism

Qualitatively, the adaptive dynamics of altruism primarily depends upon the pattern of physiological cost of altruism (see Le Galliard et al. (2003) for more details). Under the assumption of decelerating costs, ancestral selfishness can only be displaced by altruism as a result of rare, large mutations. There is a 'waiting time' for the adaptive rise of altruism that increases with the mobility rate. Only in the limiting case of a linear cost of altruism may pure selfishness remain unbeatable. This occurs mainly in species characterized by both (i) a 'strong' linear cost, for which the cost sensitivity κ is larger than a threshold equal to $\phi(1-\phi)$, which decreases as habitat connectivity increases; and (ii) a mobility rate larger than a threshold

$$m_l = b\frac{\phi(1-\phi)-\kappa}{\nu(\phi(1-\phi)-\kappa)+\kappa(1-\phi)}. \qquad (9)$$

Thus, the most unfavorable conditions for the evolution of altruism involve high cost sensitivity to altruism, high mobility, and high habitat connectivity.

In species with accelerating costs of altruism, the altruism rate evolving is lower in organisms that are more mobile. Under 'rapidly' accelerating costs (high κ and/or γ much larger than 1), the relationship between mobility and selected altruism is smooth, and at all mobility rates the selected rate of altruism is low. In contrast, under 'slowly' accelerating costs (low κ and γ close to 1) the relationship between mobility and selected altruism shows a sharp discontinuity: high levels of altruism evolve in species with low mobility whereas quasi-selfishness evolves in species with high mobility. When mobility is low, the high level of altruism that evolves can be approximated as

$$u^* = [\phi(1-\phi)/\kappa\gamma]^{1/(\gamma-1)}. \qquad (10)$$

which depends only on habitat connectivity and the parameters of the physiological cost of altruism. At intermediate mobility, the evolutionary outcome depends on the ancestral state of the population: if the ancestral altruism is low, quasi-selfishness evolves; if the ancestral altruism is sufficiently high, a high level of altruism approximated by equation (10) evolves.

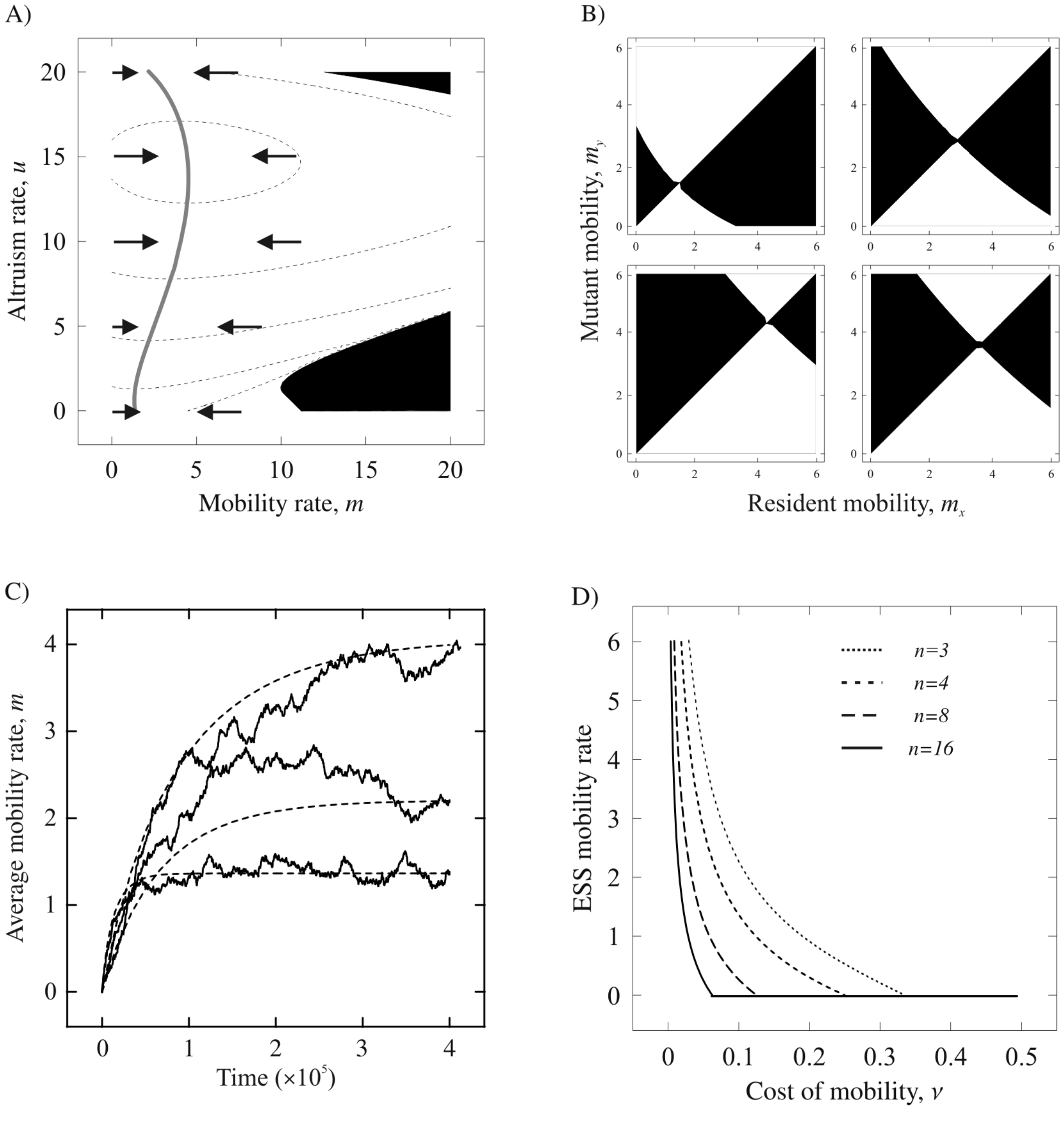


Figure 2. Adaptive dynamics of mobility. A, Singular mobility rates for an accelerating cost of altruism. Gray curve: mobility isocline; arrows indicate that the isocline is attractive. Dashed curves: contour lines of the spatial statistics $\overline{q}_{x|0}$ (local contention). Dark area: population extinction domain. Parameter values: $\gamma = 3$, $\kappa = 0.001$ and $\nu = 0.1$. B, Pairwise invasibily plots display fitness sign as a function of resident and mutant mobility rates. Dark (white) area: positive (negative) fitness. Singularities obtain as intersections between zero-fitness contour lines and the diagonal. A singularity is locally uninvadable if the fitness is negative above and below. A singularity is attractive if fitness is positive above the diagonal on the left and below the diagonal on the right. Parameter values from left to right, and from top to bottom: $u_x = 0$, $u_x = 5$, $u_x = 10$, $u_x = 17$. Other parameter values as in A. C, Average of ten independent stochastic simulations (continuous curves) and deterministic

approximation (dashed curves) at three different levels of altruism: $u_x = 0$ in the two lower curves, $u_x = 20$ in the two intermediate curves and $u_x = 10$ in the two upper curves. Mutation parameters: $k = 0.1$, $\sigma = 0.01$. D, Evolutionarily stable mobility rates with respect to the cost of mobility for different values of habitat connectivity. Other parameter values as in A.

Mobility

Mobility as a single adaptive trait always evolves toward a globally attractive and uninvadable singularity (fig. 2A). Below the evolutionary singularity, the selective pressure for opening space is higher than the pressure to reduce physiological costs, which selects for increased mobility. The intensity of this effect decreases up to the evolutionary singularity where the selection gradient vanishes. At high mobility, the selective pressure for decreasing the physiological costs of mobility favors a decreased mobility. Pairwise invasibility plots (fig. 2B) show that no mutant can invade the evolutionary singular point, which means that the attractive evolutionary singularity is also evolutionarily stable.

In purely selfish species (*u* = 0), the mobility ESS can be solved analytically and is given by

$$m^* = \frac{b(\sqrt{\nu(1-\nu)\phi(1-\phi)} - \nu(1-\nu))}{\nu(1-\nu)((1-\phi)-\nu)} \quad . \tag{11}$$

This expression solely depends on the cost of mobility, habitat connectivity, and the intrinsic birth rate. The ES mobility rate decreases with the cost of mobility *v*, and reaches $m^* = 0$ when $\nu > \phi$ (fig. 2D); it also decreases with habitat connectivity ($n = 1/\phi$, fig. 2D), and increases with the birth rate. Furthermore, *m** possesses the remarkable property of maximizing the local contention statistic, $\bar{q}_{0|0}$; thus, in selfish species, evolution of mobility alone maximizes habitat saturation around empty sites. Numerical simulations show that the same pattern applies to species at any level of altruism (*u* > 0): *m** responds to parameter variations in the same qualitative way; in addition, lower mobility is selected in response to higher mortality. Also, for all numerically tested parameter combinations, we find that *m** maximizes local contention.

The ES mobility rate *m** varies with the species' degree of altruism *u*. The empirical expectation is that more altruistic species are less mobile, but the typical pattern is more complex. First, zero mobility is selected for if the mobility cost is too high ($\nu > \phi$), irrespective of the degree of altruism. Second, whatever the type of altruism cost (decelerating or accelerating), there may be a slight decrease of *m** as *u* increases through very small values, but the singular mobility rate *m** increases with *u* over a wide range of degrees of altruism (see fig. 3A for the case of a decelerating altruism cost, fig. 4 for a linear cost, figs. 2A and 5 for accelerating costs). At very high values of *u*, *m** can decrease again with larger values of *u* in species with accelerating costs of altruism.

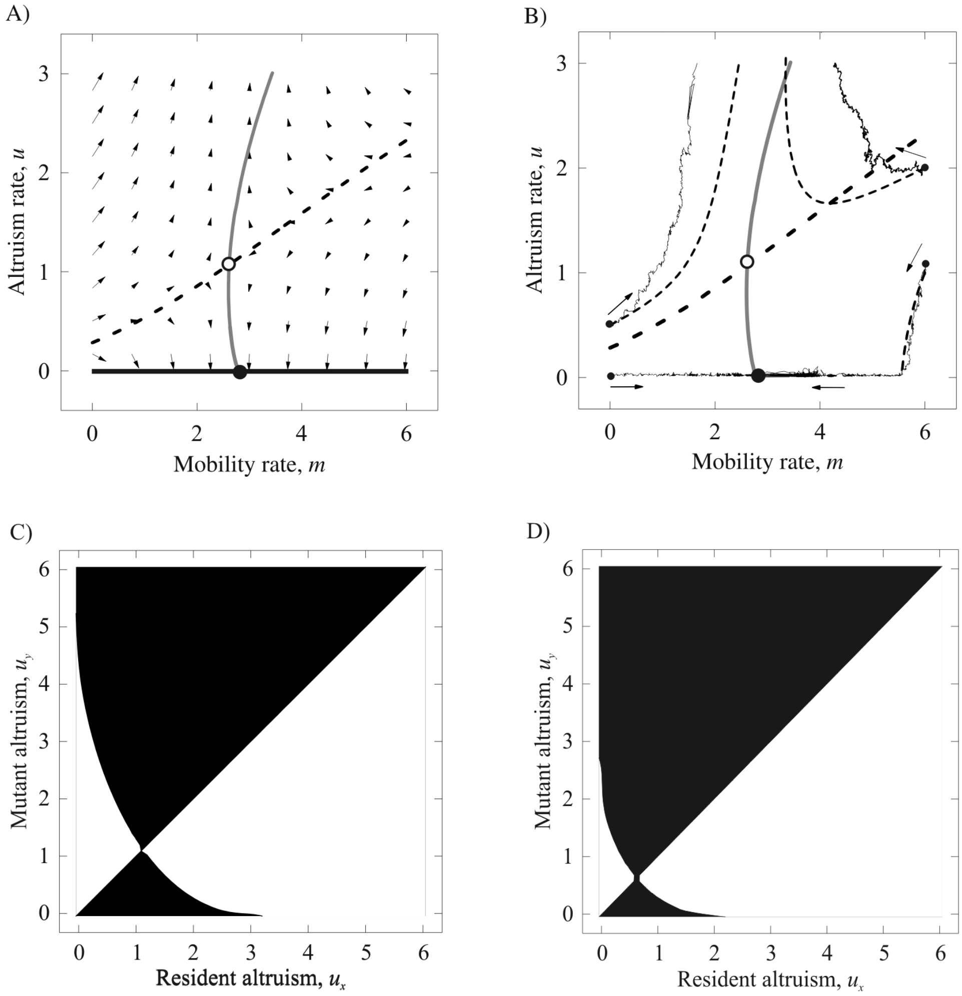


Figure 3. Coevolution of altruism and mobility with decelerating costs of altruism. A, The adaptive dynamics of altruism are bistable. For any mobility rate, with an initial value of altruism below a threshold, the evolutionary dynamics converge toward selfishness, whereas above the threshold, an evolution toward more altruism takes place. Plain gray curve: attractive mobility isocline. Dashed black curve: repelling altruism isocline. Plain black curve: attractive altruism isocline. Arrows: selection gradients. Open circle: repelling singularity. Close circle: attractive singularity. Parameters: $\gamma = 0.5$, $\kappa = 0.2$, $\nu = 0.05$. B, Average of ten stochastic simulations at four different initial conditions (continuous curve) and the deterministic predictions (dashed curves). Mutation parameters $k = 0.01$, $\sigma = 0.01$. C, D. Pairwise invasibility plots of the adaptive dynamics of altruism evaluated at the ESS mobility rate in a selfish population. Selfishness can be invaded by large investments in altruism (dark area), but low costs of mobility increase the threshold for altruism to invade. C. Same parameters as in B. C. Same parameters as in B, except $\nu = 0.1$. Other parameters as in fig. 2.

This pattern can be understood from the selective pressures that operate on *m*, as given by equation (6) and explained in fig. 1 and Appendix D. Equation (6) shows that local aggregation and the altruism rate have opposite effects on the intensity of the selective pressure to open space. Furthermore, local aggregation itself depends on the altruism rate, which sets an indirect effect of the latter on this selective pressure. At extremely low values of m, the dependency of local aggregation on u is weak. Therefore, as u increases, its direct, negative effect predominates and m^* tends to decrease. Over a range of larger u values, local aggregation rises rapidly with u, so that the indirect effect of u dominates over its direct effect: more mobility is selected for. As u becomes very large, the case of an accelerating cost of altruism causes a substantial reduction in natality, hence a decrease of local aggregation. Therefore, a larger value of u leads to the evolution of less mobility through both direct and indirect effects on local saturation.

ADAPTIVE CO-EVOLUTION OF ALTRUISM AND MOBILITY

We now consider the joint adaptive dynamics of mobility and altruism. Our study of evolutionary trajectories develops from the model described by the canonical equation (1). In general, the two isoclines calculated from this equation cross at a single attractive and evolutionarily stable singularity (ESS) of the co-adaptive dynamics, denoted by (u^*, m^*). The main conclusions of our analysis are tested against numerical simulations of an individual-based model that avoid all approximations assumed by the canonical equation (Appendix A).

Origin of altruism

To investigate conditions under which altruism can evolve from a purely selfish state, we use our model under the assumption of a decelerating cost of altruism. Altruism is indeed under the most stringent conditions to appear in species characterized by decelerating costs (Le Galliard et al. 2003). Also, in agreement with the classical empirical view, we assume that the selfish, ancestral state typically involves highly mobile individuals. Starting from selfishness associated with high mobility, the co-adaptive dynamics involves a first phase during which mobility decreases toward the critical value m^* given by equation (11) (see figs. 3A, B). The point (m^*,0) in the trait space is a halt for the deterministic dynamics predicted by equation (1). However, the stochasticity of the underlying individual process generates a different pattern. In a population where mutations may be large occasionally, mutants characterized by a large degree of altruism will eventually arise by chance, and displace the selfish resident (figs. 3C, D). Therefore, the evolutionary trajectory will sooner or later take off from the point (m^*,0). It can be seen numerically that the threshold on u increases as m^* increases. According to equation (10), this means that the waiting time for altruism to evolve is determined by the cost of mobility, habitat connectivity, and the intrinsic birth rate (in addition to the characteristics of the mutation process: mutation rate and mutation step variance, see fig. 7B in Le Galliard et al. 2003). The waiting time is shorter as the cost of mobility or habitat connectivity increases, or in species with a smaller birth rate (figs. 3C, D).

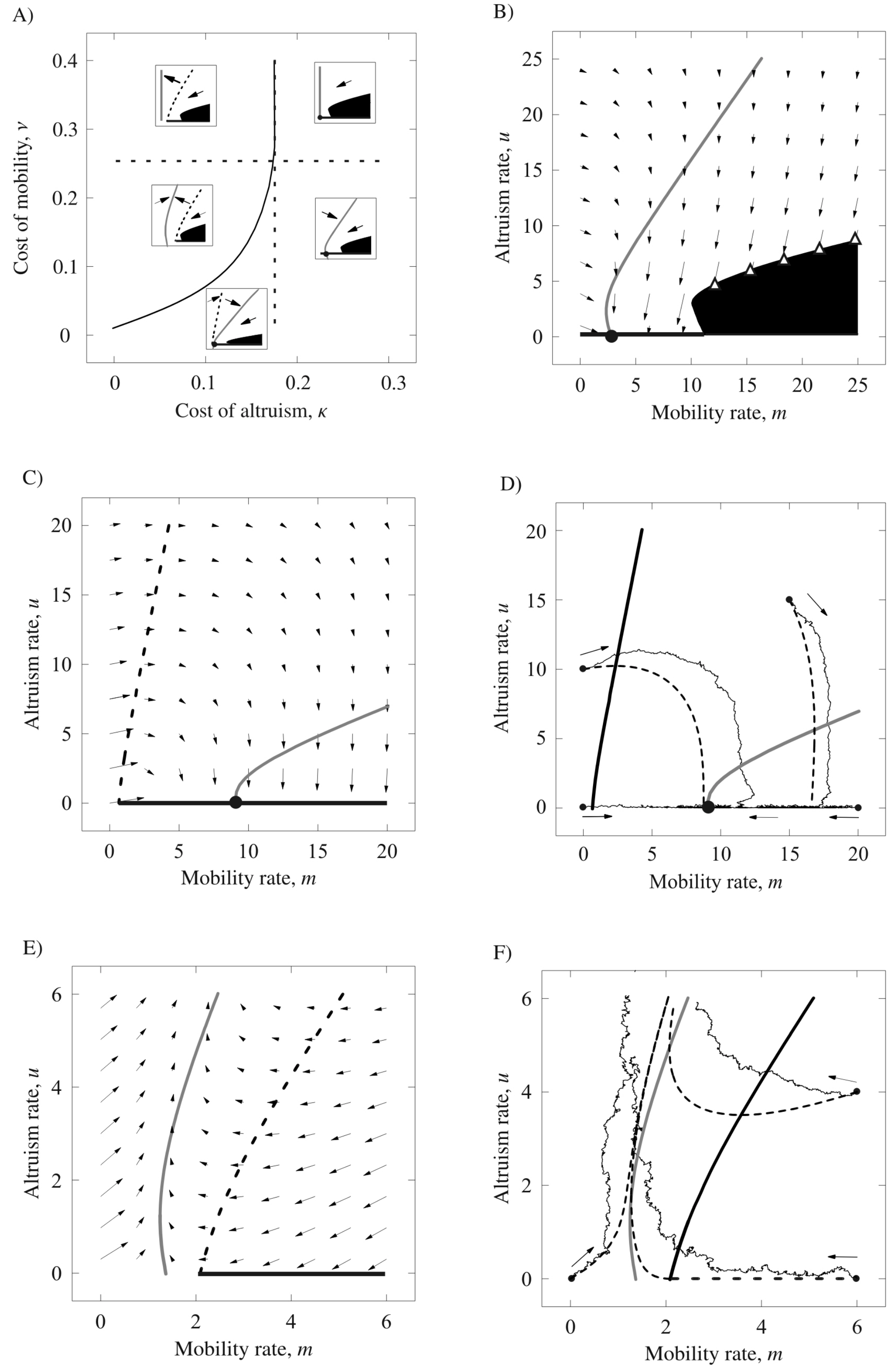


Figure 4. Coevolution of altruism and mobility with a linear cost of altruism. A, Classification of the adaptive dynamics according to the costs parameters *κ* and *ν*. Insets show the relative positions of the altruism (dashed) and mobility (gray) isoclines, the direction of evolution (arrows) and the population extinction domain (dark). In B-D, close circles indicate attractive ESSs. In D and F, small circles indicate ancestral states, and arrows give the direction of evolution. B, Convergence to selfishness under high cost sensitivity to altruism, $\kappa > \phi\,(1-\phi)$. Evolutionary suicide (triangles) can be observed when adaptive trajectories collide with the population extinction

boundary. This occurs when ancestral mobility is low and altruism is high, or when ancestral mobility is high and altruism is low. Parameter values: $\kappa = 0.25$, $\nu = 0.05$. C, Convergence to selfishness under low cost sensitivity to altruism, $\kappa < \phi\,(1-\phi)$, and low costs of mobility. The evolutionary change in low mobility ancestors involves first higher altruism together with increased mobility, until high mobility sets the stage for the secondary loss of altruism. Parameter values: $\kappa = 0.15$, $\nu = 0.01$. D, Stochastic trajectories. Parameter values as in C. E, Divergence to more altruism under low cost sensitivity to altruism, $\kappa < \phi\,(1-\phi)$, and high costs of mobility. Selection against mobility drives the divergence toward more altruism. Once altruism rises, mobility can increase secondarily, unless the physiological cost of mobility is too strong (i.e., $\nu > \phi$). For initial combinations of high mobility and low altruism, evolutionary suicide can occur. Parameter values: $\kappa = 0.1$, $\nu = 0.1$. F, Stochastic trajectories. Average of ten stochastic simulations (continuous curve), and deterministic approximation (dashed curves). Parameter values as in E. Mutation parameters for (D) and (F): $k = 0.01$; $\sigma = 0.05$ (D), $\sigma = 0.01$ (F). Other parameter values as in fig. 2.

Only in the limiting case of a linear pattern of altruism cost may selfishness be permanently uninvadable (fig. 4A). This occurs when the altruism cost parameter κ is large (fig. 4B), or when κ is small and the mobility cost ν is small too (figs. 4C, 4D). In this case, altruism may initially rise through small mutational steps provided that the ancestral state is not too mobile. Yet the trajectory of altruism evolution eventually reverts, heads back to the selfish state and homes in at the mobility ESS m^*, where no small or large mutation can invade (results not shown). In contrast, if κ is small and ν is large enough, selfishness is readily displaced by altruism even through infinitesimal mutations, as a result of selection for lower mobility (figs. 4E, F). Thus, the cost of mobility has a major impact on the origin of altruism, either determining whether the displacement of selfishness is possible (linear costs of altruism), or the timescale over which the evolution of altruism develops (decelerating costs of altruism).

Evolutionary dynamics of social traits

Once the evolutionary rise of altruism from a selfish and highly mobile ancestor is initiated, the assumption of an accelerating cost of altruism becomes more realistic. Then all possible evolutionary dynamics unfold along a continuum bounded by two archetypal templates, each involving two distinctive evolutionary phases. One template corresponds to species with a slowly accelerating cost of altruism (low κ and γ close to 1). Then the first phase is characterized by the evolution of less mobility while altruism shows little change; at the same time, local aggregation is enhanced (figs. 5A, B, C). During the second phase, altruism rises along with some increase in mobility (fig. 5C). How this second phase ends depends upon the cost of mobility. In the case of a high cost of mobility, the evolutionary trajectory simply heads to the ESS (which is a stable node equilibrium). In the case of a moderate cost of mobility, the trajectory tends to spiral around the ESS (which is a stable focus equilibrium in this case, figs. 5A, B). The eco-evolutionary feedback causes the level of local aggregation to parallel the damping oscillations of the adaptive traits. The ES altruism rate assumes higher values in relation with a larger cost of mobility. Under this pattern, evolutionary suicide can be observed when the evolution toward selfishness at high mobility causes convergence to the extinction boundary (fig. 5B).

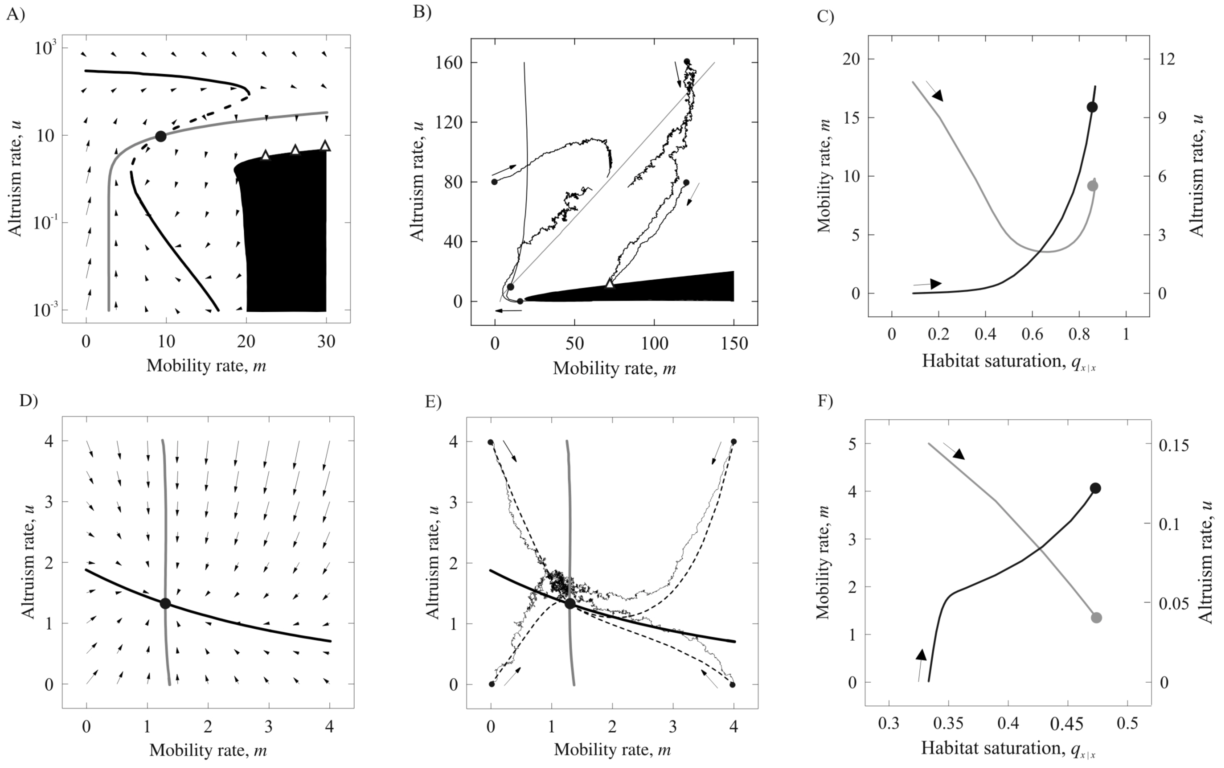


Figure 5. Coevolution of altruism and mobility with an accelerating cost of altruism. In all panels, close circles indicate attractive ESSs, arrows give the direction of evolution. In A and B, dark areas indicate population extinction. A, Convergence to a stable focus under slowly accelerating costs of altruism and intermediate costs of mobility. Parameter values: $\gamma = 1.2$, $\kappa = 0.05$, $\nu = 0.05$. B, Stochastic trajectories. Trajectories differ quantitatively from the deterministic approximation, but remain qualitatively similar. Stochasticity due to the finite population size and the randomness of mutational steps induces contingency: starting from the same mobile, altruistic ancestor, trajectories can either converge to the focus or collide with the extinction boundary (triangle). C, Relationship between mobility, altruism and habitat saturation (local aggregation) in the course of adaptive evolution. Mobility converges non monotonically to the stable focus (gray curve). Altruism remains at low levels as habitat saturation increases, and takes off secondarily (black curve). Parameter values as in A. D, Convergence to a stable node under rapidly accelerating costs of altruism. Parameter values: $\gamma = 2$, $\kappa = 0.05$, $\nu = 0.1$. E, Stochastic trajectories. Parameter values as in D. F, Relationship between mobility, altruism and habitat saturation during adaptive changes. Mobility converges monotonically to the stable node (gray curve). The evolution of altruism involves a first phase of increase while habitat saturation remains low, and increases more secondarily along with habitat saturation (black curve). Parameter values as in D, except $\kappa = 0.5$. Mutation parameters for stochastic trajectories: $k = 0.01$ and $\sigma = 0.01$. Other parameter values as in fig. 2.

The other dynamical template is specific of species with rapidly accelerating costs of altruism (figs. 5E, F). During the first phase of the evolutionary dynamics, the degree of altruism rises while mobility and the level of local aggregation remain essentially constant. The second phase drives the system to the ESS. It is characterized by a marked decrease in mobility, possibly along with a further increase in altruism, while local aggregation is enhanced significantly (fig. 5F). In this scenario, the ES altruism rate is usually low.

Adaptive patterns of altruism and mobility

Variations in the model parameters cause the joint ESS (m^*, u^*) to vary. The result is a correlation curve between u^* and m^* that represents the adaptive pattern of trait covariation in response to some underlying physiological, life-history, or environmental change. Here we analyze the covariation of u^* and m^* in species characterized by accelerating costs of altruism. More specifically, we analyze the correlation patterns obtained in response to life-history variation (i.e. on birth and death rates), and variation in constraints on mobility (i.e. in n and v). These patterns make predictions that could be tested empirically by comparing different populations of the same species (e.g. sharing the same life history, but differing in habitat structure), or different species (e.g. sharing the same type of habitat but differing in life-history traits). We focus on the following issues: When should we expect a negative correlation (or trade-off) to emerge between selected altruism and mobility? Are positive correlations or non-monotonic relationships possible, and which type of underlying changes would be sufficient to produce them?

The intrinsic birth rate b influences the evolution of mobility and altruism via an effect on habitat saturation (Appendix D). As b increases, local aggregation increases, which selects for more mobility and more altruism. Yet the adaptive response of mobility has the secondary effect of opposing the rise of local aggregation, thus selecting for less altruism. The net result can be documented by means of numerical simulations (fig. 6A): u^* decreases while m^* increases among more fecund species. However, the effect is significant only at low values of b, and in species incurring a low cost of altruism (small κ). Thus, from an empirical point of view, we should expect little effect of fertility to be detectable in the relationship between altruism and mobility at evolutionary equilibrium.

The intrinsic death rate d affects directly both local aggregation and relatedness. Increasing d reduces local aggregation, thus selecting for less mobility and less altruism. However, the former effect may be offset by a direct, positive impact of d on the intensity of the selective pressure for mobility (cf. eq. [6]); also, the latter effect may be softened by the non-monotonicity of the relationship between d and relatedness (cf. eq. [8]). Again, numerical simulations are needed to predict the net effect. Figure 6B displays the general pattern: a negative correlation between u^* and m^*, such that less altruism and more mobility occur at evolutionary equilibrium in long-lived species (smaller *d*). However, as for the result of a change in fertility, the effect of mortality variation is significant only in species incurring a low cost of altruism.

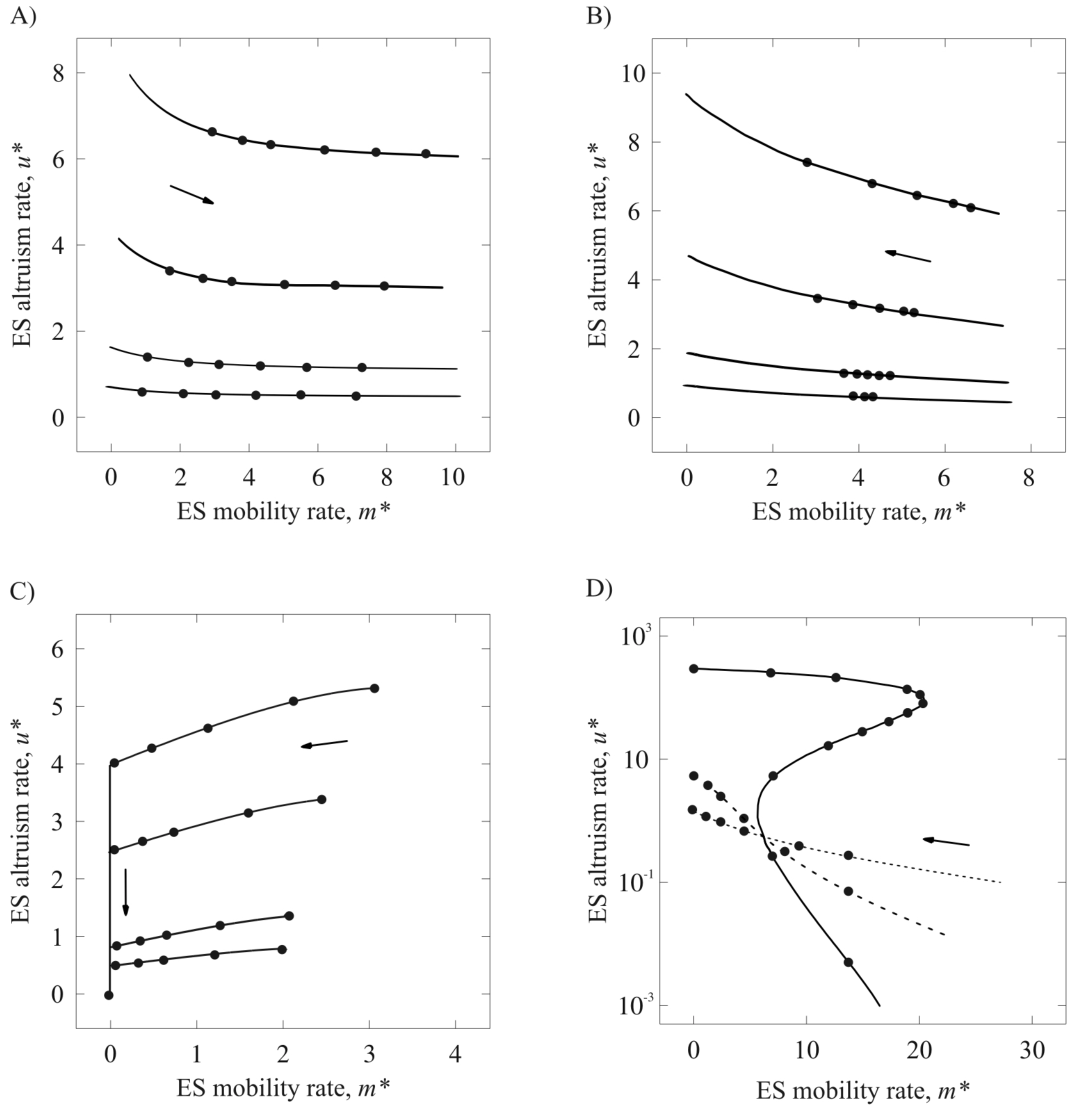


Figure 6. Adaptive correlations between altruism and mobility under accelerating costs of altruism. Arrows indicate the effect of increasing the underlying parameter, and dots correspond to specific values of the parameter. A, Effect of variation in the birth rate, *b*. The effect is displayed at different values of the cost parameter *κ*. Parameter values: $\gamma = 2$, $\nu = 0.1$. B, Effect of variation in the death rate, *d*. The effect is displayed at different values of the cost parameter *κ*. Parameters: $\gamma = 2$, $\nu = 0.1$, $b = 6$. In A and B, $\kappa = 0.01$, $\kappa = 0.02$, $\kappa = 0.05$, $\kappa = 0.1$ from top to bottom. C, Effect of habitat connectivity, *n*. The effect is displayed at different values of the cost parameter *κ*. Parameter values: $\gamma = 2.5$, $\nu = 0.1$; $\kappa = 0.005$, $\kappa = 0.01$, $\kappa = 0.05$, $\kappa = 0.1$ from top to bottom.. D, Effect of the cost of mobility, ν. The effect is displayed at three different levels of cost acceleration: $\gamma = 1.2$ (plain curve), $\gamma = 1.5$ (dashed curve), and $\gamma = 2$ (dotted curve). Parameter values: $\kappa = 0.05$. Other parameters as in fig. 2.

Variation in *n* or *v* may reflect different environmental constraints on individual mobility. As habitat connectivity *n* increases, selection for altruism becomes weaker (cf. eq. [7]), as well as selection for mobility. The former effect results from a decrease in habitat saturation when habitat connectivity increases all else being equal, which impacts negatively on the selective pressure to open space among more mobile mutants (cf. eq.[6]). These changes produce a positive correlation between altruism and mobility at evolutionary equilibrium (fig. 6C). In a triangular habitat ($n = 3$), selected altruism and mobility are maximal, whereas with a mean-field interaction ($n \to \infty$) adaptation always leads to a selfish, sessile behavior.

Increasing the cost of mobility *v* selects directly for less mobility, which increases mutant relatedness, thereby promoting the evolution of more altruism. In a range of very low values of *v*, mobility can evolve to high levels, which leads to low relatedness and therefore hampers the evolution of altruism. The resulting pattern is a negative, yet rather flat correlation between selected altruism and mobility (fig. 6D, *nota bene*: the altruism rate is shown on a log-scale). At very high values of *v*, mobility evolves to low levels, begetting high relatedness and the evolution of high altruism rate. Again the resulting pattern is flat, because the adaptive increase in *u* in response to increasing *v* is severely limited by the altruism cost acceleration. If the cost of altruism is slowly accelerating, however, the pattern is reverted over a range of intermediate values of *v*. Promoting the evolution of more altruism by increasing the cost of mobility then causes a marked increase of local aggregation. In turn, higher local saturation exerts a selective pressure for mobility which exceeds the accrued cost of mobility. Thus, the selective interaction between altruism and mobility favors more altruism along with more mobility (fig. 6D).

DISCUSSION

We have used the notion of spatial invasion fitness to model the adaptive dynamics of altruism and individual mobility, thereby setting up a unifying framework for the evolution of social traits. Our analysis backs up the empirical view that habitat saturation is a critical node of the selective interaction between the two traits (Emlen 1982; Koenig et al. 1992; Lambin et al. 2001). The "habitat saturation hypothesis" states that constraints on independent breeding favors philopatry and helping, and has provided a fruitful concept for approaching the evolution of social traits from the empirical end. Our theory leads to reexamining the basis and scope of this hypothesis, and clarifies the selective pathways whereby habitat saturation influences and becomes influenced by the evolution of social traits. Empirical analyses and recent models have predicted correlative patterns of altruism and mobility in response to different ecological constraints or demographic profiles. This study allows us to recast these various predictions in a single theoretical framework and to uncover alternatives that will hopefully stimulate further empirical research.

The notion of spatial invasion fitness

Defining invasion fitness for spatial ecologies is no trivial matter (Ferrière and Le Galliard 2001). Starting from demographic and behavioral processes operating at the individual level and locally between close neighbors, the invasion exponent of a simple system of correlation equations for the dynamics of a mutant population provides a tractable solution (van Baalen 2000a). The notion of spatial invasion fitness allows one to derive an explicit relationship between distinct components of selection on the one hand, and the characteristics of the individuals and their interactions on the other. Numerical simulations of individual-based models confirm that the spatial invasion fitness can predict qualitatively, and often quantitatively, the dynamics of the stochastic mutation-selection process.

The mathematical derivation of spatial invasion fitness proceeds by averaging over space the transition rates of pairs. This amounts to looking at the local structure of the mutant population as homogeneously replicated across the whole (infinite) lattice. The non-homogeneous distribution of the pairs containing mutants, induced by the finite size of the mutant population and the non-typical clustering pattern that may develop at the earliest stage of invasion, might require to incorporate correction terms (van Baalen 2000a). There is an interesting parallel to be drawn with the theory of evolutionary games in continuous space. In this context, the initial clustering of mutants requires to define fitness not from space averages of individual traits, but as the speed at which the front of a mutant cluster moves forward and propagates mutants through (Ellner et al. 1998; Ferrière and Michod 1996).

The habitat saturation hypothesis

Habitat saturation has long been put forward as a key hypothesis to explain the evolution of social behavior. The "habitat saturation hypothesis" was originally intended to explain the evolution of cooperative breeding in birds (Brown 1978; Emlen 1982; Koenig et al. 1992), and is now underlying theories for the evolution of delayed dispersal (Kokko and Lundberg 2001; Perrin and Lehmann 2001) and reproductive skew models of animal societies (Reeve et al. 1998). The general view is that habitat saturation drives the evolution of philopatry and altruism. By offering an explicit mathematical framework to deal with the interplay of social behavior and population dynamics, our analysis uncovers the whole complexity of the selective pathways whereby habitat saturation is involved in the evolution of social traits.

The habitat saturation hypothesis assumes that sociality evolves in two steps, which requires first the evolution of philopatry, and then the evolution of cooperation (Emlen 1997; Helms Cahan et al. 2002). As sites available for immigration and reproduction are rarely available, individuals competing for such vacancies are expected to incur strong costs of dispersal due to floating and queuing before gaining access to a territory. Thus, habitat saturation is predicted to favor delayed dispersal, conditions under which the cost of local crowding can be ameliorated by cooperating rather than simply competing (Kokko and Lundberg 2001; Pen and Weissing 2000). What causes habitat saturation in the first place? The scenario of 'ecological constraints' asserts that environmental factors constrain mobility to low levels, hence local crowding. Such environmental factors may involve habitat structure, physical predicaments to movement, or a large physiological cost of moving (Jarvis et al. 1994; Russell 2001). The 'life-history hypothesis' assumes that habitat saturation is more likely to occur in species with low mortality, in which the turnover of breeding sites is assumed to be slow (Arnold and Owens 1998).

Our analysis highlights a rather different evolutionary scenario. First, we show that there are two distinct components to habitat saturation, which play complementary roles in the evolution of social traits. "Local aggregation" ($\overline{q}_{x|0}$) measures habitat saturation *around individuals*, in line with the original definition by (Emlen 1982) of habitat saturation measuring the proportion of neighboring territories available for dispersal and reproduction. "Local contention" ($\overline{q}_{x|0}$) measures habitat saturation *around vacant sites*; it is oppositely related with the degree of clustering ($\overline{q}_{0|0}$), i.e. how isolated groups of neighbors are. Like in the ecological constraints model, a high cost of dispersal and low habitat connectivity are important determinants of the evolution of local aggregation and local contention. However, our model emphasizes that habitat saturation is a consequence of the evolution of low mobility, rather than the primary selective factor for that evolution. In other words, philopatry is a direct adaptive response to environmental constraints and physiological costs, rather than to habitat saturation.

In fact, neither the maximization of local aggregation nor that of local contention appear to be critical features of the co-evolutionary process. In single-trait evolution, however, the mobility rate evolves along with maximizing local contention—a prediction qualitatively similar to the finding that the number of competitors for territories (the limited resource) can be maximized by the evolution of habitat choice strategies (Kokko et al. 2001). Yet this remarkable maximization principle disappears when altruism evolves concomitantly. Furthermore, the evolution of low mobility and strong aggregation does not appear as an obligate evolutionary step toward sociality. Our model unravels the alternative scenario of a population initiated in the selfish-highly mobile ancestral state that first evolves a substantial degree of altruism while aggregation remains low; adaptive evolution secondarily favors less mobility, which may lead to strong aggregation. Such a scenario is expected when the cost of altruism is rapidly accelerating and the cost of mobility is low, in which case the altruism rate eventually selected should be low.

One key feature of our theory is that habitat saturation is not treated as a fixed parameter, but as a pair of dynamical variables that close the eco-evolutionary feedback loops entangling altruism and mobility. Local aggregation and local contention are, respectively, the pivotal factors of the two eco-evolutionary feedback loops cycling through altruism, and mobility. When both traits co-evolve, local aggregation turns out to be the dominant mediator of the selective interaction between both traits. Local aggregation responds antagonistically to evolutionary change of altruism and mobility, which in return affects the selective pressures acting on both traits. Such essential evolutionary feedbacks and selective interactions have been ignored in most previous models of social evolution (but see Kokko and Lundberg 2001).

Correlative patterns of social traits

Habitat saturation models predict that, at evolutionary equilibrium, (i) altruism and mobility should correlate negatively, and (ii) more altruism, hence less mobility, should be observed in populations characterized by stronger constraints on dispersal or lower mortality. Our theory, however, shows that the evolutionary outcome cannot be predicted solely from the effect of habitat saturation and its hypothesized underlying ecological or demographic determinants. This is because (i) habitat saturation is a dynamic variable entangled in the eco-evolutionary feedbacks involving altruism and mobility; (ii) the adaptive change of either trait also has a direct influence on the selection gradient of the other trait; and (iii) life-history traits (birth and death rates) have effects on the evolutionary dynamics independently of their influence on habitat saturation.

As a consequence, we find that selected altruism correlates positively with the cost of mobility and negatively with habitat connectivity; and we predict a positive correlation between selected altruism and selected mobility in response to changes in habitat connectivity or in the cost of mobility within a range that excludes extremely low and high values. The finding that low habitat connectivity or high cost of mobility selects for more altruism in our model suggests that comparative studies should find consistent relationships between physiological and habitat constraints on dispersal, and levels of cooperation. Some recent intra-specific comparisons in vertebrates (birds, mammals) have reported a negative effect of landscape connectivity on investment in helping (e.g., Russell 2001; Spinks et al. 2000). Also, in the group of African mole rats (Bathyergidae), cooperative breeding has been linked to the scarce and heterogeneous distribution of resources in arid landscapes, which results in high costs of mobility (Jarvis et al. 1994). In agreement with our findings, the comparative analysis of sociality (as measured by reproduction skew) yields a rough correlation between costs of mobility and cooperation, with the eusocial species culminating in correspondence with the most arid environment (Faulkes and Bennett 2001; Jarvis et al. 1994).

Empirical data relating altruism and mobility are scant, especially because quantitative assessments of dispersal abilities in social and asocial species are difficult to obtain. Phylogenetic comparative analyses of social traits in birds are still insufficient to test our prediction that more cooperation, as an adaptive response to ecological constraints, could be associated with higher levels of mobility. However, the observation that the correlation patterns between dispersal and cooperative breeding depends on the taxonomic level at which the analysis is performed (Arnold and Owens 1999) strongly warrants further analyses. The occurrence of a dispersing morph in captive colonies of the eusocial naked mole rat *Heterocephalus glaber* (O'Riain et al. 1996) could also be the manifestation of an adaptive association between strong altruism and a special ability to disperse. The fact that this dispersing morph participates little in cooperative activities in their new colonies further suggests that constraints on mobility might generate disruptive selective pressures on the social traits, leading to evolutionary branching and a stable genetic polymorphism of selfish-mobile and altruistic-sessile phenotypes. Although the evolutionary branching of social traits was not observed in our study, stable coexistence of different social strategies was first hypothesized by van Baalen and Rand (1998), and has been observed in cellular automaton models involving regular lattices (den Dulk and Brinkers 2000; Koella 2000).

We predict each life-history trait (death rate and intrinsic birth rate) to have, in isolation, little influence on the selected combination of altruism and mobility. On the one hand, we expect an increase in the intrinsic birth rate to drive a decrease in altruism and an increase in mobility, although the predicted pattern is fairly flat and probably difficult to detect in real data. On the other hand, more altruism is expected to evolve among species with the highest mortality rates. Comparative analyses in birds have attempted to relate social behavior with nestling mortality and adult mortality (Hatchwell and Komdeur 2000). It was found that nestling mortality had no detectable influence on the distribution of social characters (Poiani and Pagel 1997), in agreement with our prediction that the intrinsic birth rate (which can be seen as combining reproductive potential and offspring mortality) is likely to have undetectable effects. The analysis of the whole available phylogeny of birds yields a pattern of stronger cooperation along with lower adult mortality (Arnold and Owens 1998). This empirical pattern supports our finding of an effect of the death rate, but opposes the direction of the effect that we predict. The pattern could be recovered in our model, however, under the assumption that lower mortality trades-off across species with lower natality, which is known to occur in birds (Arnold and Owens 1998). This suggests that, in general, covariation of life-history traits are important to understand adaptive patterns of social traits.

Co-evolution versus single-trait evolution

The study of correlative patterns was made possible by considering the joint adaptive evolution of social traits. The fact that adaptive evolution takes place in a two-dimensional trait space has indeed major consequences on the evolutionary trajectory of either trait. One basic structural reason is that evolutionary trajectories on a more-than-one dimensional adaptive landscape can by-pass fitness valleys which would block the population if only one dimension was available to evolutionary change.

The dimensionality of the trait space has a major influence on the evolutionary onset of altruism under the assumption of a weak linear cost of altruism. With only the altruism trait evolving, a selfish and mobile ancestor is always uninvadable (Le Galliard et al. 2003). In contrast, assuming that mobility can co-evolve, selfishness can be readily displaced, even through infinitesimal mutations, provided that the cost of mobility is high enough. When the cost of mobility is low, altruism is expected to evolve as a single trait in selfish and poorly mobile ancestors, whereas traits co-evolution drives an adaptive rise of mobility, which prevents altruism to gain a permanent foothold in the population. Therefore, evolutionary constraints on mobility (as opposed to mere low mobility) are critical for altruism to evolve and maintain.

The coevolutionary dynamics also have considerable impact on the evolution of mobility. As a single trait, the evolution of mobility is driven by the selective pressure to open space for mutants during invasion, and opposed by physiological costs. The strength of the former is directly and indirectly (through local aggregation) modulated by the degree of altruism. First, more altruism weakens the positive selective pressure to open space for mutants, which emphasizes that the so-called benefits of philopatry operate against the evolution of dispersal (Perrin and Goudet 2001; Stacey and Ligon 1991). Second, and less intuitively, altruism influences the spatial structure of the population, by increasing local aggregation, which selects for more mobility. When both traits evolve, the selective pressure induced by the benefits of philopatry will be opposed by the enhanced aggregation experienced by more altruistic phenotypes, which in turn favors higher mobility rates. Typically, this synergistic selective interaction between altruism and mobility impacts species characterized by slowly accelerating costs of altruism and moderate costs of mobility (see fig. 5A). In such species, mobility selected through the coevolutionary process can be considerably higher than that predicted in a selfish species, and considerably lower than that predicted in a highly altruistic species. Thus, neglecting the propensity for altruism to co-evolve with mobility can lead to dramatically underestimate or overestimate the level of mobility that can be favored by natural selection.

The dimensionality of the trait space also has a marked effect on the evolutionary dynamics of altruism when the physiological cost is slowly accelerating. In this case, intermediate degrees of altruism are evolutionarily unstable when the altruism is the only trait evolving. In the co-evolutionary scenario, the strong evolutionary attractiveness of the mobility rate that maximizes fitness for each of these altruism trait values suffices to turn one mobility-altruism phenotype into an attractive strategy. This ESS still bears the footprint of the altruism one-dimensional instability in the fact that it behaves as a focus to evolutionary trajectories that spiral around it. This geometrical property has consequences when interpreting variation in social behavior across populations or species. Even if populations originate in the same ancestral state and share the same physiological, demographic and ecological features, they may present radically different suites of social traits should they be observed at different epochs of their evolutionary history. The spiraling dynamics around an ESS entails indeed that the same trajectory will display consecutively high and low levels of mobility and altruism in all four possible combinations. This is yet one more complication that could inform comparative analyses confronting the lack of regularities in patterns of social traits and potential correlates (Arnold and Owens 1999).

Concluding remarks

The "habitat saturation hypothesis", the "ecological constraint model", the "life history hypothesis" represent multiple attempts at singling out simple and general factors of social evolution. Each approach has contributed to a better understanding of the evolution of sociality. By integrating some of their key ingredients, our model leads to the conclusion that no simple determinism should be expected for the origin of social behavior or the evolution of strong cooperative interaction. This emphasizes that inferences from empirical or theoretical studies based on univariate analyses are likely to be hindered by the very complexity and diversity of factors involved in the evolution of social traits (Crespi and Choe 1997). However, some general principles apply: physiological or ecological constraints on mobility are essential to explain the origin of altruism; all evolutionary trajectories can be related to only two archetypes, contrasting routes to sociality; adaptive patterns of covariation among social traits could be understood in response to multivariate changes.

Eco-evolutionary feedbacks and selective interactions are key to the co-evolutionary dynamics of social traits. Taking them into account allows us to address one of the currently most debated issue in the biology of social behavior: whether the high relatedness predicted by Hamilton's original kin selection theory (1964), and found between interacting individuals of several social species, is the direct consequence of physiological or ecological constraints on dispersal, or that of more involved mechanisms of active assortment, involving communication, cognition, and habitat choice (Hamilton 1975). We offer the alternative views that in some social systems, both limited mobility and strong altruism can form the *joint* adaptive response to a complex web of multiple, interacting selective mechanisms; and that in other social systems, the dynamical structure of the population can cause evolution to promote high mobility without compromising the likelihood of passive assortments between altruistic individuals.

These conclusions are illustrative of a wealth of evolutionary dynamics structurally rooted in the multi-dimensionality of the adaptive trait space. Most evolutionary modeling of quantitative characters has concentrated on single traits so far. Hopefully this study will foster the appreciation that multidimensional adaptive evolution can proceed in radically different directions, and will pave the way toward the incorporation of important genetic factors (like genetic correlations between traits or trait-dependent genetic variation) into theories of phenotypic evolution. Multidimensional models of evolutionary dynamics should eventually allow one to make more robust and testable predictions, and to assess the scope of adaptive factors across broader ranges of biological phenomena.

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LITERATURE CITED

Arnold, K. E., and I. P. F. Owens. 1998. Cooperative breeding in birds: a comparative test of the life-history hypothesis. Proceedings of the Royal Society London B 265:739-745.

Arnold, K. E., and I. P. F. Owens. 1999. Cooperative breeding in birds: the role of ecology. Behavioral Ecology 10:465-471.

Brown, J. L. 1978. Avian communal breeding systems. Annual Review of Ecology and Systematics 9:123-155.

Choe, J. C., and B. J. Crespi. 1997. The evolution of social behavior in insects and arachnids. Cambridge University Press, Cambridge.

Clutton-Brock, T. H., D. Gaynor, R. Kansky, A. D. C. MacColl, G. McIlrath, P. Chadwick, P. N. M. Brotherton, J. M. O'Riain, M. Manser, and J. D. Skinner. 1998. Costs of cooperative behaviour in suricates (*Suricata suricatta*). Proceedings of the Royal Society London B 265:185-90.

Cockburn, A. 1998. Evolution of helping behavior in cooperatively breeding birds. Annual Reviews of Ecology and Systematics 29:141-177.

Cohen, D., and U. Motro. 1989. More on optimal dispersal: taking into account the cost of dispersal mechanism. The American Naturalist 134:659-663.

Crespi, B. J. 2001. The evolution of social behavior in microorganisms. Trends in Ecology and Evolution 16:178-183.

Crespi, B. J., and J. C. Choe. 1997. Explanation and evolution of social systems *in* J. C. Choe and B. J. Crespi, eds. The evolution of social behavior in insects and arachnids. Cambridge University Press, Cambridge.

Day, T., and P. D. Taylor. 1998. Unifying genetic and game theoretic models of kin selection for continuous traits. Journal of Theoretical Biology 194:391-407.

den Dulk, P., and M. Brinkers. 2000. Evolution of altruism in viscous populations: effects of altruism on the evolution of migrating behavior. Pp. 457-466 *in* M. Schoenauer, K. Deb, X. Yao, E. Lutton, J. J. Merelo and H.-P. Shwefel, eds. Parallel problem solving from nature. Springer, Berlin.

Dieckmann, U., and R. Law. 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. Journal of Mathematical Biology 34:579-612.

Ellner, S. P., A. Sasaki, Y. Haraguchi, and H. Matsuda. 1998. Speed of invasion in lattice population models:pair-edge approximation. Journal of Mathematical Biology 36:469-484.

Emlen, S. T. 1982. The evolution of helping. I. An ecological constraints model. The American Naturalist 119:29-39.

Emlen, S. T. 1997. Predicting family dynamics in social vertebrates. Pp. 228-253 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. Blackwell Science, Oxford.

Eshel, I. 1983. Evolutionary and continuous stability. Journal of Theoretical Biology 103:99-111.

Faulkes, C. G., and N. C. Bennett. 2001. Family values: group dynamics and social control of reproduction in African mole-rats. Trends in Ecology and Evolution 16:184-190.

Ferrière, R., and J.-F. Le Galliard. 2001. Invasion fitness and adaptive dynamics in spatial population models. Pp. 57-79 *in* J. Clobert, E. Danchin, A. A. Dhondt and J. Nichols, eds. Dispersal. Oxford University Press, Oxford.

Ferrière, R., and R. Michod. 1996. The evolution of cooperation in spatially heterogeneous populations. The American Naturalist 147:692-717.

Frank, S. A. 1995. Mutual policing and repression of competition in the evolution of cooperative groups. Nature 377:520-522.

Frank, S. A. 1998. Foundations of social evolution. Princeton University Press, Princeton.

Geritz, S. A. H., E. Kisdi, G. Meszena, and J. A. J. Metz. 1998. Evolutionary singular strategies and the adaptive growth and branching of the evolutionnary tree. Evolutionary Ecology 12:35-57.

Gillespie, D. T. 1976. A general method for numerically simulating the stochastic time evolution of coupled chemical reactions. Journal of Computational Physics 22:403-434.

Grafen, A. 1984. Neutral selection, kin selection, and group selection *in* J. R. Krebs and N. B. Davies, eds. Behavioral Ecology. Blackwell, Oxford.

Hamilton, W. D. 1964a. The genetical evolution of social behaviour, I. Journal of Theoretical Biology 7:1-16.

Hamilton, W. D. 1964b. The genetical evolution of social behaviour, II. Journal of Theoretical Biology 7:17-52.

Hamilton, W. D. 1975. Innate social aptitudes of man: an approach from evolutionary genetics. Pp. 133-155 *in* R. Fox, ed. Biosocial Anthroplogy. Wiley, New York.

Hatchwell, B. J., and J. Komdeur. 2000. Ecological constraints, life history traits and the evolution of cooperative breeding. Animal Behaviour 59:1079-1086.

Helms Cahan, S., D. T. Blumstein, L. Sundström, L. Liebig, and A. Griffin. 2002. Social trajectories and the evolution of social behaviour. Oikos 96:206-216.

Jarvis, J. U. M., M. J. O'Riain, N. C. Bennett, and P. W. Sherman. 1994. Mammalian eusociality: a family affair. Trends in Ecology and Evolution 9:47-51.

Kelly, J. K. 1992. Restricted migration and the evolution of altruism. Evolution 46:1492-1495.

Kelly, J. K. 1994. The effect of scale dependent processes on kin selection: mating and density regulation. Theoretical Population Biology 46:32-57.

Koella, J. 2000. The spread of altruism versus the evolutionary response of egoists. Proceedings of the Royal Society London B 267:1979-1985.

Koenig, W. D., F. A. Pitelka, W. J. Carmen, R. L. Mumme, and M. T. Stanback. 1992. The evolution of delayed dispersal in cooperative breeders. The Quaterly Review of Biology 67:111-150.

Kokko, H., and P. Lundberg. 2001. Dispersal, migration, and offspring retention in saturated habitats. The American Naturalist 157:188-202.

Kokko, H., W. J. Sutherland, and R. A. Johnstone. 2001. The logic of territory choice: Implications for conservation and source-sink dynamics. The American Naturalist 157:459-463.

Lambin, X., J. Aars, and S. B. Piertney. 2001. Dispersal, intraspecific competition, kin competition and kin facilitation: a review of the empirical evidence. Pp. 110-122 *in* J. Clobert, E. Danchin, A. Dhondt and J. D. Nichols, eds. Dispersal. Oxford University Press, Oxford.

Le Galliard, J.-F., R. Ferrière, and U. Dieckmann. 2003. The adaptive dynamics of altruism in spatially heterogeneous populations. Evolution 57:1-17.

Leimar, O. In press. Multidimensional convergence stability and the canonical adaptive dynamics *in* U. Dieckmann and J. A. J. Metz, eds. Cambridge Studies in Adaptive Dynamics. Cambridge University Press, Cambridge.

Marrow, P., U. Dieckmann, and R. Law. 1996. Evolutionary dynamics of predator-prey systems: an ecological perspective. Journal of Mathematical Biology 34:556-578.

Matsuda, H., N. Ogita, A. Sasaki, and K. Sato. 1992. Statistical mechanics of population - The lattice Lotka-Volterra model. Progress of Theoretical Physics 88:1035-1049.

Maynard Smith, J., and E. Szathmary. 1995. The major transitions in evolution

Metz, J. A. J., R. M. Nisbet, and G. S. A. H. 1992. How should we define fitness for general ecological scenarios ? Trends in Ecology and Evolution 7:198-202.

Michod, R. E. 1999. Darwinian dynamics - Evolutionary transitions in fitness and individuality. Princeton University Press, Princeton.

Mittledorf, J., and D. S. Wilson. 2001. Population viscosity and the evolution of altruism. Journal of Theoretical Biology 204:481-496.

Morris, A. 1997. Representing spatial interactions in simple ecological models. University of Warwick, Coventry.

O'Riain, M. J., and S. Braude. 2001. Inbreeding versus outbreeding in captive and wild populations of naked mole-rats. Pp. 143-154 *in* J. Clobert, E. Danchin, A. A. Dhondt and J. D. Nichols, eds. Dispersal. Oxford University Press, Oxford.

O'Riain, M. J., J. U. M. Jarvis, and C. G. Faulkes. 1996. A dispersive morph in the naked mole-rat. Nature 380:619-621.

Pen, I., and F. J. Weissing. 2000. Towards a unified theory of cooperative breeding: the role of ecology and life history re-examined. Proceedings of the Royal Society London B 267:2411-2418.

Perrin, N., and J. Goudet. 2001. Inbreeding, kinship, and the evolution of natal dispersal. Pp. 123-142 *in* J. Clobert, J. D. Nichols, E. Danchin and A. A. Dhondt, eds. Dispersal. Oxford University Press, Oxford.

Perrin, N., and L. Lehmann. 2001. Is sociality driven by the costs of dispersal of the benefits of philopatry? A role for kin-discrimination mechanisms. The American Naturalist 158:471-483.

Poiani, A., and M. Pagel. 1997. Evolution of avian cooperative breeding: comparative tests of the nest predation hypothesis. Evolution 51:226-240.

Queller, D. C. 1992. Does population viscosity promote kin selection? Trends in Ecology and Evolution 7:322-324.

Rand, D. 1998. Correlation equations and pair approximation for spatial ecologies. Pp. 100-143 *in* J. Mc Glade, ed. Advanced ecological theory. Blackwell Science.

Reeve, H. K., S. T. Emlen, and L. Keller. 1998. Reproductive sharing in animal societies: reproductive incentives or incomplete control by dominant breeders ? Behavioral Ecology 9:267-278.

Russell, A. F. 2001. Dispersal costs set the scene for helping in an atypical avian cooperative breeder. Proceedings of the Royal Society London B 268:95-99.

Spinks, A. C., J. U. M. Jarvis, and N. C. Bennett. 2000. Comparative patterns of philopatry and dispersal in two common mole-rat populations: implications for the evolution of mole-rat sociality. Journal of Animal Ecology 69:224-234.

Stacey, P. B., and J. D. Ligon. 1991. The benefits-of-philopatry hypothesis for the evolution of cooperative breeding : variation in territory quality and group size effects. The American Naturalist 137:831-846.

Taylor, P. D. 1992. Inclusive fitness in a homogeneous environment. Proceedings of the Royal Society London B 249:299-302.

Thorne, B. L. 1997. Evolution of eusociality in termites. Annual Review of Ecology and Systematics 28:27-54.

van Baalen, M. 2000a. Contact networks and the evolution of virulence: implications for management *in* U. Dieckmann, J. A. J. Metz and W. Sabelis, eds. Virulence management. Cambridge University Press.

van Baalen, M. 2000b. Pair approximation for ecological interactions on different geometries. Pp. 359-387 *in* U. Dieckmann, R. Law and J. A. J. Metz, eds. The geometry of ecological interactions: simplifying spatial complexities. Cambridge University Press, Cambridge.

van Baalen, M., and D. Rand. 1998. The unit of selection in viscous populations and the evolution of altruism. Journal of Theoretical Biology 193:631-648.

West, S. A., M. G. Murray, C. A. Machado, A. S. Griffin, and E. A. Herre. 2001. Testing Hamilton's rule with competition between relatives. Nature 409:510-513.

West, S. A., I. Pen, and A. S. Griffin. 2002. Cooperation and competition between relatives. Science 296:72-75.

Wilson, D. S., G. B. Pollock, and L. A. Dugatkin. 1992. Can altruism evolve in purely viscous populations? Evolutionary Ecology 6:331-341.

Wilson, E. O. 1975. Sociobiology: the new synthesis. Harvard University Press, Cambridge.

Wolfram, S. 1991. Mathematica: a system for doing mathematics by computer. Addison-Wesley, Redwood City, California.

APPENDIX A

We simulate the evolutionary process on a random lattice with periodic boundaries and 900 sites. The simulation starts by distributing individuals of an ancestral phenotype randomly over half of the lattice. Mutations generate variability with a probability k per birth event. The mutant phenotype is obtained by adding a mutation effect drawn randomly from a normal probability distribution, with zero mean and mutational variance σ^2. When a negative value is produced, the mutant phenotype is reset to zero. We use the minimal process method to simulate the time-continuous stochastic dynamics of the population (Gillespie 1976).

APPENDIX B

Notations of the models are defined in Appendix E. We consider a random, regular network with a large number of homogeneous sites, and a dimorphic population of mutants, called y, and residents, denoted by x. A mutant located at a site z on the lattice is affected by birth, death and movement, respectively

$$\begin{aligned} & b_y(z)=(b+\sum_{j=(x,y)}\phi\, u_j\, n_{j|y}(z)-C(u_y,m_y))\phi\, n_{o|y}(z)\,, \\ & d_y(z)=d\,, \qquad \text{(B1)} \\ & m_y(z)=m\phi n_{0|y}(z)\,. \end{aligned}$$

To derive the mean path of the mutant population size, we average the birth and death rates described in equation (B1) over all sites of the lattice occupied by the mutant, which gives

$$\frac{dN_y}{dt}=((b-C(u_y,m_y))\phi\, n_{0|y}-d)\,N_y+\sum_{j=(x,y)}\phi^2 u_j\sum_z n_{j|y}(z)\,n_{0|y}(z)\,, \qquad \text{(B2a)}$$

where $n_{0|y}$ is the average of $n_{0|y}(z)$ over the lattice. The third expression in (B2a) is a cross-product between random variables describing alternative neighborhoods of a mutant individual. Assuming that sites are distributed according to a multinomial probability distribution law and that the neighbors of pairs of sites can be considered as independent (Morris 1997), this term simplifies in

$$\sum_z n_{j|y}(z)\,n_{0|y}(z)=N_y\;\; n\,(n-1)\,q_{j|y}\;q_{0|y}\,, \qquad \text{(B2b)}$$

where $q_{k|y} = n_{k|y} / n$ is the average local frequency of type *k* sites neighboring a mutant. Then, the mean path of the mutant population size follows

$$\frac{dN_y}{dt} = ((b + \sum_{j=(x,y)} (1-\phi) u_j q_{j|y} - C(u_y, m_y)) q_{0|y} - d) N_y = \lambda_y N_y . \tag{B2c}$$

which involves the configurations of pairs of sites through $q_{k|y}$ terms.

We now derive the rates of three events affecting pairs. Following the notations given by van Baalen and Rand (1998), we call α_{ij} the average rate at which type *i* enters a pair *0j* when $j \neq i$, β_i the average rate at which type *i* enters a pair *0i*, and δ_{ij} the average rate of loss of type *i* from *ij* pairs. We detail only the derivation of the third rate. In this case, a pair *ij* with site *i* located at *z* can change to a *0j* pair by a dispersal event or by a death event with the per capita rate

$$\delta_{ij}(z) = d + m_i \phi n_{0|ij}(z) . \tag{B3a}$$

Averaging over the lattice leads to the average rate

$$\bar{\delta}_{ij} = \sum_{ij} \delta_{ij}(z) = (d + m_i \phi n_{0|ij}) N_{ij} , \tag{B3b}$$

and assuming that $n_{k|ij} = (n-1) q_{k|i}$, gives

$$\bar{\delta}_{ij} = (d + m_i (1-\phi) q_{0|i}) N_{ij} = \delta_i N_{ij} , \tag{B3c}$$

The second rate involves either a birth inside the pair, either a birth or a dispersal from a type *i* neighbor connected to the empty site of the pair, and is given by

$$\begin{aligned} \bar{\beta}_i = \beta_i N_{0i} &= (b + \sum_j u_j (1-\phi) q_{j|i} - C(u_i, m_i)) \phi N_{0i} \\ &(b + \sum_j u_j (1-\phi) q_{j|i} - C(u_i, m_i)) + m_i)(1-\phi) q_{i|0} N_{0i} \end{aligned} . \tag{B4}$$

The third rate involves the birth or dispersal of a type *i* neighbor connected to the empty site of the pair, and is given by

$$\bar{\alpha}_{ij} = \alpha_i N_{ij} = (b + \sum_j u_j (1-\phi) q_{j|i} - C(u_i, m_i)) + m_i)(1-\phi) q_{i|0} N_{ij} = \alpha'_i q_{i|0} N_{ij} . \tag{B5}$$

A closed dynamical system describing the pair dynamics is obtained by bookkeeping the three types of events using equations (B3c), (B4) and (B5)

$$\begin{aligned} \frac{dN_{0y}}{dt} &= (\alpha'_y q_{0|0} - \beta_y - \delta_y) N_{0y} + \delta_x N_{xy} + \delta_y N_{yy} \\ \frac{dN_{xy}}{dt} &= (\alpha_x + \alpha'_y q_{0|0}) N_{0y} - (\delta_x + \delta_y) N_{xy} \\ \frac{dN_{yy}}{dt} &= 2\beta_y N_{0y} - 2\delta_y N_{yy} \end{aligned} . \tag{B6}$$

APPENDIX C

In the case of a resident population, equilibria can be found using monomorphic versions of (B2c) and (B6). In general, the resident population converges to one stable equilibrium spatial structure ($\bar{q}_{x|x}$, $\bar{q}_{0|0}$). After (B2c), the non-trivial population equilibrium $\bar{q}_{x|x}$ satisfies a quadratic equation $((b+u_x(1-\phi)\bar{q}_{x|x}-C(u_x,m_x))(1-\bar{q}_{x|x})-d=0$, and after (B6), $\bar{q}_{0|0}=\alpha'_x/\delta_x$. If b is sufficiently larger than d, the resident population is non-viable when $\Delta<0$, where Δ denotes the discriminant of the quadratic equation.

APPENDIX D

Let $\tilde{q}_{0|y}$, $\tilde{q}_{x|y}$ and $\tilde{q}_{y|y}$ denote the pseudo-equilibrium correlation structure of the mutant population during invasion. These terms are the steady states of (B6) when x is a resident type at ecological equilibrium and y is a rare mutant type, which gives

$$\begin{aligned}&(\bar{\alpha}_x+\tilde{\alpha}'_y\bar{q}_{0|0})\tilde{q}_{0|y}-(\tilde{\delta}_y+\bar{\delta}_x+\tilde{\lambda}_y)\tilde{q}_{x|y}=0\\&2\tilde{\beta}_y\tilde{q}_{0|y}-(2\tilde{\delta}_y+\tilde{\lambda}_y)\tilde{q}_{y|y}=0\end{aligned}\quad . \tag{D1}$$

Noting that $\tilde{q}_{y|0}=0$ when the mutant is rare, the non-linear system involves three unknowns ($\tilde{q}_{0|y}$, $\tilde{q}_{x|y}$ and $\tilde{q}_{y|y}$) and two equations, along with the constraint $\tilde{q}_{0|y}=1-\tilde{q}_{x|y}-\tilde{q}_{y|y}$. The non-linear system (D1) can be used to evaluate numerically the spatial invasion fitness defined by equation (5) in the main text.

A first-order Taylor expansion of the first coordinate of the spatial invasion fitness reads

$$s_x(x+\varepsilon)=s_x(x)+\varepsilon\left.\frac{\partial s_x(y)}{\partial y}\right|_{y=x}+o(\varepsilon). \tag{D2a}$$

For the degenerate mutant, $y = x$, we can solve analytically the non-linear system (B7). This yields the solutions $\tilde{q}_{0|y}=\bar{q}_{0|x}$, $\tilde{q}_{y|y}=\bar{q}_{y|y}$ after equation (8) in the text, and $s_x(y)=s_x(x)=0$. For a slightly deviant mutant phenotype $u_y=u_x+\varepsilon$, then $\tilde{q}_{0|y}=\bar{q}_{0|x}+a\varepsilon$ and $\tilde{q}_{y|y}=\bar{q}_{y|y}+b\varepsilon$ where a measures the marginal variation of empty space in a mutant's neighborhood relative to a resident and b the marginal gain or loss of relatives. The first coordinate of the spatial invasion fitness then reads

$$s_x(x+\varepsilon)=\varepsilon\bar{q}_{0|x}\left((1-\phi)\bar{q}_{y|y}-a\left((1-\phi)u_x-\frac{d}{\bar{q}_{0|x}^{2}}\right)-\frac{C(u_y,m_x)-C(u_x,m_x)}{\varepsilon}\right). \tag{D2b}$$

We solved the system (B7) with the help of the computer package Mathematica (Wolfram 1991) to obtain symbolic expressions for *a*. Our numerical simulations showed that *a* terms are negligible, which leads to the equation (6) in the main text (see also Le Galliard et al. 2003).

We can also consider a slightly perturbed mobility phenotype, i.e., $m_y = m_x + \varepsilon$, $\tilde{q}_{0|y} = \overline{q}_{0|x} + a\varepsilon$ and $\tilde{q}_{y|y} = \overline{q}_{y|y} + b\varepsilon$. A first-order Taylor expansion of the second coordinate of the spatial invasion fitness then reads

$$s_x(x+\varepsilon) = \varepsilon \overline{q}_{0|x} \left(\left(\frac{d}{\overline{q}_{0|x}^{\,2}} - (1-\phi)u_x \right) a - \frac{C(u_x, m_y) - C(u_x, m_x)}{\varepsilon} \right). \qquad \text{(D3)}$$

Symbolic evaluation of the expression *a* in (D3) yielded a complicated formula indicating that *a* was affected directly by mobility, altruism rate, death rate, cost of mobility, and habitat connectivity, and also indirectly by the effects of all model parameters on the resident spatial statistics $\overline{q}_{x|x}$ and $\overline{q}_{0|0}$. Therefore, we conducted numerical sensitivity analyses of the selection components over a large range of model input values to understood the details of the selective interactions. The general pattern found was that the *a* term was primarily sensitive to changes in mobility rates (with a negative feedback of *m* on this selection component), while the multiplicative bracketed term was primarily sensitive to changes in altruism rate, life history traits, and habitat structure through both direct and indirect effects mediated by habitat saturation $\overline{q}_{x|x}$ (see text and fig. 1 for detailed explanations of the direction of these effects).

APPENDIX E

Table E1. Parameter definitions.

Parameter	Definition
N	Total size of the network
n	Neighborhood size (habitat connectivity)
$\phi = 1/n$	Probability to draw a connection at random within a given neighborhood
b	Intrinsic per capita birth rate
d	Intrinsic per capita death rate
m	Intrinsic per capita mobility rate (*adaptive trait*)
u	Intrinsic per capita rate of investment in altruism, or altruism rate (*adaptive trait*)
$C(u,m)$	Cost of altruism and mobility impacting the birth rate
κ	Cost sensitivity to the altruism rate
γ	Cost acceleration to the altruism rate
ν	Cost sensitivity to the mobility rate
$n_{k\|i}(z)$	Number of sites *k* neighboring of a site *i* at location *z* (*random variable*)
$n_{k\|ij}(z)$	Number of sites *k* neighboring a site *i* located at *z* within a pair *ij* (*random variable*)
N_i	Number of sites in state *i*
N_{ij}	Number of pairs in state *ij*
$q_{i\|j}$	Local frequency of sites *i* neighboring a *j* site
$q_{i\|jk}$	Local frequency of state *i* sites neighboring a site *j* within a pair in state *jk*

Note: The subscripts *i*, *j*, and *k* indicate the state of a site of the lattice. The parameters under selection (altruism and mobility) have subscript *x* if they refer to a resident population and *y* if they refer to a mutant population.

CHAPITRE 4 – RELATIONS MERE A ENFANTS ET DISPERSION NATALE

"Overall, the available evidence in favour of kin competition influencing dispersal is little more than anecdotal, and morevover is restricted to vertebrates. As more data on the genetic structure of populations become available, a clearer picture may emerge. Despite the paucity of empirical evidence for kin competition, individuals in most species must experience some level of competition with their immediate relatives. However, the contribution of kin and global competition is undetermined in most cases." X. Lambin, J. Aars & S. Piertney dans *Dispersal*. 2001. p. 119.

LES INTERACTIONS MERE A ENFANTS AFFECTENT LA DISPERSION NATALE CHEZ UN LEZARD

Jean-François Le Galliard, Régis Ferrière & Jean Clobert

RESUME

Les interactions entre apparentés génèrent des pressions de sélection fortes sur la dispersion. Récemment, une étude corrélative sur le lézard vivipare (*Lacerta vivipara*) a suggéré que la dispersion natale pouvait répondre de façon plastique aux interactions entre une mère et ses enfants. Ici, nous décrivons une expérience factorielle supportant cette observation. Deux traitements croisés ont été appliqués à des populations expérimentales du lézard vivipare : (i) présence versus absence de la mère, induisant une différence d'apparentement dans le voisinage social des enfants, et (ii) densité haute ou faible, résultant dans deux niveaux d'abondance en congénères et modulant l'effet de la présence de la mère au niveau de l'apparentement moyen de la population. La dispersion d'une même cohorte d'individus a été observée aux stades juvénile et sub-adulte. Nous avons trouvé une réponse de la dispersion natale au retrait de la mère dépendante du sexe pendant les deux stades. Pendant le stade juvénile, les filles ont dispersé plus en présence de leur mère, les fils n'étant pas affectés. Pendant le stade sub-adulte, les réponses des mâles et des femelles à la présence de la mère ont été opposées, les filles tendant à disperser plus en présence de la mère alors que les fils dispersaient moins. En plus, nous avons trouvé une relation négative entre la dispersion et la densité de la population au stade juvénile. Aucune interaction entre la densité et la présence de la mère n'a été détectée, suggérant que les réponses comportementales à l'apparentement et à la densité sont déconnectées, et que l'apparentement est évalué à une petite échelle sociale. Nous discutons le rôle de la compétition et de l'évitement de la consanguinité pour expliquer nos résultats.

Référence : Le Galliard, J.-F., Ferrière, R. et Clobert, J. 2003. « Mother-offspring interactions affect natal dispersal in a lizard ». *Proceedings of the Royal Society London B*. 270 : 1163-1169.

Mots-clés : dispersion natale, apparentement, densité, lézard vivipare

MOTHER-OFFSPRING INTERACTIONS AFFECT NATAL DISPERSAL IN A LIZARD

Jean-François Le Galliard, Régis Ferrière & Jean Clobert

PAPER AND PUBLIC ABSTRACT

Interactions between relatives operate strong selective pressures on dispersal. Recently, a correlative study in the common lizard (*Lacerta vivipara*) suggested that natal dispersal might respond plastically to mother-offspring interactions. Here, we describe a factorial experiment supporting this observation. Two crossed treatments were applied to experimental patches of the common lizard: (i) presence versus absence of the mother, inducing a difference of kinship in offspring neighbourhoods; and (ii) high versus low patch density, resulting in two levels of conspecifics abundance and modulating the effect of mother presence on the average kinship within a patch. Dispersal of the same cohort of offspring was observed at the juvenile and yearling stages. We found a sex-dependent response of offspring dispersal to the removal of the mother at the two stages. During the juvenile stage, higher dispersal was found in females in the presence of the mother, with males unaffected. During the yearling stage, the responses of both sexes to the presence of the mother opposed each other. In addition, we found a negative relationship between dispersal and patch density at the juvenile stage. No interaction between density and the presence of the mother was detected, which suggests that behavioural responses to kinship and density are disconnected and that kinship is assessed at a small social scale. We discuss the role of competition and inbreeding avoidance to explain the observed pattern.

Social interactions often involve genealogically related individuals. In most terrestrial vertebrates, the mother is present in the neighbourhood of offspring, which generates conflict and common interest between mother and offspring over the use of local resources. Here, we use an experiment to show that female offspring of a lizard disperse to avoid interactions with their mother. Male offspring were affected in the opposite way, suggesting that they behave to avoid inbreeding with their sisters. This experiment shows that social interactions within families can affect dispersal.

Reference : Le Galliard, J.-F., Ferrière, R. and Clobert, J. 2003. « Mother-offspring interactions affect natal dispersal in a lizard ». *Proceedings of the Royal Society London B*. 270: 1163-1169.

Key-words : natal dispersal, kinship, density, common lizard

INTRODUCTION

The habitat of many species tends to be fragmented (Hanski 1999). In response to habitat fragmentation, populations may develop local adaptations to local conditions, or evolve dispersal adaptations (Thomas *et al.* 1998, Ronce *et al.* 2001). Because offspring dispersal has major consequences on the demography and genetic structure of populations, understanding the selective forces driving the evolution of dispersal strategies has become an important issue at the interface of evolutionary theory, behavioural ecology and population demography.

Hamilton and May (1977) established that interactions between relatives drive the evolution of offspring dispersal in stable and homogeneous habitats. They demonstrated that dispersal can be modelled as a parental manipulation or an offspring strategy evolving under kin selection. Dispersal reduces competition between relatives, which generates some indirect genetic benefits trading against the direct costs of movement following on an Hamilton's rule (Hamilton & May 1977). Despite several developments of the original scenario to more complex spatial and demographic structures (Clobert *et al.* 2001, chapters 5, 9, 11 and 24), almost all these elaborations consider dispersal as a fixed strategy unconditional on local kinship (but see Crespi & Taylor 1990, Ronce *et al.* 1998). However, some empirical observations suggest that natal dispersal may actually depend on local relatedness. In some mammals, offspring dispersal correlates with the intensity of sib-sib competition in meadow voles (Bollinger *et al.* 1993), and dispersal of the heaviest female in a litter is a response to stronger sister-sister interactions in red-backed voles (Kawata 1987). In the common lizard, offspring dispersal decreases with lower maternal condition or during mother senescence, hence with a diminishing expected risk of competitive interactions with the mother (Massot & Clobert 1995, Léna *et al.* 1998, Ronce *et al.* 1998).

A further issue is the spatial scale at which the behavioural sensitivity to kinship is expressed, which may expand from the scale of a familial unit (Hamilton & May 1977, Ronce *et al.* 1998) to the scale of a whole patch (Crespi & Taylor 1990, Perrin & Mazalov 2000). If dispersal is a response to the expected relatedness of a patch of habitat, then the potential impact of a kin member may be diluted by the presence of non-relatives. A complete assessment of the effect of a specific relative should therefore require to control the abundance of unrelated individuals. We performed such an experiment by constructing replicated populations of the common lizard (*Lacerta vivipara*). We studied the effect of the presence of the mother on offspring dispersal during two successive life-history stages (juvenile and yearling). Local kinship was manipulated by swapping mothers between different populations, while other populations acted as controls (mothers released with their offspring). Local density was manipulated independently by doubling the number of unrelated individuals released into half of the populations. This treatment produced two contrasting levels of patch density and also two levels of patch relatedness when the mother was present, high relatedness at low density and low relatedness at high density. The two treatments were crossed so that we could investigate (i) whether juvenile dispersal responded to the presence of the mother, (ii) whether juvenile dispersal depended upon local crowding, and (iii) whether the effect of maternal presence was mediated by the level of local crowding.

METHODS

Model organism

The common lizard, *Lacerta vivipara* (Jacquin, 1787), is a viviparous species inhabiting humid habitats across Eurasia. Populations can be structured into three distinct life-history stages: juveniles (year born), yearlings (one-year old) and mature adults. Individuals share overlapping home ranges, as evidenced by the absence of any obvious spatial segregation in natural populations (Clobert *et al.* 1994). Juveniles originate from annual clutches of offspring laid synchronously during June or July. Hatching begins quickly after parturition, juveniles are autonomous at birth and offspring dispersal starts within 10 days of age (Clobert *et al.* 1994). Most dispersal occurs during the two first life-history stages, both in natural and experimental populations (Clobert *et al.* 1994, Boudjemadi *et al.* 1999). Moreover, laboratory trials have demonstrated that offspring discriminate maternal olfactory cues at birth (Léna *et al.* 2000).

Running of the experiment

The experiment was conducted on a sample of individually marked lizards collected from their natural habitats in June 1999 and released at our experimental site in July. Offspring dispersal away from the experimental populations was monitored daily until the end of December 2000. This procedure allowed us to estimate dispersal at the juvenile stage during 1999 and at the yearling stage during 2000.

(i) Collection. During June 1999, lizards were sampled in a natural habitat on the Mont Lozère (France, Lozère, 44°27'N, 3°44'E) before translocation at the Field Station of Foljuif (Seine-et-Marne, 48°17'N, 2°41'E). Altogether we collected 144 gravid females, 96 adult males and 240 yearlings to establish background populations. Lizards were individually marked, measured for length and weight, and maintained in plastic terraria at the Field Station with food and water provisioned regularly until the laying of gravid females (mid-July). Offspring were individually marked by toe clipping and measured for length and weight. Gender was determined by counting ventral scales (Lecomte *et al.* 1992). We assumed that yearlings and adults were initially unrelated to each other.

(ii) Experimental system. Sixteen experimental enclosures were constructed, each with a squared patch (10 × 10 m) and a one-way, 20 m long corridor (Fig. 1). Dispersal was defined as the movement out from an initial patch along a one-way corridor. This structure corresponds to both the

size of natural home range and the minimum dispersal distance measured in natural populations (Boudjemadi *et al.* 1999). Enclosures were closed to avian predators by nets and to intrusive mammals by daily trapping. However, we were unable to preclude intrusions of greater white-toothed shrews (*Crocidura russula*) in five of our enclosures in 1999 (Fig. 1). In 2000, efficient traps (Ugglan, Grahnab™, Sweden) were used inside and on the outskirts of the enclosures, so that predation was prevented in all enclosures.

(iii) Experimental design. In July 1999, we initiated our experimental system with a bifactorial design. We manipulated the initial density and crossed this factor with the mother presence-absence (kinship) using enclosure as a replicate (Fig. 1). We contrasted eight low-density patches (14 yearlings and adult males, 6 females, 36.5 offspring ± 2.4 s.e.) with eight high-density patches (28 yearlings and adults, 12 females, 71.2 offspring ± 3.9 s.e.). We maintained a similar population age- and sex-structure in all patches. Starting densities were chosen to frame the estimated carrying capacity of our experimental habitat (Lecomte pers. com.). We applied the kinship treatment by replacing the mother of each litter with an unrelated, unfamiliar adult female in half of the experimental populations. This swapping was conducted just prior to introduction to avoid familiarisation. In the remaining half of the populations, offspring were introduced with their mother. The kinship effect was crossed with the density manipulation such that four population replicates were initiated for each combination of the two factors (Fig. 1).

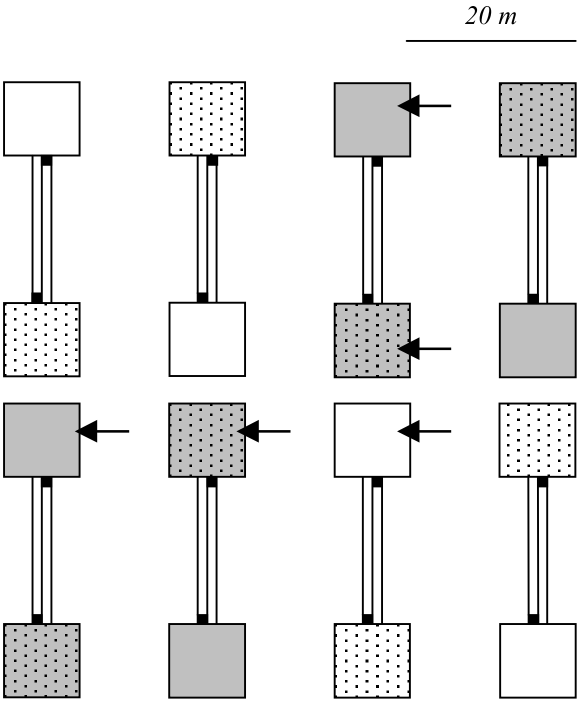

Figure 1. Experimental design. Grey indicates enclosures where the mother of each litter was replaced with an unfamiliar adult female. Otherwise, all offspring were introduced with their mother. Dotting indicates populations that were initiated at high density. Arrows indicate enclosures affected by predation in 1999, and excluded from the statistical analyses. Pitfalls traps at the end of each one-way corridor are represented in black.

(iv) Introduction and monitoring. Individuals were randomly allocated to the experimental populations. Yearlings and adult males were released during the same day in July 1999, and siblings

were released with their mother or a surrogate female following birth. The randomisation procedure used to introduce the lizards was effective at producing an initially homogeneous set (homogeneity tests for population size and individual characteristics among treatments, all $p>0.4$). Philopatric individuals were monitored by hand recaptures during sessions in August and September 1999, and in April, August and September 2000, with multiple attempts per session (usually three independent days). This robust design allowed very efficient capture (estimated capture probability ranging from 0.80 to 0.98 per session). Dispersers were caught systematically in a trap located at the corridor extremities (checked daily), identified and immediately released in a new enclosure adjacent to the trap (Fig. 1). This methodology generated a simple metapopulation of two patches mutually coupled by migration.

Data analysis

Dispersers were defined as individuals caught at least once during a year within a corridor pitfall trap. Philopatric individuals were considered as the remaining set of individuals, excluding the non captured individuals, which were either dead or philopatric (Boudjemadi *et al.* 1999). Two separate analysis were conducted at the two life-history stages using the same cohort of offspring. Indeed, the sample of individuals used to model dispersal was not the same due to mortality from the juvenile to the yearling stage.

Data were analysed using generalised linear mixed models (GLMM) in SASv8.02 (Littell *et al.* 1996). The timing of dispersal was modelled with the MIXED procedure, which amounts to specify a gaussian error distribution and an identity link function in the GLMM framework. The average per family was used as a response variable. Dispersal status was modelled with the GLIMMIX macro, using a binomial error term, a logit link function and individual dispersal status (philopatry or dispersal) as a response variable (Littell *et al.* 1996). The GLMM approach described the clustering of individual observations, owing to the fact that populations were nested within treatments (Fig. 1), and accounted for the presence of both fixed treatment effects and random replicate effects (Littell *et al.* 1996). Estimations and test statistics were calculated with a restricted maximum likelihood approach. Statistical inferences for the fixed part of the model were obtained from type III F statistics and two-tailed tests. The assumptions of those models were investigated by the analysis of residuals. In the case of binomial dispersal data, no significant overdispersion was detected (Chi-square tests, $p > 0.05$).

Body condition was calculated as the residual from a linear regression of body mass against body size. Body condition and body size were not independent of offspring gender (body condition: $F_{1,700} = 141.9$, $p < 0.001$; body size: $F_{1,700} = 16.1$, $p < 0.001$). Males were on average more corpulent at birth than females, while females were larger than males. Therefore, we accounted for both covariates in the analysis of offspring dispersal. The fixed part of the models included the two experimental factors, individual covariates (length, body condition, gender) and interactions. The random part of the model included the effects of populations nested within the treatments. Model selection was conducted by backward simplification of the fixed effects. Populations affected by predation were excluded from all analyses, although this did not modify the nature and significance of kinship effects.

Table 1. Selected generalised linear mixed models describing offspring dispersal at the juvenile stage (476 observations) and at the yearling stage (*b*, 214 observations) depending on offspring body condition, gender, kinship treatment and density manipulation. The model also included the random effect of patch identity nested within combinations of kinship and density treatments.

Juvenile dispersal predictors	**F statistic** $_{\text{ndf, ddf}}$	**P value**
Body condition at birth	$5.32_{1,462}$	0.02
Gender	$5.79_{1,462}$	0.03
Kinship	$2.10_{1,7}$	0.19
Gender × Kinship	$3.66_{1,462}$	0.06
Density	$6.09_{1,7}$	0.04
Density × Kinship	$0.08_{1,7}$	0.78
Yearling dispersal predictors	**F statistic** $_{\text{ndf, ddf}}$	**P value**
Body condition in spring	$0.27_{1,199}$	0.60
Gender	$2.68_{1,199}$	0.10
Kinship	$0.05_{1,7}$	0.83
Gender × Kinship	$5.80_{1,199}$	0.02
Body condition × Kinship	$9.19_{1,199}$	0.003
Density	$0.93_{1,7}$	0.37
Density × Kinship	$0.01_{1,7}$	0.93

RESULTS

Effects of treatments on dispersal chronology

There was no evidence that dispersal chronology was affected by our manipulation during the two first stages of offspring lifetime. Juveniles dispersal was bimodal with a first, early dispersal period (age 2 to 25 days, $n = 23$) and a second more important dispersal period at an older age (30 to 60 days, $n = 55$). This bimodality was not affected by experimental treatments (logistic regression, density: $\chi_1^2 = 0.001$, $p = 0.97$; kinship: $\chi_1^2 = 0.04$, $p = 0.85$, $n = 78$). Similarly, age at dispersal did not differ between treatments (density: $F_{1,7} = 0.13$, $p = 0.73$; kinship: $F_{1,7} = 0.68$, $p = 0.44$, $n = 78$). Most yearlings movements occurred before June-July. The chronology of these movements was not influenced by the experimental treatments (density: $F_{1,7} = 0.01$, $p = 0.92$; kinship: $F_{1,7} = 2.57$, $p = 0.15$, $n = 42$).

Effects of treatments on dispersal status

In 1999, juvenile dispersal was affected by body condition, gender, a marginal interaction between gender and kinship, and density (Table 1). First, dispersal was associated with higher corpulence at birth than philopatry (dispersal: 0.005 ± 0.002, $n = 65$; philopatry: –0.008 ± 0.009, $n = 411$), and males disperse more on average than females (odds male : odds female = 1.22). Second, females dispersed more in the presence of their mother than in the presence of a surrogate adult female (female sample, kinship: $F_{1,7} = 6.53$, $p = 0.04$, $n = 236$), whereas male offspring displayed no significant response to the presence of the mother (male sample, kinship: $F_{1,7} = 0.01$, $p = 0.94$, $n = 240$). Together, these two different responses generated the marginal interaction between gender and kinship detected in the selected model (Fig. 2a). Third, the effect of density on patch dispersal was significant. Contrary to expectations, a lower dispersal was observed in the high-density treatment (Fig. 2b). There was no indication that density interacted with kinship (Table 1) nor with the sex-dependent response (gender × kinship × density: $F_{1,460} = 1.17$, $p = 0.28$).

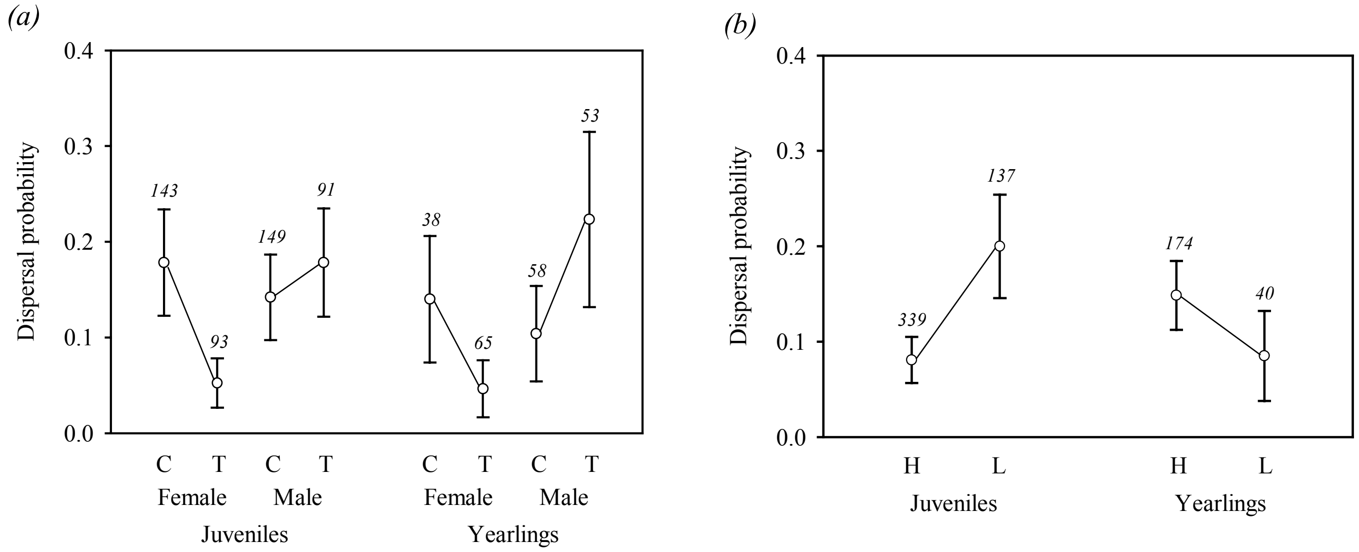

Figure 2. a. Kinship treatment, offspring gender and dispersal. During the juvenile stage (1999) and the yearling stage (2000), the presence of the mother had a significant effect on the sex-biased dispersal. C: Control, offspring introduced with their mother. T: Mother removal treatment, offspring introduced with a surrogate mother. b. Patch density and offspring dispersal. Dispersal decreased at high density during the juvenile stage, but was unaffected during the yearling stage. H: High-density patches. L: Low-density patches. Values are back-transformed from the GLMM presented in Table 1 (least-square means ± s.e.). Numbers indicate sample sizes.

In the following, the study of yearling dispersal showed persistent effects of the kinship treatment modulated by gender and by spring body condition, whereas the effect of density disappeared (Table 1). The interaction between kinship and gender originated from the fact that females displayed a response to the presence of the mother opposite to that of male offspring (Fig. 2a), albeit both responses were not significant (males, $F_{1,7} = 0.88$, $p = 0.38$, $n = 111$; females, $F_{1,7} = 1.09$, $p = 0.33$, $n = 103$). Males tended to disperse more in the absence of the mother, while females tended to disperse more in the presence of the mother. Also, irrespective of gender, the body condition of dispersers was higher than the residents' in the presence of the mother, whereas the reverse was observed when an unrelated female replaced the true mother (Table 2). Finally, the effect of density was not significant (Fig. 2b), and did not interact with kinship (Table 1) nor with the sex-dependent response (gender × kinship × density: $F_{1,197} = 1.72$, $p = 0.19$).

Table 2. Body condition according to the dispersal status and the presence of the mother at the yearling stage (offspring dispersing at the juvenile stage were excluded from the analysis). The first line indicates average value across gender, and data are also illustrated separately for females (F) and males (M) in the next lines. Dispersers were more corpulent than philopatric individuals when the mother was present ($F_{1,7} = 4.94$, $p = 0.03$), but less corpulent when the mother was replaced by a surrogate female ($F_{1,7} = 4.63$, $p = 0.03$). Bracketed numbers indicate sample sizes. C: Control, offspring introduced with the mother. T: Mother removal treatment, offspring introduced with a surrogate mother.

	C	T
Philopatry	- 0.002 ± 0.018 ($n = 82$)	- 0.009 ± 0.012 ($n = 99$)
	F: - 0.036 ± 0.018	F: - 0.056 ± 0.015
	M: 0.020 ± 0.017	M: 0.059 ± 0.017
Dispersal	0.125 ± 0.062 ($n = 14$)	- 0.033 ± 0.042 ($n = 19$)
	F: 0.053 ± 0.09	F: - 0.174 ± 0.08
	M: 0.177 ± 0.08	M: 0.032 ± 0.04

DISCUSSION

Our experiment demonstrates that maternal presence has a significant effect on sex-biased dispersal from natal patch. During the juvenile stage, higher dispersal was found in female offspring in the presence of the mother, while males were unaffected. This result lends experimental explanation to some correlations observed between the intensity of mother-offspring interactions and natal dispersal under natural conditions in the same species. For example, Ronce *et al.* (1998) observed that old females had lower annual survival than young females, which should decrease the likelihood of future mother-offspring interactions for offspring born from older females. This maternal ageing was associated with a stronger female offspring philopatry, while male dispersal was not affected (Ronce *et al.* 1998). Other maternal effects, possibly reflecting potential mother-offspring interactions, have been shown to influence offspring dispersal in the same species, including food availability (Massot & Clobert 1995), parasitism (Sorci *et al.* 1994) and hormonal stress (de Fraipont *et al.* 2000, Meylan *et al.* 2002). Such correlations could result from proximal constraints of producing different types of offspring, independently from the ultimate cause involving mother-offspring interactions that we manipulated here. Nevertheless, our experimental suppression of all mother-offspring interactions yields exactly the behavioural response observed in Ronce *et al.*'s (1998) correlative study. These concordant results go to prove that female offspring disperse to avoid competitive interactions with the mother in this species. Whether the behaviour documented here results from offspring control on dispersal, or from parental manipulation of offspring behaviour (Hamilton & May 1977, Ronce *et al.* 1998) is difficult to assess. Against the latter hypothesis, we know at least that adults female do not seem to demonstrate any particular behaviour forcing offspring to leave their natal environment (Clobert *et al.* 1994, Léna *et al.* 1998).

The fact that male behaviour was unaffected at the juvenile stage and opposed the female response at the yearling stage requires alternative explanations. Despite offspring have been shown experimentally to suffer from competition with adults (Massot *et al.* 1992), competitive interactions may differ between sexes. For example, females tend to compete for resources, while males tend to compete for mates (Pilorge 1987, Massot *et al.* 1992). Thus, young males might suffer less from competition with the mother than young females. Additionally, males may avoid potential mating with their sisters rather than their mother, and therefore adopt a dispersal strategy opposite to that of their sisters (Massot & Clobert 2000). Indeed, the likelihood that one brother and one sister of an average clutch (3 males and 3 females) both survive to sexual maturity is high (0.11-0.30, with survival data from Massot *et al.* 1992), and exceeds the risk of inbreeding with mother (0.07-0.20, with survival data from Ronce *et al.* 1998). This scenario (female dispersal to avoid competition with mother, and male philopatry to avoid inbreeding with sisters) would match a model of sex-biased dispersal evolving under the joint influences of kin competition and inbreeding avoidance by Perrin and Mazalov (2000).

Dispersers' phenotypes and behaviour are not random. This is particularly evident in some species with dispersing morphotypes, but less extreme differences are also found in many other species (Swingland 1983). Our experiment showed that when dispersal occurred in response to presence of the mother, dispersers were more corpulent than philopatric individuals, which confirms previous observations (Clobert *et al.* 1994, Léna *et al.* 1998). On the contrary, when offspring were released with a surrogate mother, yearling dispersers were leaner than yearling residents. This result supports the view that individuals can differ morphologically depending on their dispersal strategy, but indicates that the actual dispersal decision is made by the offspring in response to proximal cues of potential kin competition.

In species exhibiting intense intraspecific competition, natal dispersal is expected to be positively density-dependent (e.g., Aars *et al.* 2000). In contrast, we found that offspring dispersal was inversely related to local crowding during the juvenile stage. Such a response may indicate constraints of habitat saturation acting on dispersal (see review in Lambin *et al.*, 2001). This would imply that the cost of movement and settlement are higher at high density in natural populations and limit emigration (e.g., Jones *et al.* 1988). A density manipulation in the field has indeed shown that social fences can prevent immigration (Massot *et al.* 1992). Also, it is possible that some individuals choose their habitat based on the presence of conspecifics (conspecific attraction, Stamps 1991). This hypothesis would apply if the fitness of individuals increases with density (e.g., Allee effect) or if settlement costs are reduced in the presence of conspecifics (Greene & Stamps 2001). Finally, our transfer to an unfamiliar environment might have increased the benefits of the public information offered by neighbouring conspecifics as opposed to the individual private information (Valone 1989, Danchin *et al.* 2001). For example, offspring may cue on conspecifics to learn the location of suitable habitats (Stamps 1991). As private and public information built up in the population, one would expect the response to density to vary over time, as was observed here (see also, Clobert et al. *in press*). Whether negative density-dependence in our experiment reflects information sharing, social attraction, or habitat saturation remains to be established.

Local crowding did not influence the response of female and male offspring to the presence of the mother. Therefore, offspring did not react to relatedness at the level of the whole patch but at a smaller scale, such as that of a family unit. This behaviour suggests that assuming a few relatives per patch (Hamilton & May 1977, Ronce *et al.* 1998) is not an unrealistic modelling hypothesis to describe natal dispersal in our species. Alternatively, other lizard species might assess relatedness at different social scales and using different proximate cues. For example, recent evidences gathered in the side-blotched lizard (*Uta stansburiana*) indicate that dispersal promotes the local aggregation of genetically similar individuals irrespective of genealogy (Sinervo *et al.* 2001, Sinervo pers. com.). Thus, identity by state rather than by descent may also influence dispersal behaviour. More generally, the additivity between the effects of crowding and kinship suggests that the evolution of dispersal responses to both factors might have taken place along two independent pathways. This calls for a more detailed investigation of the distinct physiological and behavioural mechanisms involved in both responses (Dufty *et al.* 2002).

CONCLUSION

Evolutionary theory has long shown that kin interactions can be important in the evolution of dispersal. Following the parallel made by Hamilton and May (1977) between dispersal and altruism, theoretical studies of the evolution of kin recognition in cooperative species (Agrawal 2001) can be used to predict that kinship-dependent dispersal is likely to evolve. Our experimental results provide evidence for a more complicated scenario of a sex-dependent relationship between mother presence and natal dispersal. The direction of the relationship (here positive in females, negative in males) is likely to depend upon the relative influences of kin competition, inbreeding and kin cooperation (Perrin & Mazalov 2000, Lambin *et al.* 2001). Indeed, whereas the first two effects seem to prevail in our species, studies with some mammals and birds indicate that different responses may actually exist when costs of inbreeding or benefits of kin cooperation dominate (Cockburn *et al.* 1985, Wolff 1992, Lambin *et al.* 2001).

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REFERENCES

Aars, J., Johannesen, E. & Ims, R. A. 2000 Population dynamic and genetic consequences of spatial density-dependent dispersal in patchy populations. *Am. Nat.* **155**, 252-265.

Agrawal, A. F. 2001 Kin recognition and the evolution of altruism. *Proc. R. Soc. London Ser. B* **268**, 1099-1104.

Bollinger, E. K., Harper S. J. & Barrett, G. W. 1993 Inbreeding avoidance increases dispersal movements of the meadow vole. *Ecology* **4**, 1153-1156.

Boudjemadi, K., Lecomte, J. & Clobert, J. 1999 Influence of connectivity on demography and dispersal in two contrasting habitats: an experimental approach. *J. Anim. Ecol.* **68**, 1207-1224.

Clobert, J., Massot, M., Lecomte, J., Sorci, G., de Fraipont, M. & Barbault, R. 1994 Determinants of dispersal behavior: the common lizard as a case study. In *Lizard Ecology: Historical and Experimental Perspectives* (ed. L. J. Vitt & E. R. Pianka), pp. 183-206. Princeton: Princeton Univ. Press.

Clobert, J., Danchin, E., Dhondt, A. A. & Nichols, J. D. 2001 *Dispersal.* Oxford: Oxford Univ. Press.

Clobert, J., Ims, R. & Rousset, F. *In press* Dispersal and the metapopulation concept. In *Ecology, genetics, and the evolution of metapopulation* (ed., I. Hanski & O., Gaggiotti), chapter 8. San Diego: Academic Press.

Cockburn, A., Scott, M. P. & Scotts, D. J. 1985 Inbreeding avoidance and male-biased dispersal in *Antechinus spp.* (Marsupialia: Dasyuridae). *Anim. Behav.* **33**, 908-915.

Crespi, B. J. & Taylor, P. D. 1990 Dispersal rates under variable patch density. *Am. Nat.* **135**, 48-62.

Danchin, E., Heg, D. & Doligez, B. 2001 Public information and breeding habitat selection. In *Dispersal* (ed. J. Clobert, E. Danchin, A. A. Dhondt & J. D. Nichols), pp. 243-258. Oxford: Oxford Univ. Press.

de Fraipont, M., Clobert, J., John-Alder, A. H. & Meylan, S. 2000 Increased pre-natal maternal corticosterone promotes philopatry of offspring in common lizards *Lacerta vivipara. J. Anim. Ecol.* **69**, 404-413.

Dufty, A., Clobert, J. & Møller, A. P. 2002 Hormones, developmental plasticity and adaptation. *Trends Ecol. Evol.* **17**, 190-196.

Greene, C. M. & Stamps, J. 2001. Habitat selection at low population densities. *Ecology* **82**, 2091-2100.

Hamilton, W. D. & May, R. M. 1977 Dispersal in stable habitats. *Nature* **269**, 578-581.

Hanski, I. 1999 *Metapopulation ecology.* Oxford: Oxford University Press.

Jones, W. T., Waser, P. M., Eliott, L. F. & Link, N. E. 1988 Philopatry, dispersal, and habitat saturation in the banner-tailed kangaroo rat, *Dipodomys spectabilis*. *Ecology* **69**, 1466-1473.

Kawata, M. 1987 The effect of kinship on spacing among female red-backed voles, *Clethrionomys rufocanus bedfordiae. Oecologia* **72**, 115-122.

Lambin, X., Aars, J. & Piertney, S. B. 2001 Dispersal, intraspecific competition, kin competition, and kin facilitation: a review of the empirical evidence. In *Dispersal* (ed. J. Clobert, E. Danchin, A. A. Dhondt & J. D. Nichols), pp. 110-122. Oxford: Oxford Univ. Press.

Lecomte, J., Clobert, J. & Massot, M. 1992 Sex identification in juveniles of *Lacerta vivipara. Amphibia-Reptilia* **13**, 21-25.

Léna, J.-P., Clobert, J., de Fraipont, M., Lecomte, J. & Guyot, G. 1998 The relative influence of density and kinship on dispersal in the common lizard. *Behav. Ecol.* **9**, 500-507.

Léna, J.-P., de Fraipont, M. & Clobert, J. 2000 Affinity towards maternal odour and offspring dispersal in the common lizard. *Ecol. Let.* **3**, 300-308.

Littell, R. C., Milliken, G. A., Stroup, W. W. & Wolfinger, R. D. 1996 *SAS® System for Mixed Models.* Cary: SAS Institute, Inc.

Massot, M., Clobert, J., Pilorge, T. & Barbault, R. 1992 Density dependence in the common lizard: demographic consequences of a density manipulation. *Ecology* **73**, 1742-1756.

Massot, M. & Clobert, J. 1995 Influence of maternal food availability on offspring dispersal. *Behav. Ecol. Sociobiol.* **37**, 413-418.

Massot, M. & Clobert, J. 2000 Processes at the origin of similarities in dispersal behaviour among siblings. *J. Evol. Biol.* **4**, 707-719.

Meylan, S., Belliure, J., Clobert, J. & de Fraipont, M. 2002. Stress and body condition as prenatal and postnatal determinants of dispersal in the Common Lizard (*Lacerta vivipara*). *Horm. Behav.* **42**, 319-326.

Perrin, N. & Mazalov, V. 2000 Local competition, inbreeding, and the evolution of sex-biased dispersal. *Am. Nat.* **155**, 116-127.

Pilorge, T. 1987 Density, size structure, and reproductive characteristics of three populations of *Lacerta vivipara* (Sauria: Lacertidae). *Herpetologica* **43**, 345-356.

Ronce, O., Clobert, J. & Massot, M. 1998 Natal dispersal and senescence. *Proc. Natl. Acad. Sci. USA* **95**, 600-605.

Ronce, O., Olivieri, I., Clobert, J. & Danchin, E. 2001. Perspectives for the study of dispersal evolution. In *Dispersal* (ed. J. Clobert, E. Danchin, A. A. Dhondt & J. D. Nichols), pp. 110-122. Oxford: Oxford Univ. Press.

Sinervo, B., Bleay, C. & Adamopolou, C. 2001 Social causes of correlational selection and the resolution of a heritable throat color polymorphism in a lizard. *Evolution* **55**, 2040-2052.

Sorci, G., Massot, M. & Clobert, J. 1994 Maternal parasite load increases sprint speed and philopatry in female offspring of the common lizard. *Am. Nat.* **144**, 153-164.

Stamps, J. 1991 The effect of conspecifics on habitat selection in territorial species. *Behav. Ecol. Sociobiol.* **28**, 29-36.

Thomas, C. D., Hill, J. K. & Lewis, O. T. 1998. Evolutionary consequences of habitat fragmentation in a localized butterfly. *J. Anim. Ecol.* **67**, 485-497.

Swingland, I. R. 1983 Intraspecific differences in movement. In *The Ecology of Animal Movement* (ed. I. R. Swingland & P. J. Greenwood), pp. 102-115. Oxford: Clarendon Press.

Valone, T. J. 1989 Group foraging, public information and patch estimation. *Oikos* **56**, 357-363

Wolff, J. O. 1992 Parents suppress reproduction and stimulated dispersal in opposite-sex juveniles white-footed mice. *Nature* **359**, 409-410.

ADDENDA

This part presents some unpublished results on the emigration behaviour of yearlings and adults (and not only juveniles), on the immigration behaviour (and not only emigration), on delayed density dependence on dispersal, and on the correlation between offspring affinity toward maternal olfactory cues and natal dispersal in 1999.

Dispersal of yearling and adult cohorts

There is no evidence that dispersal chronology of yearlings and adults is affected by our manipulations during the two years of the study. For example, yearling movements are regularly spread over the summer and insensitive to manipulations in 1999 (density: $F_{1,6} = 0.49$, $P = 0.51$; kinship: $F_{1,6} = 0.07$, $P = 0.81$; gender: $F_{1,14} = 4.06$, $P = 0.06$, $n = 24$). At the same time, the chronology of adult movements is not influenced by treatments (density: $F_{1,8} = 1.05$, $P = 0.34$; kinship: $F_{1,8} = 1.87$, $P = 0.21$, $n = 33$).

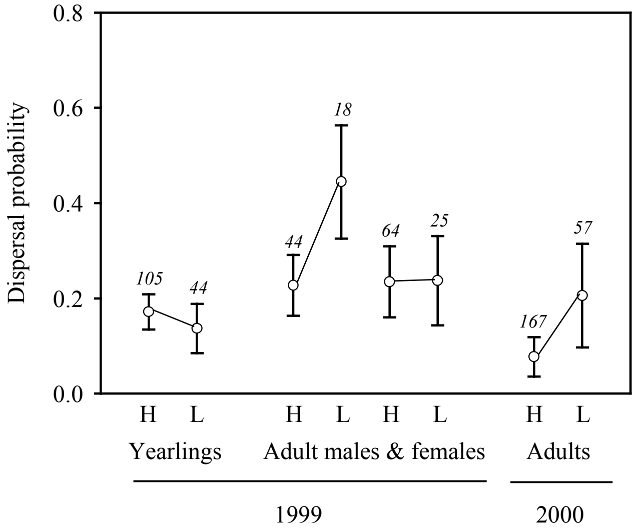


Figure A1. Population density and dispersal probability in 1999 for yearlings, adult males, and adult females, and in 2000 for adults. H: High-density patches. L: Low-density patches. Values are back-transformed from the GLMM discussed in the text (least-square means ± s.e.). Numbers indicate sample sizes.

Mother-offspring interactions have no detectable effect on dispersal probability of yearlings and adults in 1999 and in 2000 (e.g., effect of offspring removal on adult female dispersal probability in 1999: $F_{1,9} = 0.08$, $P = 0.79$, $n = 89$; Offspring absence: 0.19 ± 0.09, Offspring presence: 0.16 ± 0.06). Therefore, we only report the effects of population density on dispersal probability during the two years of the study. In 1999, we analysed yearlings and adults separately. Yearling dispersal is not influenced by conspecifics density ($F_{1,9} = 0.28$, $P = 0.61$, $n = 149$, Fig. A1). Adult male dispersal in 1999 is not affected by population density ($F_{1,9} = 2.73$, $P = 0.13$, $n = 62$, Fig. A1), but the trend is for increased philopatry at high density. Adult females dispersal in 1999 is also density-independent ($F_{1,9} = 0.97$, $P = 0.35$, $n = 89$, Fig. A1). Pooling together adult males and females yields increased power, but the results remained unchanged (Density: $F_{1,9} = 2.78$, $P = 0.13$, odds Low density : odds High density = 2.25; Gender:$F_{1,139} = 0.28$, $P = 0.61$, odds Male : odds Female = 2.05 , $n = 151$, Fig. A1). We then compared patterns of density-dependent dispersal among age and gender classes in 1999. This analysis shows that the difference between juveniles, yearlings and adults suggested by separate analyses was close to significance (Table A1).

In 2000, we analysed together yearlings and adult cohorts to investigate patterns of density-dependent dispersal on mature individuals, accounting for age and gender effects. There is no effect of density on dispersal probability (Density: $F_{1,9} = 1.68$, $P = 0.23$, odds Low density : odds High density = 3.11; Age: $F_{1,211} = 0.03$, $P = 0.86$, odds Two years old : odds Three years old = 1.07; Gender: $F_{1,211} = 14.91$, $P = 0.0002$, odds Male : odds Female = 4.77, $n = 224$). The tendency is for a higher dispersal probability at low population density (Fig. A1).

Table A1. Selected generalised linear mixed models describing dispersal probability in 1999 depending on age and gender classes, and density manipulation (*n = 776*). The model also included the random effect of patch nested within density treatments.

Lizard dispersal predictors	**F statistic $_{\text{ndf, ddf}}$**	**P value**
Density	$1.54_{1,\,9}$	0.21
Age class	$3.37_{1,\,758}$	0.03
Gender	$2.88_{1,\,758}$	0.09
Age class × Gender	$2.82_{2,\,758}$	0.06
Age class × Density	$2.48_{2,\,758}$	0.08

Immigration

The maximal number of individual moves between patches in 1999 is equal to 3, and 27 individuals did not settle in their patch of arrival. The occurrence of settlement is not affected by age (G-test, $G_2 = 0.52$, P = 0.77) which allowed us to analyse pooled data. The probability of settlement is not affected by density in the arrival patch ($\chi^2_{1\text{df}} = 0.02$, P = 0.90, *n = 164*). The trend is for a higher probability of settlement in high density patches (87%) than in low density patches (70%). Among transient individuals leaving their arrival patch, the time spent in the arrival patch is not affected by patch density ($F_{1,10} = 0.77$, P = 0.40, *n = 27*). Although less powerful, separate analyses within each age class give similar results (treatment level analysis, Student's and contingency tests, all P > 0.3). Multiple moves are more pronounced in 2000, with a maximum of 5 moves during the year by the same adult male. The probability of settlement after a dispersal event is not influenced by density (density: $\chi^2_{1\text{df}} = 1.0$, P = 0.32, *n = 102*, pooled sample). The time elapsed between the initiation of two movements is similar in arrival patches at high versus low density for all age classes (treatment level analysis, Student's tests, all P > 0.9). Therefore, we found no evidence that settlement was influenced by the density of the arrival patch.

Delayed density dependence

Individuals captured at the end of experiment were classified (i) according to the patch densities encountered during the two years of the study (cohorts introduced in 1999), leading to four classes of life histories depending on whether patch density was low or high during the first or the second year of the experiment, or (ii) according to the patch densities encountered during the last year of the study (cohort born locally in 2000). These individuals were then introduced into unfamiliar populations in 2001, which enabled us to test the effects of past history on dispersal following release in this new environment (see chapter 5 for a description of the standard population structure). The effect of maternal history on natal dispersal can also be tested because offspring born from adult females captured at the end of the experiment were randomly introduced in standard enclosures. Significant effects of the life history type would indicate delayed density dependence.

When dispersal behaviour is compared among life history types, we find no differences in adults (GLMM on dispersal probability including patch of release as a random effect, Life history type: $F_{3,92} = 1.49$, $P = 0.22$; Age class: $F_{2,92} = 1.38$, $P = 0.26$; Gender: $F_{1,92} = 3.41$, $P = 0.07$, $n = 107$, Table A2) and yearlings (Life history type: $F_{1,32} = 2.41$, $P = 0.13$; Gender: $F_{1,32} = 4.12$, $P = 0.05$, $n = 43$, Table A2). The effect of maternal history on offspring dispersal is also not significant (Life history type: $F_{3,162} = 1.25$, $P = 0.29$; Gender: $F_{1,162} = 2.22$, $P = 0.14$, $n = 175$, Table A2).

Table A2. Delayed density dependence on dispersal probability in adults and yearlings captured at the end of the experiment and in offspring born from adult females captured at the end of the experiment. Dispersal probability was measured following release in new, unfamiliar enclosures. Results are the back-transformed least-square means (± s.e.) from the GLMM described in the text. Life history is given as first year of study – second year of study.

Age class	Life history type	Dispersal probability
Adults	Low density – Low density	0.27 ± 0.11 ($n = 21$)
	Low density – High density	0.10 ± 0.07 ($n = 23$)
	High density – Low density	0.10 ± 0.08 ($n = 10$)
	High density – High density	0.07 ± 0.04 ($n = 53$)
Yearlings	Low density	0.05 ± 0.04 ($n = 18$)
	High density	0.17 ± 0.09 ($n = 25$)
Offspring	Low density – Low density	0.124 ± 0.06 ($n = 54$)
	Low density – High density	0.064 ± 0.05 ($n = 19$)
	High density – Low density	0.105 ± 0.06 ($n = 22$)
	High density – High density	0.215 ± 0.08 ($n =80$)

Maternal olfactory cue and natal dispersal

Affinity toward maternal odour was assessed in 1999 to determine whether olfactory maternal cue is a proximal factor involved in dispersal behaviour depending on mother presence (Léna and de Fraipont 1998). Less than eight hours after laying, mothers were isolated for 36 hours in dry individual terraria (17 × 11 × 12 cm high) covered with blotting paper. About 1 ½ h before the start of each experiment, each of a control blot and the maternal odour blot was randomly assigned under one of two cardboard shelters, symmetrically disposed in a clean, empty terrarium (25 × 15 × 15 cm high). After introducing around 5 a.m. one juvenile per terrarium with a maternal odour, all terraria were heated during two hours using a 25W incandescent bulb to trigger movements. Juvenile behaviour (outside a shelter, inside a control shelter, or inside a shelter with maternal odour) was recorded at 12 a.m. and 8 p.m.

Results from this laboratory experiment allows us to analyse the effects of behaviour and kinship on dispersal status. The choice of a shelter and the affinity toward a maternal odour conditional on shelter choice were analysed independently (Table A3). In the morning, there are more individuals outside the shelters ($G_1 = 37.94$, $P < 0.001$), but overall there is no significant preference toward a maternal cue (midnight: $G_1 = 0.16$, $P = 0.31$; early morning: $G_1 = 0.11$, $P = 0.26$). There is no association between affinity toward the maternal cue, kinship and dispersal status (e.g., at midnight, kinship: $\chi^2_{1df} = 0.15$, $P = 0.70$; odour choice: $\chi^2_{1df} = 0.62$, $P = 0.43$; odour choice × kinship: $\chi^2_{1df} = 1.44$, $P = 0.23$, *n = 460*). In contrast, the relationship between dispersal and shelter use is somewhat modulated by kinship (e.g., on early morning observations, kinship: $\chi^2_{1df} = 0.45$, $P = 0.50$; shelter use: $\chi^2_{1df} = 1.60$, $P = 0.20$; shelter use × kinship: $\chi^2_{1df} = 3.88$, $P = 0.05$, *n = 537*). Individuals refusing a shelter tend to disperse more than individuals using a shelter in the presence of the mother, whereas the reversed is observed in the absence of the mother (Table A3). Analysing the number of observations under a shelter during two consecutive observations (Léna et al. 2000) instead of shelter use yields a similar pattern (kinship: $\chi^2_{1df} = 3.14$, $P = 0.10$; number of visits outside a shelter: $\chi^2_{1df} = 1.57$, $P = 0.21$; number of visits × kinship: $\chi^2_{1df} = 4.74$, $P = 0.03$, *n = 537*).

Table A3. Summary statistics of the experiment characterising shelter choice, affinity toward maternal cue and dispersal behaviour in 1999 (midnight and early morning observations). Predicted dispersal probability from two GLMM (see text for details) with behaviour and kinship as explanatory variables (logit scale, ± s.e.), using all recaptured individuals from the early morning observation. Similar results were obtained with midnight observations. C, Control. T, Mother removal treatment.

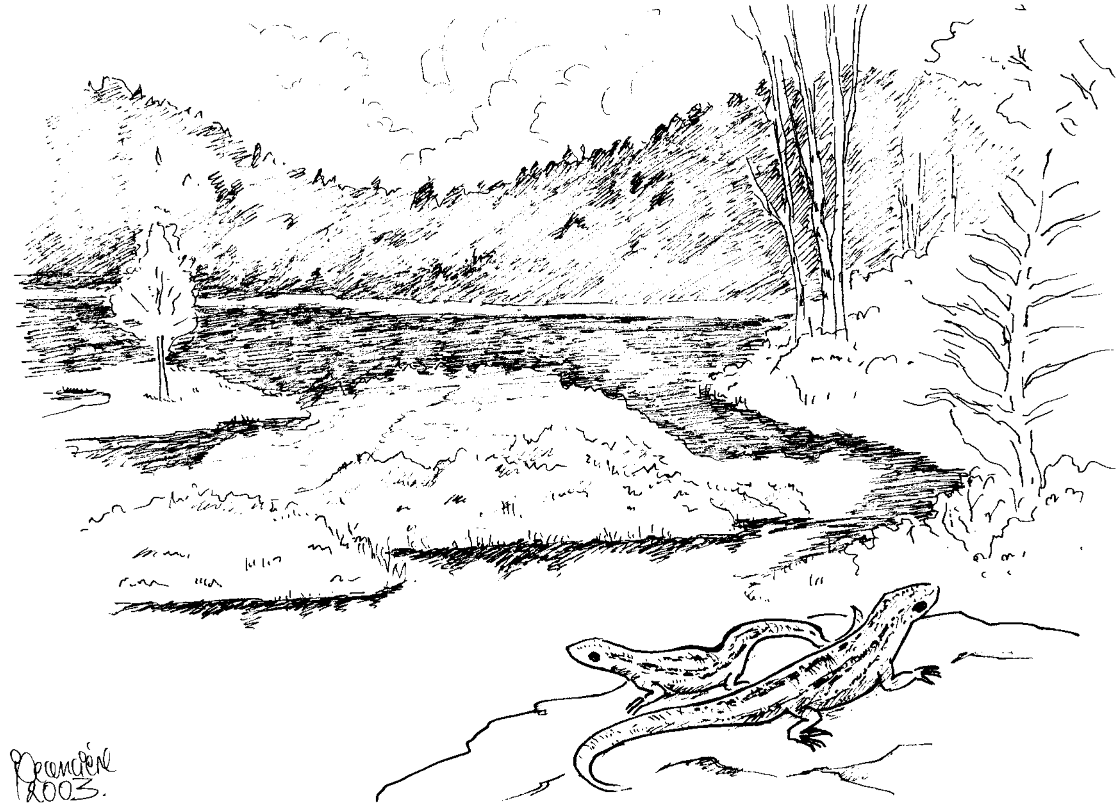
	Midnight *(n=537)*	Early morning *(n=537)*	Dispersal probability	
			C	T
Outside the shelter	*77*	*160*	-1.63 ± 0.4 (*n = 89*)	-2.57 ± 0.54 (*n = 71*)
Inside a shelter	*460*	*377*	-1.85 ± 0.34 (*n = 209*)	-1.58 ± 0.34 (*n = 168*)
Control shelter	*236*	*193*	-1.90 ± 0.37 (*n = 106*)	-1.38 ± 0.36 (*n = 87*)
Odour shelter	*224*	*184*	-1.77 ± 0.36 (*n = 103*)	-1.71 ± 0.38 (*n = 81*)

Addenda literature

Léna, J.-P. & de Fraipont, M. 1998 Kin recognition in the common lizard. *Behav. Ecol. Sociobiol.* **42**, 341-347.

Léna, J.-P., de Fraipont, M. & Clobert, J. 2000 Affinity towards maternal odour and offspring dispersal in the common lizard. *Ecol. Let.* **3**, 300-308.

CHAPITRE 5 – IMMIGRATION DANS LES METAPOPULATIONS



“According to the classical metapopulation concept, which Levins established, all local populations have a substantial probability of extinction, and hence the long-term persistence of the species can occur at the regional or metapopulation level. Alternatively, migration among local populations might affect their dynamics, as in the case of voles and their predators. […] The metapopulation concept appeals to ecologists because the world is patchy, has always been so, and is sadly becoming, for many species, ever more patchy.” I. Hanski dans *Metapopulation ecology*. 1999. p. 2-3.

IMMIGRATION DANS LES METAPOPULATIONS: CONSEQUENCES COMPORTEMENTALES ET DEMOGRAPHIQUES CHEZ LE LEZARD VIVIPARE

Jean-François Le Galliard, Régis Ferrière & Jean Clobert

RESUME

Ce chapitre présente une manipulation de métapopulations du lézard vivipare (*Lacerta vivipara*) pour évaluer les conséquences comportementales et démographiques de l'immigration dans des habitats vides (colonisation) ou des habitats occupés (augmentation). Le traitement expérimental implique soit deux habitats occupés connectés pour créer les conditions de l'augmentation, soit un habitat occupé connecté à un habitat vide pour créer les conditions de la colonisation. Les conséquences de la manipulation ont été évaluées sur les comportements de dispersion et de fixation, et sur les paramètres démographiques des résidents et des immigrants de trois classes d'âge (juvéniles, sub-adultes, et adultes).

Deux composantes de la valeur sélective des juvéniles – croissance et reproduction – ont été augmentées lors de la colonisation. Une plus forte croissance pendant la colonisation a permis aux immigrantes de se reproduire plus tôt que dans un habitat occupé. Aucun effet sur la croissance et la reproduction des sub-adultes et des adultes n'a été détecté. La probabilité de fixation n'a pas été influencée par la présence de congénères dans l'habitat d'arrivée, en accord avec l'hypothèse d'une fixation aléatoire. La relation entre la condition corporelle des sub-adultes et leur statut de dispersion dépendait de la présence de congénères dans l'habitat connecté au sein de la même métapopulation, signifiant que différents individus ont dispersé dans différentes structures de métapopulation.

Cette expérience met en évidence les effets de l'immigration sur l'individu et la population chez le lézard vivipare. Au niveau de l'individu, nos résultats suggèrent que la colonisation génère une pression de sélection positive sur l'évolution de la dispersion des juvéniles, mais pas pour les sub-adultes et les adultes. Ceci pourrait expliquer que la dispersion soit essentiellement natale chez cette espèce. Au niveau de la population, aucun effet de Allee n'a été détecté dans les habitats colonisés. Au contraire, une compétition réduite a conduit à un recrutement reproductif plus élevé et, conjointement à un flux d'immigration, a permis une croissance plus forte de la population. Ceci pourrait contribuer à la capacité de l'espèce à développer et maintenir une distribution géographique large.

Référence : Le Galliard, J.-F., Ferrière, R. and Clobert, J. « Immigration within metapopulations: behavioral and demographic consequences in the common lizard ». Soumis à *Ecology*, avec du matériel supplémentaire inclus.

IMMIGRATION WITHIN METAPOPULATIONS: BEHAVIORAL AND DEMOGRAPHIC CONSEQUENCES IN THE COMMON LIZARD

Jean-François Le Galliard, Régis Ferrière & Jean Clobert

ABSTRACT

This paper presents a manipulation of two-patch metapopulations of the common lizard (*Lacerta vivipara*) to assess the behavioral and demographic consequences of immigration into initially empty patches (colonization) versus immigration into occupied patches (augmentation). The experimental treatment involves two types of metapopulations: two initially occupied patches connected to create the condition for augmentation, and an occupied patch connected to an initially empty patch to create the condition for colonization. Consequences of manipulation on dispersal behavior, settlement behavior, and demographic parameters in residents and immigrants of three age classes (juveniles, yearlings, and adults) were measured.

Two fitness components – growth and reproduction – were positively influenced in juveniles by colonization, whereas no difference between immigrants to occupied patches and residents was observed. Faster growth during colonization allowed female immigrants to reproduce earlier than females immigrating into occupied patches. No effect on growth and reproduction was detected in yearlings or adults. Settlement probability was not influenced by conspecifics presence, in agreement with the hypothesis of random settlement and against the hypotheses of social attraction or social fence. The relationship between yearlings body condition and dispersal status was affected by conspecifics presence in the connected patch of the same metapopulation, meaning that different yearlings disperse depending upon metapopulation structure.

This experiment highlights the effects of immigration at the individual and population levels in the common lizard. At the individual level, our results suggest that colonization generates a positive selective pressure on the evolution of dispersal in juveniles, but not in yearlings or adults. This might provide an ultimate explanation for why dispersal occurs primarily at the offspring stage in this species. At the population level, no Allee effect was detected in colonized patches; rather, reduced competition resulting in increased reproductive recruitment and immigration led to higher population growth in colonized patches. This may contribute to the species' capacity to develop and maintain a wide geographic distribution.

Reference : Le Galliard, J.-F., Ferrière, R. and Clobert, J. « Immigration within metapopulations: behavioral and demographic consequences in the common lizard ». Submitted to *Ecology*, with supplementary material included.

INTRODUCTION

Dispersal is under the influence of multiple selective pressures, arising from social interactions (Perrin and Goudet 2001), and heterogeneity in space and time (Gadgil 1971). In homogeneous populations (no temporal or spatial variability), several lines of theoretical work and empirical research on vertebrates have shown that dispersal patterns are affected by the costs and benefits of emigration, resulting from interactions between relatives (kin competition, risks of inbreeding) and from intraspecific competition (see review in Lambin et al. 2001). However, fragmented populations are likely to be heterogeneous to some degree, in which case dispersal strategies can also be affected by the costs and benefits associated with the immigration phase of dispersal (Ims and Yoccoz 1997). Even in uniform landscapes, spatial and temporal variation in population size must arise among and within patches due to demographic and environmental stochasticity. The classical notion of a metapopulation offers a simple approximation of this process by contrasting two patch states: empty (following on extinction and prior to recolonization) versus occupied (Levins 1969). In this context, immigration results either in the *augmentation* of the recipient population when the arrival patch is already occupied by conspecifics, or in *colonization* if the arrival patch is initially empty of congeners (Ebenhard 1991, Ims and Yoccoz 1997). Understanding the proximate and ultimate factors of dispersal therefore requires that the immigration behaviour is evaluated and compared between these two types of patches. This study is aimed at gaining experimental insights into the demographic costs and benefits, as well as the behavioral consequences, of augmentation versus colonization.

Understanding the demographic costs and benefits of colonization is important because dispersal from occupied to empty patches of habitat is a key factor of metapopulation persistence and of the invasion of new habitats (Levins 1969, Ebenhard 1991). Augmentation can be beneficial if conspecifics decrease the costs of settlement in unfamiliar habitats (review in Greene and Stamps 2001, Fig. 1C). For example, in lizards, the location and behavior of resident individuals can be used by immigrants to select suitable basking and feeding sites (Stamps 1987, 1988). Positive density dependence due to reproductive interactions (Allee effects) also reduce the demographic success of adult colonizers, hence the potential for invading new habitats and metapopulation persistence (e.g., Veit and Lewis 1996, Stephens and Sutherland 1999). However, immigration into already occupied patches can increase the costs of intra-specific competition directly with individuals already present in the patch, and later with other immigrants (review in Lambin et al. 2001, Fig. 1B). For example, enclosed prairie voles (*Microtus ochrogaster*) emigrating in empty patches survive and reproduce better than residents in occupied patches (Johnson and Gaines 1985, 1987). Immigrants might also suffer from asymmetric competition with settled individuals due to a prior-resident advantage arising from familiarity with the habitat or social dominance (Anderson 1989, Massot et al. 1994). To our knowledge, however, no experiment has attempted to tease apart these different effects by comparing the fitness of immigrants in situation of colonization versus augmentation.

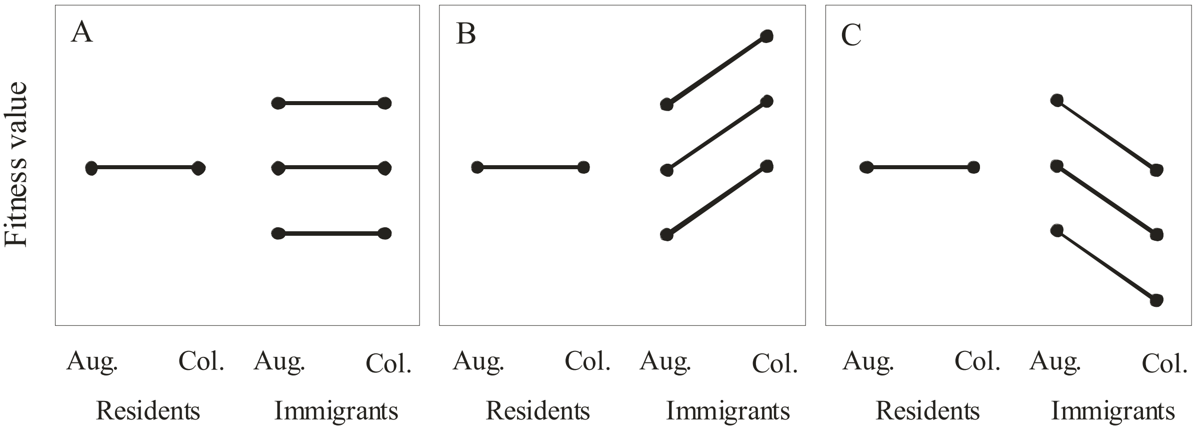


Figure 1. Three scenarios predicting the dependence of fitness on immigration status (resident versus immigrant) and immigration context (Aug.: augmentation, Col.: colonization). A. Neutral colonization scenario. Difference between residents and immigrants can be revealed by a significant effect of immigration status (top: immigrants fitness higher than residents, middle: no difference, bottom: residents fitness higher than immigrants). B. Beneficial colonization scenario. C. Costly colonization scenario.

The settlement behavior of dispersers may also differ during augmentation and colonization (Doligez et al. 2003). Such differences could affect metapopulation dynamics because settlement behavior influences colonization rate and rescue effects (Smith and Peacock 1990). Individuals may adopt three distinct patch choice strategies depending on their sensitivity to conspecifics. First, settlement could be independent of habitat occupancy, such as most metapopulation models assume (random settlement, Levins 1969). Second, settlement could involve a preference for already occupied sites, as a response to the presence of conspecifics (social attraction, Stamps 1991), or to the public information provided by the performance of conspecifics (Valone 1989). Immigrants would then cue on patch reproductive performance, assessed by the density of juveniles present in a patch (Danchin et al. 2001). A contrasting, third settlement strategy could involve aggressive interactions or competition with residents (Hestbeck 1982), resulting in repulsion for already occupied sites (social fence). Social attraction has been investigated at the individual level, and it has been found that individuals can cue on conspecifics presence for territory choice (Stamps 2001). Population level studies have only recently been initiated, and patch selection based on public information has been suggested in several birds (Frederiksen and Bregnballe 2001, Boulinier et al. 2002, Doligez et al. 2002). However, only two population-level experiments have addressed the effect of conspecifics presence on settlement in vertebrates (Danielson and Gaines 1987, Gundersen et al. 2002), and both concluded to the occurrence of social repulsion (but see Meadows and Campbell (1972) for patch level social attraction in marine invertebrates). Yet, these studies were based on the translocation of individuals into established populations, meaning that they investigated emigration following artificial transfer rather than immigration following voluntary dispersal (*sensu* Ims and Yoccoz 1997). Such an approach may be problematic to assess the effect of conspecifics presence on settlement behavior of dispersers in natural populations. Indeed, dispersers are not a random subset of the population (e.g., Swingland 1983, Massot et al. 1994), and dispersers are often engaged in social interactions with conspecifics that differ from residents (e.g., Holekamp 1986, Léna et al. 2000). Therefore, population-level experiments comparing dispersers are needed to understand the dependence of settlement choice on conspecifics presence.

Here, we report the results of an experiment designed to analyze the effect of conspecifics presence on immigration success and dispersal behavior in juveniles, yearlings, and adults of the common lizard (*Lacerta vivipara* Jacquin). We established two types of experimental units: in O─O units, two connected occupied patches created the condition for augmentation; in O─E units, an occupied patch connected to an empty patch created the condition for colonization. We monitored emigration, as measured by the probability of leaving the patch of introduction, and immigration, as the probability to settle in the arrival patch. We measured several life history traits (growth, survival and female reproduction), and analyzed their dependence upon immigration status (resident or immigrant) and treatment. As shown in Figure 1, the relationship between fitness, immigration status and treatments can be of one of three kinds: “neutral colonization”, “beneficial colonization”, or “costly colonization” scenario. The analysis of life history components allows us to identify the scenario that provides the best interpretation of our data.

METHODS

Model system

The experimental system consisted of 7 metapopulation units located in a meadow at the Ecological Research Center of Foljuif (Seine et Marne, 48°17’N, 2°41’E). Each unit consists of two patches of enclosed habitat (10 m × 10 m) connected by 20 m long one-way corridors (Fig. 2). The length of the corridors corresponds to the upper-limit of a standard home range, and is used to define dispersal status in natural populations (Clobert et al. 1994). Patch size is matched to the size of the overlapping home ranges of this species, and each patch can sustain a local population (Lecomte and Clobert 1996). Enclosures are delimited by plastic walls to prevent lizards from escaping and to preclude the intrusion of terrestrial predators. Corridors allow movements of lizards from one patch to another, and are delimited by plastic walls (1 m width, 60 cm high) covered with mesh. Pitfall traps at the end of each corridor are used to capture dispersers. More details on the experimental system can be found in Lecomte and Clobert (1996) and in Boudjemadi et al. (1999).

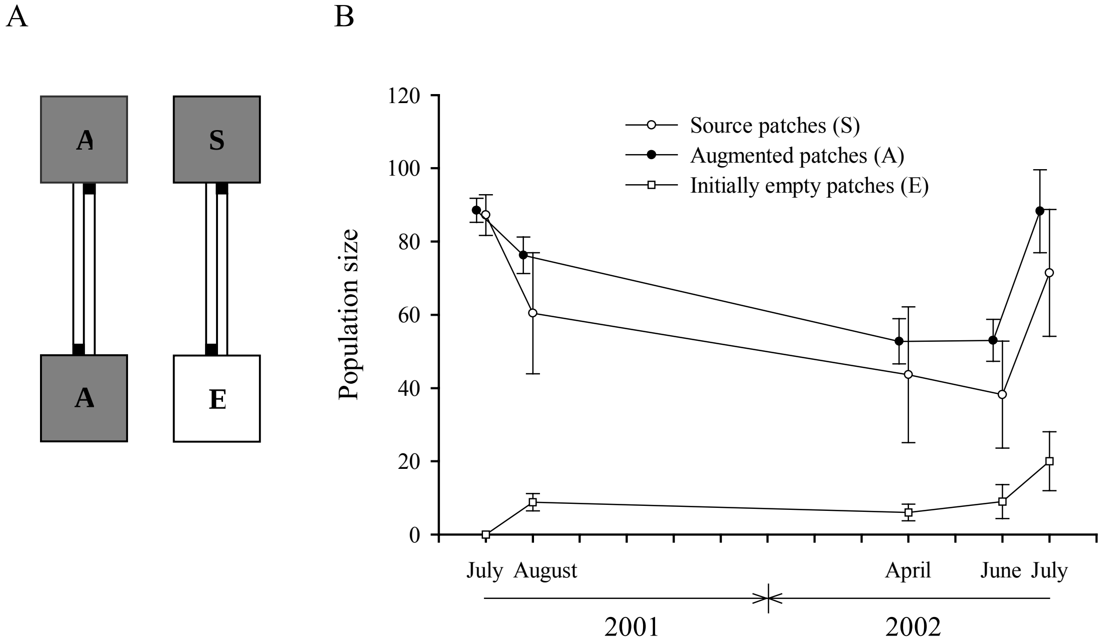


Figure 2. Experimental design and population dynamics. A. Experimental units contained either two occupied patches (A), or one occupied, source patch (S) and one empty patch (E). Pitfall traps used to monitor dispersal are the black squares at the end of each corridor. B. Population dynamics in the three types of patches (mean patch size ± SD).

Lizards can disperse from one patch to the other within the same unit, and we distinguish three classes of individuals: *residents*, *immigrants* (individuals that established in their arrival patch), and *transients* (individuals that move at least twice between patches). This characterization of movement status based on the interpatch distance fixed in the study seems appropriate. Indeed, the timing and intensity of movements events, and the morphological attributes of dispersers reported in previous studies have been found to agree with natural dispersal patterns (Lecomte and Clobert 1996, Boudjemadi et al. 1999, Le Galliard et al. 2003). Furthermore, average dispersal distances are typically smaller than the scale of landscape heterogeneity and long dispersal distances have not been reported in natural populations (Clobert et al. 1994), suggesting that habitat colonization proceeds through short-distance movements.

Experimental design

The experiment was conducted from June 2001 to June 2002. Two types of units were established at the start of the experiment: (1) two connected occupied patches to create the condition for augmentation (denoted by O─O, 2 replicates), or (2) one occupied patch connected to one empty patch to create the condition for colonization (O─E, 5 replicates). This design generated three different types of patches (Fig. 2): augmented patches, occupied from the start of the experiment and connected to similar, occupied patches (denoted by A, 4 replicates); source patches, occupied at the start of the experiment and connected to an initially empty patch (S, 5 replicates); and initially empty patches (E, 5 replicates). We treated these patches as independent observations in patch-level analyses, although pairs of patches are connected in the same unit.

Lizards were initially captured in June 2001. One sample came from populations maintained at the Ecological Research Center of Foljuif since 1999 (60 m a.s.l., sample called Fo, $n = 175$). A second sample came from one natural population in the Cévennes, Southern France (1400-1600 m a.s.l., sample called Cv, $n = 164$). All lizards were kept in individual terraria until females gave birth to autonomous offspring. The length (to the nearest mm) and body mass (to the nearest mg) of neonates were recorded, and their sex was determined by counting ventral scales (Lecomte et al. 1992). Each individual was given a unique code by toe-clipping. We initiated the experimental populations during June and July 2001 by releasing individuals into patches with which they had no prior familiarity. Sixteen yearlings were released in each population, involving 3 Fo males, 5-8 (5.8 ± 0.8 SD) Cv males, 2 Fo females and 3-6 (5.2 ± 0.8 SD) Cv females. There was no difference in the yearling sex-ratio between treatments ($\chi^2_1 = 0.35$, $P = 0.55$). Five adult males from each sample were also released in each population. Finally, ten post-gravid females along with their offspring and one non-reproductive female were released per patch (7-8 Fo and 3-4 Cv females per patch). Body size, body condition, introduction date, sex-ratio, and litter size did not differ between treatments at the start of the experiment (ANOVAs of treatment effect, all $P > 0.3$).

Census and sampling effort

Dispersal was monitored daily from July to November 2001 and from March to June 2002 by inspecting pitfall traps located at the end of each corridor. Dispersers were measured for body size and body mass, and released in the other patch of the same unit during the same day. Patches were monitored by hand recaptures in August 2001 (three recapture days), September 2001 (one recapture day), and April 2002 (two recapture days). All individuals were captured in June 2002. Individuals never caught in a dispersal trap were considered as residents provided they were captured at least once. We recorded body size and body mass at each capture. At the end of the experiment, all lizards were isolated in individual terraria, and the brood characteristics of gravid females were recorded. The data set contained 790 individuals released at the start of the experiment, 645 individuals and 2102 captures in 2001, and 446 individuals and 1131 captures in 2002.

Statistical comparisons of individual behaviour and life history traits between treatments assume adequate sampling, such that capture probabilities during the censuses have to be high and unbiased. To check these assumptions, we estimated capture probability from capture-recapture data (not seen, captured as resident, or captured as an immigrant) in August 2001, September 2001, and April 2002, assuming capture of all lizards in June 2002. We modeled capture probability as a function of treatment, season and unit within treatment using multi-strata models (Nichols et al. 1992). Results indicated significant seasonal changes in capture probability in all age classes (all $P < 0.01$), but no significant difference between treatments (all $P > 0.36$). Capture probabilities were high in August (> 90 %) compared to September (> 60 %) and April (> 75 %). Therefore, comparisons between treatments were conducted on data from the censuses of August 2001 and June 2002.

Statistical methods

We estimated population size per age and sex classes in August 2001 and April 2002 using capture-recapture models (Otis et al. 1978). Patch demography was then compared between treatments with repeated measurements analyses using August 2001 as an offset term. Patch was included as a random effect. Temporal variation was modeled as a within-patch effect using a variance-covariance structure minimizing the Aikake Index Criterion of the model (Littell et al. 1996). We used logistic analyses for age and sex structure, and log-linear models for population size.

For movements and life history traits, individuals were used as observations in hierarchical models incorporating random replicate effects nested within treatments. When we compared residents and immigrants between augmentation and colonization, unit was used as a replicate and transients were excluded from the analyses. When we compared residents between augmented and source patches, or immigrants between augmented and empty patches, patch was used as a replicate. Dispersal and settlement probabilities were modeled with mixed-effect logistic regressions (Littell et al. 1996). Individual covariates (body size, body condition, sex, geographic origin) and their interactions with treatment were included in these analyses because they have been shown to influence dispersal (Massot et al. 2002). For dispersers, we also analyzed the time between introduction and emigration, and, for transients, the time spent in the immigration patch.

We compared individual growth, survival, and reproduction between treatments. First, we calculated daily growth rates as body size differences between two measurements divided by the time separating them. Variation in body condition was also measured, but females were excluded from these analyses because of the confounding effects of reproductive burden. Body condition was computed within each session as the residual of the linear regression of body mass against body size. Second, we measured survival probability by assuming that individuals not captured in 2001 and in June 2002 were dead. Third, we assessed female reproduction in June 2002 by palpation of vitellogenic eggs. Clutch size was measured as the total number of eggs laid, and relative clutch mass was calculated as the mass loss during parturition divided by the post-parturition body mass. We measured the number of vitellogenic eggs, aborted eggs, and viable eggs per clutch to calculate the proportion of fertilized eggs within total clutch, and the proportion of viable eggs within fertilized eggs. The sex-ratio, body size, and body condition of neonates were also measured. Continuous life history traits were analyzed with mixed-effects linear models (Littell et al. 1996). Because these models assume homoscedasticity and normality of residuals, we checked the residuals with Shapiro-

Wilk tests of normality and with Bartlett's tests for homogeneity of variance. Assumptions were met in all models presented here. Binary life history traits (e.g., survival probability) were modeled with mixed-effects logistic regressions. In all cases, the full model included treatment effects, individual covariates (body size, body condition, sex, geographic origin) and interactions, and selection was done through backward elimination of the non-significant terms. Results are given as mean ± SE unless otherwise stated.

Table 1. Influences of treatment (augmentation or colonization) or patch type (augmented, source, or initially empty) on dispersal and settlement probabilities.

Movement type	Predictor effect	Test statistic	P value
Summer dispersal	Age	$F_{2,633} = 1.49$	$P = 0.23$
	Sex	$\mathbf{F_{1,633} = 7.47}$	$\mathbf{P = 0.006}$
	Treatment	$F_{1,7} = 0.47$	$P = 0.51$
Spring dispersal	**Age**	$\mathbf{F_{2,457} = 3.14}$	$\mathbf{P = 0.04}$
	Sex	$\mathbf{F_{1,457} = 8.63}$	$\mathbf{P = 0.003}$
	Patch type	$F_{2,11} = 1.44$	$P = 0.28$
Summer settlement	Age	$F_{2,103} = 0.81$	$P = 0.45$
	Sex	$F_{1,103} = 2.01$	$P = 0.16$
	Treatment	$F_{1,7} = 0.32$	$P = 0.59$

In addition to the fixed terms, the generalized linear mixed models also included a random replicate effect (unit for treatment or patch for patch type). Significant terms are highlighted in bold.

RESULTS

Population dynamics

All initially empty patches became colonized within one month (Fig. 2), and no colonized patch went extinct during the experiment. Starting one month after introduction, the average population size of colonized patches was 7.3 ± 3.5 SD individuals, a value lower than in initially occupied patches ($F_{2,11} = 31.4$, $P < 0.001$, contrast between initially empty and occupied patches: $P < 0.001$). There was no detectable difference between population sizes of source and augmented patches (S: 47.4 ± 18.2 SD individuals, A: 60.7 ± 12.6 SD individuals, contrast: $P = 0.23$). Also, from one month after introduction onward, sex structure and age structure remained similar in initially empty, source and augmented patches (patch specific proportion of males: $F_{2,11} = 0.93$, $P = 0.42$; patch specific proportion of juveniles: $F_{2,11} = 1.37$, $P = 0.29$).

Population growth rate before reproductive recruitment in July 2002 was calculated as the log-transformed ratio of population size in June 2002 to population size in August 2001. Populations declined in augmented patches (per capita growth rate: $r = -0.37 \pm 0.08$) and in source patches ($r = -0.49 \pm 0.06$), while colonized populations increased at the same time ($r = 0.26 \pm 0.26$; effect of patch type: $F_{2,11} = 5.34$, $P = 0.02$; Fig. 2). The reproductive recruitment rate was measured as the number of juveniles produced per female in the patch. The reproductive recruitment rate was higher in colonized patches (3.02 ± 0.58), than in source patches (2.04 ± 0.39) and augmented patches (1.43 ± 0.22; contrast between initially empty and occupied patches: $P = 0.04$). Therefore, the population growth rate from August 2001 to the end of offspring recruitment was again higher in colonized patches ($F_{2,11} = 6.95$, $P = 0.01$). At the metapopulation level, units growth rates calculated from the time of introduction to the end of offspring recruitment did not differ between treatments (O—O units: $r = -0.006 \pm 0.39$, O—E units: $r = 0.01 \pm 0.10$; treatment effect: $F_{1,5} = 0.02$, $P = 0.89$).

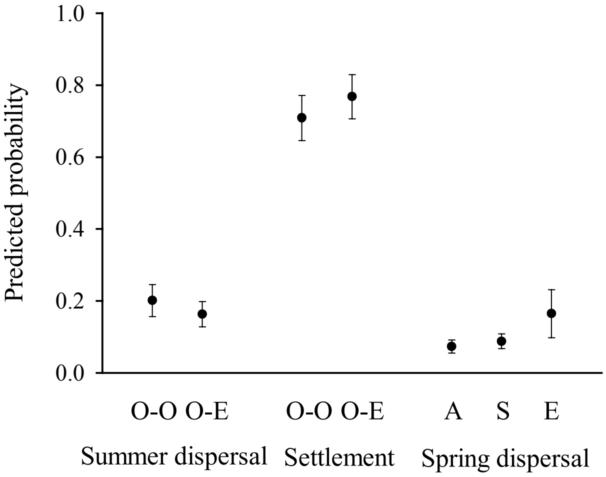


Figure 3. Effect of manipulation on dispersal and settlement probability during 2001 (O—O: augmentation, O—E: colonization), and effect of patch type on dispersal during spring 2002 (A: augmented patches, S: source patches, and E: initially empty patches). Values are back-transformed predicted means ± SE derived from the models described in Table 1.

Dispersal probability

Of the 790 lizards introduced, 645 individuals were captured at least once and 116 individuals (52 juveniles, 27 yearlings, and 37 adults) moved at least once before hibernation. Overall, males were 1.8 times more likely to leave the introduction patch than females. However, dispersal probability was not different between source patches and augmented patches (Table 1, Fig. 3). Of the 474 individuals surviving after hibernation, 45 individuals (12 juveniles, 13 yearlings, and 20 adults) moved at least once before the end of the experiment. Because these individuals were captured in all patches, it was possible to compare dispersal probability between source, augmented, and colonized patches (Table 1, Fig. 3). Yearlings and adults dispersed earlier than juveniles (juveniles: 4 may ± 5.1 days, yearlings: 9 April ± 5.5, adults: 7 April ± 4.3; $F_{2,97} = 9.95$, $P < 0.001$; contrast between juveniles and yearlings-adults = 26.2 days ± 6.17, $P = 0.002$). Also, yearlings and adults dispersed more than juveniles during spring (odds yearlings and adults : odds juveniles = 2.31, $P = 0.02$), and dispersal was male-biased (odds male : odds female = 2.86). However, movement probability was not affected by patch type (Table 1, Fig. 3).

We also analyzed the effects of individual covariates on dispersal probability with multiple logistic regressions. In 2001, there was no effect of juvenile body size, sex, maternal origin, clutch size, or maternal body size on dispersal probability (all $P > 0.08$), but there was a correlation between dispersal status and juvenile body condition at birth ($F_{1,303} = 8.74$, $P = 0.003$). Juvenile dispersers were more corpulent at birth than residents irrespective to the treatment (Residents: -0.0016 ± 0.001, $n = 280$; Dispersers: 0.009 ± 0.003, $n = 52$). In yearlings, dispersal was affected by an interaction between body condition at introduction and experimental treatment ($F_{1,120} = 4.96$, $P = 0.03$). Yearling dispersers tended to be more corpulent at introduction than yearling residents in O─O units (odds ratio per condition unit = 2.19, $P = 0.19$), while they tended to be leaner at introduction in O─E units (odds ratio = -3.77, $P = 0.07$, Table 2). Adult dispersal probability was correlated with body size, sex and geographic origin irrespective to experimental treatment in the multiple regression. Adult dispersers had larger body size than residents (odds ratio per mm = 1.22 , $F_{1,170} = 7.72$, $P = 0.006$), adult males dispersed more than females (odds males : odds females = 10.7, $F_{1,170} = 11.95$, $P < 0.001$), and adults from the local area dispersed more than individuals translocated from the mountain area (odds Fo : odds Cv = 3.25, $F_{1,170} = 6.53$, $P = 0.01$).

Settlement probability

Settlement observations during 2001 involved 87 immigrants that settled in their patch of arrival, and 29 transients that returned to their patch of introduction. Across all age and sex classes, settlement probability was not different between augmented and initially empty patches (Table 1). Nevertheless, settlement probability in initially empty patches might have been influenced by the timing of dispersal in the course of the colonization process as predicted by a social attraction hypothesis (Stamps 1991). Against this hypothesis, late dispersers were not more likely to settle in an initially empty patch than early dispersers (logistic regression of settlement probability on the rank of arrival in an empty patch, $F_{1,44} = 0.25$, $P = 0.62$).

Table 2. Initial body condition for resident, immigrant and transient yearlings in the two treatments.

Type	Augmentation	Colonization
Residents	-0.04 ± 0.03 (*n = 46*)	0.04 ± 0.02 (*n = 58*)
Dispersers	0.04 ± 0.05 (*n = 13*)	-0.06 ± 0.05 (*n = 14*)
Immigrants	0.14 ± 0.06 (*n = 8*)	-0.09 ± 0.05 (*n = 10*)
Transients	-0.12 ± 0.05 (*n = 5*)	0.03 ± 0.11 (*n = 4*)

Data are means ± SE, and sample size is indicated in brackets

Due to the low number of transients (15 juveniles, 9 yearlings, and 5 adults spread over 12 different patches), we analyzed the effect of morphology on settlement probability ignoring patch identity. For juveniles, there was no difference between body size, body condition, and origin of immigrants and transients (all P > 0.33). For yearlings, transients were heavier at introduction than immigrants in colonized patches, whereas the opposite was observed in augmented patches (Settlement status × Patch type effect: χ^2_1 = 9.12, P = 0.002; Table 2). Transient adults were leaner than immigrants at introduction irrespective to the type of patch (Settlement status effect χ^2_1 = 4.00, P = 0.04; Transients: -0.43 ± 0.11, *n = 7*, Immigrants: 0.10 ± 0.12, *n = 30*).

Table 3. Effects of conspecific presence on the timing of dispersal and transience.

Time spent in	Predictor effect	Test statistic	P
Introduction patch	**Age**	**$F_{2,102} = 12.01$**	**P < 0.001**
	Sex	**$F_{1,102} = 5.90$**	**P = 0.02**
	Age × Sex	**$F_{2,102} = 3.19$**	**P = 0.045**
	Treatment	$F_{1,7} = 0.56$	P = 0.48
Arrival patch	Age	$F_{2,19} = 0.94$	P = 0.41
	Sex	$F_{1,19} = 1.53$	P = 0.23
	Treatment	**$F_{1,5} = 7.35$**	**P = 0.042**

The time spent in a patch is calculated as the number of days between arrival in and departure from a patch. Significant terms are highlighted in bold.

Timing of dispersal and settlement

Time spent within a patch before dispersal was found to be similar between augmented and source patches (Table 3). Adult and yearling males dispersed earlier than adult and yearling females (contrast between females and males = 17.8 days ± 5.65, P = 0.002), but juvenile males and juvenile females had similar timing of dispersal (contrast between females and males = -3.9 days ± 6.48 , P = 0.55). In the case of transients, the time spent within the patch of arrival before moving back was not affected by age or sex, but depended on the presence of conspecifics (Table 3). Transients stayed longer in initially empty patches than in augmented patches (31.1 ± 5.9 days versus 10.8 ± 4.8 days).

Growth

Changes in body size were studied in both sexes, but body condition was investigated only in males due to the confounding effects of reproduction (see *Reproduction* section). The body condition of male juveniles was not affected by treatment or immigration status (all P > 0.11). The body size growth was sex-dependent in all age classes (Table 4), and females had higher growth rates than males. In a linear mixed-effect model of juvenile body growth, the interaction between treatment and immigration status was significant (Table 4). As predicted by the beneficial colonization scenario (see Fig. 1B), the annual growth rate was higher for immigrants in O—E units than for immigrants in O—O units and for residents (independent contrasts, all adjusted $P < 0.05$, Fig. 4), and immigrants in O—O units were not different from residents ($P > 0.13$). The growth rates of juvenile immigrants before dispersal were not different between source and augmented patches ($F_{1,6} = 1.02$, $P = 0.35$, *n = 52*), whereas growth following settlement until the end of 2001 was higher in colonized patches than in augmented patches ($F_{1,5} = 31.45$, $P = 0.002$, *n = 19*) and over the study year ($F_{1,4} = 7.81$, $P = 0.05$, *n = 24*). A comparison among siblings within families with at least one resident and one immigrant led to the same result (Immigration status × Treatment effect: $F_{1,49} = 5.08$, $P = 0.03$).

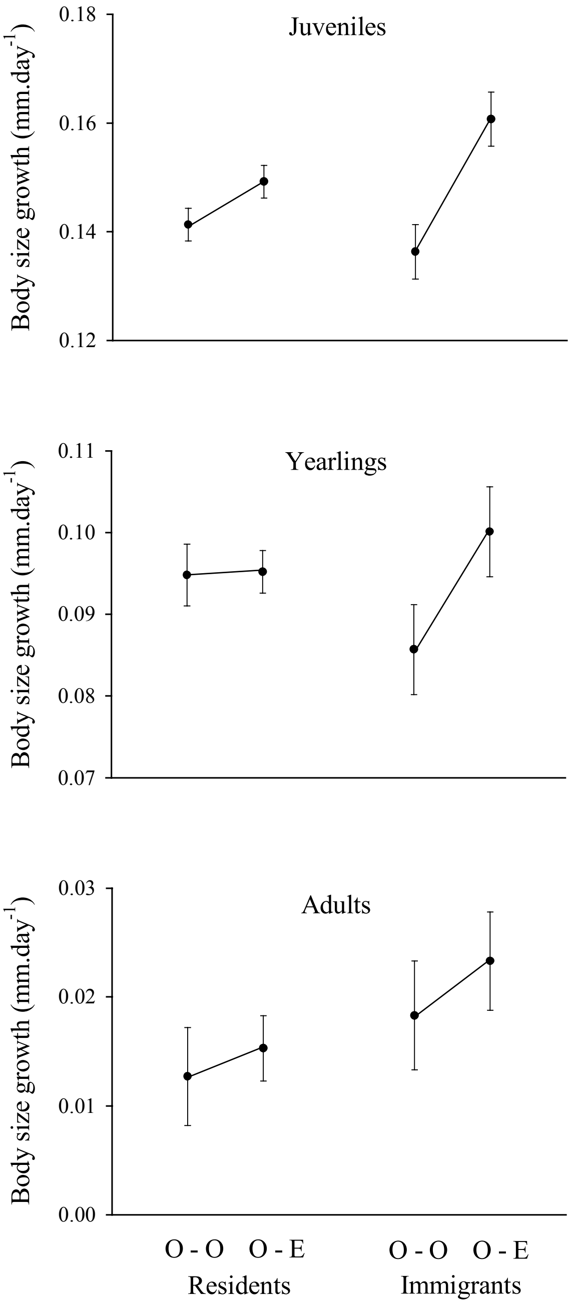


Figure 4. Annual body size growth depending on immigration status (residents or immigrants) and manipulation within juveniles, yearlings and adults (O—O: augmented units, O—E: colonization units). Data are predicted means ± SE after the models described in Table 4.

Table 4. Demographic consequences of colonization versus colonization on annual growth in body size.

Age cohort	Predictor effect	Test statistic	P
Juveniles	**Sex**	**$F_{1,118} = 10.2$**	**P = 0.002**
	Immigration status	$F_{1,118} = 0.85$	P = 0.36
	Treatment	**$F_{1,5} = 7.47$**	**P = 0.04**
	Immigration status × Treatment	**$F_{1,118} = 7.08$**	**P = 0.009**
Yearlings	**Sex**	**$F_{1,73} = 46.75$**	**P < 0.001**
	Immigration status	$F_{1,73} = 0.31$	P = 0.58
	Treatment	$F_{1,5} = 2.06$	P = 0.21
	Immigration status × Treatment	$F_{1,73} = 3.54$	P = 0.06
Adults	**Sex**	**$F_{1,113} = 86.6$**	**P < 0.0001**
	Immigration status	**$F_{1,113} = 6.92$**	**P = 0.01**
	Treatment	$F_{1,5} = 0.45$	P = 0.53
	Immigration status × Treatment	$F_{1,113} = 0.20$	P = 0.66

Statistical models included a random unit effect, and a random family effect in the case of juveniles. Significant terms are highlighted in bold.

Among yearlings, immigration status and experimental treatment tended to affect annual growth rates in body size (Table 4), and the trend was concordant with the beneficial colonization hypothesis (see Figs. 4 and 1B). Among adults, immigrants grew more than residents over the study year independently of treatment (Table 4, Fig. 4). Annual variation in body condition of yearling males was not affected by the treatment (P > 0.20). The annual variation in body condition of adult males was influenced by an interaction between immigration status and treatment ($F_{1,47} = 8.39$, P = 0.006, 57 observations). As predicted by the benefits of colonization hypothesis (see Fig. 1B), body condition of male immigrants increased in O─E units relative to O─O units (contrast between colonization and augmentation units = 0.56 ± 0.18, P = 0.016, Fig. 5). Treatment had no effect on body condition variation for adult male residents (P = 0.95).

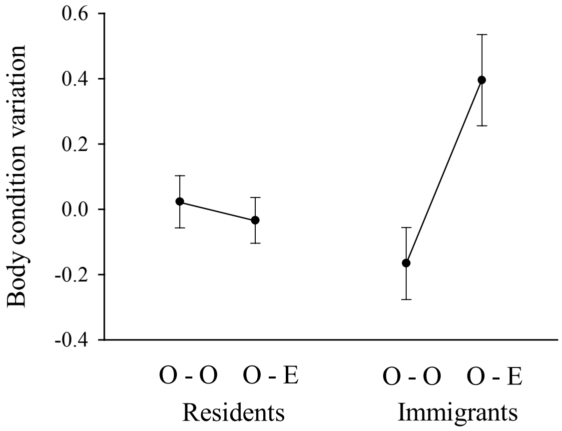


Figure 5. Annual variation in the body condition of adult males depending on immigration status (residents or immigrants) and experimental treatment (O─O: augmentation, O─E: colonization). Data are predicted means ± SE after the model described in the text.

Survival

Individuals that were captured during 2001 but not in June 2002 were assumed to be dead at the end of the experiment. The post-summer survival of juveniles, yearlings or adults was not influenced by experimental treatment and immigration status (Table 5). Annual survival was affected by age, and juveniles had lower annual survival than yearlings and adults (odds juveniles : odds yearlings and adults = 0.49, $P < 0.001$).

Table 5. Demographic consequences of colonization versus colonization on post-summer survival.

Predictor effect	Test statistic	P
Age	**$F_{2,595} = 15.43$**	**$P < 0.001$**
Sex	$F_{1,595} = 0.001$	$P = 0.98$
Immigration status	$F_{1,595} = 0.76$	$P = 0.38$
Treatment	$F_{1,5} = 0.10$	$P = 0.76$
Immigration status × Treatment	$F_{1,595} = 0.06$	$P = 0.81$

Significant terms are highlighted in bold.

Reproduction

In a logistic regression model of the proportion of females maturing before the age of one year, the interaction between treatment and immigration status was significant ($\chi^2_1 = 5.84$, $P = 0.02$). As predicted by beneficial colonization scenario (Fig. 1B), the proportion of mature females before the age of one year was independent of treatment in residents (Fisher's exact test, $P = 0.50$, $n = 94$), whereas this proportion was higher in colonized patches than in augmented patches in immigrants (Fisher's exact test, $P = 0.01$, $n = 11$; Table 6). Because age at first reproduction can depend on post-natal growth, we tested whether reproduction of immigrants was correlated with rates of body growth. In agreement with this hypothesis, the post-natal growth in body size was positively correlated with the probability of reaching maturity before the age of one year ($\chi^2_1 = 34.52$, $P < 0.001$). When the post-natal growth rate was accounted for, the interaction between treatment and immigration status had no effect on the probability to reach maturity ($\chi^2_1 = 1.28$, $P = 0.26$).

Among yearling and adult females, some females were not gravid at the end of the experiment. The probability that a female older than one-year was gravid increased with female body size ($F_{1,97} = 12.63$, $P < 0.001$), but was not affected by the treatment ($F_{1,5} = 1.03$, $P = 0.36$), the immigration status ($F_{1,97} = 0.87$, $P = 0.35$) or the interaction between treatment and immigration status ($F_{1,97} = 1.72$, $P = 0.19$, Table 6). Clutch size, laying date and reproductive effort were all influenced by female body size ($P < 0.001$), but not by the treatment or the immigration status ($P > 0.24$). The 92 gravid females produced a total of 480 eggs among which 40 were not fertilized and 63 were fertilized but not viable. Fertilization success, developmental success, clutch sex-ratio, offspring body size and offspring condition at birth were not influenced by experimental treatment or immigration status of the mother (all $P > 0.23$).

Table 6. Probability for females to reproduce in the two treatments.

Age cohort	Type	Augmentation	Colonization
Less than one-year old	Resident	0.08 [0.03, 0.19] (*n* = *51*)	0.14 [0.06, 0.28] (*n* = *43*)
	Immigrant	0.0 (*n*= *5*)	0.83 [0.37, 0.97] (*n* = *6*)
More than one-year old	Resident	0.80 [0.65, 0.90] (*n* = *41*)	0.74 [0.60, 0.85] (*n* = *51*)
	Immigrant	0.50 [0.16, 0.84] (*n* = *6*)	0.89 [0.48, 0.98] (*n* = *9*)

Results are the predicted means and 95% CI after the models described in the main text. Sample size is in parentheses.

DISCUSSION

In contrast with most previous experiments, our manipulation of experimental two-patch metapopulations involved the spatial structure of the population rather than the structure of the habitat (Hansson 1991, Clobert et al. 2001). By comparing individuals moving into occupied patches and those moving into initially empty patches, our study outlines the behavioral and demographic consequences of colonization versus augmentation in the common lizard.

Costs and benefits of immigration

Two fitness components (rates of body growth and female reproduction) were enhanced for offspring immigrating into initially empty patches compared to offspring immigrating into occupied patches, whereas the latter did not differ from resident offspring. No difference between colonization and augmentation was detected in yearling and adult immigrants. These results agree with the "beneficial colonization" scenario for juveniles, and with the "neutral colonization" scenario for yearlings and adults (Fig. 1).

These effects of colonization can be explained by intraspecific competition. It has been shown in the common lizard that population density influences competition for food, space or social partners (Massot et al. 1992, Lecomte et al. 1994), especially through natal growth (Massot et al. 1992). Therefore, competitive interactions and social stress were probably reduced in colonized patches, resulting in enhanced body growth. Furthermore, prey availability was affected by initial patch occupancy: at the end of the experiment, the abundance of spiders (one of the main preys of the common lizard, see Avery 1962) was double along 10 m length transects in initially empty patches (22.2 ± 1.7 spiders per transect) compared to initially occupied patches (11.2 ± 1.2; $F_{1,10} = 22.2$, $P <$

0.001). This suggests that the short-term benefits of colonization are tightly associated with a greater food availability. The fact that the growth of yearlings and adults was not influenced in the same way as juveniles growth may reflect the lower growth plasticity of older lizards. Also, older individuals occupy a dominant position in the population, and may be less sensitive to competition than juveniles (Lecomte et al. 1994).

The increase in body growth within colonized patches caused most female juveniles immigrating into initially occupied patches to reach a critical body size before the age of one year, enabling them to reproduce earlier than females immigrating into occupied patches. This experimental shift in the age of reproduction parallels the spatial variation described in this species. Along an altitudinal gradient, constraints on thermal opportunities delay reproduction from the age of one year in lowland populations up to the age of three years in most upland populations (Bauwens et al. 1986). Our results suggest further that age at first reproduction is plastic within lowland habitats depending on the intensity of competition experienced during post-natal growth.

Nevertheless, the short-term benefits of colonization evidenced here could be offset by delayed fitness costs. First, some intraspecific comparisons suggest that post-natal growth and reproductive efforts can decrease future survival in lizards (e.g., Olsson and Shine 2002). Second, compared to more than one-year old females, less than one-year old females laid their clutches significantly later (contrast between less and more than one-year old females = 12.5 ± 2.5 days, $P < 0.001$), produced smaller (contrast = -0.94 ± 0.33 mm, $P < 0.001$), and leaner offspring (contrast = -0.019 ± 0.006, $P = 0.001$). The delay between young and old females relates to the fact that smaller females keep growing during the spring before they are receptive to mates, whereas larger females engage in mating right after hibernation (Bauwens and Verheyen 1985). The correlation between maternal age and offspring phenotype may originate from allometric constraints due to the lower sizes of abdominal cavity and pelvic girdles in younger females (Sinervo 1999). Delayed laying, and lower offspring condition and size are likely to be associated with poor offspring survival in the common lizard (Sorci and Clobert 1999). To assess the importance of these compensatory effects, we computed lifetime reproductive success of two life-history strategies: reproduction before the age of one year, and reproduction after the age of one year (Fig. 6). We neglected the fact that benefits of colonization can affect females later in life. We assumed that earlier reproduction trades off with reduced reproductive recruitment, due to lower fecundity and lower offspring survival, and with some unknown reduction in survival to adulthood. The model gave us an upper bound on survival costs of earlier reproduction such that benefits of colonization hold through entire life:

$$S_1 - S'_1 \leq \frac{B_1}{B_2} \times (1 - S_2) \cong 0.20,$$

where terms and estimations are explained in Figure 6. To our knowledge, such a decrement of female yearling survival has never been recorded in any demographic study of the common lizard (Massot et al. 1992, Sorci et al. 1996, Ronce et al. 1998).

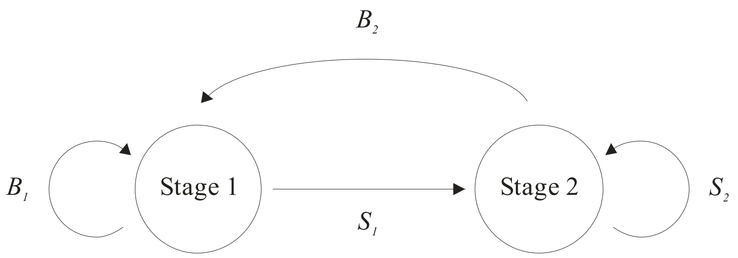


Figure 6. Two-stage life cycle of female common lizards with less than one-year old (stage 1) and more than one-year old individuals (stage 2). Parameters are reproductive recruitment rates (B) and survival (S). Reproductive recruitment involves stage-specific fecundity (F) and offspring survival (s), and is given by $B_i = F_i \times s_i$ Lifetime reproductive success (R_0) is given by $R_0 = B_1 + S_1 B_2 / (1 - S_2)$. Parameters for females maturing after one year are: $F_1 = 0$, $F_2 = 5.2$, $S_2 = 0.70$, and $S_1 = 0.60$. Parameters for females maturing before one year are: $F_1 = 4.1$, $F_2 = 5.2$, $S_2 = 0.70$, and S'_1. Offspring survival s can be estimated from offspring phenotype and independent data on phenotypic selection gradients, which yields $s_1 = 0.36$ and $s_2 = 0.44$ (Le Galliard et al. *unpublished manuscript*[3]).

The significant short-term increase in offspring immigrant fitness associated with colonization has implications for our understanding of the dispersal patterns in the common lizard. Theory suggests that a negative relationship between fitness and patch occupancy, jointly with a variability in patch density favor the evolution of dispersal (Gadgil 1971, Olivieri et al. 1995). The second condition is likely to be fulfilled in our species since there is significant variation of population density at the spatial scale over which most dispersal occurs (Massot *personal communications*). This experiment provides evidence that the first condition applies to dispersing offspring, which suggests that opportunities of colonization could generate a significant selective pressure for the evolution of natal dispersal in this species. The non significant effect of colonization on adult and yearling immigrants further indicates that selective pressures associated with colonization decrease with age. This result might explain why dispersal occurs primarily at the offspring stage in this species (Clobert et al. 1994).

Settlement behavior

Settlement probability was not influenced by conspecifics presence in the patch of immigration. Transient individuals stayed longer in colonized patches than in augmented patches. Conspecifics presence affected the body condition of yearling immigrants, which were leaner in colonized than in augmented patches, but not the morphology of juvenile and adult immigrants. Nevertheless, low sample sizes in transients call for caution when interpreting these comparisons. Thus, although some qualitative differences between settlement in augmented and colonized patches were detected, our

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[3] See Appendix 5 of the thesis

results are more consistent with the hypothesis of random settlement flow (Levins 1969) than with the hypotheses of conspecific attraction (Stamps 1991) or social fence (Hestbeck 1982).

Social fence phenomena have been illustrated by studies of dispersal movements in saturated populations of small mammals and cooperatively breeding birds (Lambin et al. 2001). In these species, it has been suggested that habitat saturation increases the frequency of aggressive social interactions, raises the risks of predation during movement, and decreases the opportunities of independent breeding (e.g., Jones et al. 1988, Komdeur 1992, Gundersen et al. 2002). The absence of any sign of social fence in our study is somewhat puzzling, since it has been found in the common lizard that competition increases with habitat saturation (Massot et al. 1992), that habitat occupancy affects the success of immigration (this study), and that habitat density decreases transience (Massot et al. 1992).

Conspecifics attraction has received support from studies of habitat selection at different spatial scales, ranging from territory to patch or colony choice (Stamps 1991, Danchin et al. 2001). Recent models have shown that cueing on conspecifics can bring some benefits (Greene and Stamps 2001, Doligez et al. 2003). For example, comparing random settlement with settlement strategies based on conspecifics presence or success, Doligez et al. (2003) found that a random strategy is generally defeated, unless habitat quality is really unpredictable. However, these models do not account for the spatial and temporal constraints acting on settlement neither for the fitness costs of habitat rejection. In our species, mobility is restricted and exploration requires to move over distances that are not less than effective dispersal distances. Thus, it is possible that the random settlement strategy observed here could reflect constraints on habitat choice (Stamps 2001).

Metapopulation effects

We created two types of metapopulations by releasing individuals either in one patch or in both patches within a unit, implying two levels of metapopulation density (low in colonization O—E units, high in augmentation O—O units), and two types of interpatch flow (unidirectional in colonization units, bi-directional in augmentation units). Demographic and behavioral processes acting at the metapopulation level could produce differences in the population dynamics, and the dispersal behavior or life history traits of residents between colonization and augmentation units. Indeed, effects of metapopulation structure dispersal in juvenile common lizards were found in a study by Boudjemadi et al. (1999) in response to a manipulation of interpatch connectivity. In our study, we found no effect of treatment on units demography, and life history of residents. Also, emigration probabilities from the patches of introduction were not influenced by metapopulation manipulation. However, the analysis of dispersers morphology indicated that metapopulation structure affected the body condition of yearling dispersers. This response could have been mediated by the arrival of unfamiliar immigrants inducing different social interactions in augmented patches compared to source patches.

Colonization and patch demography

Colonized patches had a higher per capita growth rate from early colonization to the end of the study than other types of patches. This was essentially due to immigration from source patches more than compensating for local mortality. Thus, the success of the settlement phase of colonization was facilitated by an immigration flow acting like the general rescue effect (Brown and Kodric-Brown 1977).

In colonized patches, rescue effects were not cancelled out by reduced reproductive recruitment resulting from Allee effects. On the contrary, reproductive recruitment was higher in colonized patches than in previously occupied patches. It may be that founder populations exceeded any critical threshold density for Allee effects to occur, or that the size of our enclosures was too small to induce such effects. However, Allee effects could also have been cancelled by alternative mate search strategies, population structure, or qualitative aspects of dispersal. First, low population density in colonized patches may be compensated for by higher male mobility during the mating season, increasing the operational sex-ratio and decreasing the risk of Allee effects (e.g., South and Kenward 2001). Second, dispersal can affect the sex ratio of colonized patches, because males dispersed more than females while settlement was independent of sex. Perhaps the low power of the corresponding tests explains why this did not lead to significant sex structure differences at the population level. However, colonized patches tended to host more males than other patches (male proportion, E: 0.61 ± 0.05, S: 0.53 ± 0.02, A: 0.53 ± 0.02), and were often subject to male immigration during the mating period (2.4 males per patch). These differences have the potential to mitigate a negative impact of low population density on mating dynamics. Third, any other qualitative difference between dispersers and residents, such as the body condition differences detected in our study, is likely to influence small populations resulting from colonization events, and make them different from small populations resulting from the decrease of formerly large populations.

Overall, reduced competition resulting in increased reproductive recruitment and continuous immigration flow from source patches led to higher population growth in colonized patches than in other patches. Such dynamics may explain the capacity of the common lizard to maintain a large geographic distribution. The species is indeed one of the most widespread lizard on Earth, with populations distributed from northern Spain up to the Arctic circle, from western Europe to the Sakkaline Island, and from sea level up to 3,000 m elevation (Heulin and Guillaume 1989). Also, accelerated colonization could have contributed to the Quaternary invasions of the whole Eurasia by populations of viviparous common lizards spreading from its main southeastern refuges during the ice ages (Surget-Groba et al. 2001).

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LITERATURE CITED

Anderson, P. K. 1989. Dispersal in rodents: a resident fitness hypothesis.

Avery, R. A. 1962. Notes on the ecology of *Lacerta vivipara* L. British Journal of Herpetology **3**:165-170.

Bauwens, D., B. Heulin, and T. Pilorge. 1986. Variation spatio-temporelle des caractéristiques démographiques dans et entre populations du lézard *Lacerta vivipara*. Pages 531-536 *in* J.-M. Legay, editor. Biologie des populations. CNRS, Université Claude Bernard Lyon I.

Bauwens, D., and R. F. Verheyen. 1985. The timing of reproduction in the Lizards *Lacerta vivipara* : differences between individual females. Journal of Herpetology **19**:353-364.

Boudjemadi, K., J. Lecomte, and J. Clobert. 1999. Influence of connectivity on demography and dispersal in two contrasting habitats: an experimental approach. Journal of Animal Ecology **68**:1207-1224.

Boulinier, T., N. G. Yoccoz, K. D. McCoy, K. E. Eriskstad, and T. Tveraa. 2002. Testing the effect of conspecific reproductive success on dispersal and recruitment decisions in a colonial bird: design issues. Journal of Applied Statistics **29**:509-520.

Brown, J. H., and A. Kodric-Brown. 1977. Turnover rates in insular biogeography: effect of immigration on extinction. Ecology **58**:445-449.

Clobert, J., E. Danchin, A. A. Dhondt, and J. D. Nichols, editors. 2001. Dispersal. Cambridge University Press, Cambridge.

Clobert, J., M. Massot, J. Lecomte, G. Sorci, M. de Fraipont, and R. Barbault. 1994. Determinants of dispersal behavior: the common lizard as a case study. Pages 183-206 *in* L. J. Vitt and E. R. Pianka, editors. Lizard ecology. Historical and experimental perspectives. Princeton University Press, Princeton.

Danchin, E., D. Heg, and B. Doligez. 2001. Public information and breeding habitat selection. Pages 243-258 *in* J. Clobert, E. Danchin, A. A. Dhondt, and J. D. Nichols, editors. Dispersal. Cambridge University Press, Cambridge.

Danielson, B. J., and M. S. Gaines. 1987. The influences of conspecific and heterospecific residents on colonization. Ecology **68**:1778-1784.

Doligez, B., C. Cadet, E. Danchin, and T. Boulinier. 2003. When to use public information for breeding habitat selection? The role of environmental predictability and density dependence. Animal Behaviour.

Doligez, B., E. Danchin, and J. Clobert. 2002. Public information and breeding habitat selection in a wild bird population. Science **297**:1168-1170.

Ebenhard, T. 1991. Colonization in metapopulations: a review of theory and observations. Journal of the Linnean Society **42**:105-121.

Frederiksen, M., and T. Bregnballe. 2001. Conspecific reproductive success affects age of recruitment in a great cormorant, *Phalacrocorax carbo sinensis*, colony. Proceedings of the Royal Society London B **268**:1519-1526.

Gadgil, M. 1971. Dispersal: population consequences and evolution. Ecology **52**:253-261.

Greene, C. M., and J. A. Stamps. 2001. Habitat selection at low population densities. Ecology **82**:2091-2100.

Gundersen, G., H. P. Andreassen, and R. A. Ims. 2002. Individual and population level determinants of immigration success on local habitat patches: an experimental approach. Ecology Letters **5**:294-301.

Hansson, L. 1991. Dispersal and connectivity in metapopulations. Biological Journal of the Linnean Society **42**:89-103.

Hestbeck, J. B. 1982. Population regulation of cyclic mammals: the social fence hypothesis. Oikos **39**:157-163.

Heulin, B., and C. Guillaume. 1989. The geographical distribution of oviparous populations of *Lacerta vivipara*. Revue d'Ecologie - La Terre et la Vie **44**:283-289.

Holekamp, K. E. 1986. Proximal causes of natal dispersal in Belding's ground squirrels (*Spermophilus beldingi*). Ecological Monographs **56**:365-391.

Ims, R. A., and N. G. Yoccoz. 1997. Studying transfer processes in metapopulations: emigration, migration, and colonization. Pages 247-265 *in* I. A. Hanski and M. E. Gilpin, editors. Metapopulation biology: ecology, genetics, and evolution. Academic Press, San Diego.

Johnson, M. L., and M. S. Gaines. 1985. Selective basis for emigration of the prairie vole, *Microtus ochrogaster:* open field experiments. Journal of Animal Ecology **54**:399-410.

Johnson, M. L., and M. S. Gaines. 1987. The selective basis for dispersal of the prairie vole, *Microtus ochrogaster*. Ecology **68**:684-694.

Jones, W. T., P. M. Waser, L. F. Eliott, and N. E. Link. 1988. Philopatry, dispersal, and habitat saturation in the banner-tailed kangaroo rat, *Dipodomys spectabilis*. Ecology **69**:1466-1473.

Komdeur, J. 1992. Importance of habitat saturation and territory quality for evolution of cooperative breeding in the Seychelles warbler. Nature **358**:493-495.

Lambin, X., J. Aars, and S. B. Piertney. 2001. Dispersal, intraspecific competition, kin competition and kin facilitation: a review of the empirical evidence. Pages 110-122 *in* J. Clobert, E. Danchin, A. Dhondt, and J. D. Nichols, editors. Dispersal. Oxford University Press, Oxford.

Le Galliard, J.-F., R. Ferrière, and J. Clobert. 2003. Mother-offspring interactions affect natal dispersal in a lizard. Proceedings of the Royal Society London B.

Lecomte, J., and J. Clobert. 1996. Dispersal and connectivity in populations of the common lizard *Lacerta vivipara*: An experimental approach. Acta Oecologica **17**:585-598.

Lecomte, J., J. Clobert, and M. Massot. 1992. Sex identification in juveniles of *Lacerta vivipara*. Amphibia-Reptilia **13**:21-25.

Lecomte, J., J. Clobert, M. Massot, and R. Barbault. 1994. Spatial and behavioural consequences of a density manipulation in the common lizard. Ecoscience **1**:300-310.

Léna, J. P., F. M. De, and J. Clobert. 2000. Affinity towards maternal odour and offspring dispersal in the common lizard. Ecology Letters **3**:300-308.

Levins, R. 1969. Some demographic and genetic consequences of environmental heterogeneity for biological control. Bulletin of the Entomological Society of America **15**:237-240.

Littell, R. C., G. A. Millinken, W. W. Stroup, and R. D. Wolfinger. 1996. SAS systems for mixed models. SAS Institute, Cary, North Carolina.

Massot, M., J. Clobert, J. Lecomte, and R. Barbault. 1994. Incumbent advantage in common lizards and their colonizing ability. Journal of Animal Ecology **63**:431-440.

Massot, M., J. Clobert, P. Lorenzon, and J.-M. Rossi. 2002. Condition-dependent dispersal and ontogeny of the dispersal behaviour: an experimental approach. Journal of Animal Ecology **71**:253-261.

Massot, M., J. Clobert, T. Pilorge, J. Lecomte, and R. Barbault. 1992. Density dependence in the common lizard: demographic consequences of a density manipulation. Ecology **73**:1742-1756.

Meadows, P. S., and J. I. Campbell. 1972. Habitat selection by aquatic invertebrates? Pages 271-382 *in* S. S. Russell, R. L. Chazdon, and A. P. Smith, editors. Advances in marine biology. Academic Press, London.

Nichols, J. D., J. R. Sauer, K. H. Pollock, and J. B. Hestbeck. 1992. Estimating transition probabilities for stage-based population projection matrices using capture-recapture data. Ecology **73**:306-312.

Olivieri, I., Y. Michalakis, and P.-H. Gouyon. 1995. Metapopulation genetics and the evolution of dispersal. The American Naturalist **146**:202-228.

Olsson, M., and R. Shine. 2002. Growth to death in a lizard. Evolution **56**:1867-1870.

Otis, D. L., K. P. Burnham, G. C. White, and D. R. Anderson. 1978. Statistical inference from capture data on closed animal populations. Wildlife Monographs:1-135.

Perrin, N., and J. Goudet. 2001. Inbreeding, kinship, and the evolution of natal dispersal. Pages 123-142 *in* J. Clobert, J. D. Nichols, E. Danchin, and A. A. Dhondt, editors. Dispersal. Oxford University Press, Oxford.

Ronce, O., J. Clobert, and M. Massot. 1998. Natal dispersal and senescence. Proceedings of the National Academy of Sciences USA **95**:600-605.

Sinervo, B. 1999. Mechanistic analysis of natural selection and a refinement of Lack's and William's principles. The American Naturalist **154**:S26-S42.

Smith, A. T., and M. M. Peacock. 1990. Conspecific attraction and the determination of metapopulation colonization rates. Conservation Biology **4**:320-323.

Sorci, G., and J. Clobert. 1999. Natural selection on hatchling body size and mass in two environments in the common lizard (*Lacerta vivipara*). Evolutionary Ecology Research **1**:303-316.

Sorci, G., J. Clobert, and Y. Michalakis. 1996. Cost of reproduction and cost of parasitism in the common lizard, *Lacerta vivipara*. Oikos **76**:121-130.

South, A. B., and R. E. Kenward. 2001. Mate finding, dispersal distances and population growth in invading species: a spatially explicit model. Oikos **95**:53-58.

Stamps, J. A. 1987. Conspecifics as cues to territory quality: a preference of juveniles lizards (*Anolis aeneus*) for previously used territories. The American Naturalist **129**:629-642.

Stamps, J. A. 1988. Conspecifics attraction and aggregation in territorial lizards. The American Naturalist **131**:329-347.

Stamps, J. A. 1991. The effect of conspecifics on habitat selection in territorial species. Behavioral Ecology and Sociobiology **28**:29-36.

Stamps, J. A. 2001. Habitat selection by dispersers: integrating proximate and ultimate approaches. Pages 230-242 *in* J. Clobert, E. Danchin, A. Dhondt, and J. Nichols, editors. Dispersal. Cambridge University Press, Cambridge.

Stephens, P. A., and W. J. Sutherland. 1999. Consequences of the Allee effect for behaviour, ecology, and conservation. Trends in Ecology and Evolution **14**:401-405.

Surget-Groba, Y., B. Heulin, C.-P. Guillaume, R. S. Thorpe, L. Kupriyanova, N. Vogrin, R. Maslak, S. Mazzotti, M. Venczel, I. Ghira, G. Odierna, O. Leontyeva, J. C. Monney, and N. Smith. 2001. Intraspecific phylogeography of *Lacerta vivipara* and the evolution of viviparity. Molecular Phylogenetics and Evolution **18**:449-459.

Swingland, I. R. 1983. Intraspecific differences in movement. Pages 102-115 *in* I. R. Swingland and P. J. Greewood, editors. The ecology of animal movement. Clarendon Press, Oxford.

Valone, T. J. 1989. Group foraging, public information, and patch estimation. Oikos **56**:357-363.

Veit, R. R., and M. A. Lewis. 1996. Dispersal, population growth, and the Allee Effect: Dynamics of the House Finch Invasion of eastern North America. The American Naturalist **148**:255-274.

CHAPITRE 6 – SEXE RATIO DE LA POPULATION ET DISPERSION

"In many species, one sex is philopatric, settling near its birthplace. The other sex tends to disperse earlier and further from the birthplace. In mammals, males are usually the dispersive sex, while in birds females are more likely to disperse. The sex that disperses appears to represent an interplay between selection for inbreeding avoidance and a tendency of the sex that gains more from resource defence to remain philopatric." A. Cockburn, S. Legge, & M. C. Double in *Sex ratios: concept and research methods*. 2002.

EFFETS DU SEXE RATIO ADULTE DE LA POPULATION SUR LA DISPERSION DU LEZARD VIVIPARE

Jean-François Le Galliard
Projet en collaboration avec Patrick Fitze, Jean Clobert & Régis Ferrière

RESUME DE L'ETUDE

Chez les espèces polygynes, Greenwood (1980, 1983) a prédit que les mâles dispersent pour éviter la compétition locale pour les partenaires, alors que les femelles sont en compétition pour les ressources et bénéficient de la philopatrie. Dans cette étude, nous testons si la compétition intrasexuelle pour les ressources et les partenaires affecte la dispersion chez un lézard à reproduction polygynandrique. Nous avons manipulé la sexe ratio adulte de la population chez le lézard vivipare (*Lacerta vivipara*) pour créer des populations biaisées vers les mâles, créant les conditions d'une forte compétition intrasexuelle chez les mâles et d'une faible compétition intrasexuelle chez les femelles, et des populations biaisées vers les femelles. Nous avons mesuré la dispersion dans trois classes d'âge (juvéniles, sub-adultes, et adultes) pendant la récupération de l'effort reproducteur (été), et pendant l'investissement dans les appariements et la gestation (printemps). Notre étude donne des résultats confirmant partiellement le scénario envisagé par Greenwood. Premièrement, le biais sexuel de la dispersion natale n'est pas affecté par la sexe ratio adulte de la population. Par contre, la manipulation a induit la dispersion de jeunes lézards de différentes conditions corporelles à la naissance pendant l'été. Deuxièmement, la perturbation de la sexe ratio adulte a affecté la dispersion de reproduction des femelles pendant l'été, mais pas les mouvements de reproduction des mâles. La concordance entre l'augmentation de la dispersion des femelles adultes dans les populations biaisés en faveur du même sexe et la période de restauration des ressources après la gestation suggère que les femelles dispersent pour éviter la compétition pour les ressources plutôt que la compétition pour les partenaires.

Référence : Ces données n'ont pas été publiées, soumises à publication ou révisées par mes collaborateurs.

Mots-clés : sexe ratio, compétition pour les partenaires, compétition pour les ressources, lézard vivipare

EFFECTS OF POPULATION ADULT SEX-RATIO ON DISPERSAL IN THE COMMON LIZARD

Jean-François Le Galliard
Project in collaboration with Patrick Fitze, Jean Clobert & Régis Ferrière

STUDY ABSTRACT

In polygynous species, Greenwood (1980, 1983) predicted that males disperse to avoid mate competition, while females compete for resources. In this study, we test whether intrasexual competition for resources and mates affects dispersal patterns in a polygynandrous lizard. We manipulated adult sex-ratio in populations of the common lizard (*Lacerta vivipara*) to create male-biased population structures with strong intrasexual competition among males and low intrasexual competition among females, and female-biased population structures. We monitored dispersal across three age classes (juveniles, yearlings, and adults) during reproductive recovery (summer) and during mating and gestation (spring). Our study supports partially Greenwood's scenario. First, sex-biased natal dispersal was not affected by adult sex-ratio. Rather, the manipulation induced different offspring to disperse during summer, as shown by the influence of treatment on the relationship between offspring body condition and natal dispersal probability. Second, sex ratio manipulation affected breeding dispersal in females during summer, but not male breeding movements. The concordance between the increase of adult females dispersal in populations biased toward the same sex and the recovery period following parturition suggests that females dispersed to avoid competition for resources rather than competition for mates.

Reference : These data have not been published, submitted or reviewed by my collaborators.

Key-words : sex ratio, mate competition, resource competition, common lizard

SHORT INTRODUCTION

Sex-biased dispersal occurs in many species during natal and breeding movements (*sensu* Greenwood, 1980). This pattern is generally efficient to reduce inbreeding, because if one sex disperses, then incestuous matings between residents are less likely (Johnson and Gaines, 1990). Although inbreeding avoidance is important to explain the evolution sex-biased dispersal (e.g., Pusey, 1987; Wolff, 1992; Wolff and Plissner, 1998), several empirical and theoretical studies have suggested that sex-biased dispersal stems from the interaction between inbreeding avoidance and other causes (Dobson and Jones, 1985; Greenwood, 1980, 1983; Perrin and Goudet, 2001; Perrin and Mazalov, 2000). Indeed, the relationship between sex-biased dispersal and inbreeding depression depends systematically upon the mating system. In species with poor male investment in parental care, strong female investment in reproduction, and polygyny, such as many mammals, males usually disperse more than females. In species with symmetric investment in parental care between males and females and monogamy, such as many birds, females usually disperse more than males (Dobson, 1982; Greenwood, 1980; Moore and Ali, 1984; Wolff, 1994).

Familiarity with the local territory and competitive asymmetry between the sexes have been put forward to explain this covariation (Greenwood, 1980; Perrin and Goudet, 2001). Territory defence by males and resource defense by females result in benefits of familiarity with the local territory in males for birds and in females for mammals, respectively, hence in the philopatric sex (Greenwood, 1980). Furthermore, in polygynous species, males compete among themselves to acquire mates (local mate competition, Hamilton, 1967), while females compete among themselves to acquire resources (local resource competition, Clarke, 1978). If local resources are not limited, this asymmetry has the potential to induce male dispersal and female philopatry (Perrin and Goudet, 2001; Perrin and Mazalov, 2000). Although the covariation between sex-biased dispersal and mating systems among high-order taxa and among species substantiates the roles of familiarity and competitive asymmetry (see review in Perrin and Goudet, 2001), relatively few studies have documented the effects of intrasexual competition on sex-biased dispersal within a single species (e.g., Lambin, 1994).

In this study, we investigate the effects of intrasexual competition on natal and breeding dispersal in the common lizard (*Lacerta vivipara*), a polygynandrous species without prolonged bonding. This mating system can promote symmetrical roles between the sexes (Andersson, 1994; Kvarnemo and Ahnesjö, 1996). However, in the common lizard, natal dispersal is slightly male-biased, breeding dispersal is male-biased, males disperse during matings, and females disperse during the summer period when they acquire local resources to recover their reproductive efforts (Clobert et al., 1994; Massot, 1992). This suggests that males disperse to avoid local mate competition, while females disperse to avoid resource competition, as in the polygynous system envisioned by Greenwood (1980, 1983). Moreover, local mate competition among males can be expected because (i) males contribute only a few sperm cells to reproduction, do not guard females and do not provide parental cares (Clobert et al., 1994); (ii) males are involved in endurance and contest competition before and during matings (Lecomte, 1993; Lecomte et al., 1994); and (iii) adult males growth and survival are insensitive to the overall population density, suggesting weak resource competition among breeding males (Massot et al., 1992; Pilorge, 1987). Local resource competition among females is likely to occur because (i) females invest more time and energy in reproduction than males, due to the burden of gestation (Avery, 1975; Bauwens and Thoen, 1981; Sorci et al., 1996); (ii) females do not compete aggressively for males during the breeding season (Lecomte, 1993); and (iii) competition reduces females reproductive success (Massot et al., 1992; Pilorge, 1987). Therefore, a high proportion of adults from one sex in the population should promote natal and breeding dispersal of the same sex individuals, as a way to escape intrasexual competition.

To test the effects of intrasexual competition on dispersal, we manipulated adult sex-ratio in several experimental populations of the common lizard to create male-biased population structures, setting the conditions for strong intrasexual competition among males and low intrasexual competition among females (hence, high intersexual competition in females), or female-biased population structures. We separated our study in two periods corresponding with mating and investment in reproduction (spring), and with recovery from reproduction (summer). We hypothesised (i) that each sex should disperse to avoid intrasexual competition, (ii) that female dispersal should be associated with resource competition avoidance during the recovery period, and (iii) that male dispersal should be associated with mate competition avoidance during the mating period.

EXPERIMENTAL SYSTEM

The experimental set up consists of enclosed populations located in a natural meadow at the Ecological Research Station of Foljuif (Seine et Marne, 60 m a.s.l.). In this study, twelve experimental patches of natural habitat (10 × 10 m) were used, each patch being extended by one 20 m length corridor ending with a pitfall trap (see Fig. 1). The patch mimics a natural home range and can sustain a small local population of common lizards for several years, because the spacing system is not territorial in this species (Le Galliard et al., 2003b). Corridors allow movements of lizards out of one patch. The length of the corridor is also used to define dispersal movements in natural populations, and has been found to be efficient to characterise dispersal in several past studies (Boudjemadi et al., 1999; Le Galliard et al., 2003b; Lecomte and Clobert, 1996). More details on the experimental system can be found in Boudjemadi (1999) and in Lecomte and Clobert (1996).

MODEL SPECIES

The common lizard *Lacerta vivipara* (Jacquin 1787) is a small (50-70 mm adult snout-vent size, 15-25 mm offspring size), and viviparous species inhabiting humid habitats (e.g., peatbogs, heathlands, meadows) across Eurasia. In natural populations, three age classes can be distinguished: juveniles (year born individuals), yearlings (one year old), and adults (equal to or more than two years old, Pilorge and Castanet, 1981). Age at first reproduction is a plastic trait that depends on thermal opportunities and competition for food (Bauwens et al., 1986; Massot et al., 1992). In our experimental system, maturity can be reached before the age of one year, and all individuals are mature at two years old (Boudjemadi et al., 1999).

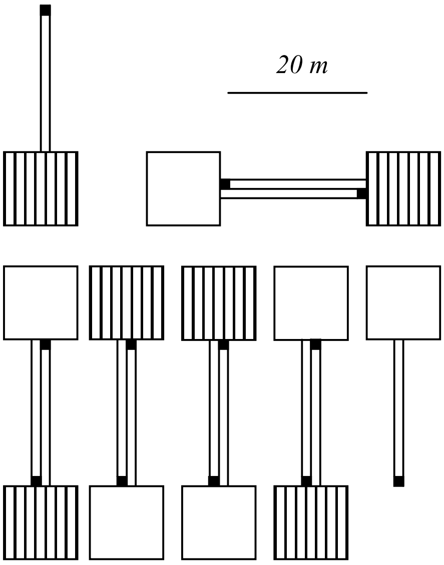

Figure 1. Experimental design and the allocation of experimental treatments from 2002 to 2003. Dashing refers to populations with a female-biased adult sex-ratio, and blank refers to populations with a male-biased adult sex-ratio. Black dotes indicate pitfall traps.

The activity season spreads from February to November (Boudjemadi, 1999). Adult and yearling individuals hibernate first (October), while juveniles delay hibernation up to the start of November. Activity begins from mid-February to early March, where most active individuals are adult males. The early emergence of adult males is linked with the establishment of a social hierarchy and with the ending of the spermatogenetic cycle (Bauwens et al., 1989; Lecomte, 1993; Olsson and Madsen, 1998). Adult females and yearlings emerge from hibernation between the end of March and the beginning of April. Matings can be observed during the month of April, and multiple matings usually occur in experimental populations. The average multipaternity level in a female clutch is around 2 fathers per clutch, a pattern concordant with observations derived from several natural populations of the same species (Laloi et al. submitted). Parturitions take place on average two months following matings, therefore from the end of June to the end of July. Females lay a clutch of 5 shell-less eggs on average (range 1-12), and offspring hatch during the same day. Offspring are autonomous at birth and the mother does not provide direct parental care. Dispersal movements usually start in a few days following birth (de Fraipont et al., 2000).

EXPERIMENTAL DESIGN

Spatial design

The experiment was conducted from July 2002 to June 2003 using twelve populations. In one half of the system, we initiated the populations with a female-biased adult sex-ratio, whereas we initiated the populations with a male-biased adult sex-ratio in the other half (Fig. 1). The spatial distribution of treatments among populations within the experimental system was chosen to optimise interspersion, especially along the edges of the system and with respect to an experiment done one year ago with the same enclosures (see chapter 5, Hurlbert, 1984).

Release protocol

Populations were initiated with juveniles, yearlings, and adults from natural populations of the Cévennes mountain range (South France, 1500 m a.s.l., see Clobert et al., 1994). Two different samples of yearlings and adults were used depending on when these individuals have been translocated at our site in the past (Le Galliard et al., 2003a, 2003b). Some lizards belonged to a cohort introduced in 2001 and acclimated during one year in 14 different populations (type A individuals, *n = 60* yearlings, *n = 96* adults). The others belonged to a cohort introduced in 1999, and acclimated during more than one year in 16 populations (type B individuals, *n = 84* yearlings, *n = 120* adults). This classification was accounted for during the experiment because translocation effects have been detected in the past (Boudjemadi, 1999). Furthermore, two different samples of juveniles were used. A first sample was obtained from gravid females maintained at the experimental site (type A and B mothers, *n = 324*). A second sample was obtained from gravid females of natural populations from the Cévennes (type C mothers, *n = 197*). All individuals were allocated to populations to which they had no prior familiarity, and juveniles were introduced in different populations than their mother to avoid the confounding effects of the presence or absence of the mother (Le Galliard et al., 2003b).

Table 1. Age and sex structure at the start of the experiment. Number of juveniles is given as mean ± SD.

Age class	Male-biased treatment (*N = 6*)		Female-biased treatment (*N = 6*)	
	Males	Females	Males	Females
Juveniles	21.3 ± 0.5	21.8 ± 0.7	21 ± 0.6	22.3 ± 1.1
Yearlings	6	6	6	6
Adults	14	4	4	14

Yearlings and adults were captured from the 18 to the 25 May, and all individuals were kept in the laboratory under similar conditions. Yearlings were isolated in individual terraria (17 × 11 × 12 cm) distributed on five shelves. Adults were isolated in larger plastic boxes (25 × 15 × 15 cm) and distributed randomly on four shelves. Food was provided once per four days, and included crickets (*Acheta domestica*) and mealworms larvae (*Pyralis sp.*) complemented with calcium (calcium Bone Aid^{TM}, T-Rex) and vitamin (Tonivit, TVM Laboratory). Basking opportunities were provided from 9h to 12h and from 14h to 17h local time. A heating ensured a thermal gradient encompassing the thermal breadth of the common lizard (van Damme et al., 1986). At parturition, offspring were isolated from their mother and kept during two days before introduction. Offspring sex was determined by the counting of ventral scales (Lecomte et al., 1992). Individuals were measured by the same person for snout-vent length (nearest mm, here called body size), tail length (nearest mm) and body mass (nearest mg). Body condition was calculated as the residual of a linear regression of body mass against body size.

The timing of introduction was dependent upon age. Twelve yearlings were introduced in each population on the 7 June, including 2 type-A males, 4 type-B males, 3 type-A females, and 3 type-B females (Table 1). A total of 42 to 44 juveniles was also introduced in each enclosure from the 15 June to the 27 July, including a half of each sex and nearly one third of type-C individuals. There was no difference between treatment in the sex or origin structure of this sample at the start of the experiment ($p > 0.90$). Eighteen adults were introduced in each population on the 11 July. In male-biased populations, we introduced 14 adult males and 4 adult females. In female-biased populations, we introduced 14 adult females and 4 adult males (Table 1). The female-biased sex-structure equals the equilibrium sex ratio of natural populations in the Cévennes mountain range, where females average 60-70 % of the adult population (Massot et al., 1992). Population structure of adults was matched between treatments for age (two years old versus more than two years old individuals, chi-square tests, $p > 0.65$) and for origin (type-A versus type-B individuals, $p > 0.49$). At the start of the experiment, body size, tail size, and body condition were similar between treatments across all age classes (ANOVA on treatment effects, all $p > 0.25$).

Individual census

Corridors were closed up to the 16 July to avoid exploratory movements of yearlings and adults following introduction (Boudjemadi, 1999). Dispersal was then monitored daily up to November 2002, and from March to June 2003 by inspecting pitfall traps at the end of each corridor. Each disperser was introduced in a new, unfamiliar enclosure randomly chosen within the same treatment during the same day. In addition, we captured lizards in August 2002 (three capture days), September 2002 (one capture day), and June 2003, where all individuals were captured. We analysed movements separately before and after hibernation. During each study period, individuals captured in pitfall traps were called *dispersers*, while individuals caught in habitat patches during demographic censuses but never caught in pitfall traps were called *residents*. Among dispersers, lizards which settled in their patch of arrival were called *immigrants*, while lizards which did not settle in their patch of arrival but disperse to another patch were called *transients*.

DATA ANALYSIS

We asked whether adult population sex-ratio affects dispersal movements during the two periods by modelling (i) dispersal probability (residency versus dispersal), (ii) settlement probability (immigration versus transience), and (iii) the timing of movements. In the latter case, we defined two types of events: the residency time of dispersers, calculated as the number of days from introduction in a patch to dispersal, and the transience time, calculated as the number of days from arrival in a patch to dispersal. Data were analysed with generalised linear mixed models (GLMM) in SAS, using patch as a random effect (Littell et al., 1996). Dispersal and settlement probabilities were modelled with the GLIMMIX macro, using a binomial error term and a logit link function (McCullagh and Nelder, 1989). When we modelled dispersal probability, we accounted for the effects of treatment, sex, as well as body length, body condition, origin and age, and all pairwise interactions. Indeed, these covariates have been found to influence natal and breeding dispersal in past studies on the common lizard (Le Galliard et al., 2003b; Léna et al., 1998; Massot and Clobert, 2000; Massot et al., 2002; Meylan et al., 2002). The chronology of dispersal was modelled with the MIXED procedure, which specifies a gaussian error distribution and an identity link function. Estimations and test statistics were obtained from restricted maximum likelihood. Statistical inferences for the fixed part of the model are given as type III F statistics for two-tailed tests. Model selection was conducted by backward simplification. Models assumptions were fulfilled in all analyses presented here.

RESULTS

Movements during the recovery period

A total of 681 individuals was captured at least once following release in our enclosed populations and 77 individuals dispersed from these patches (54 juveniles, 6 yearlings, and 17 adults). Overall, dispersal probability was dependent upon age class, and also marginally upon sex (GLMM including patch as a random effect, Age class effect: $F_{2,666} = 7.71$, $p < 0.001$; Sex effect: $F_{1,666} = 3.61$, $p = 0.06$, $n = 681$). Females were 1.59 [0.98 , 2.57] more likely to disperse than males across all age classes, a pattern somewhat surprising given the slightly male-biased dispersal in natural populations (Massot, 1992; Massot and Clobert, 1998). Juveniles were more likely to disperse than yearlings and adults across all sex classes (Tukey's contrasts, $p < 0.02$, odds juveniles : odds yearlings and adults = 3.0), while yearlings disperse as frequently as adults ($p = 0.42$).

The dependence of dispersal probability upon treatment and individual covariates was analysed separately for juveniles and adults, because yearling movements were too rare. In juveniles, the adult sex-ratio of the population did not influence sex-biased dispersal (GLMM with patch as a random effect, Sex-ratio effect: $F_{1,10} = 0.23$, $p = 0.64$; Sex effect: $F_{1,323} = 1.20$, $p = 0.27$; Sex-ratio × Sex effect: $F_{1,323} = 0.09$, $p = 0.77$, $n = 340$). Offspring males were as likely as females to disperse from male-biased than from female-biased populations (Fig. 2). The model had a significant interaction between offspring body condition at birth and population sex-ratio (Offspring condition effect: $F_{1,323} = 0.45$, $p = 0.50$; Sex-ratio × Offspring condition effect: $F_{1,323} = 4.22$, $p = 0.04$). In female-biased populations, residents were heavier at birth than dispersers (Residents: 0.0007 ± 0.001, Dispersers: -0.004 ± 0.003, $p = 0.04$). In male-biased populations, dispersers tended to be heavier than residents (Residents: -0.001 ± 0.002, Dispersers: 0.007 ± 0.003, $p = 0.45$). An additive effect of offspring origin was found, with offspring introduced directly from the mountain area (type C mothers) dispersing less than offspring born from females of the experimental site (Offspring origin effect: $F_{1,323} = 8.30$, $p = 0.004$, odds type C : odds type A-B = 0.34). In adults, the sex-dependent breeding dispersal was affected by the sex-ratio manipulation (logistic regression, Sex-ratio effect: $\chi^2 = 0.62$, $p = 0.43$; Sex effect: $\chi^2 = 2.25$, $p = 0.13$; Sex-ratio × Sex effect: $\chi^2 = 4.16$, $p = 0.04$, $n = 207$), whatever the age, origin and body size of lizards (all $p > 0.79$). In adult females, dispersal probability was higher in female-biased than in male-biased populations ($\chi^2 = 5.63$, $p = 0.02$, $n = 103$, Fig. 2). Adult males were not affected by the manipulation ($\chi^2 = 0.12$, $p = 0.73$, $n = 104$).

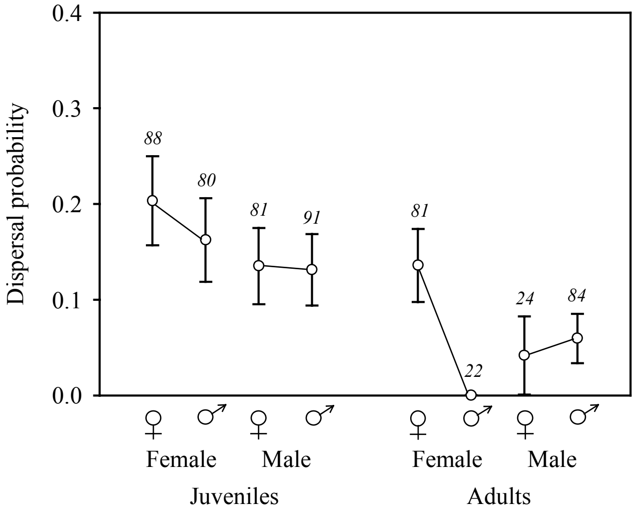

Figure 2. Effect of sex and adult sex-ratio manipulation on dispersal probability in juveniles and adults before wintering. ♀: female-biased populations, ♂: male-biased populations. Values are back-transformed from the GLMMs discussed in the text (least-square means ± s.e.). Numbers indicate sample sizes.

From the 77 dispersers, only 20 individuals did not establish before wintering in their patch of arrival (15 juveniles, 3 yearlings, and 3 adults). The overall settlement probability was not affected by age ($\chi^2 = 2.69$, $p = 0.26$), and there was only enough observations to conduct analyses in juveniles. In this age class, males and females had similar settlement probabilities across treatments ($p > 0.05$), and adult sex-ratio had a marginal effect on settlement probability ($\chi^2 = 3.33$, $p = 0.07$). The trend was for a lower settlement probability in female-biased than in male-biased populations (Female-biased: 0.52, $n = 29$; Male-biased: 0.84, $n = 25$).

To study dispersal chronology, we used the day when corridors were opened as a reference for individuals introduced prior to corridors opening, and introduction as a reference for offspring introduced after corridors opening. The time spent in the introduction patch was not influenced by the sex-ratio manipulation (ANOVA on individual observations with patch as a random effect, Age effect: $F_{2,62} = 10.24$, $p < 0.001$; Sex effect: $F_{1,62} = 1.52$, $p = 0.22$; Sex-ratio effect: $F_{1,10} = 0.62$, $p = 0.45$, $n = 77$). Juveniles and adults dispersed at the same time following introduction (Juveniles: 43.1 days ± 3.2, Adults: 33.9 days ± 5.5, $p = 0.29$), while yearlings dispersed earlier than juveniles and adults (contrast = 36.5 days ± 9.2, $p < 0.001$). The transience time within an arrival patch was not affected by the manipulation (Wilcoxon's rank test, $p = 0.86$).

Movements during the mating period

A total of 385 individuals was captured at the end of the experiment and 27 individuals dispersed after hibernation (12 juveniles, 6 yearlings, and 9 adults). Overall, dispersal probability was not affected by age or sex (GLMM including patch as a random effect, Age class effect: $F_{2,370} = 0.22$, $p = 0.81$; Sex effect: $F_{1,370} = 1.59$, $p = 0.21$, $n = 385$). The dependence of dispersal probability on treatments was analysed for juveniles on one side and for adults and yearlings on the other side.

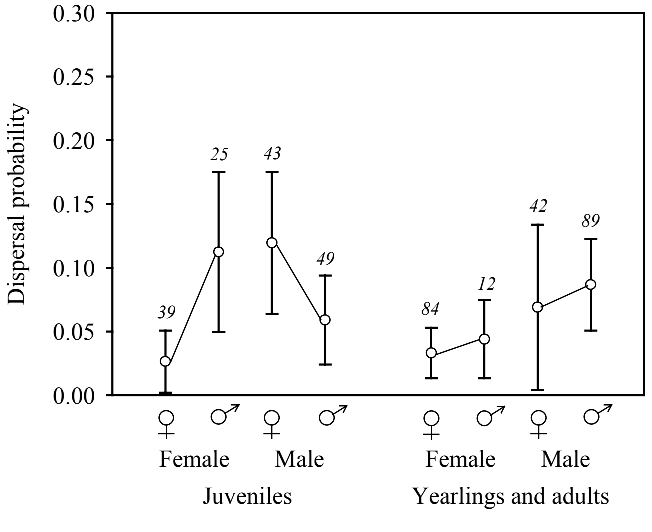

Figure 3. Effect of sex and adult sex-ratio manipulation on dispersal probability in juveniles and in adults and yearlings after wintering. ♀: female-biased populations, ♂: male-biased populations. Values are back-transformed from the GLMMs discussed in the text (least-square means ± s.e.). Numbers indicate sample sizes.

In juveniles, the adult sex-ratio of the population tended to influence sex-biased dispersal (GLMM with patch as a random effect, Sex-ratio effect: $F_{1,10} = 0.25$, $p = 0.63$; Sex effect: $F_{1,142} = 0.47$, $p = 0.27$; Sex-ratio × Sex effect: $F_{1,142} = 3.39$, $p = 0.07$, $n = 156$). Contrary to our expectations, male juveniles tended to disperse more from female-biased populations, while female juveniles tended to disperse more from male-biased populations (Fig. 3). The sex-dependent breeding dispersal of yearlings and adults was not affected by the sex-ratio manipulation (GLMM with patch as random effect, Sex-ratio effect: $F_{1,10} = 0.88$, $p = 0.37$; Sex effect: $F_{1,215} = 0.17$, $p = 0.68$; Sex-ratio × Sex effect: $F_{1,215} = 0.00$, $p = 0.97$, $n = 229$, Fig. 3).

The timing of these dispersal events during spring varied with age and population sex-ratio (ANOVA on individual observations with patch as a random effect, Age effect: $F_{1,15} = 22.39$, $p < 0.001$; Sex-ratio effect: $F_{1,10} = 5.19$, $p = 0.05$, $n = 27$). Yearlings and adults dispersed earlier than juveniles (contrast = 23 ± 5.1 days), and all age classes tended to disperse earlier from female-biased than from male-biased populations (contrast = 11.3 ± 5.13 days).

SHORT CONCLUSION

Our study supports partially Greenwood's scenario for the evolution of sex-biased dispersal. First, sex-biased natal dispersal was found to be independent of intrasexual competition avoidance before and after wintering. Rather, we observed that sex-ratio manipulation induced different offspring to disperse, as shown by the fact that treatment affected the relationship between offspring body condition and natal dispersal probability, and that males (resp. females) tended to disperse more from female-biased (resp. male-biased) populations during the mating period. Second, sex ratio manipulation affected breeding dispersal in females, but not in males. The concordance between the response of adult females to the sex ratio manipulation and the recovery period following parturition further suggests that females dispersed to avoid local competition for resources rather than local competition for mates. This suggestion deserves complementary experiments where both factors could be disentangled.

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REFERENCES

Andersson M, 1994. Sexual selection. Princeton: Princeton University Press.

Avery RA, 1975. Clutch size and reproductive effort in the lizard *Lacerta vivipara* Jacquin. Oecologia 19:165-170.

Bauwens D, Heulin B, Pilorge T, 1986. Variation spatio-temporelle des caractéristiques démographiques dans et entre populations du lézard *Lacerta vivipara*. In: Biologie des populations (Legay J-M, ed). Université Claude Bernard Lyon I: CNRS; 531-536.

Bauwens D, Thoen C, 1981. Escape tactics and vulnerability to predation associated with reproduction in the lizard *Lacerta vivipara*. Journal of Animal Ecology 50:733-743.

Bauwens D, van Damme R, Verheyen RF, 1989. Synchronization of spring molting behaviour with the onset of mating behavior in male lizards, *Lacerta vivipara*. Journal of Herpetology 23:89-91.

Boudjemadi K, 1999. Influence de la connexion entre populations sur leur viabilité: une approche expérimentale de la fragmentation des habitats. Paris: Paris VI.

Boudjemadi K, Lecomte J, Clobert J, 1999. Influence of connectivity on demography and dispersal in two contrasting habitats: an experimental approach. Journal of Animal Ecology 68:1207-1224.

Clarke AB, 1978. Sex ratio and local resource competition in a promisian primate. Science 201:163-165.

Clobert J, Massot M, Lecomte J, Sorci G, de Fraipont M, Barbault R, 1994. Determinants of dispersal behavior: the common lizard as a case study. In: Lizard ecology. Historical and experimental perspectives. (Vitt LJ, Pianka ER, eds). Princeton: Princeton University Press; 183-206.

de Fraipont M, Clobert J, John AH, Meylan S, 2000. Increased pre-natal maternal corticosterone promotes philopatry of offspring in common lizards *Lacerta vivipara*. Journal of Animal Ecology 69:404-413.

Dobson FS, 1982. Competition for mates and predominant juvenile male dispersal in mammals. Animal Behaviour 30:1183-1192.

Dobson FS, Jones WT, 1985. Multiple causes of dispersal. The American Naturalist 126:855-858.

Greenwood PJ, 1980. Mating systems, philopatry and dispersal in birds and mammals. Animal Behaviour 28:1140-1162.

Greenwood PJ, 1983. Mating systems and evolutionary consequences of dispersal. In: The ecology of animal movement (Swingland IR, Greenwood PJ, eds). Oxford: Clarendon Press; 116-131.

Hamilton WD, 1967. Extraordinary sex ratios. Science 156:477-488.

Hurlbert SH, 1984. Pseudoreplication and the design of ecological field experiments. Ecology 54:187-211.

Johnson ML, Gaines MS, 1990. Evolution of dispersal: theoretical models and empirical tests using birds and mammals. Annual Review of Ecology and Systematics 21:449-480.

Kvarnemo C, Ahnesjö I, 1996. The dynamics of operational sex ratios and competition for mates. Trends in Ecology and Evolution 11.

Lambin X, 1994. Natal philopatry, competition for resources, and inbreeding avoidance in Towsend's voles (*Microtus townsendii*). Ecology 75:224-235.

Le Galliard J-F, Ferrière R, Clobert J, 2003a. Immigration within metapopulations: behavioral and demographic consequences in the common lizard. Submitted to Ecology.

Le Galliard J-F, Ferrière R, Clobert J, 2003b. Mother-offspring interactions affect natal dispersal in a lizard. Proceedings of the Royal Society London B.

Lecomte J, 1993. Rôle du comportement dans l'organisation et la régulation des populations de lézards vivipares. Paris: Paris XI.

Lecomte J, Clobert J, 1996. Dispersal and connectivity in populations of the common lizard *Lacerta vivipara*: An experimental approach. Acta Oecologica 17:585-598.

Lecomte J, Clobert J, Massot M, 1992. Sex identification in juveniles of *Lacerta vivipara*. Amphibia-Reptilia 13:21-25.

Lecomte J, Clobert J, Massot M, Barbault R, 1994. Spatial and behavioural consequences of a density manipulation in the common lizard. Ecoscience 1:300-310.

Léna JP, Clobert J, de Fraipont M, Lecomte J, Guyot G, 1998. The relative influence of density and kinship on dispersal in the common lizard. Behavioral Ecology 9:500-507.

Littell RC, Millinken GA, Stroup WW, Wolfinger RD, 1996. SAS systems for mixed models. Cary, North Carolina: SAS Institute.

Massot M, 1992. Movement patterns of the common lizard (*Lacerta vivipara*) in relation to sex and age. In: Proceedings of the Sixth Ordinary General Meeting of the Societas Europea Herpetologica (Korsos Z, Kiss I, eds). Budapest: Hungary Natural History Museum; 315-319.

Massot M, Clobert J, 1998. Similarities in dispersal behaviour among siblings in the common lizard (*Lacerta vivipara*). In: Proceedings of the Ordinary General Meeting of the Societas Europea Herpetologica (Miaud C, Guyétant R, eds). Le Bourget du Lac / France; 315-323.

Massot M, Clobert J, 2000. Processes at the origin of similarities in dispersal behaviour among siblings. Journal of Evolutionary Biology 13:707-719.

Massot M, Clobert J, Lorenzon P, Rossi J-M, 2002. Condition-dependent dispersal and ontogeny of the dispersal behaviour: an experimental approach. Journal of Animal Ecology 71:253-261.

Massot M, Clobert J, Pilorge T, Lecomte J, Barbault R, 1992. Density dependence in the common lizard: demographic consequences of a density manipulation. Ecology 73:1742-1756.

McCullagh P, Nelder JA, 1989. Generalized linear models, second ed. London: Chapman & Hall.

Meylan S, Belliure J, Clobert J, de Fraipont M, 2002. Stress and body condition as prenatal and postnatal determinants of dispersal in the common lizard (*Lacerta vivipara*). Hormones and Behavior 42.

Moore J, Ali R, 1984. Are dispersal and inbreeding avoidance related? Animal Behaviour 32:94-112.

Olsson M, Madsen T, 1998. Sexual selection and sperm competition in reptiles. In: Sperm competition and sexual selection (Birkhead TR, Møller AP, eds). London: Academic Press; 503-577.

Perrin N, Goudet J, 2001. Inbreeding, kinship, and the evolution of natal dispersal. In: Dispersal (Clobert J, Nichols JD, Danchin E, Dhondt AA, eds). Oxford: Oxford University Press; 123-142.

Perrin N, Mazalov V, 2000. Local competition, inbreeding, and the evolution of sex-biased dispersal. The American Naturalist 155:116-127.

Pilorge T, 1987. Density, size structure, and reproductive characteristics of three populations of *Lacerta vivipara* (Sauria: Lacertidae). Herpetologica 43:345-356.

Pilorge T, Castanet J, 1981. Détermination de l'âge dans une population naturelle du Lézard vivipare (*Lacerta vivipara* Jacquin 1787). Acta Oecologia 2:3-16.

Pusey AE, 1987. Sex-biased dispersal and inbreeding in birds and mammals. Trends in Ecology and Evolution 10:295-299.

Sorci G, Clobert J, Michalakis Y, 1996. Cost of reproduction and cost of parasitism in the common lizard, *Lacerta vivipara*. Oikos 76:121-130.

van Damme R, Bauwens D, Verheyen RF, 1986. Selected body temperatures in the lizard *Lacerta vivipara*: variation within and between populations. Journal of Thermal Biology 11:219-222.

Wolff JO, 1992. Parents suppress reproduction and stimulated dispersal in opposite-sex juveniles white-footed mice. Nature 359:409-410.

Wolff JO, 1994. More on juvenile dispersal in mammals. Oikos 71:349-352.

Wolff JO, Plissner JH, 1998. Sex biases in avian natal dispersal: an extension of the mammalian model. Oikos 83:327-330.

DISCUSSION GENERALE

HETEROGENEITE SPATIALE

HETEROGENEITE LOCALE ET REGIONALE

L'hétérogénéité spatiale résulte de la distribution discrète des individus et des habitats de l'espèce. A l'échelle locale, la limitation des capacités de mouvement, de reproduction et d'interaction sociale génère une forme d'agrégation spatiale (chapitres 1-3). Cette agrégation spatiale a des effets perceptibles sur la dynamique écologique d'une espèce sociale. A l'échelle régionale, l'hétérogénéité spatiale est imposée par l'environnement, même si ses propriétés peuvent être dynamiques.

Notre travail montre que la compétition générée par l'hétérogénéité locale de la population n'empêche pas l'évolution d'un altruisme de reproduction fort. Ce résultat reste valide indépendamment de la structure géométrique du réseau : l'altruisme évolue plus ou moins spontanément dans un réseau structuré dans l'espace comme dans un réseau aléatoire. En fait, une géométrie régulière semble favoriser l'émergence de phénotypes plus altruistes (Figure 1). Ces différences posent la question de l'évolution conjointe du voisinage. Un modèle récent qui considère l'évolution de la distance d'interaction sociale et de la distance de dispersion sur un réseau fournit un premier élément de réponse. L'altruisme peut alors coexister avec l'égoïsme parce que les altruistes maintiennent des liens sociaux à courte distance qui les séparent dans l'espace des égoïstes qui évoluent des liens sociaux à longue distance (Koella 2000). Cependant, ce modèle repose sur des conditions restrictives d'itération du cycle de vie et sur des structures spatiales à grande échelle qui peuvent être rapidement détruites par la variabilité démographique. Lorsque l'hétérogénéité de la population est plus locale, un polymorphisme peut émerger par un processus continu de mutation-sélection autour du foyer de la dynamique adaptative des deux traits pour peu que les mutations soient orientées le long d'une corrélation génétique négative (voir Figure 5 du chapitre 3). Ce branchement évolutif amène à la coexistence entre une espèce sociale sédentaire et une espèce asociale mobile par branchement évolutif (*sensu* Geritz et al. 1998) comme prévu par van Baalen et Rand (1998).

Au contraire, **la compétition générée par la structuration régionale de la population constitue un frein à l'évolution de la socialité** que la dispersion soit globale (modèles en îles) ou locale (modèles en stepping-stone, Queller 1992; Taylor 1992a; West et al. 2002). Cependant, pour une population qui prendrait la forme d'une grille de sites voisins comme dans notre réseau structuré, Wilson et al. (1992) ont aussi observé une incapacité des altruistes à prendre le dessus sur les égoïstes. Il est donc évident que notre résultat ne traduit pas uniquement des différences d'échelle spatiale de l'hétérogénéité de la population (locale *versus* régionale). Nous discuterons plus loin plusieurs mécanismes qui peuvent contribuer à diminuer la compétition entre apparentés et expliquer l'évolution de la socialité dans notre modèle.

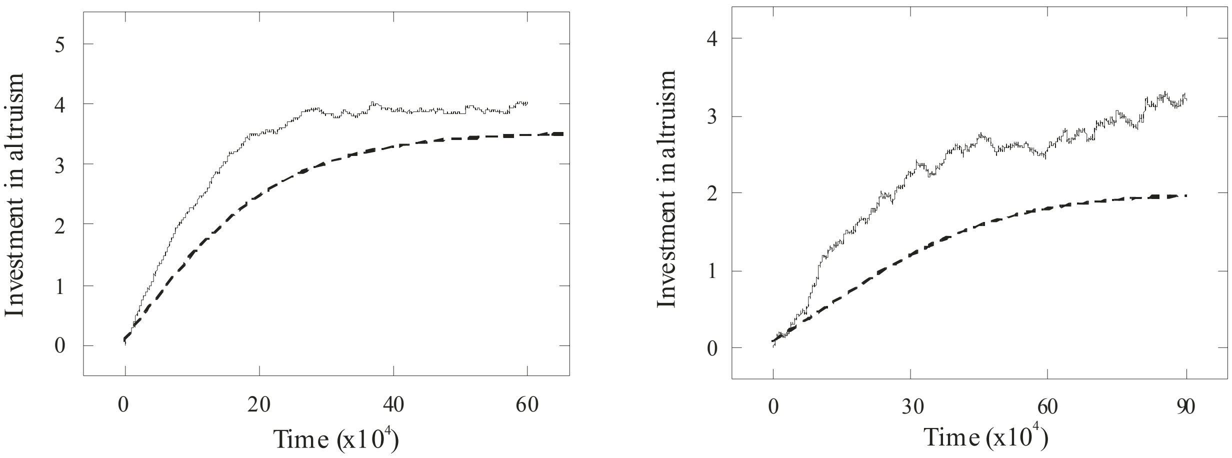

Figure 1. Evolution de la coopération dans un réseau structurée dans l'espace où les liens sont tirés entre les plus proches voisins selon une structure carrée et non aléatoire. Coûts de type accélérant selon les mêmes paramètres de simulation que la Figure 7F du chapitre 2 pour *m=0* à gauche et *m=6* à droite. L'altruisme évolue à des niveaux sensiblement plus élevés que dans un réseau aléatoire dans ce cas, comme pour d'autres combinaisons de paramètres.

DE L'HETEROGENEITE LOCALE AUX PRESSIONS DE SELECTION

Il n'est pas évident de prédire à priori l'effet de l'hétérogénéité locale sur les dynamiques adaptatives de l'altruisme et de la mobilité, et d'expliquer ces dynamiques à l'aide des pressions de sélection (Ferrière et Le Galliard 2001). **D'une part, l'hétérogénéité locale induit à la fois des éléments de parenté (les interactions coopératrices et compétitives se font préférentiellement entre individus apparentés) et des éléments de variabilité (la variabilité spatiale et temporelle émerge de la stochasticité des évènements démographiques)**. Notre approche se situe donc à la frontière entre les modèles de sélection de parentèle, qui ignorent en général la dynamique écologique de la population, et les modèles de dynamique adaptative, qui étudient les conséquences de la structuration spatiale et démographique de la population en supposant un apparentement nul (chapitre 1). **D'autre part, la description de l'invasion de la population mutante dans une population structurée dans l'espace pose plusieurs problèmes techniques** : le mutant est globalement rare mais localement abondant et la structure spatiale de la population mutante varie lors de la dynamique d'invasion (Ferrière et Michod 1996; Rousset et Billiard 2000). En utilisant l'*approximation des paires* du processus d'invasion décrite par van Baalen (2000), nous sommes arrivés à formuler une expression analytique de la valeur sélective qui clarifie les pressions de sélection agissant sur l'altruisme et la mobilité. Cette approximation est basée sur l'hypothèse de la mise en place d'une structure transitoire d'invasion lors de la phase initiale de rareté du mutant, en considérant que la population du mutant est distribuée de façon homogène sur le réseau (Ferrière et Le Galliard 2001). Les nombreuses comparaisons que nous avons menées entre les dynamiques adaptatives et des simulations numériques suggèrent que la distribution réellement locale du mutant induit peu de changements qualitatifs et favorise quantitativement l'altruisme (voir chapitres 1-3). La structure locale de la population mutante provoque vraisemblablement une plus forte agrégation des mutants que sous l'hypothèse de l'approximation des paires (Ellner et al. 1998). Remarquons cependant que ces approximations sont sensibles à la structure géométrique du réseau et au cycle de vie considéré (Morris 1997) : notre relatif succès n'est donc pas généralisable. Dans l'attente de nouveaux résultats analytiques, on serait donc tenter de recommander de toujours combiner l'approche analytique à une série de simulations.

Une fois considéré le processus d'invasion, la valeur sélective se dérive comme le taux de croissance exponentielle de la population mutante. **Le gradient de sélection sur l'altruisme permet d'obtenir une règle marginale de Hamilton du type** $\partial B \times r > \partial C$ où ∂B indique le gain marginal indirect et ∂C le coût marginal direct (voir chapitre 2). **La mesure correcte de l'apparentement est alors la probabilité qu'un voisin d'un mutant soit lui-même un mutant :** $r = \overline{q}_{y|y}$. Cet apparentement est mesuré dans une phase transitoire de l'invasion où le mutant est globalement rare et établi à un pseudo-équilibre spatial. La pression de sélection liée à la diminution des sites vides dans le voisinage d'un mutant plus altruiste s'oppose à l'évolution de la coopération, mais elle quantitativement négligeable (chapitre 2). L'évolution de la coopération passe alors par une forme de sélection de parentèle impliquant la proximité spatiale des mutants. **Le gradient de sélection sur la mobilité implique une première pression de sélection pour ouvrir de l'espace dans le voisinage du mutant, modulé par un terme impliquant la saturation de l'habitat et les traits d'histoire de vie de la population résident, et une deuxième pression de sélection pour réduire les coûts de la mobilité**. L'évolution de la mobilité est donc motivée par une diminution de l'intensité de la compétition locale pour l'espace entre mutants. C'est donc *a priori* la compétition pour l'espace entre mutants qui est diminuée par l'évolution de plus forts taux de mobilité, ce qui implique que la mobilité évolue en partie par une forme de sélection de parentèle. Cependant, toutes choses étant égales par ailleurs, une plus forte saturation de l'habitat de la population résidente augmente cette pression de sélection, même si cette saturation de l'habitat n'agit pas en soit comme un moteur de l'évolution de la mobilité. De plus, la saturation de l'habitat de la population résident constitue le filtre principal des effets des traits d'histoire de vie, de la structure de l'habitat et du phénotype altruiste sur l'évolution de la mobilité. Remarquons cependant que, du fait que la pression de compétition locale ne puisse s'écrire sous une forme compacte, nous nous sommes limités à une analyse numérique des différents termes du gradient de sélection et nos explications restent qualitatives (voir chapitre 3). Cette analyse démontre de la difficulté à mettre en évidence les pressions de sélection impliqués dans l'évolution de la dispersion dans un environnement hétérogène et variable (voir aussi Cadet et al. 2003).

INTERACTIONS SOCIALES ENTRE APPARENTES

Les structures sociales sont menacées par un risque accru de compétition locale, et ce risque peut annuler les bénéfices à la coopération locale entre apparentés. Pourtant, de très nombreuses sociétés coopératrices existent sous la forme de populations structurées où les risques de compétition entre proches génétiques sont élevés (Annexe 1). Face à ce dilemme, deux solutions sont possibles. Pour certains auteurs, la coopération émerge de bénéfices directs liés à la réciprocité, à l'acquisition d'un territoire, à l'apprentissage ou aux avantages de la vie en groupe plutôt que de bénéfices génétiques indirects. En ce sens, la synthèse de Clutton-Brock (2002) sous-entend que les comportements sociaux reflètent un *altruisme faible*. Une telle forme d'altruisme peut émerger dans un contexte de compétition entre apparentés. Charge alors aux empiristes de nous fournir des mesures plus précises des coûts et des bénéfices de l'altruisme, une tâche qui peut s'avérer bien compliquée (Annexe 1). Pour d'autres auteurs, à la suite des synthèses de Queller (1992, 1994), il faut rechercher des mécanismes permettant de découpler la compétition et la coopération entre apparentés. Je vais donc discuter ce point dans les paragraphes qui suivent.

LA STRUCTURE DU CYCLE DE VIE

Les modèles de sélection de parentèle supposent un cycle de vie itéropare (un seul évènement de reproduction) et une alternance entre stades du type « interactions sociales, reproduction, dispersion, puis compétition ». Les individus interagissent socialement, ce qui se traduit par des différences de reproduction annuelle. Après la reproduction, tous les adultes meurent et laissent la place aux juvéniles qui dispersent globalement ou localement entre les habitats. La compétition a lieu entre les juvéniles pour les places disponibles. Un bon exemple de ce cycle de vie est décrit dans les modèles en îles ou en stepping-stone de Taylor (1992a, 1992b).

Une première altération du cycle de vie consiste à **modifier la position respective des évènements de dispersion et de reproduction** (Perrin et Lehmann 2001). Dans un cycle de vie où la dispersion a lieu avant la reproduction, la compétition locale entre les jeunes s'effectue dans un voisinage plus large que la coopération, et l'altruisme devrait évoluer. Un cas possible est celui d'un insecte à développement larvaire et à cycle de vie annuel (Perrin et Lehmann 2001) : la coopération a lieu entre les larves au sein d'une famille, et les adultes dispersent après la métamorphose avant de se reproduire. Une situation similaire a été analysée par Kelly (1994) pour un altruisme affectant la survie des individus.

Une deuxième modification consiste à **autoriser la survie et plusieurs évènements de reproduction après le premier, ou itéroparité** (Taylor et Irwin 2000). L'itéroparité augmente l'apparentement local entre adultes, alors que la dispersion contrôle le niveau de compétition entre apparentés. L'altruisme peut alors évoluer sous certaines conditions, et une augmentation de la survie adulte favorise la coopération (Irwin et Taylor 2001, Taylor et Irwin 2000). Le cas d'un cycle de vie itéropare permet par ailleurs d'envisager deux contextes démographiques : *l'altruisme de reproduction*, qui affecte la natalité, et *l'altruisme de survie*, qui affecte la mortalité. Les résultats de Irwin et Taylor (2000, 2001) démontrent que l'altruisme de reproduction évolue sous des conditions moins restreintes que l'altruisme de survie dans des populations fragmentées (voir aussi Nakamaru et al. 1997, 1998; van Baalen et Rand 1998). L'interprétation fournie par ces auteurs est que l'altruisme de reproduction s'exporte par la dispersion des juvéniles, alors que l'altruisme de survie augmente la compétition locale si les adultes ne dispersent pas. Le contraste évolutif entre altruisme de reproduction et altruisme de survie devrait donc être moins marquée si les adultes dispersent.

Dans notre cas, la **dynamique adaptative de l'altruisme est affectée par les conséquences démographiques des bénéfices et des coûts de l'altruisme** (chapitre 2). Des analyses préliminaires comparant un altruisme de reproduction à un altruisme de survie montrent que (i) la pression de sélection pour limiter la compétition locale entre altruistes n'est plus négligeable quand les bénéfices et les coûts de l'altruisme affectent la mortalité plutôt que la natalité, et que (ii) l'altruisme de survie évolue plus difficilement que l'altruisme de reproduction. La structure du réseau explique en partie ces résultats. En augmentant la natalité d'un voisin, on favorise la production de jeunes en dehors de son propre voisinage (pour peu que le réseau soit aléatoire). Au contraire, en diminuant la mortalité de ses congénères, on baisse l'intensité du flux démographique de son voisinage et l'évolution de l'altruisme de survie se heurte à l'effet négatif de la compétition locale (West et al. 2002). Il est intéressant de constater que l'altruisme de reproduction semble plus répandue que l'altruisme de survie, en accord avec les prédictions de tous ces modèles (Annexe 1).

LA STRUCTURE DE L'HABITAT

Dans une population structurée en îles, la compétition entre apparentés a lieu à la même échelle spatiale que la coopération. **Une façon de limiter les chevauchements entre la compétition et la coopération consiste alors à séparer les échelles spatiales de la coopération et de la compétition**. Une première alternative est d'élargir l'échelle spatiale de la compétition. On pourrait ainsi envisager une régulation de la densité de la population à l'échelle de plusieurs groupes de coopération (Kelly 1994). Par exemple, la coopération a lieu entre plusieurs membres de la même famille (des larves d'insectes qui se défendent mutuellement) et la compétition a lieu entre ces familles sur la même plante. Certains travaux montrent que ceci revient à diminuer l'effet délétère de la compétition entre les apparentés sur l'évolution de la coopération (Kelly 1994; Queller 1994 ; West et al. 2002). **Une seconde alternative repose sur le principe que « les voisins de mes voisins ne sont pas forcément mes voisins »**. Dans un réseau social, les voisinages de compétition et d'interaction se trouvent ainsi dissociés par la géométrie de l'habitat (chapitre 2, van Baalen 2000).

L'ELASTICITE DE L'ENVIRONNEMENT

La dynamique de population des modèles de sélection de parentèle conduit à une saturation de l'habitat qui annihile toute stochasticité, sauf via la dérive génétique (e.g., Frank 1998; Taylor et Frank 1996). Dans les populations naturellement fragmentées, la petite taille des populations se traduit en réalité par une variabilité démographique locale dans la composition et l'effectif de la population, et par une variabilité régionale dans l'occupation des habitats (voir chapitre 5 et Annexe 3). **Il y a ici un paradoxe à vouloir envisager la structuration génétique locale engendrée par la fragmentation de l'habitat en ignorant les conséquences démographiques de cette fragmentation**.

En terme sélectif, la saturation de l'habitat conduit à une *inélasticité* de l'environnement : les populations altruistes sont localement plus productives et elles sont donc défavorisées par une compétition locale plus forte pour un même espace disponible (e.g., Kelly 1994). Plusieurs auteurs ont suggéré **des formes d'élasticité qui permettraient à l'environnement de s'étendre pour accommoder le surplus de descendants produits par un groupe plus altruiste** (Taylor 1992b). C'est le cas dans une population en croissance exponentielle, même si cette situation ne peut pas éternellement perdurer du fait de la régulation locale par la densité. De plus, la possibilité que l'altruisme puisse évoluer parce que les groupes altruistes sont plus aptes à fonder et développer des populations pourrait être compromise par des effets de Allee. On pourrait donc s'attendre à des interactions entre la coopération locale, la compétition locale et les mécanismes démographiques. Une deuxième possibilité consiste à homogénéiser les chances d'établissement des jeunes entre les différentes populations. Kelly (1992) a ainsi construit un modèle de dispersion complète où le regroupement local alterne avec une phase de mélange complet, mais ce modèle détruit l'apparentement local (voir aussi Goodnight 1992). Finalement, **l'habitat peut s'ouvrir spontanément sous l'effet de la stochasticité démographique, de la stochasticité environnementale, ou de catastrophes locales** (Ferrière et Le Galliard 2001). La capacité de l'altruisme à exploiter ces ouvertures dépendra de l'échelle de la dispersion et de la compétition, et de l'impact d'un changement adaptatif de l'altruisme sur ce processus stochastique. Dans notre modèle, l'ouverture de l'habitat est facilitée par la mortalité individuelle (chapitres 1-2). Si l'altruisme affecte uniquement la natalité, alors l'évolution de l'altruisme n'a pas d'effet sur ce processus et la capacité d'exportation de l'altruisme se maintient au cours de l'accroissement adaptatif de l'investissement altruiste. Au contraire, si l'altruisme affecte la mortalité, alors une augmentation de l'investissement altruiste agit directement contre ce processus. La stochasticité environnementale peut alors devenir nécessaire à l'évolution de la coopération (Mittledorf et Wilson 2001). **L'effet de la stochasticité sur la diminution de la compétition locale et l'évolution de la coopération dépendrait donc du type de stochasticité et des mécanismes démographiques de la coopération**. Remarquons toutefois que cette démarcation nette entre altruisme de reproduction et de survie repose sur l'hypothèse forte de l'absence d'un trade-off entre natalité et mortalité (Roff 1992). Par ailleurs, il serait intéressant de tester ces hypothèses pour des populations dérivées du modèle en îles de la sélection de parentèle, par exemple en utilisant un cadre théorique récemment développé pour mesurer la valeur sélective dans des métapopulations structurées (Metz et Gyllenberg 2001).

LA FLEXIBILITE DU COMPORTEMENT

Un autre découplage de la sphère de coopération et de compétition implique des processus comportementaux par lesquels un individu choisit son partenaire de coopération ou de compétition sur la base d'un critère de ressemblance corrélé avec la proximité génétique (e.g., Komdeur et Hatchwell 1999). Par exemple, une discrimination du partenaire social peut permettre de restreindre la coopération entre les résident du même fragment d'habitat : le voisinage de compétition reste inchangé alors que le voisinage de coopération devient plus court (Perrin et Lehmann 2001, Annexe 1). On ignore cependant dans quelle mesure la discrimination des proches génétique a évolué conjointement à la structuration sociale de l'espèce, et si la discrimination est un préalable à l'évolution des organisations sociales.

Un évitement des proches génétiques pourrait aussi être impliquée dans les phénomènes de dispersion ou de choix de l'habitat (Le Galliard et al. 2003b; Sinervo et Clobert 2003). Ces choix influenceraient en retour les risques d'interférences compétitives futures entre apparentés. Contrairement au népotisme, un évitement de la compétition entre apparentés par des mécanismes de dispersion a cependant le désavantage de diminuer l'apparentement, donc de défavoriser la coopération entre apparentés. Une forte structure d'apparentement pourrait tout de même se maintenir si on considère deux étapes de dispersion : une étape de dispersion conduisant à un évitement des apparentés pendant la phase de compétition, puis une association spatiale entre les apparentés pendant la phase de coopération.

CONCLUSION

Cette synthèse entrouvre brièvement les portes des interactions complexes qui existent entre cycles de vie, structuration socio-spatiale, stochasticité, discrimination des proches génétiques, et l'évolution des comportements altruistes. Une étude plus poussée des ces interactions nécessiterait de mettre en place une hiérarchie de modèles.

Cette complexité pose aussi un certain nombre de questions générales qui restent sans réponse à l'heure actuelle. Tout d'abord, on peut se demander si la socialité évolue globalement au cours de la phylogénie en réponse à de multiples pressions de sélection venant moduler l'intensité des interactions sociales entre apparentés, ou si de nombreux comportements sociaux évoluent indépendamment en réponse spécifique à chaque pression de sélection, la socialité n'étant alors qu'une propriété émergente. De la même façon, on peut s'interroger sur la multiplicité des facteurs génétiques, écologiques et démographiques impliqués dans le maintien de la coopération : la socialité constitue telle un problèmes avec trop de solutions ? Ainsi, pour Alexander et al. (1991), l'émergence de l'eusocialité dépend à la fois de ressources basiques qui seraient élastiques, divisibles, améliorables, persistantes, protectrices, et défendables, et du type de risques de prédation … Une solution empirique à ce surdimensionnement est d'envisager des approches statistiques à plusieurs facteurs de l'évolution de la socialité, alors que la théorie doit se charger de mieux définir les liens causaux entre histoire de vie, environnement, génétique et socialité (Crespi et Choe 1997). Finalement, on peut s'interroger sur le rôle fondamental qu'on prête à l'altruisme dans l'apparition de la majorité des formes de socialité. Est-ce que l'altruisme est l'état ancestral ou l'état dérivé par rapport à l'organisation sociale ? La socialité ne pourrait elle pas être le sous-produit des stratégies de mobilité, soit parce que la socialité émerge en conséquence de fortes contraintes sur la capacité d'émigration comme l'envisage la voie sub-sociale de l'évolution de la coopération (Brockmann 1997; Helms Cahan et al. 2002), ou soit parce que l'association entre partenaires sociaux résulte des processus de la sélection de l'habitat et du partenaire de reproduction, comme Danchin et Wagner (1997) l'envisagent pour la colonialité ? Cette complication oblige à adopter une approche historique et à clairement dissocier les causes de la socialité (e.g., choix de l'habitat) de ces conséquences (e.g., avantages à la vie en groupe, structuration spatiale). C'est dans cette perspective que nous avons envisagé l'effet de l'évolution de la mobilité sur l'émergence de la coopération (chapitre 3).

LES CAUSES SOCIALES DE LA DISPERSION

Cette partie illustre et discute les effets des **propriétés généalogiques et écologiques** des voisinages sociaux sur la dispersion natale et de reproduction du lézard vivipare. Les conséquences sur la dynamique locale et régionale de la population sont aussi explicitées. Ce travail est mis en rapport avec le développement récent d'approches multi-factorielles de la dispersion, et avec les travaux traitant des mécanismes de choix de l'habitat.

INTERACTIONS ENTRE PROCHES GENETIQUES

Une augmentation de la dispersion des filles et une diminution de la dispersion des fils sont observées en présence de la mère (chapitre 4). **Ceci suggère que les filles dispersent pour éviter la compétition avec la mère, alors que les fils évitent la consanguinité avec leurs sœurs**. Des calculs simples, basés sur les valeurs des paramètres du cycle de vie de l'espèce, montrent en effet que le risque d'une rencontre entre un frère et une sœur à l'âge adulte est plus élevé en moyenne que le risque de rencontre entre une mère et son fils. Dans tous les cas, cette expérience suggère qu'il n'existe pas une raison unique à la dispersion des jeunes mais que plusieurs causes sont à l'origine de la dispersion natale (Dobson et Jones 1985 ; Gandon et Michalakis 2001). Il me semble important d'insister dans cette discussion sur trois points de ces résultats et de leur interprétation.

Le premier point concerne la structure logique de l'expérience (voir aussi la discussion de l'Annexe 3). Au cours de notre manipulation, la présence de la mère a été manipulée à l'échelle de l'enclos en gardant ou en substituant toutes les mères biologiques. L'absence des mères dans l'environnement pourrait être perçue comme un signal général de mauvaise qualité de l'environnement, d'une forte prédation ou d'une émigration intense. Cette manipulation n'est donc pas absente de tout biais d'interprétation. Plus généralement, il s'avère bien souvent difficile de déconnecter un élément particulier du déterminisme du comportement de dispersion (Ims et Hjermann 2001). Les sources d'informations véhiculées par la présence de la mère sont ainsi discutées par Léna (1999) et Meylan et al. (2002) : la présence de la mère indiquerait à la fois un risque fort de compétition entre apparentés, un bénéfice par des relations de familiarité, un risque plus élevé de croisements consanguins futurs pour les fils, ou des différences de qualité de l'habitat.

Le deuxième point de discussion concerne la suggestion selon laquelle la dispersion des filles serait expliquée par un évitement de la compétition avec la mère, une suggestion qui s'accorde avec le comportement de dispersion natale du lézard vivipare (Massot et Clobert 1995; Ronce et al. 1998; Sorci et al. 1994). En présence de la mère, on prédirait une augmentation de la dispersion natale sous le contrôle de la mère, qui force des jeunes à disperser afin d'éviter de diminuer la valeur sélective de ses enfants, ou sous le contrôle des jeunes, qui se risquent dans des mouvements de dispersion afin d'éviter de diminuer la valeur sélective de leur mère (Ronce et al. 1998). Même si la structure de l'expérience ne permet pas de déterminer l'acteur qui contrôle le comportement, on peut chercher le scénario le plus vraisemblable. D'une part, les classes d'âge les plus jeunes du lézard vivipare sont les plus sensibles à la densité dépendance, via la croissance natale et l'âge à première reproduction (Le Galliard et al. 2003a; Massot et al. 1992). Ceci induit une asymétrie dans les coûts à la philopatrie : le coût à rentrer en compétition avec sa mère pour une fille est plus élevée que le coût en rentrer en compétition avec ses filles pour une mère. Par ailleurs, nous avons observé une diminution de la survie des jeunes en présence de la mère. Ceci suggère que des interactions agressives aient eu lieu entre la mère et les jeunes, et que les mères ont pu activement tenter d'expulser les jeunes des enclos (voir Annexe 3). **Ces deux observations rendent plus probables l'hypothèse selon laquelle les mères ont contrôlé la dispersion des filles après leur naissance en les incitant à disperser** (*contra* Clobert et al. 1994; de Fraipont et al. 2000).

Finalement, le troisième point de cette discussion concerne la réponse des jeunes mâles, que nous avons interprétée par l'asymétrie compétitive entre sexes et l'évitement de la consanguinité avec les sœurs (voir aussi chapitre 6). Premièrement, nous suggérons que la valeur sélective des mâles est déterminée de manière critique par leurs interactions compétitives avec des mâles adultes. Cette suggestion est en accord avec le fonctionnement et la régulation par la densité des populations naturelles (Massot et al. 1992; Pilorge 1987; Pilorge et al. 1987). Deuxièmement, nous proposons que les jeunes mâles dispersent pour éviter des appariements consanguins avec leurs sœurs. Par exemple, la sexe ratio de la ponte affecte la dispersion en dépendance du sexe : les jeunes mâles dispersent plus des pontes riches en femelles, et inversement chez les jeunes femelles (Massot et Clobert 2000). Un déterminisme post-natal de la dispersion par l'évitement de la consanguinité pourrait être démontré par des échanges d'apparentés au sein des pontes.

COMPETITION

L'intérêt porté à la densité dépendance de la dispersion est justifié par l'importance de la dispersion comme un mécanisme régulateur de la dynamique des populations (Brown et Kodric-Brown 1977) et par le rôle de la compétition intraspécifique comme une cause ultime de la dispersion (Gadgil 1971). Le signe de densité dépendance de la dispersion des mammifères a été très controversée, en partie du fait de l'ignorance des mécanismes proximaux impliqués, des disparités entre les stades de la dispersion affectés, et de l'influence de l'échelle spatiale (Gaines et McClenaghan 1980; Ims et Hjermann 2001; Lambin et al. 2001). La densité peut agir comme un reflet de l'intensité de la compétition locale pour les ressources ou pour les partenaires (e.g., Denno et al. 1991), mais aussi comme un critère de qualité de l'habitat (Stamps 1991). La densité comme reflet de la compétition locale a un effet positif sur l'émigration (e.g., Aars et Ims 2000) mais un effet négatif sur l'immigration (e.g., Gundersen et al. 2002) : la réponse totale sur la dispersion dépend donc de deux processus antagonistes dont la balance change selon la covariance spatiale de la densité de l'habitat (Ims et Hjermann 2001). Certaines structures de covariance peuvent engendrer des différences de signe et d'intensité de la densité dépendance de la dispersion en fonction de l'échelle spatiale. Par ailleurs, l'importance de la densité dépendance par rapport à d'autres causes de dispersion dépend de la stabilité de l'habitat. Dans des habitats stables, comme ceux du lézard vivipare (Clobert et al. 1994), les causes sociales de la dispersion devraient dominer sur les causes environnementales.

En manipulant le niveau de densité de deux populations connectées, nous avons mis en évidence que **la dispersion natale a diminué dans les populations à haute densité pendant la première année suivant l'introduction** (chapitre 4), **suggérant soit une préférence pour les habitats densément peuplés soit une contrainte sur les mouvements d'exploration**. Par ailleurs, les effets de la densité de la population ne sont pas les mêmes sur les différentes classes d'âge, puisque la dispersion de reproduction est indépendante de la densité (chapitre 4). Le signe de la densité dépendance conduit à des échanges balancés entre fragments d'habitat qui contribuent à maintenir la variance de densité entre populations sur le moyen terme (Annexe 3). Dans le même temps, les paramètres démographiques affectant à court terme la dynamique de la population ne sont pas ou peu affectés par la densité de la population (Annexe 3). Au contraire, il a été montré que l'immigration, la survie, et la reproduction participent à très court terme à la régulation démographique d'un habitat naturellement continue (Massot et al. 1992). **La dispersion et la densité dépendance locale en populations fragmentées s'avèrent donc moins efficaces que dans un contexte de perturbation d'une population continue.**

En manipulant l’occupation d’un voisinage d’arrivée d’un dispersant, nous avons aussi mis en évidence que **la probabilité d’immigration d’un lézard est indépendante de l’occupation de l’habitat par des congénères** (chapitre 5). Le rôle stabilisant de la dispersion sur la dynamique régionale peut alors s’exprimer par l’intermédiaire d’un effet aléatoire de rescousse (Brown et Kodric-Brown 1977; Gundersen et al. 2002; Hanski 1999). A l’échelle de notre dispositif expérimental, la colonisation d’habitat est rapide et efficace : les populations fondatrices croissent du fait d’un afflux continu d’immigrants qui jouissent alors de conditions plus favorables pour leur croissance et leur reproduction (chapitre 5). L’effet rescousse se trouve donc renforcé par une augmentation locale du recrutement reproducteur au sein des populations récemment fondées : processus régionaux et locaux se combinent alors pour diminuer la variance entre populations au sein de la métapopulation.

Ces deux études suggèrent donc une **différence entre la densité dépendance de l’émigration et de l’immigration**. Cette différence pourrait refléter des contraintes sur le comportement d’immigration et l’exploration de l’habitat chez le lézard vivipare (chapitre 5). D’autre part, les différences d’intensité de la densité dépendance entre les deux expériences peuvent être expliquées par l’échelle de la perturbation : autour de la capacité de charge supposée dans la première expérience, et depuis une densité nulle jusqu’à la densité de charge du milieu dans la deuxième expérience. Ainsi, la relaxation compétitive observée au cours de la dynamique de colonisation traduit probablement le fort contraste initial de densité (mais voir les problèmes d’interprétation de la première expérience discutés dans l’Annexe 3). Ces deux points pourraient être éclaircis par des expériences testant les effets de la densité de la population aux différents stades de la dispersion, et en manipulant une gamme plus large de densité de population. Il serait alors intéressant d’identifier les paramètres démographiques impliqués dans la régulation des effectifs en fonction de la densité locale de la population et du contraste de densité à l’échelle régionale (voir par exemple Andreassen et Ims 2001).

Une partie des asymétries compétitives dans la population peut être expliquée par le rôle différent des mâles et des femelles dans la compétition (voir chapitre 6). Notre perturbation de la sexe ratio de la population confirme que ces asymétries jouent un rôle significatif dans les décisions de dispersion. Le biais sexuel de la dispersion natale n’est pas affecté par la sexe ratio adulte de la population, suggérant que **la compétition intrasexuelle entre les jeunes et les adultes n’est pas impliquée dans la dispersion natale**. Par contre, la perturbation de la sexe ratio adulte a affecté la dispersion de reproduction des femelles après introduction, ce qui suggère que **la compétition pour les ressources a incité à la dispersion du sexe en général philopatrique**.

DISPERSION, CHOIX D’HABITAT ET INFORMATION

Un point de vue empirique et théorique consiste à considérer que la dispersion découle de la sélection de l’habitat (Danchin et al. 2001; Doligez et al. 2003; Stamps 1991). Cette approche a l’avantage (i) d’expliciter clairement le rôle des *facteurs proximaux*, en particulier des critères de choix de l'habitat, (ii) de considérer le processus de dispersion dans sa dimension ontogénique, et (iii) de générer des prédictions testables par l'observation du comportement de dispersion des individus (Stamps 2001). Quels sont les changements comportementaux ou physiologiques impliquées dans la réponse des jeunes à la présence de la mère ? Les effets de la densité sur la dispersion natale sont ils directs (Aars et Ims 2000), ou indirects comme chez certains rongeurs (Andreassen et Ims 2001; Nunes et al. 1998) ? Quelle est la nature de l'information véhiculée par la présence de la mère, la densité de la population, ou la sexe ratio de la population ? Ces questions resteront ici en suspens parce que nous avons pris le parti de traiter ces mécanismes physiologiques et comportementaux comme une boîte noire. Pourtant, il me semble fondamental de mieux comprendre les modes d'action des causes sociales de la dispersion décrites dans cette thèse (Dufty et al. 2002; Ims et Hjermann 2001). Un tel agenda de recherche nécessiterait dans un premier temps de retourner dans les populations naturelles afin d'étudier l'association entre ces paramètres et la qualité de l'habitat, d'en décrire la variation spatio-temporelle, et de mettre en rapport cette variabilité avec l'échelle spatiale de la dispersion.

L'approche de la sélection de l'habitat a un deuxième intérêt. En considérant l'acte de mouvement dans sa dimension d'échantillonnage, elle voit la dispersion comme un processus de recherche, de collecte et d'échange d'informations. En bougeant d'un habitat à un autre, la dispersion déplace ainsi une histoire individuelle qui peut se donner à lire par des congénères. Dans certains contextes, cette histoire pourrait être utilisée pour refuser les interactions sociales avec les immigrants (Perrin et Lehmann 2001), mais elle peut aussi être considérée comme une source d'information intéressante sur les populations voisines. **On voit donc que la dispersion n'est pas seulement un transfert d'individus et de gènes entre populations, mais aussi un transfert d'information**.

REFERENCES

Aars, J., and R. A. Ims. 2000. Population dynamic and genetic consequences of spatial density-dependent dispersal in patchy populations. American Naturalist 155:252-265.

Alexander, R. D., K. M. Noonan, and B. J. Crespi. 1991. The evolution of eusociality. Pp. 3-44 *in* J. U. M. Jarvis and R. D. Alexander, eds. The biology of naked mole rat. Princeton University Press, Princeton.

Andreassen, H. P., and R. A. Ims. 2001. Dispersal in patchy vole populations: role of patch configuration, density dependence, and demography. Ecology 82:2911-2926.

Brockmann, H. J. 1997. Cooperative breeding in wasps and vertebrates: the role of ecological constraints *in* J. C. Choe and B. J. Crespi, eds. The evolution of social behavior in insects and arachnids. Cambridge University Press, Cambridge.

Brown, J. H., and A. Kodric-Brown. 1977. Turnover rates in insular biogeography: effect of immigration on extinction. Ecology 58:445-449.

Cadet, C., R. Ferrière, J. A. J. Metz, and M. van Baalen. 2003. The evolution of dispersal under demographic stochasticity. The American Naturalist

Clobert, J., M. Massot, J. Lecomte, G. Sorci, M. de Fraipont, and R. Barbault. 1994. Determinants of dispersal behavior: the common lizard as a case study. Pp. 183-206 *in* L. J. Vitt and E. R. Pianka, eds. Lizard ecology. Historical and experimental perspectives. Princeton University Press, Princeton.

Clutton-Brock, T. 2002. Breeding together: kin selection and mutualism in cooperative vertebrates. Science 296:69-72.

Crespi, B. J., and J. C. Choe. 1997. Explanation and evolution of social systems *in* J. C. Choe and B. J. Crespi, eds. The evolution of social behavior in insects and arachnids. Cambridge University Press, Cambridge.

Danchin, E., D. Heg, and B. Doligez. 2001. Public information and breeding habitat selection. Pp. 243-258 *in* J. Clobert, E. Danchin, A. A. Dhondt and J. D. Nichols, eds. Dispersal. Cambridge University Press, Cambridge.

Danchin, E., and R. H. Wagner. 1997. The evolution of coloniality: the emergence of new perspectives. Trends in Ecology and Evolution 12:342-347.

de Fraipont, M., J. Clobert, A. H. John, and S. Meylan. 2000. Increased pre-natal maternal corticosterone promotes philopatry of offspring in common lizards *Lacerta vivipara*. Journal of Animal Ecology 69:404-413.

Denno, R. F., G. K. Roderick, K. L. Olsmtead, and H. G. Döbel. 1991. Density-related migration in planthoppers (Homoptera:Delphacidae): the role of habitat persistence. The American Naturalist 138:1513-1541.

Dobson, F. S., and W. T. Jones. 1985. Multiple causes of dispersal. The American Naturalist 126:855-858.

Doligez, B., C. Cadet, E. Danchin, and T. Boulinier. 2003. When to use public information for breeding habitat selection? The role of environmental predictability and density dependence. Animal Behaviour

Dufty, A. M., J. Clobert, and A. P. Moller. 2002. Hormones, developmental plasticity and adaptation. Trends in Ecology and Evolution 17:190-196.

Ellner, S. P., A. Sasaki, Y. Haraguchi, and H. Matsuda. 1998. Speed of invasion in lattice population models:pair-edge approximation. Journal of Mathematical Biology 36:469-484.

Ferrière, R., and J.-F. Le Galliard. 2001. Invasion fitness and adaptive dynamics in spatial population models. Pp. 57-79 *in* J. Clobert, E. Danchin, A. A. Dhondt and J. Nichols, eds. Dispersal. Oxford University Press, Oxford.

Ferrière, R., and R. Michod. 1996. The evolution of cooperation in spatially heterogeneous populations. The American Naturalist 147:692-717.

Frank, S. A. 1998. Foundations of social evolution. Princeton University Press, Princeton.

Gadgil, M. 1971. Dispersal: population consequences and evolution. Ecology 52:253-261.

Gaines, M. S., and L. R. J. McClenaghan. 1980. Dispersal in small mammals. Annual Review of Ecology and Systematics 11:163-196.

Gandon, S., and Y. Michalakis. 2001. Multiple causes of the evolution of dispersal *in* J. Clobert, ed. Cambridge University Press.

Geritz, S. A. H., E. Kisdi, G. Meszena, and J. A. J. Metz. 1998. Evolutionary singular strategies and the adaptive growth and branching of the evolutionnary tree. Evolutionary Ecology 12:35-57.

Goodnight, K. F. 1992. The effect of a stochastic variation on kin selection in a budding-viscous population. The American Naturalist 140:1028-1040.

Gundersen, G., H. P. Andreassen, and R. A. Ims. 2002. Individual and population level determinants of immigration success on local habitat patches: an experimental approach. Ecology Letters 5:294-301.

Hanski, I. 1999. Metapopulation ecology. Oxford University Press

Helms Cahan, S., D. T. Blumstein, L. Sundström, L. Liebig, and A. Griffin. 2002. Social trajectories and the evolution of social behaviour. Oikos 96:206-216.

Ims, R. A., and D. O. Hjermann. 2001. Condition dependent dispersal. Pp. 203-216 *in* J. Clobert, E. Danchin, A. A. Dhondt and J. D. Nichols, eds. Dispersal. Oxford University Press, Oxford.

Irwin, A. J., and P. D. Taylor. 2001. Evolution of altruism in stepping-stone populations with overlapping generations. Theoretical Population Biology 60:315-325.

Kelly, J. K. 1992. Restricted migration and the evolution of altruism. Evolution 46:1492-1495.

Kelly, J. K. 1994. The effect of scale dependent processes on kin selection: mating and density regulation. Theoretical Population Biology 46:32-57.

Koella, J. 2000. The spread of altruism versus the evolutionary response of egoists. Proceedings of the Royal Society London B 267:1979-1985.

Komdeur, J., and B. J. Hatchwell. 1999. Kin recognition: function and mechanism in avian societies. Trends in Ecology and Evolution 14:237-241.

Lambin, X., J. Aars, and S. B. Piertney. 2001. Dispersal, intraspecific competition, kin competition and kin facilitation: a review of the empirical evidence. Pp. 110-122 *in* J. Clobert, E. Danchin, A. Dhondt and J. D. Nichols, eds. Dispersal. Oxford University Press, Oxford.

Le Galliard, J.-F., R. Ferrière, and J. Clobert. 2003a. Immigration within metapopulations: behavioral and demographic consequences in the common lizard. Submitted to Ecology

Le Galliard, J.-F., R. Ferrière, and J. Clobert. 2003b. Mother-offspring interactions affect natal dispersal in a lizard. Proceedings of the Royal Society London B 270:1163-1169.

Léna, J. P. 1999. Discrimination des proches génétiques et gestion de l'espace chez le lézard vivipare. Ecologie. Paris VI, Paris.

Massot, M., and J. Clobert. 1995. Influence of maternal food availability on offspring dispersal. Behavioural Ecology and Sociobiology 37:413-418.

Massot, M., and J. Clobert. 2000. Processes at the origin of similarities in dispersal behaviour among siblings. Journal of Evolutionary Biology 13:707-719.

Massot, M., J. Clobert, T. Pilorge, J. Lecomte, and R. Barbault. 1992. Density dependence in the common lizard: demographic consequences of a density manipulation. Ecology 73:1742-1756.

Metz, J. A. J., and M. Gyllenberg. 2001. How should we define fitness in structured metapopulation models? Including an application to the calculation of evolutionarily stable dispersal strategies. Proceedings of the Royal Society London B 268:499-508.

Meylan, S., J. Belliure, J. Clobert, and M. de Fraipont. 2002. Stress and body condition as prenatal and postnatal determinants of dispersal in the common lizard (*Lacerta vivipara*). Hormones and Behavior 42:319-326.

Mittledorf, J., and D. S. Wilson. 2001. Population viscosity and the evolution of altruism. Journal of Theoretical Biology 204:481-496.

Morris, A. 1997. Representing spatial interactions in simple ecological models. University of Warwick, Coventry.

Nakamaru, M., H. Matsuda, and Y. Iwasa. 1997. The evolution of cooperation in a lattice-structured population. Journal of Theoretical Biology 184:65-81.

Nakamaru, M., H. Nogami, and Y. Iwasa. 1998. Score-dependent fertility model for the evolution of cooperation in a lattice. Journal of Theoretical Biology 194:101-124.

Nunes, S., C.-D. T. Ha, E.-M. Mueke, L. Smale, and K. E. Holekamp. 1998. Body fat and time of the year interact to mediate dispersal behaviour in ground squirrels. Animal Behaviour 55:605-614.

Perrin, N., and L. Lehmann. 2001. Is sociality driven by the costs of dispersal of the benefits of philopatry? A role for kin-discrimination mechanisms. The American Naturalist 158:471-483.

Pilorge, T. 1987. Density, size structure, and reproductive characteristics of three populations of *Lacerta vivipara* (Sauria: Lacertidae). Herpetologica 43:345-356.

Pilorge, T., J. Clobert, and M. Massot. 1987. Life history variations according to sex and age in *Lacerta vivipara*. 4th Ordinary General Meeting of the Societas Europaea Herpetologica, Nijmegen 311-315.

Queller, D. C. 1992. Does population viscosity promote kin selection? Trends in Ecology and Evolution 7:322-324.

Queller, D. C. 1994. Genetic relatedness in viscous populations. Evolutionary Ecology 8:70-73.

Roff, D. A. 1992. The evolution of life histories. Chapman and Hall, New York.

Ronce, O., J. Clobert, and M. Massot. 1998. Natal dispersal and senescence. Proceedings of the National Academy of Sciences USA 95:600-605.

Rousset, F., and S. Billiard. 2000. A theoretical basis for measures of kin selection in subdivided populations: finite populations and localized dispersal. Journal of Evolutionary Biology 13:814-825.

Sinervo, B., and J. Clobert. 2003. Morphs, dispersal behavior, genetic similarity, and the evolution of cooperation. Science 300:1949-1951.

Sorci, G., M. Massot, and J. Clobert. 1994. Maternal parasite load increases sprint speed and philopatry in female offspring of the common lizard. The American Naturalist 144:153-164.

Stamps, J. A. 1991. The effect of conspecifics on habitat selection in territorial species. Behavioral Ecology and Sociobiology 28:29-36.

Stamps, J. A. 2001. Habitat selection by dispersers: integrating proximate and ultimate approaches. Pp. 230-242 *in* J. Clobert, E. Danchin, A. Dhondt and J. Nichols, eds. Dispersal. Cambridge University Press, Cambridge.

Taylor, P. D. 1992a. Altruism in viscous populations - an inclusive fitness approach. Evolutionary Ecology 6:352-356.

Taylor, P. D. 1992b. Inclusive fitness in a homogeneous environment. Proceedings of the Royal Society London B 249:299-302.

Taylor, P. D., and S. A. Frank. 1996. How to make a kin selection model ? Journal of Theoretical Biology 180:27-37.

Taylor, P. D., and A. J. Irwin. 2000. Overlapping generations can promote altruistic behavior. Evolution 54:1135-1141.

van Baalen, M. 2000. Pair approximation for ecological interactions on different geometries. Pp. 359-387 *in* U. Dieckmann, R. Law and J. A. J. Metz, eds. The geometry of ecological interactions: simplifying spatial complexities. Cambridge University Press, Cambridge.

van Baalen, M., and D. Rand. 1998. The unit of selection in viscous populations and the evolution of altruism. Journal of Theoretical Biology 193:631-648.

West, S. A., I. Pen, and A. S. Griffin. 2002. Cooperation and competition between relatives. Science 296:72-75.

Wilson, D. S., G. B. Pollock, and L. A. Dugatkin. 1992. Can altruism evolve in purely viscous populations? Evolutionary Ecology 6:331-341.