sperm number: Wilcoxon $T = 9.5$, $P = 0.83$, $n_{males} = 10$). Second, and more importantly, we found a positive correlation between mean ejaculate volume and male status ($r_s = 0.68$, $n_{males} = 10$, $P = 0.03$).

When female pre-copulatory behaviour cannot prevent copulations from suboptimal partners, females can reduce the fertilization success of an ejaculate by increasing the intensity of sperm competition[19] and by the differential ejection of sperm[3,5]. This is the first study to show that sperm ejection in the absence of any form of male manipulation is non-random and that male phenotype can consistently explain variations in sperm ejection. Although females of some species may select against the sperm of incompatible genotypes[6,20], incompatibility avoidance is unlikely to generate directional selection because it is usually inconsistent between females[21]. Artificial insemination studies investigating cryptic female choice[4] in fowl and other birds have failed to find evidence for incompatibility between the female reproductive tract or the ova and the sperm of individual males[22,23]. However, our results indicate that during natural copulation cryptic female choice may occur through a behavioural response before sperm is taken up into the sperm storage tubules, rather than through a physiological response to sperm, and, in addition, that it may be consistent between females and have a directional character.

Differential sperm ejection may allow female birds to bias sperm utilization in favour of a preferred male phenotype. Because the number of sperm reaching the sperm stores of the female is crucial in determining fertilization success in birds[24], differential sperm ejection is likely to have a strong impact on the outcome of sperm competition in this group and in other taxa[3,5]. Differential sperm ejection may allow females to influence paternity despite sexual coercion, and simultaneously reduce any costs associated with insemination and sperm storage[25,26]. □

Methods

We studied a free-living population of feral fowl of the Bankiva breed, which is morphologically and behaviourally similar to the red junglefowl. *Gallus gallus gallus*[10,27]. The investigation was conducted at the University of Stockholm from April to July 1998 when the study population consisted of 13 males and 21 females, and from April to July 1999 when the population consisted of 10 males and 13 females different from the previous year. Male hierarchy was established from the outcome of pairwise interactions[28] (male top rank = 1.83, bottom rank = 0.00). The birds were fully habituated and most copulations were observed from a distance ≤5 m. Solicited copulations were initiated by female crouching. In coerced copulations, a male mounted a female without her solicitation and this was followed by female resistance[10]. The probability of solicitation was measured as the proportion of copulations that were solicited by the female. The occurrence of sperm ejection was determined by observing whether an ejaculate was taken up or rejected through cloacal contractions within 5 s after insemination. Copulations where the ejaculate was not observed were not considered. The proportion of behaviourally successful copulations for which we could ascertain whether sperm ejection had occurred was 25.4% (87/343) in 1998. We collected natural ejaculates by allowing males to copulate with females provided with a false cloaca[29]. We collected semen with a Gilson pipette and measured semen volume (±0.5–1.0 μl) and the number of sperm using a standard method[30]. A general linear model was used to assess the importance of male status explaining the variation in the between-male probability of sperm ejection. The difference in social status and the mean probability of sperm ejection after the removal of six males were obtained as: d(status) = social status after removal − status before removal; d(ejection) = mean sperm ejection probability after removal − mean probability before removal.

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A universal pattern of mortality decline in the G7 countries

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Human lifespan has increased enormously this century[1–3]. But we remain uncertain about the forces that reduce mortality, and about the cost implications of ageing populations[4,5] and their associated social burden. The poor understanding of the factors driving mortality decline[2,6,7], and the difficulty of forecasting mortality[8–11] are due in part to the pronounced irregularity of annual to decadal mortality change[7,12]. Here we examine mortality over five decades in the G7 countries (Canada, France, Germany, Italy, Japan, UK, US). In every country over this period, mortality at each age has declined exponentially at a roughly constant rate. This trend places a constraint on any theory of society-driven mortality decline, and provides a basis for stochastic mortality forecasting. We find that median forecasts of life expectancy are substantially larger than in existing official forecasts. In terms of

the costs of ageing, we forecast values of the dependency ratio (that is, the ratio of people over 65 to working people) in 2050 that are between 6% (UK) and 40% (Japan) higher than official forecasts.

We examine long-term patterns in mortality rates, measured as central death rates $m(x,t)$ (that is, deaths in a year divided by mid-year population) in age class x in year t for both sexes. We use annual data for the period 1950–1994 for all countries except Italy (1951–1993) and Germany (1952–1990); here, Germany means the former West Germany. We use the age classes, 0 to 1 yr, 1 to 5 yr, and then 5-yr intervals up to age 105; time t is in single years. For countries except Japan, ages 85 yr and older are reported as a single lumped class; for Japan, we used accurate national data for all age classes.

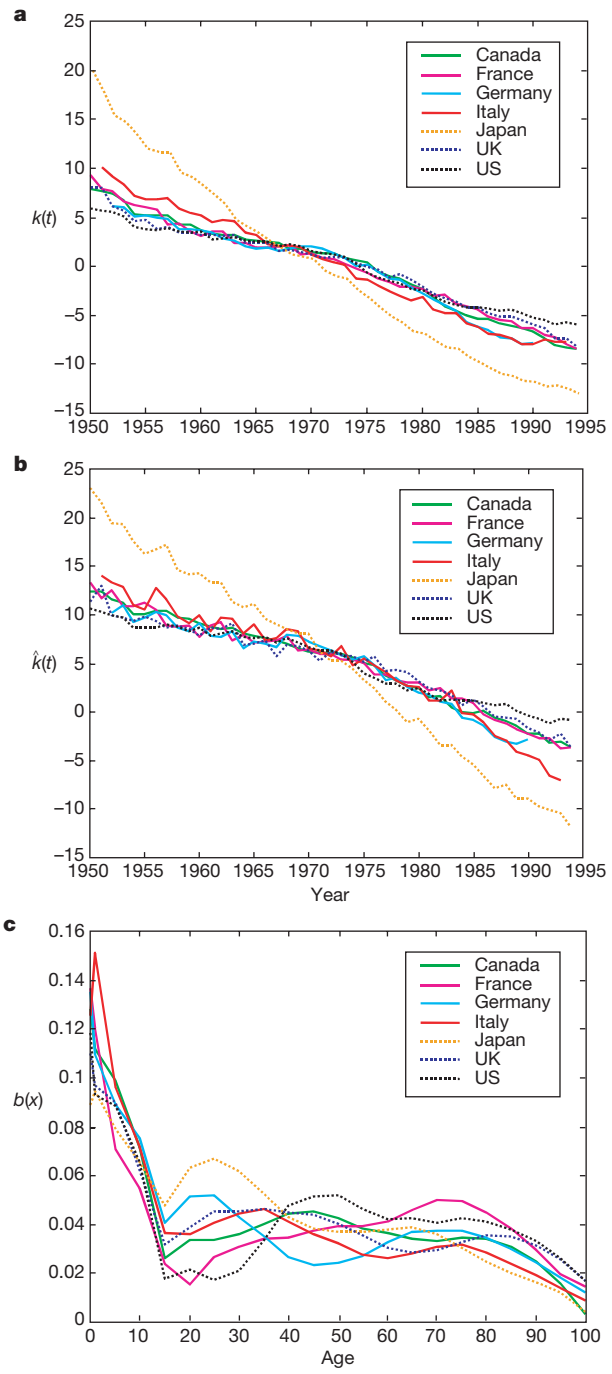

Figure 1 Dominant temporal patterns in the decline of mortality. **a**,**b**, Changes in age-specific central death rates are dominated by a single time factor $k(t)$, values of which are shown for the G7 countries (**a**). This time factor and its accompanying age factor, $b(x)$ account for over 94% of the temporal variation in the logarithm of the central death rates. To make forecasts that include variability not included in the dominant factor $k(t)$, we use a death-adjusted factor $\hat{k}(t)$ whose values are shown in **b**. **c**, The relative rate of progress against mortality at different ages. Mortality change at each age x depends on the product of the factor $k(t)$ and an age factor, $b(x)$, whose values are plotted versus age x for the G7 countries. The $b(x)$ for each country sum to one, so that the values shown indicate the relative rate of progress against mortality at different ages.

Our analysis echoes the pioneering work of Lee and Carter for the US[13]. We seek a dominant temporal 'signal' in the data—the simplest model that captures trend and variation in death rates—and to find it we use a singular-value decomposition[13–15] applied to the logarithms log $m(x,t)$[13,16,17]. For each age x we subtract the sample average $a(x)$ of the logarithm and obtain the decomposition

$$\log m(x,t) - a(x) = \Sigma_i s_i u_i(x) v_i(t)$$

The real singular values are $s_1 \geq s_2 \geq \ldots \geq 0$. The ratio of s_1^2 to the sum of squares of all singular values is the proportion of the total temporal variance in the transformed death rates that is explained by just the first term in the singular-value decomposition.

In every G7 country, the first singular value explains over 94% of the mortality variation (Table 1); therefore, we have a dominant temporal pattern, and we write

$$\log m(x,t) = a(x) + b(x)k(t) + E(x,t).$$

The single factor $k(t)$ corresponds to the dominant first singular value and captures most of the change in mortality. The far smaller variability from other singular values is $E(x,t)$. The dominant time factors $k(t)$ display highly linear long-term declines with superimposed short-term fluctuations (Fig. 1a). This linearity is supported by examination of differences (not shown) between consecutive values of $k(t)$; the term $E(x,t)$ has no long-term trend. Mortality decline at a particular age x depends on the profiles $b(x)$ (Fig. 1c): these are broadly similar but display differences attributable to social and historical factors[18]. We note that these age profiles will depend on the base period that we use for analysis: in comparing mortality change[19] before and after 1950, the pattern of decline is similar but the earlier data reflect rapid declines in infant and child mortality.

What accounts for this regular long-term decline in mortality? An argument may be made if we assume that mortality decline in this century has resulted from a sustained application of resources and knowledge to public health and mortality reduction. Societies typically allocate attention and resources in proportion to observed levels of mortality at different ages (for example, immunization programs against childhood disease). Such allocation would produce an exponential (proportional) change in mortality, although not necessarily at a constant rate over time. Over time, the rate of proportional decline depends on a balance between the level of resources focused on mortality reduction and their marginal effectiveness. Historically, the level of resources has increased over time but their marginal effectiveness has decreased over time (because,

Table 1 Proportion of variance of log $m(x,t)$ (both sexes combined) explained by the unadjusted mortality index $k(t)$ and the adjusted index $\hat{k}(t)$.

	Canada	France	Germany	Japan	Italy	UK	US
$k(t)$ (unadjusted)	0.9612	0.9428	0.9569	0.9520	0.9748	0.9479	0.9527
$\hat{k}(t)$ (adjusted)	0.9560	0.9323	0.9461	0.8912	0.9447	0.9321	0.9482

Table 2 The slope of adjusted mortality index $\hat{k}(t)$ and the value of in the adjusted model, for each country

	Canada	France	Germany	Italy	Japan	UK	US
Drift term (z)	0.3649	0.3877	0.3422	0.5043	0.7948	0.3427	0.2606
Standard error	0.3685	0.7017	0.7397	1.0367	0.7702	0.8765	0.3966

for example, we are confronted with ever more complex causes of mortality that require substantial resources or new knowledge). Our observation—that roughly constant long-run exponential rates of decline—implies that increasing level and decreasing effectiveness have balanced each other over long times. It is of course possible that the linear pattern of decline that we report has some other basis. For the future, we expect a continued increase in resources spent on mortality reduction and a growing complexity of causes of death. The balance between these could certainly shift if there were departures from history—for example, if new knowledge is discovered and translated into mortality reductions at an unprecedented rate. But this century has witnessed an amazing series of discoveries that have altered medicine and public health, and there is no compelling reason why the future should be qualitatively different. We therefore expect a continuation of the long-run historical pattern of mortality decline.

We use the long-term linear decline in $k(t)$ to forecast the expectation of life at birth, e_0, for the G7 countries. A naive forecast based on the long-run trend is not sensible because the short-term variation will accumulate over time. To incorporate short-term variation[20] we use a modified time factor defined by

$$\log m(x,t) \cong a(x) + b(x)\hat{k}(t)$$

and compute $\hat{k}(t)$ (Fig. 1b) to match historical total deaths in each year. This adjustment procedure is effective for the forecasts of life expectancy (e_0) we report here; the adjustment would have to be modified to forecast other quantities. In our view, differences in mortality trends between the sexes are mainly driven by short-term variations relative to aggregate mortality: for a sex-specific forecast we would model separately aggregate decline and the relative change between the sexes.

We use a stochastic model of decline,

$$\hat{k}(t+1) = \hat{k}(t) - z + \epsilon_t$$

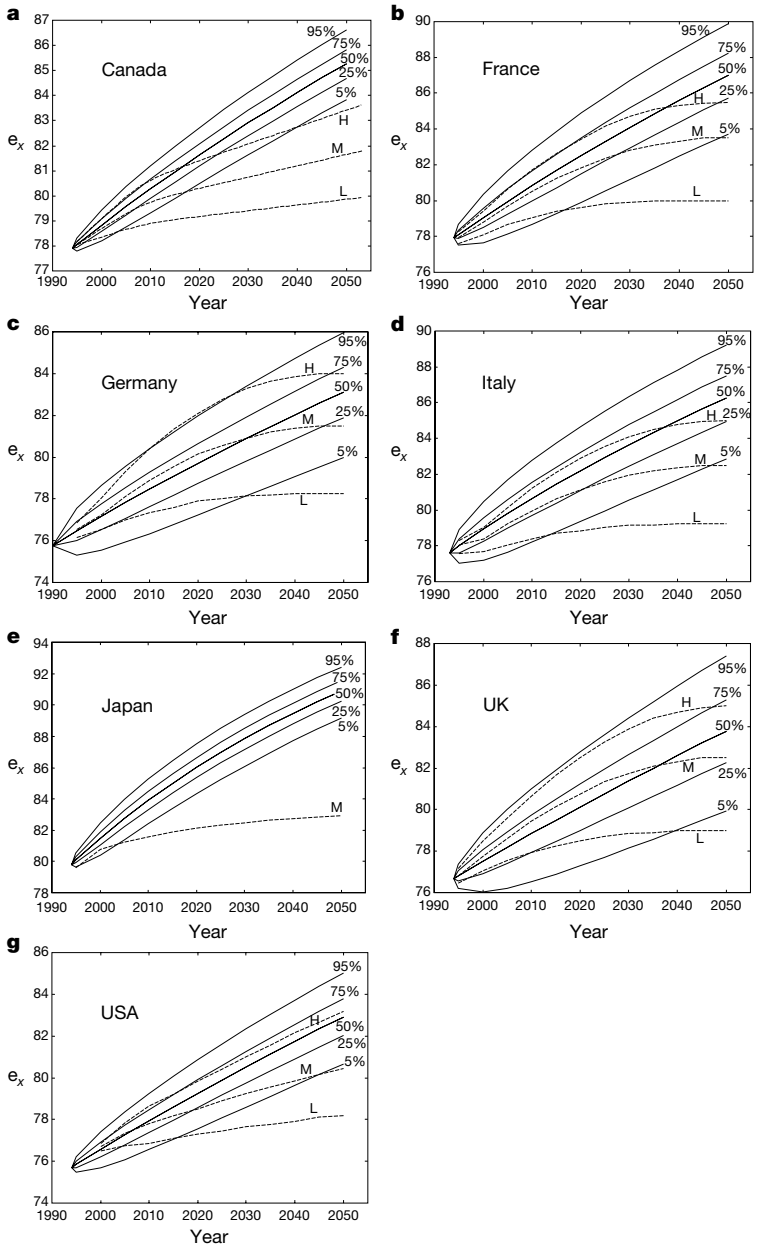

Figure 2 Forecasts of e_0, the expectation of life corresponding to mortality rates for each year from 1995 to 2050. Stochastic (solid lines) and official (dashed lines) forecasts of e_0 are shown for the G7 countries. For the stochastic forecasts, point-wise prediction intervals at the 5%, 25%, 50%, 75% and 95% levels are shown; for the official forecasts, high (H), medium (M) and low (L) outcomes are shown where they are available. Note the differences between stochastic and official forecasts in uncertainty over the span of the forecast and in the forecast levels.

Table 3 Stochastic forecasts of combined sex life expectancy at birth (e_o), compared with official forecasts

		2000		2020		2050	
	Forecast	Central	Range	Central	Range	Central	Range
Canada	S	78.83	1.24	81.64	2.23	85.26	2.78
	O	78.72	0.72	80.31	2.23	81.67	3.55
France	S	79.04	2.71	82.51	4.98	87.01	6.15
	O	78.80	1.30	81.85	3.85	83.50	5.50
Germany	S	77.16	3.07	79.71	4.75	83.12	5.97
	O	77.25	1.50	80.15	4.20	81.50	5.75
Italy	S	78.94	3.28	82.20	5.31	86.26	6.38
	O	78.40	1.35	81.15	4.05	82.50	5.75
Japan	S	81.47	2.07	86.01	3.20	90.91	3.27
	O	80.76	0.00	82.12	0.00	82.95	0.00
UK	S	77.50	2.85	80.13	5.50	83.79	7.47
	O	77.75	1.45	80.75	4.00	82.50	6.00
US	S	76.56	1.73	79.25	3.30	82.91	4.35
	O	76.70	0.35	78.50	2.55	80.45	5.00

Official forecasts have 'high', 'medium', and 'low' variants, except that there is only one official forecast for Japan[23]. For France, Germany, Italy and the UK, mortality forecasts are those published by Eurostat (Data Shop Eurostat, Luxembourg)[24]; For Canada, the forecasts follow assumptions of the Canadian Pension Plan[25,26]. S, stochastic forecast: central (50%), range (95–5%). O, official forecast: central (medium), range (high–low).

where $z > 0$, and ϵ_t is a series of independent normal stochastic disturbances. This model has been used earlier to forecast mortality[13] and population[21] for the US. In countries other than Japan, age-specific mortality over age 85 is estimated using a model schedule[7,17].

The rate of decline z (Table 2, first row) is in the range of 0.3 to 0.5 per year in the US, Canada and the European countries, and is highest in Japan at about 0.8 per year. Note that exponential decline at a constant rate implies that the absolute annual decrement in mortality will diminish over time so that, for example, the rate of decline of life expectancy will also diminish over time. There is substantial short-term variability in the annual rates of decline (Table 2, second row).

Our forecasts advance from a launch date using the stochastic model to generate a probability distribution of forecast values in each year. Figure 2 exemplifies the difference between our stochastic forecasts of e_0 and official forecasts based on scenarios. For example, the median (50th percentile of probability) stochastic forecast for France is about 3.5 yr higher in 2050 than the central official forecast. Comparisons for all countries (Table 3) show stochastic median forecasts in 2050 that are higher than official ones by an astounding 8 yr for Japan, to a relatively low 1.3 yr for the UK. Official forecasts may be strongly influenced by short-term slowdowns in mortality change or by conservative expert opinion[7,12]. To see why this matters, note that a 1-yr difference in e_0 corresponds to a difference of over 5% in the dependency ratio (population over 65 divided by population ages 20–64; we use the life-table ratio here, a good approximation for the G7). The dependency ratio is a useful direct index of the costs of ageing, so we project median dependency ratios, and thus costs of ageing, higher by between 6 and 40% than do official forecasts. Beyond 2050, this forecast divergence would increase, because virtually all official scenarios assume that mortality decline will eventually slow (or stop)[22]. Figure 2 and Table 3 also display differences in forecast uncertainty, which provides a measure of demographic and policy risk. Uncertainty grows much more quickly in the stochastic forecast, but by 2050, official 'high' and 'low' patterns spread over about the same range.

Our forecasts have large implications for the financing of public programs of old-age support and pensions, as well as private and public health insurance. In the US, for example, the finances of Social Security and medical care for the elderly will be driven by the ageing of the baby boom in the next two decades, but the longer term finances will turn on the sustained increase in human life span. The demographic forecasts that shape policy analyses related to old-age support should reflect the regularity of mortality change that we describe. □

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Ultrasensitive pheromone detection by mammalian vomeronasal neurons

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The vomeronasal organ (VNO) is a chemoreceptive organ that is thought to transduce pheromones into electrical responses that regulate sexual, hormonal and reproductive function in mammals[1–5]. The characteristics of pheromone signal detection by vomeronasal neurons remain unclear[3,5]. Here we use a mouse VNO slice preparation to show that six putative pheromones