whose fates have been followed since 1971. Tissue sampling for DNA analyses started in 1988. Blood samples were taken from all captured sheep until 1993 and stored in preservative at −20 °C. Sampling resumed in 1997, when hair samples were taken from all captured sheep by plucking 50–100 hairs including roots from the back or flank. Hairs were kept either in paper envelopes or plastic bags containing about 5 g of silica at room temperature. From 1998 to 2002, a tissue sample from each captured sheep was taken from the ear with an 8-mm punch. Ear tissue was kept at −20 °C in a solution of 20% dimethylsulphoxide saturated with NaCl. We sampled 433 marked individuals over the course of the study.

DNA was extracted from blood with a standard phenol–chloroform method, and from either 20–30 hairs including follicles or about 5 mg of ear tissue, using the QIAamp tissue extraction kit (Qiagen Inc., Mississauga, Ontario). Polymerase chain reaction amplification at 20 ungulate-derived microsatellite loci, 15 as described previously[5] plus MCM527, BM4025, MAF64, OarFCB193 and MAF92 (refs 25, 26), and fragment analysis were performed as described elsewhere[5]. After correction for multiple comparisons, we found no evidence for allelic or genotypic disequilibria at or among these 20 loci.

Paternity of 241 individuals was assigned by using the likelihood-based approach described in CERVUS[27] at a confidence level of more than 95% with input parameters given in ref. 5. After paternity analysis, we used KINSHIP[28] to identify 31 clusters of 104 paternal half-sibs among the unassigned offspring. A paternal half-sibship consisted of all pairs of individuals of unassigned paternity that were identified in the KINSHIP analysis as having a likelihood ratio of the probability of a paternal half-sib relationship versus unrelated with an associated $P < 0.05$ (ref. 28). Members of reconstructed paternal half-sibships were assigned a common unknown paternal identity for the animal model analyses. Paternal identity links in the pedigree were therefore defined for 345 individuals.

Animal model analyses

Breeding values, genetic variance components and heritabilities were estimated by using a multiple trait restricted-estimate maximum-likelihood (REML) model implemented by the programs PEST[29] and VCE[30]. An animal model was fitted in which the phenotype of each animal was broken down into components of additive genetic value and other random and fixed effects: $\mathbf{y} = X\mathbf{b} + Z\mathbf{a} + P\mathbf{c} + \mathbf{e}$, where $\mathbf{y}$ was a vector of phenotypic values, $\mathbf{b}$ was a vector of fixed effects, $\mathbf{a}$ and $\mathbf{c}$ were vectors of additive genetic and permanent environmental, $\mathbf{e}$ was a vector of residual values, and X, Z and P were the corresponding design matrices relating records to the appropriate fixed or random effects[18]. Fixed effects included age (factor) and the average weight of yearling ewes in the year of measurement (covariate), which is a better index of resource availability than population size because it accounts for time-lagged effects[4]. The permanent environmental effect grouped repeated observations on the same individual to quantify any remaining between-individual variance over and above that due to additive genetic effects, which would be due to maternal or other long-term environmental and non-additive genetic effects.

The total phenotypic variance (V_p) was therefore partitioned into three components: the additive genetic variance (V_a), the permanent environmental variance (V_e) and the residual variance (V_r), thus: $V_p = V_a + V_e + V_r$. Heritability was calculated as $h^2 = V_a/V_p$. The VCE[30] program returns standard errors on all variance components and ratios. Best linear unbiased predictors of individual breeding values were quantified by using REML estimates of the variance components obtained with PEST[29]. All statistical tests were conducted in SPLUS 6.1.

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Correspondence and requests for materials should be addressed to D.W.C. (d.coltman@sheffield.ac.uk).

The role of evolution in the emergence of infectious diseases

Rustom Antia[1], Roland R. Regoes[1], Jacob C. Koella[2] & Carl T. Bergstrom[3]

[1]*Department of Biology, Emory University, Atlanta, Georgia 30322, USA*
[2]*Laboratoire de Parasitologie Evolutive, Université Pierre et Marie Curie, 75252 Paris, France*
[3]*Department of Biology, University of Washington, Seattle, Washington 98195, USA*

It is unclear when, where and how novel pathogens such as human immunodeficiency virus (HIV), monkeypox and severe acute respiratory syndrome (SARS) will cross the barriers that separate their natural reservoirs from human populations and ignite the epidemic spread of novel infectious diseases. New pathogens are believed to emerge from animal reservoirs when ecological changes increase the pathogen's opportunities to enter the human population[1] and to generate subsequent human-to-human transmission[2]. Effective human-to-human transmission requires that the pathogen's basic reproductive number, R_0, should exceed one, where R_0 is the average number of secondary infections arising from one infected individual in a completely susceptible population[3]. However, an increase in R_0, even when insufficient to generate an epidemic, nonetheless increases the number of subsequently infected individuals. Here we show that,

as a consequence of this, the probability of pathogen evolution to $R_0 > 1$ and subsequent disease emergence can increase markedly.

The emergence of a disease combines two elements: the introduction of the pathogen into the human population and its subsequent spread and maintenance within the population. Ecological factors such as human behaviour can influence both of these elements, and consequently ecology has been recognized to have an important role in the emergence of disease[1,2,4]. In contrast, evolutionary factors including the adaptation of the pathogen to growth within humans and the subsequent transmission of the pathogen between humans are mostly considered in terms of changes in the virulence of the pathogen, and are often thought to have a lesser role in the initial emergence of pathogens[4]. One exception[5] suggests that immunocompromised individuals might provide "stepping stones" for the evolution of pathogens.

The successful emergence of a pathogen requires R_0 to exceed one in the new host. Only then can an introduction trigger emergence[2]. (Here we use R_0 to refer to spread in human populations, not in the natural reservoir.) If R_0 for a potential pathogen exceeds one, this scenario represents an epidemic waiting to happen. By contrast, when R_0 is initially less than one, infections will inevitably die out and there will be no epidemic unless genetic or ecological changes drive R_0 above one.

There are a number of ways in which R_0 can increase. Ecological changes such as changes in host density or behaviour can increase R_0, as can genetic changes in the pathogen population or in the population of its new host. Genetic changes in the pathogen can arise either through 'coincidental' processes such as neutral drift or coevolution of the pathogen and its reservoir host, or through adaptive evolution of the pathogen during chains of transmission in humans. Genetic changes of the new host might be more likely for domesticated or endangered species than for humans.

Here we show that factors, such as ecological changes, that increase the R_0 value of the potential pathogen to a level not sufficient to cause an epidemic (that is, R_0 remains less than one) can greatly increase the length of the stochastic chains of disease transmission. These long transmission chains provide an opportunity for the pathogen to adapt to human hosts, and thus for the disease to emerge.

Our model is illustrated in Fig. 1. Introductions occur stochastically from the natural reservoir of the pathogen, and each primary case is followed by stochastic transmission that generates a variable number of subsequent infections in the human population. We assume that the number of secondary cases follows a Poisson distribution with a mean equal to R_0. Each introduction thus forms branched chains of transmission, which stutter to extinction if $R_0 < 1$, and the pathogen cannot evolve ($\mu = 0$). The probability that the pathogen evolves and a secondary infection is caused by the mutant is equal to μ for each of the secondary infections (we note that μ incorporates not only the pathogen's mutation rate, but also its dynamics within the host and its transmissibility). We use multitype branching processes[6–9] to describe the initial spread of the infection, incorporating the evolution of the pathogen.

In the simplest case (see Fig. 2a) only one mutation is required for the R_0 of the evolved pathogen to exceed one. The probability that a single introduction evolves, causing one (or more) infections with the evolved pathogen (a filled circle in Fig. 1) before it goes extinct, depends very strongly on the R_0 of the introduced pathogen, particularly for low μ values as R_0 approaches 1. This probability is approximately linearly dependent on the rate of evolution μ.

The probability that the introduction leads to an epidemic (the 'probability of emergence') depends on the probability of evolution and the probability that the evolved infections do not go extinct due to stochastic effects. In Fig. 2b we plot the probability of emergence for three different R_0 values of the evolved strain. The probability of emergence approaches the probability of evolution when R_0 of the evolved strain is large, and is lower when the R_0 of the evolved strain

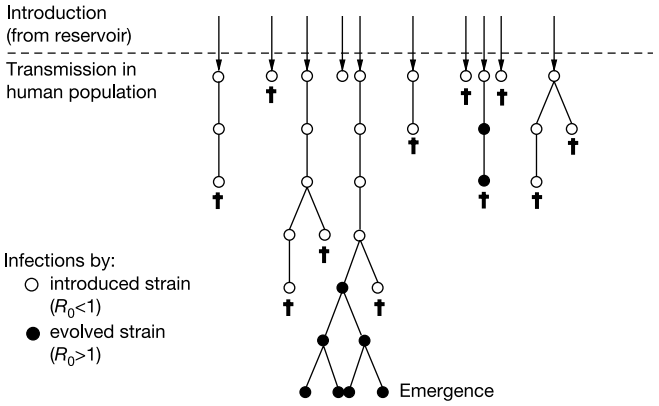

Figure 1 Schematic for the emergence of an infectious disease. Introductions from the reservoir are followed by chains of transmission in the human population. Infections with the introduced strain (open circles) have a basic reproductive number $R_0 < 1$. Pathogen evolution generates an evolved strain (filled circles) with $R_0 > 1$. The infections caused by the evolved strain can go on to cause an epidemic. Daggers indicate no further transmission.

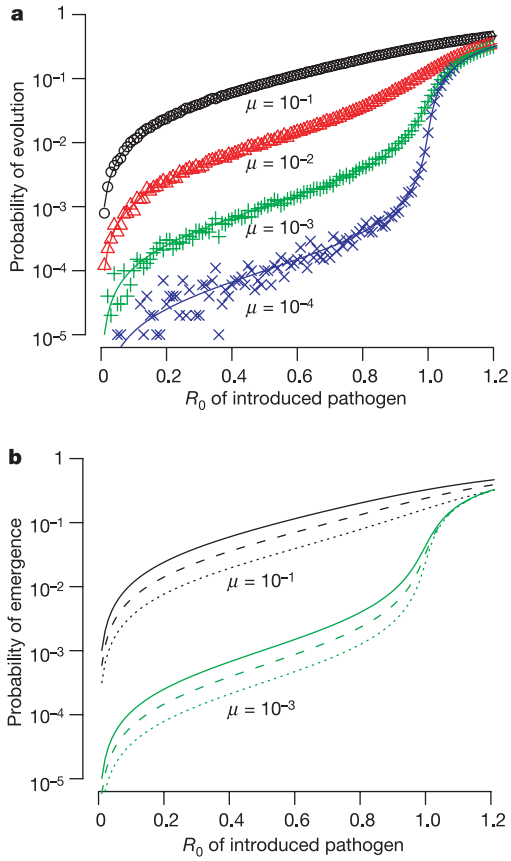

Figure 2 One-step evolution. A single change is required for the pathogen to evolve to $R_0 > 1$. **a**, The probability that an introduction leads to an infection with an evolved strain of the pathogen (filled circle in Fig. 1) is highly sensitive to R_0, and is approximately linearly dependent on the mutation rate μ. Lines correspond to numerical solutions to the branching process model (see Supplementary Information) and symbols correspond to Monte-Carlo simulations following 10^5 introductions. **b**, The probability of emergence per introduction depends on the R_0 value of the introduced pathogen and of the evolved pathogen. The solid, dashed and dotted lines correspond to the evolved pathogen having an R_0 of 1,000, 1.5 and 1.2 respectively.

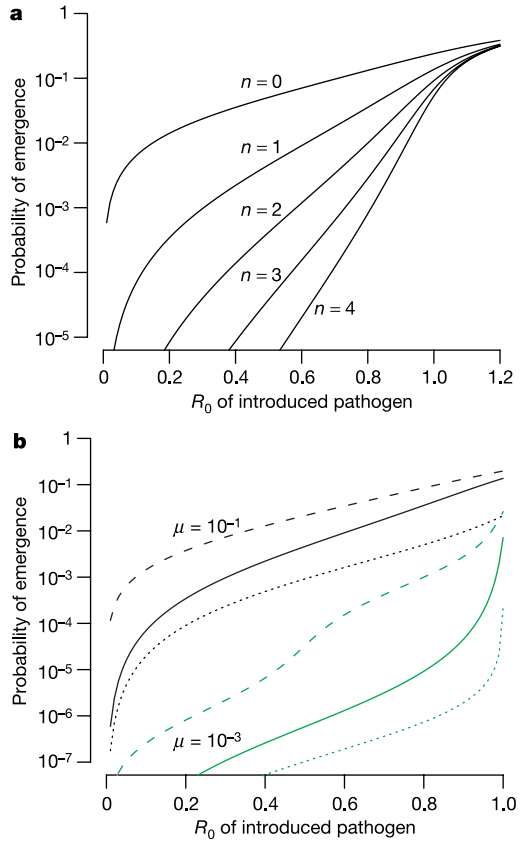

Figure 3 Multiple-step evolution. Here multiple evolutionary changes are required for evolution of the pathogen to have an $R_0 > 1$. **a**, Jackpot model with $\mu = 0.1$ and n intermediate changes each with R_0 equal to that of the introduced pathogen: increasing the number of steps (n) greatly decreases the probability of evolution, and makes it more sensitive to the R_0 of the introduced pathogen. **b**, Alternative multi-step models for the one-intermediate ($n = 1$) case. The jackpot model (solid line), additive model (dashed line) and fitness valley model (dotted line) are shown (see text for details).

is close to 1. We find that the probability of emergence depends most strongly on the R_0 of the introduced pathogen, increases approximately linearly with the mutation rate μ, and depends only modestly on the R_0 of the evolved pathogen.

We extend the simple one-step mutation model to consider the situation in which multiple evolutionary changes are required for the pathogen to attain $R_0 > 1$ in the human population. We begin with a simple scenario, which we call the jackpot model, where the R_0 of the pathogen with the intermediate mutations is the same as that of the introduced pathogen, and where only the addition of the final mutation results in an increase in R_0 to greater than one. As seen in Fig. 3, increasing the number of required evolutionary steps greatly reduces the probability of emergence and increases its sensitivity to changes in R_0. The probability of emergence is approximately proportional to the mutation rate to the power of the number of evolutionary steps required (see Supplementary Information).

The fitness landscape on which evolution occurs is important in determining the outcome[10]. Figure 3b illustrates this for the case of a single intermediate type. As should be expected, changing the jackpot model to an additive model, where the fitness of the intermediate is the average of the fitness of the introduced strain and fully evolved strain, increases the probability that the pathogen evolves to $R_0 > 1$, whereas changing it to a fitness valley model, where the fitness of the intermediate is lower than the fitness of the introduced strain, decreases the probability of emergence.

The key characteristic distinguishing our model from the conventional view is the R_0 of the introduced pathogen. In the conventional view the R_0 of these infections must be greater than one, whereas in the mechanism described here it is less than one, and evolution during the stochastic chains of transmission allows R_0 to increase above one. In the case of human infections such as HIV[1,11,12], SARS[13–17] and (potentially) monkeypox[18–21], seroprevalence studies among groups at high risk of infection from the reservoir would allow evaluation of whether crossover events are usually dead ends, as we expect in our model, or whether they are associated with a large number of secondary cases. In the case of diseases emerging into non-human populations, such as the Nipah virus, which moved from bats to pigs[22–24], it may be possible to conduct additional tests involving controlled experimental infections to estimate the R_0 (in this case of the bat Nipah virus in pigs) and to determine whether it evolves during the course of chains of transmission. Such studies may additionally help to identify pathogens that have an ability to evolve rapidly and thus have a high potential for emergence.

The framework presented here has special relevance for pathogens that have been driven to extinction by vaccination. In the case of smallpox there are probably reservoirs of related zoonoses (such as, but not restricted to, monkeypox) from which smallpox may have originated. Although the R_0 of monkeypox in the human population is clearly less than one, there are occasional chains of transmission in the human population[18–21]. As the level of herd immunity to smallpox wanes in the absence of continued vaccination, we expect an increase in R_0 of infections with monkeypox albeit to a level still less than one (the smallpox vaccine provides about 85% cross immunity against monkeypox). Our results suggest that this increase in the effective R_0 of monkeypox in the human population could markedly increase the probability of evolution of monkeypox, allowing it to emerge into a successful human pathogen (which, depending on the evolutionary trajectory followed, may be similar to or differ from smallpox).

The present study could be extended in a number of directions. These include explicitly incorporating the details of ecological interactions such as heterogeneity in the transmission in different areas and subpopulations[2,25,26,27] and incorporating genetic diversity of the pathogen in its reservoir. Finally, we note that this framework can be applied to the more general problem of biological invasions[28,29]. □

Methods

We describe the dynamics and evolution of emerging diseases as a multi-type branching process with the following probability-generating functions[6–9]:

$$f_i(s_1, s_2, \ldots, s_m) = \exp[-(1-\mu)R_0^{(i)}(1-s_i)]\exp[-\mu R_0^{(i)}(1-s_{i+1})], \quad i = 1, \ldots, m-1$$

$$f_m(s_1, s_2, \ldots, s_m) = \exp[-R_0^{(m)}(1-s_m)]$$

We calculated the extinction probabilities of the above process numerically. For details and definitions see Supplementary Information.

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Correspondence and requests for materials should be addressed to R.A. (rantia@emory.edu).

Plankton effect on cod recruitment in the North Sea

Grégory Beaugrand[1,2], Keith M. Brander[3], J. Alistair Lindley[2], Sami Souissi[1] & Philip C. Reid[2]

[1]*CNRS, UMR 8013 ELICO, Station Marine, Université des Sciences et Technologies de Lille BP 80, 62930 Wimereux, France*
[2]*Sir Alister Hardy Foundation for Ocean Science, The Laboratory Citadel Hill, Plymouth PL1 2PB, UK*
[3]*ICES, Palaegade 2–4, 1261 Copenhagen K, Denmark*

The Atlantic cod (*Gadus morhua* L.) has been overexploited in the North Sea since the late 1960s and great concern has been expressed about the decline in cod biomass and recruitment[1]. Here we show that, in addition to the effects of overfishing[1], fluctuations in plankton have resulted in long-term changes in cod recruitment in the North Sea (bottom-up control). Survival of larval cod is shown to depend on three key biological parameters of their prey: the mean size of prey, seasonal timing and abundance. We suggest a mechanism, involving the match/mismatch hypothesis[2], by which variability in temperature affects larval cod survival and conclude that rising temperature since the mid-1980s has modified the plankton ecosystem in a way that reduces the survival of young cod.

Fish stock biomass fluctuates in space and time, but the causes and mechanisms responsible for the observed variability remain poorly identified[3]. A number of studies have reported that temperature influences cod recruitment, although the relationships found were often weak for the North Sea[4]. Food quantity and quality are also essential because they influence the growth of fish larvae[2,5] and attempts have been made to establish relationships between changes in plankton (that is, availability of prey) and cod[6]. However, such studies, including the ICES/GLOBEC Cod and Climate Change programme[7], often focused on a limited number of biological parameters and no credible evidence has emerged[4,8].

Here we have simultaneously used six key biological parameters for the diet and growth of cod larvae and juveniles in the North Sea[5,9]. We considered the total calanoid copepod biomass as a quantitative indicator of food for larval cod, the mean size of calanoid copepods as a qualitative indicator of food, and the abundance (that is, mean number of individuals per sample) of the two dominant congeneric species, *Calanus finmarchicus* and *C. helgolandicus*, of the genus *Pseudocalanus* and of the taxonomic group euphausiids. These indicators were derived using data (46,777 samples) collected by the Continuous Plankton Recorder (CPR) survey[10]. We first examined long-term monthly changes (period 1958–1999) in the plankton ecosystem of the North Sea (including the Skagerrak), applying a standardized Principal Component Analysis (PCA, table years–months × indicators). The first principal component (Fig. 1, 33.71% of the total variability) revealed a clear distinction between the periods 1963–1983 and both of the periods 1984–1999 and 1958–1962. The period 1963–1983 was characterized by high abundance of prey for larval cod (positive anomalies in the biomass of calanoid copepods, in the abundance of *C. finmarchicus*, euphausiids and *Pseudocalanus* spp.) and a high mean size of calanoid copepods. In twelve of the 21 years from 1963 to 1983 cod recruitment (one-year-olds) in the North Sea was high, in parallel with positive anomalies in the plankton ecosystem (Fig. 1). Estimated recruitment during this period was

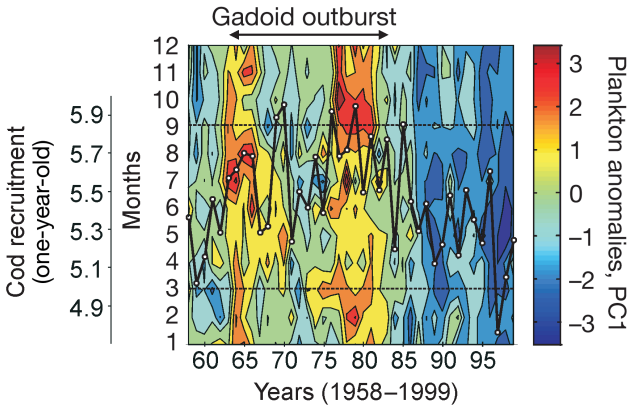

Figure 1 Long-term monthly changes (1958–1999) in the plankton index (as the first principal component, 33.78% of the total variability), resulting from analysis of the table years–months × biological indicators. The main variables related to this first principal component were, in order of importance, mean abundance (as mean number of individuals per CPR sample) of *C. finmarchicus* (normalized first eigenvector $C_m = 0.84$), euphausiids ($C_m = 0.72$), mean size of calanoid copepod ($C_m = 0.72$), *C. helgolandicus* ($C_m = -0.41$), calanoid copepod biomass ($C_m = 0.34$) and the genus *Pseudo-calanus* spp. ($C_m = 0.07$). A negative anomaly in the first principal component indicates a low value for all biological parameters with the exception of *C. helgolandicus* (opposite pattern) and *Pseudocalanus* spp. (no relationship). Cod recruitment (one-year-olds; in decimal logarithm) in the North Sea (curve in black) is superimposed with a lag of one year. The period of the 'gadoid outburst'[11] is indicated. Horizontal dashed lines indicate the period (March–September) of larval cod occurrence in the North Sea.