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A male–female longevity paradox in medfly cohorts

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Summary

1. A long-standing question in biology is whether longevity is greater in females or in males for most non-human species. This is an open question for the majority of species because little is known about the nature of the underlying mortality differences.
2. Examination of mortality data on approximately 600 000 medflies of each sex revealed a demographic paradox—male medflies possessed the higher life expectancy (average longevity) but female medflies were usually the last to die.
3. The underlying demographic cause of this incongruency was a male–female mortality crossover—females exhibited higher mortality than males to around 3 weeks, lower mortality than males from about 3–8 weeks, and mortality approximately equal to that of males thereafter.
4. The findings help explain the ambiguity of male–female longevity differences in the literature, suggest that relative male–female survival cannot be used as a proxy for sex mortality differences, shed light on sex biasing of older ages, and underscore the difficulties with comparative aspects of ageing.
5. We propose a general framework for sex–mortality differentials in which the underlying mortality factors are grouped into three interrelated categories: constitutional endowment, reproductive biology and behaviour. This framework provides conceptual structure as well as insights into how complex patterns in the sex–mortality ratio can arise.

Key-words: *Ceratitis capitata*, gender gap, sex biasing, sex mortality differentials.

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Introduction

While women generally outlive men by a margin of 4–10 years throughout the industrialized world (Stolnitz 1957; Hazzard 1986; Holden 1987; Waldron 1987), a long-standing question in biology is whether this female advantage in longevity is a general characteristic of most non-human species as well. The scientific literature contains conflicting views. For example, Hazzard (1990) states 'The greater longevity of females than males appears to have a fundamental biological basis. Studies of comparative zoology suggest that greater female longevity is virtually universal.' Hamilton & Mestler (1969) begin their paper with the statement, 'Males tend to die at an earlier age than females in most species of animals for which data are available.' Brody & Brock (1985) state, '...there are basic and fundamental questions posed by the fact that female survival [advantage] seems to be one of the most pervasive findings within the animal kingdom.' In contrast, the paper by Lints *et al.* (1983) made 218 comparisons between female and male life span in *Drosophila melanogaster* and concluded that mean life span of females exceeds that of males in only about half the cases. Smith (1989) notes, '...while there is some evidence that adult populations of many animal species contain more females than males, most of these studies do not consider survival to an age approaching the potential limit for the species as implied by the word longevity.' Gavrilov & Gavrilova (1991) state 'The hopes connected with the search for general biological mechanisms underlying these [sex] differences seem to be in vain, since, despite the widespread opinion to the contrary, the greater life span of females is not in itself a general biological regularity.'

In examining sex-specific demographic data from a

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large-scale medfly *Ceratitis capitata* (Wiedemann) life table study (experiment 3 in Carey *et al.* 1992), we discovered a paradox with respect to male–female life table traits—in 167 cohorts averaging 7200 flies each, males usually possessed the higher life expectancy but females were usually the last to die. These patterns suggested that the underlying mortality schedules for the sexes were inconsistent with several long-held assumptions about the nature of sex mortality and longevity differences in the population biology, ecology and gerontology literature (Hamilton 1948; Fisher 1958; Trivers 1972; Charlesworth 1980; Charnov 1982; Clutton-Brock & Iason 1986; Smith & Warner 1989); that the sign of mortality differences is unchanging throughout the adult life course, that females are typically longer lived than males and that sex ratio biases always favour one sex at all ages.

The demographic incongruency in medfly male–female longevity can only be explained as due to an underlying mortality crossover where age-specific death rates of one sex must be higher up to a particular age and lower thereafter (Manton, Poss & Wing 1979; Manton & Stallard 1984; Petersen & Petersen 1986; Coale & Kisker 1986). Because a demographic relationship of the type described by the medfly data has not been previously documented in a non-human species and, more generally, reliable data on age-specific mortality in most non-human species are relatively rare (Promislow & Harvey 1990; Promislow 1991), the specific objective of this research was to confirm the existence of mortality crossovers in the medfly cohorts. The broad importance of this research concerns two sex models in demography (Goodman 1953, 1967; Keyfitz 1966; Das Gupta 1973; Schoen 1978; Caswell 1989; Pollack 1986, 1987), forecasting sex differentials in mortality (Carter & Lee 1992) and comparative-studies on male–female ageing (Kitagawa & Hauser 1973; Peterson 1975; Preston 1976; Comfort 1979; Lopez & Ruzicka 1983; Partridge 1986; Greenwood & Adams 1987; Tatar & Carey 1994; Tatar, Carey & Vaupel 1994). Indeed, Brody & Brock (1985) believe that sex differences alone provide one of the most promising areas of research into longevity available to science.

Methods

Studies were conducted at the Moscamed medfly mass rearing facility located in Metapa, Chiapas, Mexico (see Vargas 1989 for technical details on fruit fly mass rearing). Three separate experiments were conducted, as originally described in Carey *et al.* (1992): medflies of both sexes were maintained in solitary confinement in experiments 1 and 2 and in grouped cages in experiment 3. Adult flies in the three experiments were maintained under the following environmental conditions. Experiment 1: continuous light, 25·2°C (±2°C), 67% relative humidity (±8%); experiment 2: 12:12 light–dark cycle, 25·6°C (±2°C), 67·5% relative humidity (±8%); experiment 3: 12:12 light–dark cycle, 24·0°C (±2°C) and 65% relative humidity (±9%). In experiment 1 a single pupa and adult food (3:1 sugar to protein dry mixture) were placed in 1-oz cups. The cups were then attached by the upper rim to the underside of a 60 cm × 90 cm screened tray which, in turn, was placed in a vertical holding rack. Water was supplied to each fly with a moist dental wick. Flies in experiment 2 were also confined alone. Conditions in this experiment differed from those in experiment 1 in three respects—3·5-ml tissue culture cells (Falcon® 24-cell units) were used rather than the 1-oz cups, sugar alone was the food source, and flies obtained water from a layer of saturated cotton placed on top of the cells. For experiment 3 pupae were sorted into one of five size classes using a pupal sorter. This enabled size dimorphism to be eliminated as a potential source of sex-specific mortality differences. Approximately 7200 medflies (both sexes) of a given size class were maintained in each of 167 mesh-covered, 15 cm × 60 cm × 90 cm aluminum cages. Adults were given a diet of sugar and water, *ad libitum*, and each day dead flies were removed, counted and their sex determined. Mortality rates were determined for a total of 21 204 individuals in experiment 1, a total of 27 181 individuals in experiment 2 and a total of 1 203 646 individuals in experiment 3.

Demographic methods used in the analysis follow those given in Chiang (1984), Manton & Stallard (1984) and Carey (1993). Three main parameters were used in the analysis: (i) age-specific mortality, q_x, defined as the fraction of individuals alive at age x dying in the interval x to $x+1$; (ii) age-specific survival, l_x, defined as the fraction of the initial number of individuals remaining alive at age x; and (iii) expectation of remaining life at age x, e_x, defined as the average remaining lifetime for an individual who survives to age x. The first two parameters computed for males and females were used to determine two ratios: (i) sex–mortality ratio, which is the age ratio of the q_x schedules for males and females; and (ii) sex–survival ratio, which is the age ratio of the l_x schedules for males and females.

Results

SEX AND AGE-SPECIFIC MORTALITY

The age-specific mortality rates for male and female medflies are given in Fig. 1 and the Appendix. Female mortality increased more rapidly at young ages than did male mortality. However, at day 16 female mortality abruptly began to level off while male mortality continued to increase, thus causing a mortality crossover at around 20 days. Male mortality peaked at the same time as female mortality and mortality in both sexes began declining at about the same age. Male and female mortality rates were similar at ages beyond 60 days.

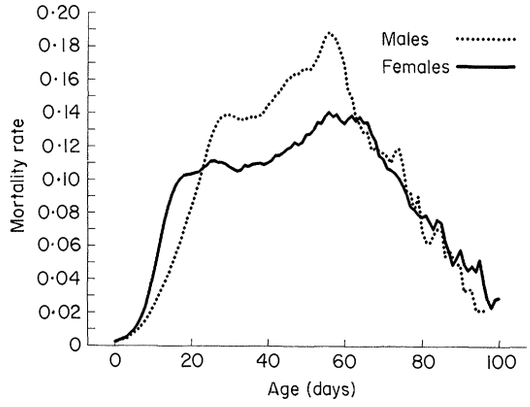

Fig. 1. Smoothed male and female age-specific mortality rates from cohorts consisting of approximately 600 000 medflies of each sex. Curves were smoothed using a 7-day running mean (geometric).

SEX MORTALITY AND SURVIVAL RATIOS

The ratio of the age-specific male and female mortality schedules showed both the relative differences in the mortality levels and the patterns of convergence, crossover and divergence (Fig. 2). The sex–mortality ratio indicated that the greatest relative difference between male and female mortality at the younger ages occurred at around day 16, when male rates differed from female rates by a factor of 0·7. After the mortality crossover, male mortality was higher than that for females by a factor of around 1·3 from 30 to 60 days. The effects of these mortality differences on the relative abundance of each sex were not offsetting, since the male advantage occurred when the rates of both sexes were relatively low whereas the female advantage occurred when the rates of both sexes were relatively high.

The numerical consequences of these mortality ratios on the relative abundance of each sex is indicated by the sex–survival ratios—the ratio of male to female survival rates. At 25 days the males outnumbered females by 1·6-fold. However, by 40 days the number of each sex was equal and at older ages the number of males was only 0·4 that of females. These trends show that age-specific sex ratio is neither fixed nor biased towards only one sex at all ages. The relationship between sex mortality rates and relative abundance reveals that the sex–survival ratio cannot be used to estimate sex–mortality differentials. Three comparisons illustrate this point: (i) from ages 0 to 14 the sex–survival ratio was constant and near unity, but the proportional sex–mortality differences were high and increasing; (ii) at 40 days the proportional mortality differences were again high (though reversed) and constant, however the male–female ratio was near unity and decreasing; and (iii) at ages greater than 60 days the cohort was strongly female biased but the sex–mortality differences were virtually non-existent.

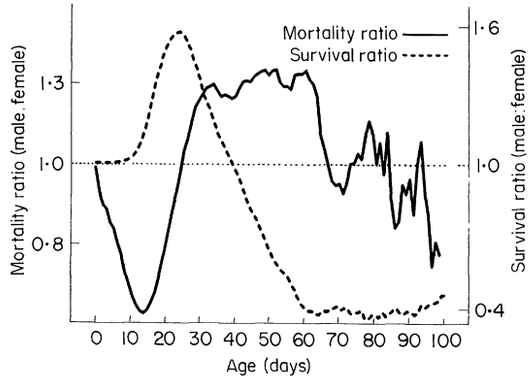

Fig. 2. Sex–mortality ratios for medflies (male–female age-specific mortality ratios) using the smoothed rates shown in Fig. 1 and the sex–survival ratio (ratio of male–female survival schedules).

COHORT VARIABILITY

Evidence of the occurrence of male–female mortality crossovers in most of the 167 cages is shown in Fig. 3. The clustering of points above the isometric (diagonal) line for mortality rates at 10 days reveals that female mortality exceeded that of males in nearly 90% of all cages at this early age. In contrast, the clustering of points below the isometric line for mortality at 30 days indicates that male mortality exceeded that for females in over 95% of all cages at this later age.

Further evidence of the widespread occurrence of mortality crossovers is given in Fig. 4, which shows sex-specific expectation of life at ages 0 and 30. The expectation of life at eclosion (age 0) for males exceeded that for females in over 95% of all cages, but life expectancy at day 30 for females exceeded that for males in over 90% of the cages. The net result of

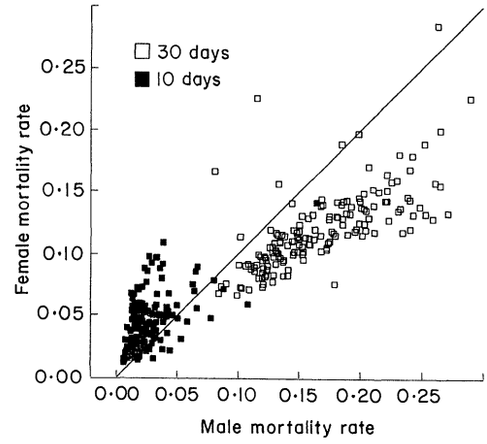

Fig. 3. Male versus female mortality rates at 10 and 30 days for 167 medfly cohorts of approximately 3600 individuals of each sex. The diagonal line is the isometric line, where male = female mortality. Thus a point above the line indicates that female mortality within the cage was higher at the specified age than was male mortality at that age. Sex differences in mortality among all cages at both 10 and 30 days were statistically significant ($P < 0{\cdot}001$).

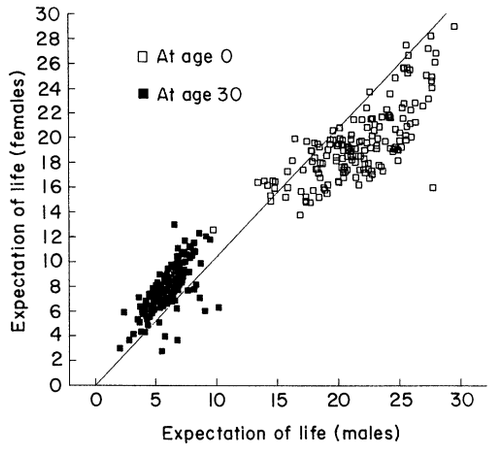

Fig. 4. Male versus female expectations of remaining life at 0 and 30 days for 167 medfly cohorts of approximately 3600 individuals of each sex. The diagonal line is the isometric line, where male = female expectation of life. Thus a point above the line indicates that female expectation of life at the specified age within the cage was higher than was male expectation of life at that age. Sex differences in expectations of life among all cages at 0 and 30 days were statistically significant ($P < 0{\cdot}001$).

the crossover was to bias the cohort in favour of females at older ages. The last fly to die in a cage was four times more likely to be a female than a male (Fig. 5).

COHORTS MAINTAINED IN SOLITARY CONFINEMENT

A male–female mortality crossover and female bias at older ages was also evident in mortality data from the two experiments in which medflies were maintained in solitary confinement, as shown in Fig. 6. In both cases male mortality was less than female mortality until 18–22 days when the mortality crossover occurred—female mortality was then lower than male mortality. The male : female ratio at older ages ranged from 0·7 to 0·8 at older ages for both experiments, as shown in the survival ratios. These findings for medflies that were held in uncrowded environments and were not allowed to mate or reproduce suggest that the male–female mortality crossover and female bias at older ages is due to differences in the basic biology of the sexes and is not unique to conditions for either mated flies or those maintained in groups. The combined effects of mating and density, as observed in the mortality data for flies maintained in cages, amplifies the sex–mortality differential but does not change its fundamental pattern.

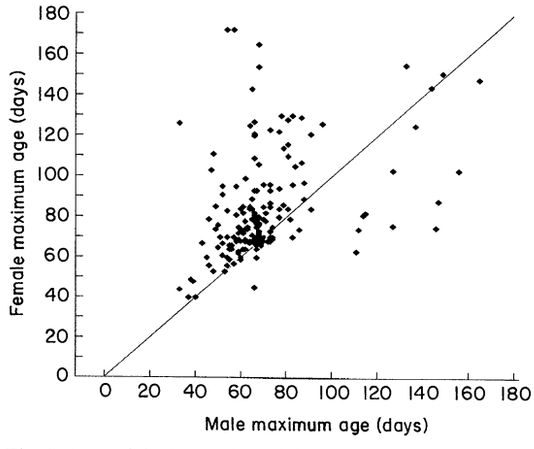

Fig. 5. Ages of the last male and the last female to die in each of 167 medfly cohorts of approximately 3600 individuals of each sex. The diagonal line is the isometric line, where male = female oldest age. Thus a point above the line indicates that the last female to die within the cage was older than the last male to die. Sex differences among all cages in oldest age attained were statistically significant ($P < 0{\cdot}001$).

MORTALITY CROSSOVER EXPLANATIONS

There are two possible explanations for the male–female mortality crossovers. The first explanation is that the mortality crossover could be an artifact of compositional change in the male and female subpopulations due to demographic heterogeneity (Vaupel, Manton & Stallard 1979; Vaupel & Carey 1993). As populations age, they become more selected because individuals with higher death rates will die out in greater numbers than those with lower death rates, thereby transforming the population into one consisting mostly of individuals with low death rates

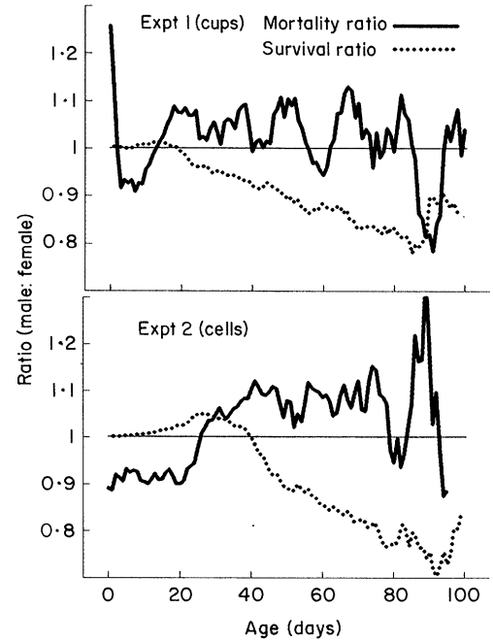

Fig. 6. Sex–mortality and survival ratios for medflies reared in solitary confinement. Approximately 21 000 individuals were used in experiment 1 (top) and approximately 27 000 individuals were used in experiment 2 (bottom).

(Rogers 1992). This explanation is also referred to as the 'cohort-inversion model' which is based on the concept that cohorts experiencing particularly hard or good times early in life will respond inversely later in life (Hobcraft, Menden & Preston 1982; Elo & Preston 1992). Thus the possibility exists that male–female mortality rates crossed because of heterogeneity at the cohort level. Perhaps females at emergence were, on average, frailer than males and there was also greater variance in female frailty; there may have been a relatively large proportion of frail females and of robust females compared with males.

A second explanation is that biological differences between males and females existed at the individual level and were manifested as differences in age-specific mortality. These sex-specific differences could include mating behaviour (Matthews & Matthews 1978), physiology (Engelmann 1970; Chapman 1971), reproductive costs (Reznick 1985; Tatar *et al.* 1994) and hormonal activity (Engelmann 1968). A finding that suggests the existence of differences at the individual level is the male–female survival crossover at 40 days (Fig. 2). The reason for the importance of the survival ratio is that it represents the cumulative mortality experience of the population. If the cumulative mortality advantage is eliminated, as is the case with the male and female survival shown in Fig. 2, then a mortality selection model cannot be a complete explanation of the mortality differentials (Manton & Stallard 1984).

Manton & Stallard (1984) believe that as a complement to the concept of crossover, attention should be paid to the concept of a peak mortality differential—the age at which the greatest proportional difference exists between the age-specific mortality rates for the two populations. They suggest that it is quite possible that an explanation of the peak differential may serve to explain mortality convergence and crossover. They state, 'This is because once it is understood why the peak differential occurs at that point in the age range, the later convergence and crossover of the two mortality curves may turn out to be a natural consequence of the mortality dynamics involved in the explanation.'

It is likely that the age patterns for the medfly sex–mortality ratio are at least partly due to differences in both physiological and behavioural costs of reproduction (e.g. gonadal activity and mating), as reflected in two peaks in mortality differentials. The first peak in the sex–mortality ratio occurred at about 2 weeks, when mortality was lower for males than for females; egg production in females is greatest from 7 to 14 days (Vargas 1989). The second peak was flatter than the first and occurred at around 30 days. This was after the mortality crossover and extended through ages when male mortality exceeded female mortality; this period occurred when female egg production was low or nil (Carey, Yang & Foote 1988) and male mating activity was reduced.

Discussion

A GENERAL FRAMEWORK FOR SEX–MORTALITY DIFFERENTIALS

Despite the ambiguity of much of the data in the non-human biological literature, explanations for differences in male–female mortality have been framed around the assumption that males of most species experience higher mortality rates than females. The two most common explanations (Trivers 1972; Smith & Warner 1989) on why males (putatively) have shorter lifespans are (i) behavioural aspects where males of many species are at higher risk due to different life-history requirements than females, such as mate finding and territory defence; and (ii) the chromosomal hypothesis where, it is suggested, females have an advantage because in most species females are the homogametic sex (XX) whereas males are the heterogametic sex (XY). It is argued that having two X chromosomes is advantageous because the X chromosome is three times the size of the Y chromosome and contains far more expressed genetic information, most of which is for functions and molecules unrelated to the female geneotype (Montagu 1974; Sandberg 1983; Smith & Warner 1989; Greenwood & Adams 1987; Zuk 1990).

In contrast to the ambiguity of male–female mortality differences in the biological literature, human data on sex mortality differentials in the demographic literature are unequivocal—females experience lower mortality than males at virtually all ages in almost every contemporary society (Hazzard 1986; Keyfitz & Flieger 1990). Two explanations have been proposed to account for the female longevity advantage in humans (Wingard 1984). The first is a biological explanation, that women are biologically 'more fit' than men. This explanation includes the chromosomal hypothesis as well as arguments related to the protective effects of female sex hormones such as oestrogen or to the deleterious effect of testosterone in males (Alexander & Stimson 1988; Hazzard 1990; Hazzard & Applebaum-Bowden 1990). The second explanation for why women outlive men concerns social, lifestyle and environmental factors—men behave in ways more damaging to health, such as smoking, consuming alcohol, encountering occupational hazards and violence. It has been suggested that the cigarette smoking sex differential may account for over half of the sex differential in longevity in the USA (Hazzard 1986).

In light of reports in both the demographic and the biological literature on sex–mortality differentials, as well as the results of the current study on the medfly, we believe that the underlying mechanisms for sex–mortality differentials can be grouped into three interrelated and co-evolved categories. The first category is 'constitutional endowment', which includes all structural, physiological, endocrinological and immu-

nological factors affecting the ability of individuals of each sex to resist disease, stress, physical challenge and deterioration. This category is concerned with overall 'fitness' and includes the direct and indirect effects of chromosomal differences between males and females. The second category concerns factors associated with sex-specific 'reproductive biology', including the effects of male and female hormones, gonadal development and production of eggs or offspring. This group of factors is concerned with processes typically classified as costs of reproduction (Reznick 1985; Stearns 1992); for example, virgin insects typically exhibit lower mortality rates than individuals which mate and reproduce (Partridge & Farquhar 1981; Partridge 1986; Carey, Krainacker & Vargas 1986; Partridge & Fowler 1992; Tatar *et al.* 1994). The third category of factors that influence the sex–mortality differential is 'behavioural predispositions'. These include behavioural traits evolved to maintain territories as well as the 'high risk–high stakes' strategy of males of many species for locating, competing for and defending mates (Zuk 1990).

Factors interact within and between categories. For example, gender-related exposure to parasites will be affected by differences in male and female behaviours (Bundy 1989; Tinsley 1989). Once infected the immunological response (endowment) of each sex will modulate survival of parasites which, in turn, is influenced to a large degree by sex hormones (Alexander & Stimson 1988). The interaction of the three sex-specific factors—endowment, reproductive biology and behaviour—determine the overall 'susceptibility to death' for each sex which, when filtered through environmental, biological and other factors, produces a 'probability of death'. The two concepts—susceptibility and probability—are not equivalent (Kannisto 1991). This is because mortality often runs counter to constitutional frailty due to behavioural factors. As Kannisto (1991) notes, boys die of accidents more frequently than girls, not because boys are more frail but because they take greater risks; reproducing females often experience higher death rates than males of the same age due to the high cost of offspring production and not due to differences in frailty, *per se*. In both cases, however, the sex differentials in risk-taking or in costs often diminish with age. Consequently differences in frailty or endowment may account for most of the sex mortality differential at older ages, whereas differences in both behaviour and reproductive biology between the sexes may account for the largest proportion of the sex mortality differential at younger ages, *ceteris paribus*. The combination of all factors will ultimately determine the overall 'sex survival differential' at advanced ages because survival is cumulative; differential mortality at young ages will affect the relative survival to older ages and thus influence the sex bias at advanced ages.

DEMOGRAPHIC IMPLICATIONS OF THE MORTALITY CROSSOVER

Discovery of a male–female mortality crossover has several implications in population biology, demography and gerontology. One implication involves the specific question of male–female longevity differences. Without an awareness of the nature of the male–female mortality schedules, and specifically the mortality crossover, sex differences in longevity could be interpreted in three ways: (i) that males are longer lived if life expectancy at emergence is used as the longevity criterion; (ii) that females are longer lived if oldest age attained is the longevity criterion; and (iii) that males and females are equally long lived if the cohort numbers are used as the longevity criterion to day 10, when the numbers of each sex were approximately equal, or several days before and after day 40, when the sex ratio was at or near 50 : 50 due to the mortality crossover. Indeed, ambiguous and conflicting reports on longevity sex differentials are common in the literature (Lints *et al.* 1983; Clutton-Brock & Iason 1986; Smith & Warner 1989; Ehrlich, Launer & Murphy 1984).

A second implication is that the results cast doubt on the assumption that sex ratio is biased toward only one sex throughout the life course of most species. The consequence of the mortality crossover around day 20 and its persistence through day 60 was to shift the sex ratio from a male bias at young ages to a female bias at middle ages. Females were the last to die, not because female mortality was lower at the older ages, but because mortality rates of both sexes were low and mortality differentials were small at advanced ages. This aspect is important because it sheds light on the dynamics of sex biasing. For example, the sex ratio at birth in humans is 1·05 (male:female) in most developed countries such as the USA. However, by 30 years of age the sex ratio is 50:50 due to excess male mortality. By age 85 females outnumber males by 3:1 (Taeuber & Rosenwaike 1992).

A third assumption that is that sex ratio cannot be used as a proxy for sex mortality differentials for two reasons: (i) survival differences not only lag behind mortality differences, but may result from mortality patterns that are exceedingly complex, as shown in Fig. 2. This complexity is not revealed in survival differences because survival is cumulative; and (ii) growing populations contain smaller fractions of their total membership in the older age classes than do stationary or declining ones. Therefore, a population in which a male–female mortality crossover exists may be biased toward one sex when it is increasing but biased toward the other sex when it is decreasing (Keyfitz 1985). Thus shifts in population sex ratio may reflect changes in growth rate and not changes in relative male–female mortality patterns.

A fourth implication concerns the population

biology of species when two sexes are considered. For example, Werren & Charnov (1978) demonstrated that there are exceptions to the argument by Fisher (1958) that differential mortality between sexes will have no effect on the equilibrium 1:1 sex ratio. Specifically they showed that, because most populations in nature are not at a demographic equilibrium (stability), selection can favour genes which result in the temporary overproduction of one or the other sex. Another example of an implication in population biology of differential mortality between sexes is the finding of Caswell & Weeks (1986) who demonstrated that complex bifurcation patterns may occur in populations if male and female survival differences exist and there is interstage competition for mates. This finding is important because, as Caswell (1989) notes, selection on the primary sex ratio when there is no such equilibrium adult sex ratio is an unsolved question.

A final implication is that discovery of the mortality crossover challenges long-held views on ageing research, including the nature of senescence and the use of certain life-table parameters. For example, the mortality crossover makes it impossible to neatly classify two populations according to any of a number of demographic metrics widely used in ageing studies. If the relative rate of change in mortality with age is used to compare senescence rates between two populations, then senescence for males relative to females is lower from ages 0–20 days, higher from 21–60 days and the same from 60 days onward. If life expectancy differences at eclosion are used as the criteria for differences in ageing rates, then female medflies age more rapidly than males. However, the majority of the 2·5-day gap in male–female life expectancies at eclosion can be explained as due to sex–mortality differences at the relatively youthful ages between 11 and 20 days (Carey 1993).

That the complex dynamics of sex–mortality differentials in non-human species has previously been unrecognized is of little surprise. This is because the vast majority of life-table studies on non-human species are based on relatively small numbers of individuals, because determining sex-specific mortality and sex ratio in the field is exceedingly difficult and because most life-history studies have been concerned historically with survival rather than mortality differences. As Ehrlich *et al.* stated (1984): 'Thus a seemingly simple thing like sex ratio is, in detail, quite complex both to define and to estimate.'

It is doubtful that sex–mortality differentials observed in the laboratory for any species including the medfly would be similar to those in the field. Unlike populations in the field which usually consist of individuals in a variety of different ages, the laboratory medflies were maintained as same-aged cohorts within each cage at densities far higher than would ever be experienced in nature. Consequently extrapolation of specific findings such as crossover age to field situations is probably not valid. However, the general finding that male–female mortality rates may crossover under some circumstances is important in a broader context and raises the likely possibility that sex-specific differences in life tables for many species may be far more complicated than previously realized. Indeed, whether males or females live longer may be equivocal in some species. However, as Zuk (1990) notes, generalization of specific results on male–female mortality differences from one species, that is adapted to a particular set of circumstances, to other species, is probably risky. We believe that future biological research focusing on causal mechanisms underlying convergence, crossover and divergence of male–female mortality rates with age will be more important to understanding gender differences in ageing than will a continuing quest to demonstrate the universality of a female longevity advantage.

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Appendix

Survival and age-specific mortality rates (q_x) for approximately 600 000 medflies of each sex for experiment 3. [$q_x = 1-(N_{x+1}/N_x)$]

	Males		Females	
Age (x)	Number living (N_x)	Age-specific mortality (q_x)	Number living (N_x)	Age-specific mortality (q_x)
0	598 118	0·00000	605 528	0·00000
1	598 118	0·00150	605 528	0·00138
2	597 220	0·00413	604 693	0·00388
3	594 752	0·00495	602 346	0·00520
4	591 806	0·00572	599 214	0·00704
5	588 423	0·00659	594 996	0·00847
6	584 548	0·00884	589 954	0·01070
7	579 382	0·01014	583 644	0·01450
8	573 509	0·01420	575 184	0·01862
9	565 364	0·01735	564 472	0·02633
10	555 554	0·02350	549 610	0·03620
11	542 496	0·02561	529 713	0·05040
12	528 604	0·03100	503 016	0·06014
13	512 216	0·03869	472 764	0·08070
14	492 399	0·04090	434 612	0·08898
15	472 261	0·04806	395 941	0·10106
16	449 563	0·05328	355 926	0·10400
17	425 609	0·06250	318 911	0·10161
18	399 008	0·06852	286 506	0·10229
19	371 667	0·07441	257 199	0·10027
20	344 010	0·08405	231 410	0·10452
21	315 095	0·09198	207 224	0·10413
22	286 111	0·09764	185 645	0·10424
23	258 176	0·10454	166 293	0·10789
24	231 186	0·11243	148 351	0·10677
25	205 193	0·12195	132 511	0·10629
26	180 170	0·13795	118 426	0·11763
27	155 315	0·14703	104 496	0·11363
28	132 479	0·14571	92 622	0·12237
29	113 176	0·14356	81 288	0·10637
30	96 928	0·13644	72 641	0·10107
31	83 703	0·13222	65 299	0·10757
32	72 636	0·13060	58 275	0·09965
33	63 150	0·13960	52 468	0·10542
34	54 334	0·14146	46 937	0·10595
35	46 648	0·13900	41 964	0·11291
36	40 164	0·13281	37 226	0·11108
37	34 830	0·14643	33 091	0·12061
38	29 730	0·12879	29 100	0·10165
39	25 901	0·14019	26 142	0·10971
40	22 270	0·13601	23 274	0·10712

Appendix (*Continued*)

	Males		Females	
Age (x)	Number living (N_x)	Age-specific mortality (q_x)	Number living (N_x)	Age-specific mortality (q_x)
41	19 241	0·14932	20 781	0·10813
42	16 368	0·15457	18 534	0·10856
43	13 838	0·15956	16 522	0·11730
44	11 630	0·16148	14 584	0·12994
45	9752	0·15330	12 689	0·12263
46	8257	0·15744	11 133	0·11075
47	6957	0·15998	9900	0·11939
48	5844	0·17728	8718	0·12377
49	4808	0·16452	7639	0·11441
50	4017	0·17376	6765	0·13821
51	3319	0·16270	5830	0·12075
52	2779	0·16877	5126	0·13305
53	2310	0·16364	4444	0·14311
54	1932	0·15269	3808	0·12237
55	1637	0·19914	3342	0·14123
56	1311	0·19375	2870	0·14530
57	1057	0·21381	2453	0·13412
58	831	0·20818	2124	0·15113
59	658	0·18997	1803	0·14920
60	533	0·15009	1534	0·12516
61	453	0·13245	1342	0·13040
62	393	0·15013	1167	0·11654
63	334	0·13772	1031	0·13288
64	288	0·10417	894	0·15324
65	258	0·17829	757	0·16513
66	212	0·11321	632	0·12500
67	188	0·11702	553	0·14286
68	166	0·09639	474	0·10759
69	150	0·15333	423	0·11584
70	127	0·06299	374	0·08556
71	119	0·10924	342	0·12281
72	106	0·16981	300	0·10333
73	88	0·10227	269	0·11152
74	79	0·10127	239	0·10460
75	71	0·07042	214	0·09813
76	66	0·19697	193	0·10363
77	53	0·07547	173	0·06358
78	49	0·04082	162	0·09259
79	47	0·06383	147	0·06803
80	44	0·09091	137	0·05839
81	40	0·02500	129	0·09302
82	39	0·12821	117	0·07692
83	34	0·05882	108	0·09259
84	32	0·03125	98	0·07143
85	31	0·03226	91	0·06593
86	30	0·10000	85	0·03529
87	27	0·11111	82	0·09756

Appendix (*Continued*)

Age (x)	Males		Females	
	Number living (N_x)	Age-specific mortality (q_x)	Number living (N_x)	Age-specific mortality (q_x)
88	24	0·04167	74	0·08108
89	23	0·00000	68	0·00000
90	23	0·08696	68	0·04412
91	21	0·00000	65	0·01538
92	21	0·00000	64	0·09375
93	21	0·09524	58	0·06897
94	19	0·00000	54	0·03704
95	19	0·05263	52	0·05769
96	18	0·00000	49	0·02041
97	18	0·00000	48	0·02083
98	18	0·00000	47	0·06383
99	18	0·00000	44	0·00000
100	18	0·00000	44	0·00000
101	18	0·00000	44	0·00000
102	18	0·00000	44	0·09091
103	18	0·00000	40	0·02500
104	18	0·00000	39	0·05128
105	18	0·00000	37	0·02703
106	18	0·00000	36	0·02778
107	18	0·00000	35	0·00000
108	18	0·00000	35	0·02857
109	18	0·00000	34	0·02941
110	18	0·05556	33	0·03030
111	17	0·05882	32	0·03125
112	16	0·00000	31	0·06452
113	16	0·06250	29	0·03448
114	15	0·06667	28	0·00000
115	14	0·07143	28	0·03571
116	13	0·00000	27	0·00000
117	13	0·00000	27	0·00000
118	13	0·00000	27	0·00000
119	13	0·00000	27	0·03704
120	13	0·00000	26	0·07692
121	13	0·00000	24	0·04167
122	13	0·07692	23	0·08696
123	12	0·00000	21	0·00000
124	12	0·00000	21	0·09524
125	12	0·00000	19	0·15789
126	12	0·16667	16	0·06250
127	10	0·00000	15	0·06667
128	10	0·00000	14	0·07143
129	10	0·00000	13	0·15385
130	10	0·00000	11	0·00000

Appendix (*Continued*)

Age (x)	Males		Females	
	Number living (N_x)	Age-specific mortality (q_x)	Number living (N_x)	Age-specific mortality (q_x)
131	10	0·00000	11	0·00000
132	10	0·10000	11	0·00000
133	9	0·00000	11	0·00000
134	9	0·00000	11	0·00000
135	9	0·00000	11	0·00000
136	9	0·11111	11	0·00000
137	8	0·00000	11	0·00000
138	8	0·00000	11	0·00000
139	8	0·00000	11	0·00000
140	8	0·00000	11	0·00000
141	8	0·12500	11	0·09091
142	7	0·00000	10	0·10000
143	7	0·14286	9	0·11111
144	6	0·00000	8	0·00000
145	6	0·33333	8	0·00000
146	4	0·25000	8	0·00000
147	3	0·00000	8	0·12500
148	3	0·33333	7	0·00000
149	2	0·00000	7	0·00000
150	2	0·00000	7	0·14286
151	2	0·00000	6	0·00000
152	2	0·00000	6	0·00000
153	2	0·00000	6	0·16667
154	2	0·00000	5	0·20000
155	2	0·50000	4	0·00000
156	1	0·00000	4	0·00000
157	1	0·00000	4	0·00000
158	1	0·00000	4	0·25000
159	1	0·00000	3	0·00000
160	1	0·00000	3	0·00000
161	1	0·00000	3	0·00000
162	1	0·00000	3	0·00000
163	1	0·00000	3	0·00000
164	1	1·00000	3	0·33333
165	0		2	0·00000
166	0		2	0·00000
167	0		2	0·00000
168	0		2	0·00000
169	0		2	0·00000
170	0		2	0·00000
171	0		2	1·00000
172	0		0	1·00000