Theor Appl Genet (1988) 76:870–879

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Computer simulation of genetic control. Comparison of sterile males and field-female killing systems

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Received March 22, 1988; Accepted June 29, 1988
Communicated by J. S. F. Barker

Summary. A computer program, GENCON, designed to simulate genetic control using field-female killing systems, is described. These systems incorporate sex-linked translocations and conditional lethal mutations. Genetic death in field populations is caused by semisterility of the translocation and by homozygosis of the mutations in females and non-translocation males of field origin. Simulations using the program compare the effectiveness, in populations regulated by density, of genetic control using this type of system with control using sterile-male release. At high release rates, sterile males cause more rapid suppression and earlier eradication than sex-linked translocation strains. However, if releases are interrupted before eradication, the rate of recovery of density-dependent populations is more rapid following sterile-male release than following suppression with translocation strains. In such populations, the cumulative population suppression (number of individuals killed) is greater with translocation-strain release than with sterile-male release. At low release rates, sex-linked translocation strains can be much more effective at suppressing and eradicating density-dependent populations than sterile males. In continental Australia, eradication of the sheep blowfly *Lucilia cuprina* is probably not practicable. A suppression campaign using sex-linked translocation strains could yield a higher benefit to cost ratio than one using sterile males.

Key words: Genetic control – Simulation – Translocation – Sterile male – *Lucilia cuprina*

Introduction

The sheep blowfly, *Lucilia cuprina*, is a serious myiasis pest of sheep in Australia (Beck et al. 1985). For over two decades this species has been the subject of research programs aimed at development of genetic methods for its control (Foster and Whitten 1974). As in many species of higher Diptera, the male sex is determined by the presence of a Y chromosome and crossing over in males is rare or absent (Ullerich 1963; Foster et al. 1980). In such species, strains can be constructed in which autosomal mutations can be combined with Y-autosome translocations, so that males are phenotypically wild-type while females express the mutations. This allows preferential removal or killing of females during rearing for autocidal control programs (Whitten 1969; Whitten and Foster 1975). There are now numerous examples of pest species to which this principle has been applied (reviewed by Robinson 1983).

Whitten et al. (1977) proposed the use of sex-linked translocations to construct female-killing systems which operate in the field. In *L. cuprina*, flies that are homozygous for recessive eye colour and many other mutations are viable in the laboratory but inviable under field conditions. Overflooding of natural populations with flies heterozygous for such mutations can lead to substantial genetic death from homozygosis of the mutations. Combining autosomal recessive mutations in repulsion with appropriate sex-linked translocations permits the synthesis of strains in which all males are heterozygous and all females homozygous for deleterious mutations. Because such females do not survive to reproductive maturity in the field, both sexes can be released with impunity. Matings by the released males to wild females transfer the mutations to their daughters (Fig. 1 a). In subsequent generations, matings of heterozygous females with released males result in homozygosis of the mutations in a large portion of their female offspring (Fig. 1 b). The combined effects of semisterility of the rearrangement (due to production of aneuploid sperm by translocation-

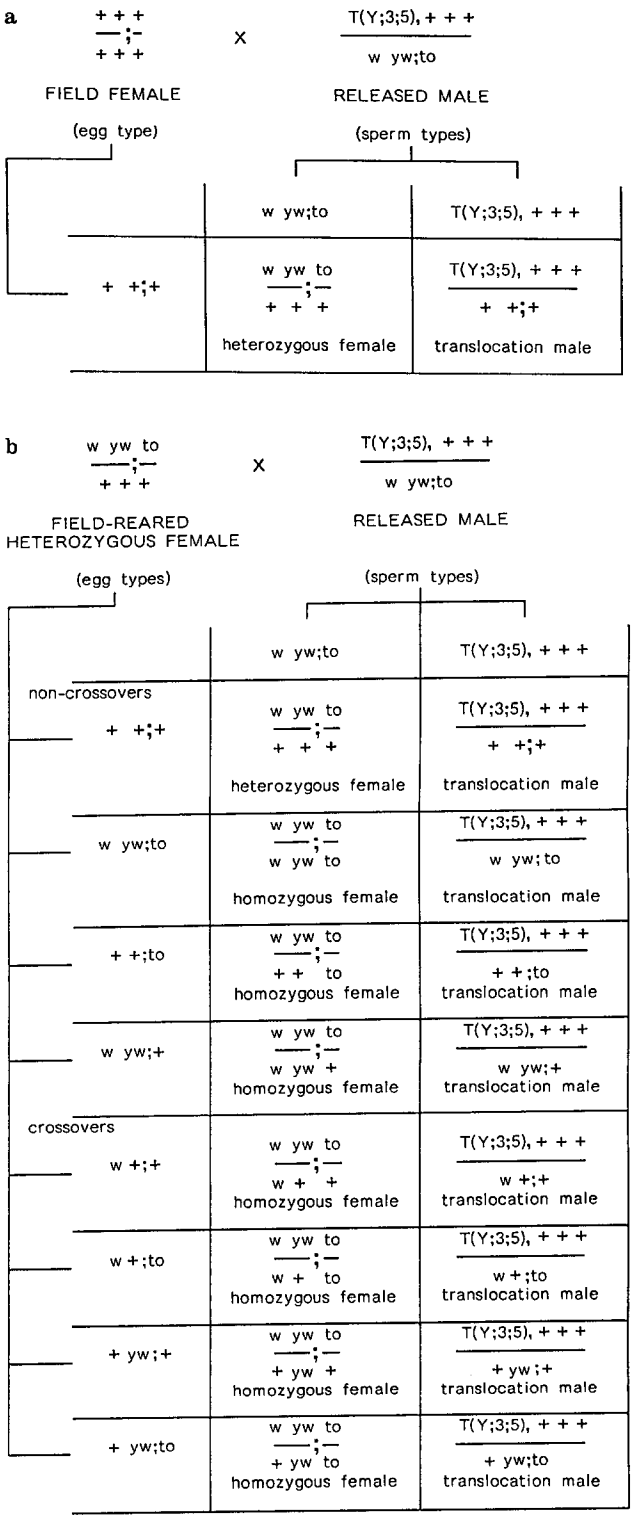

Fig. 1. **a** Transmission of the *L. cuprina* eye-colour mutations *w,yw* and *to* to the daughters of field-females mated by released translocation males; **b** production of homozygous daughters in matings of field-reared heterozygous females by released males

bearing males), and homozygosis of the mutations in their descendents can lead to high levels of genetic death in target populations (Whitten 1979; Foster et al. 1985).

In predicting and assessing the impact of such pest control methods, the effects of many factors, in addition to genetic death, on population size and fecundity must also be considered. In *L. cuprina* populations, for example, weather strongly influences oviposition rates (Wardhaugh et al. 1988) and the survival of post-feeding immature stages (Dallwitz 1984), both of which affect the reproductive capacity of fly populations. It also appears likely that the rate of population increase is density-dependent. Remedial measures taken by farmers are probably related to frequency and intensity of infestation of sheep (Foster et al. 1975). Finally, theoretical and field studies have shown that any type of genetic control program is likely to be heavily influenced by migration (Dietz 1976; McKenzie 1976, 1977; Prout 1978).

Because of the complexity of both the genetic system and the factors influencing population size, the GENCON series of simulation programs has been developed to facilitate planning and evaluation of genetic control systems based on sex-linked translocations and conditional lethal mutations. In this paper we describe simulations using an experimental version of GENCON, GC1, to explore the suppressive potential of semisterile sex-linked translocation strains (T-strains) in comparison with fully sterile males. The results are discussed with reference to the sheep blowfly ecosystem in Australia.

Materials and methods

The simulation program GC1

General description. Starting with a test population of finite size and using discrete generations, GC1 computes the combined effects of matings by released males and their descendents, natural or contrived rates of population increase, and migration, on genotype frequencies, genetic death, and population size.

Assuming that mating is random, the program pairs each possible female genotype with each male genotype. As each type of mating is selected, four genetic subroutines are called: (1) PSPERM assigns gamete probabilities to the male genotype; (2) PROVA assigns gamete probabilities to the female genotype; (3) ZYGOTE assigns zygote probabilities based on the gamete probabilities generated by PSPERM and PROVA; then (4) GENEXT computes the numerical contribution of each mating type to the next generation.

Genetic computations. The subroutines PSPERM, PROVA, and ZYGOTE are encoded using empirical data on the linkage relationships of the mutations concerned. This enables simulation of releases of males carrying particular heterozygous combinations of conditional-lethal mutations, linked in repulsion with an appropriate Y-autosome translocation. The genotype of the released males is specified in the input data as an integer corresponding to genotype codes embodied in the genetic subroutines.

The genetic subroutines used in the present report have been encoded specifically for simulation of genetic control using any

combination of the *L. cuprina* eye colour mutations white (w), yellowish (yw) and topaz (to), heterozygous in repulsion with any appropriate Y-autosome translocation. The mutations w and yw are 53.9 map units apart on chromosome 3, and to is on chromosome 5 (Foster et al. 1981). This model can thus simulate strains with translocations between the Y chromosome and chromosome 3 (T(Y;3)), the Y and chromosome 5 (T(Y;5)), or between all three chromosome (T(Y;3;5)). So far, several field trials of strains carrying T(Y;3;5) translocations have been conducted (Foster et al. 1985; R. J. Mahon, T. L. Woodburn, and G. G. Foster, unpublished).

For mutations normally carried on different chromosomes where these chromosomes are involved in the translocation, the program assumes random assortment when they are transmitted through females or non-translocation males, and complete pseudolinkage when transmitted through translocation males. For linked mutations on the chromosomes in the translocation, the crossover frequency in females is specified as data and the program computes recombination when transmission is through females. As meiotic recombination in males is much less frequent (Foster et al. 1980), the program assumes complete linkage through males.

Sterility. Semisterility of the translocation-bearing males is defined as 1.0-fertility, where fertility of these males is defined as the number of female offspring produced from a cross between wild-type females and translocation males, divided by the number of female offspring from a cross between wild-type females and non-translocation males (Foster et al. 1985).

Sex ratio. The sex-ratio characteristic of the offspring of a particular rearrangement is used in subroutine PSPERM. This is specified as the proportion of males among the chromosomally balanced offspring of wild-type females which have mated with translocation-bearing males.

Genetic death. Genetic death is defined as the proportion of female zygotes killed each generation due to homozygosis of mutations and semisterility caused by the rearrangements (gametic aneuploidy). These values are calculated as follows:

translocation death $TD = (Mt/M) \times S$
mutational death $MD = Nm/Nf$
total genetic death $GD = TD + MD(1\text{-}TD)$

where Mt = no. of females mated by translocation-bearing males, M = total no. of matings, S = semisterility of the translocation, Nm = no. of mutant female offspring, and Nf = no. of female offspring.

Release numbers. The number of males released each generation is specified. Releases of either fully or partially sterilized non-translocation males can be simulated. In the latter case, the program separates released males into sterile and fertile equivalents, and adds the fertile equivalents to the native males.

Mating competitiveness. The mating competitiveness of released and field-reared translocation-bearing males (Foster et al. 1985) is specified separately. Competitiveness of native (non-translocation) males is defined as 1.00. Competitiveness, as used in the program, is the product of the relative probability of survival from emergence to mating and the relative probability of mating success.

Breeding conditions. Ecological breeding conditions are summarized in a parameter RATINC, which is defined as the rate of increase (number of female offspring per female) in each generation for field females mated by field males under the environmental conditions and population size prevailing at the time. This parameter can be either specified as an input data array (e.g. to permit the use of RATINC values estimated from ecological studies of field populations), or it can be calculated by the program (e.g. to allow population density to determine RATINC). When a mathematical model of *L. cuprina* populations (currently being developed) is available, it is anticipated that it will be possible to calculate RATINC from data inputs such as weather, farm management practices and insecticide resistance frequencies.

Density dependence. Density-dependent populations were simulated using the relationship:

$$RATINC = R_{max} \times K/(K + (R_{max} - 1) \times N)$$

where R_{max} is the maximum possible rate of increase in the population, N is the adult female population density in any given generation, and K is the equilibrium population density. This formula is modified from that of Prout (1978), whose analysis was based on survival in immatures (larvae) that was largely density-dependent. Our formula assumes that the density-dependent rate of increase is related to adult population size.

In *L. cuprina* myiases, larval survival is independent of larval density in untreated sheep (Dallwitz 1987), but is reduced by insecticidal treatment of sheep by farmers (McKenzie and Whitten 1982), the frequency of which is influenced mainly by larval infestation rates. The latter are determined by adult fly densities in conjunction with the effects of weather on oviposition rates (Wardhaugh et al. 1988) and sheep susceptibility (Graham and Egerton 1968; Watts et al. 1979). Since weather and sheep susceptibility are not considered in this paper, infestation rates were assumed to be proportional to adult fly densities. This biological difference from Prout's assumptions does not alter the release to wild ratios required to suppress populations to the critical unstable equilibrium densities, below which eradication is inevitable assuming no immigration (Prout 1978 and personal communication).

Data outputs. Program GC1 computes and presents both ecological and genetic results in both tabular and graphical form for each generation. Ecological outputs include: (1) population size (number of breeding females); (2) number and proportion of native females; (3) number and proportion of field-reared translocation and native non-translocation males, and released translocation males; (4) number and proportion of immigrant males and females; and (5) RATINC values (if calculated by the program). Genetic outputs include: (1) mating type frequencies in the mating population (native females plus immigrant virgin females) or breeding population (i.e. mating females plus immigrant mated females); (2) effective male genotype proportions (i.e. corrected for competitiveness of released or field-reared translocation males) in the mating and breeding populations; and (3) female genotype proportions.

Simulations

Strain characteristics. In all the simulations presented in this paper, certain characteristics of the T-strains were standard. Sex ratio equalled 0.5. The crossover frequency between the linked mutations w and yw was 0.4744. This is the average frequency of single crossover progeny observed in *L. cuprina* for mutations 50 or more map units apart (N = 19,210, data from Foster et al. 1981 and unpublished). The competitiveness of released translocation or sterilized males, and that of field-reared translocation males, was assumed to be equal to that of field males, i.e. was set at 1.0.

Ecological conditions. Simulated test populations were either under density-dependent or density-independent population regulation. The density-independent populations had a constant rate of increase of 1.0 in the absence of releases (i.e. RATINC = 1.0). Density-dependent populations were given maximum R values (R_{max}) of 10 or 20. The equilibrium population density K was set at one million breeding females in all simulations.

Although neither setting RATINC values at unity nor allowing density to be the sole determinant of RATINC (in the ranges of 1–10 or 1–20) accurately reflects ecological conditions in populations of *L. cuprina,* we believe the results obtained are sufficiently general for purposes of illustration. Average *springtime* rates of increase for *L. cuprina* populations in south eastern New South Wales have been observed over approximately a tenfold range, from as low as 3.5-fold (Vogt et al. 1985) to approximately 30-fold (K. G. Wardhaugh, unpublished). Although springtime rates of increase can thus exceed the maximum RATINC values employed in the present paper, they are not maintained indefinitely. Midsummer and winter rates of increase are regularly quite a bit less than the minimum RATINC value of 1.0 used in this report (Vogt et al. 1985).

In most simulations, migration was set at zero. In simulations to investigate the effect of migration, a proportion of each genotype of the test population was removed from the population each generation. The same proportion of wild-type flies was transferred to the test population from a hypothetical external population, whose size was equal to the predicted size of the test population in the absence of releases.

Releases. All simulations involved releases against an initial population of one million field females and one million field males. The simulation was continued for 20 or 40 generations. Releases were made either continuously (i.e. once each generation) or as specified in the text. The initial release was always made in the first generation. Subsequent releases, where made, always involved the same number of males each generation as in the initial release (i.e. 'hard' releases as used by Prout 1978).

Results

Inherited vs non-inherited sterility

The effects of releasing males with 25%, 50%, 75% or 100% non-inherited sterility (such as that induced by irradiation or chemosterilants), or T-strains carrying no mutations with 25%, 50%, or 75% inherited semisterility are summarized in Fig. 2. Releases against both density-independent and density-dependent populations (R_{max} = 10) were simulated with continuous release at an initial release ratio (IRR) of 1:1.

In the density-independent case, the rates of population decline were largely determined by the level of sterility of the released males. The rates of decline were more rapid with T-strains than with partially sterilized males of equal sterility. The rate of genetic death rose asymptotically towards the sterility of each release type as the field males were eliminated from the population. The rise was more rapid in the case of inherited semisterility because the field-reared translocation-bearing descendents of the released males also contributed to genetic death.

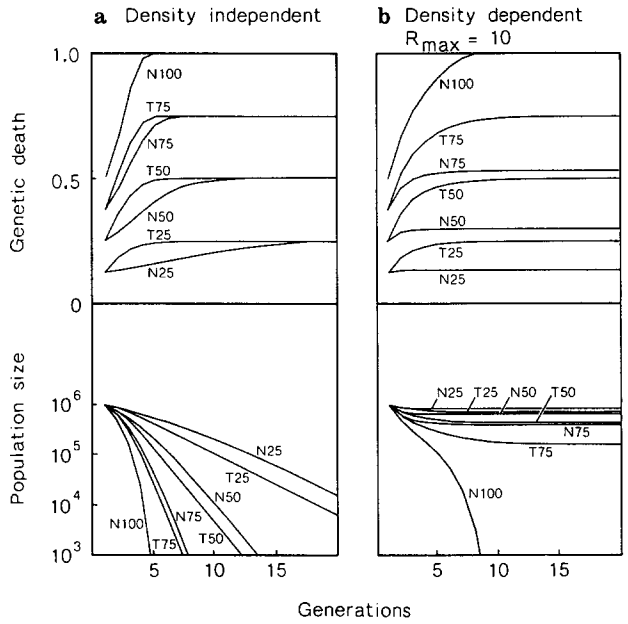

Fig. 2 a and b. Genetic death levels and population trends with releases of translocation males (inherited semisterility) and fully or partially sterilized non-translocation males (non-inherited sterility); numerals indicate percent sterility, T = translocation, N = non-translocation

In the density-dependent case, the density-reactive effect overcame the effect of the releases at all levels of inherited and non-inherited sterility tested, except 100%. In more formal terms, none of the treatments except release of fully sterile males reduced the population below the unstable equilibrium size, at which eradication would be inevitable (Prout 1978). Only reductions in population size of less than tenfold were achievable with translocation-bearing males under these release and ecological conditions. In contrast, the 100%-sterile males reduced the population 1000-fold within eight generations of the start of releases. Substantial differences in the levels of genetic death were evident between the inherited and non-inherited sterility. With T-strain releases, genetic death rates rose rapidly to the semisterility level of the T-strains, because of accumulation of field-reared translocation-bearing males at the expense of non-translocation males in these populations. The maximum rates of genetic death achieved were considerably lower with partially sterilized non-translocation males, particularly at low sterility values.

Effect of mutations in combination with inherited semisterility

The effects of including conditional lethal mutations in the T-strains are summarized in Fig. 3. Translocation semisterilities of 0%, 25%, 50% and 75% in combination with 1(w), 2(w, to) or 3 (w, to, yw) mutations are compared to the effect of releasing 100%-sterile males.

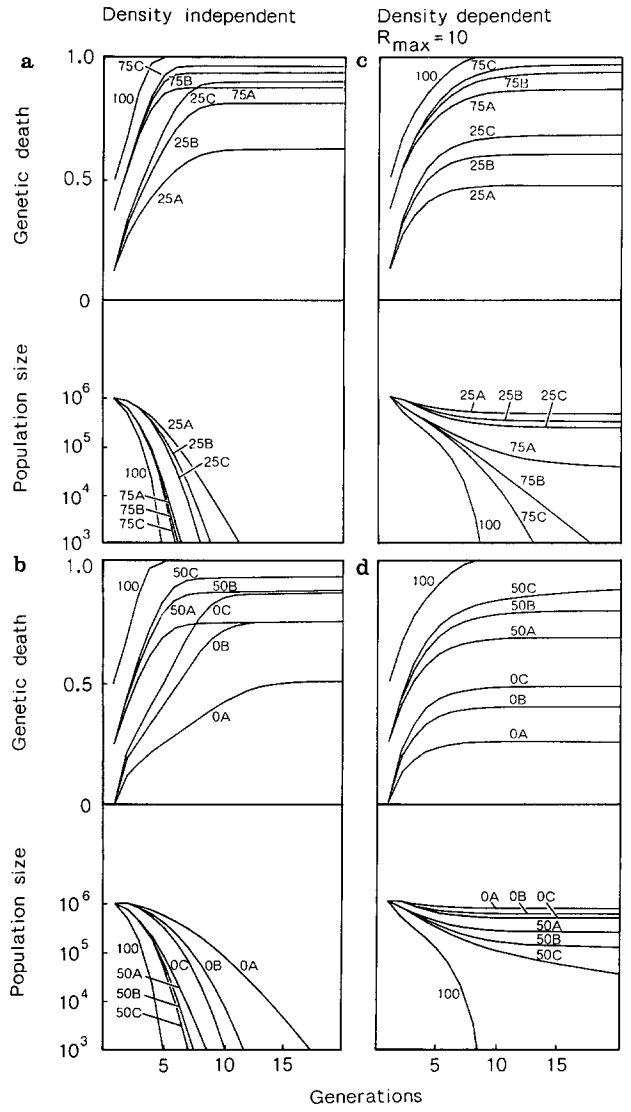

Fig. 3a–d. Genetic death levels and population trends with releases of fully sterile (100%) males or translocation males heterozygous for one, two or three mutations and with (**a, c**) 25% or 75% semisterility, or **b, d** zero or 50% semisterility; **a, b** density-independent, **c, d** density-dependent ($R_{max} = 10$); A = 1 mutation (*w*), B = 2 mutations (*w, to*), C = 3 mutations (*w, yw, to*)

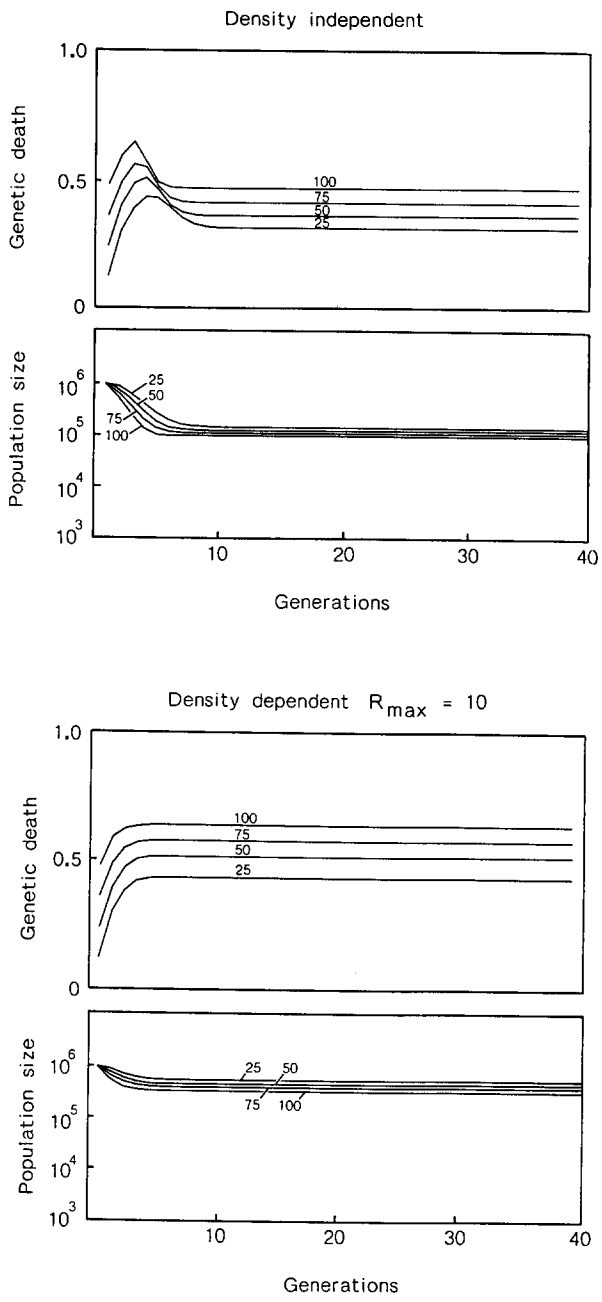

Fig. 4. Effect of 5% immigration on genetic death and populations suppression by translocation males (25%, 50%, 75% semisterility) of fully sterile (100%) males heterozygous for three mutations

In the density-independent case (Fig. 3a and b), each additional mutation increased the rate of population decline characteristic of a T-strain with a particular level of translocation semisterility. Strains combining semisterility with mutations caused much greater suppression than strains with either semisterility (Fig. 2) or mutations alone.

In the density-dependent case, addition of two or three mutations to the 75%-semisterile translocation (Fig. 3c), or three mutations to the 50%-semisterile translocation (Fig. 3d) causes sufficient genetic death to overcome the reactive effect of the density dependence [i.e. suppress the population below the unstable equilibrium size (Prout 1978)], leading eventually to elimination of the population. The extra effect of the mutations on the level of genetic death was considerable in all cases.

Effect of immigration

The effect of transferring 5% of the males and mated females from an external population into the test population each generation is illustrated in Fig. 4, for both

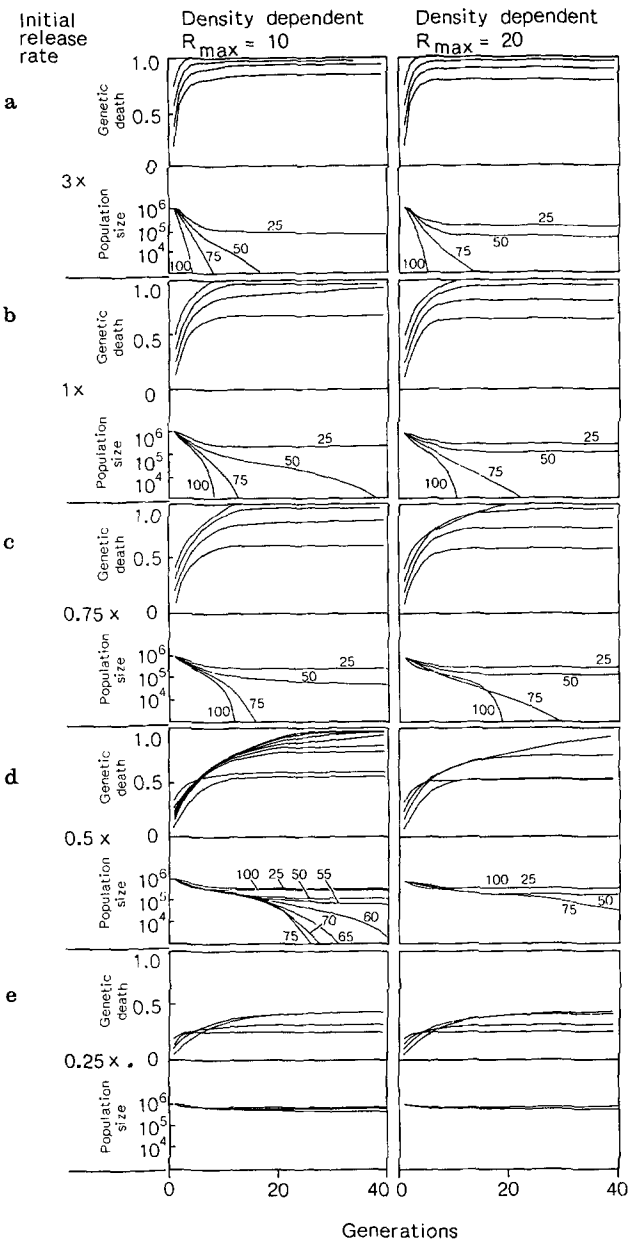

Fig. 5a–e. Effect of variation of release rate on genetic death and suppression of density-dependent population by translocation males heterozygous for three mutations (various semisterilities, as indicated by numerals) or sterile males (100%)

density-independent and density-dependent ($R_{max}=10$) populations. With all levels of immigration of mated females, any release regime would fail to eradicate the test population (Prout 1978).

Effect of release rates on relative effectiveness of T-strain and sterile male releases

All interstrain comparisons so far have involved continuous releases with IRR = 1:1. In these examples the T-strains were less effective at reducing the population than sterile males. In order to examine the effects of other release rates and density-dependence levels, continuous release of either sterile or T(Y;3;5)/w yw to males with IRR between 3:1 and 0.25:1 were applied to populations under two levels of density dependence ($R_{max}=10$ or 20) (Fig. 5).

At release rates of 3:1, 1:1 and 0.75:1, fully sterile males were more effective than T-strain males in terms of rate of suppression of test populations (Fig. 5a–c). Under these release conditions the effectiveness of the T-strains increased with increasing semisterility. The 25%-semisterile T-strain was incapable of overcoming the density-reactive effects even at the highest release rate.

With release rates at 0.5:1 the effectiveness of most T-strains was greater than that of sterile males (Fig. 5d). Against a density-dependent population with $R_{max}=10$, the 50%-semisterile T(Y;3;5)/w yw to strain gave lower population densities than sterile males, while the 75%-semisterile T-strain eliminated the population. Additional simulations with this ecological and release regime revealed that translocation semisterility of 55% was insufficient to overcome the density-reactive effect, whereas 60% or higher semisterilities eventually led to eradication (Fig. 5d). At IRR = 0.25:1, little effect on population density was observed with any type of released male (Fig. 5e).

In populations with $R_{max}=20$, eradication was not achieved with any strain using an IRR of 0.5:1. The 50%- and 75%-semisterile T-strains were approximately equivalent in terms of population suppression, and were more effective than sterile males.

Persistence of effects on populations after cessation of releases

Two examples in Fig. 6 illustrate the effect of the persistence of genetic death in density-dependent populations ($R_{max}=10$), following cessation of releases of T-strain males. All of these density-dependent populations returned to the equilibrium level after cessation of releases. This occurred most rapidly with sterile males and more slowly with decreasing translocation sterility. After cessation of releases of sterile males, genetic death rate dropped immediately to zero, whereas in the T-strain trials, genetic death declined more slowly, persisting for many generations. The decline in the rate of genetic death was more rapid for the T-strains with higher semisterility, reflecting more rapid elimination of field-reared translocations from these populations.

Releases for seven generations at IRR = 1:1 caused a 316-fold maximum reduction in population size with sterile males, and 21-, 7.2- and 3.4-fold reductions with 75%-, 50%- and 25%-semisterile T(Y;3;5)/w yw to males

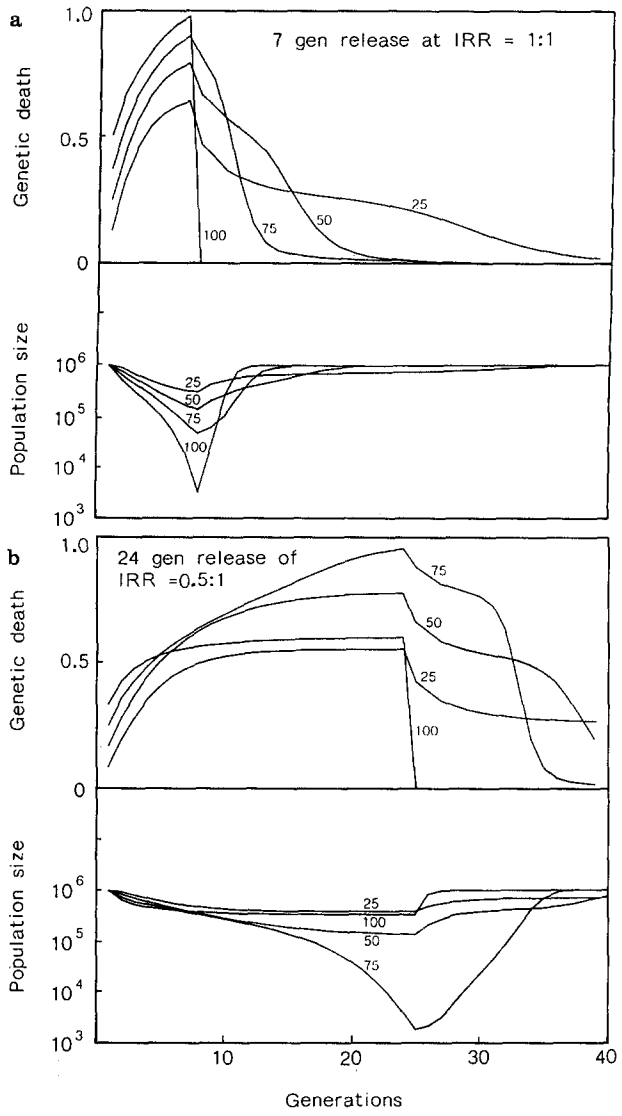

Fig. 6a and b. Effect of cessation of releases after build-up of genetic death in density-dependent populations by releases of translocation males heterozygous for three mutations (25%, 50%, 75% semisterility) or sterile males (100%)

(Fig. 6a). If the impact of releases in measured in terms of the population reduction summed across generations, the T-strains had a greater impact on the population. The cumulative population reductions thus measured over 40 generations were:

sterile males = 7.8 million
75%-semisterile T-strain = 8.9 million
50%-semisterile T-strain = 9.9 million
25%-semisterile T-strain = 10.9 million

With lower release rates over a longer period, the persistence of genetic death and population suppression following T-strain releases can be very marked. In the example shown in Fig. 6b (24 generations of releases at IRR = 0.5:1), both the 50%- and 75%-semisterile T-strains outperformed sterile males in both maximum suppression and total genetic death. The cumulative population reductions over 40 generations in this case were:

sterile males = 14.9 million
75%-semisterile T-strain = 27.3 million
50%-semisterile T-strain = 25.2 million
25%-semisterile T-strain = 17.7 million

Note that the values given for the 25%-semisterile T-strains and for the 50%-semisterile T-strain at the lower IRR do not indicate the full suppressive potential, since substantial genetic death was still occurring at the termination of these runs (Fig. 6b).

The decline in genetic death rates after cessation of T-strain releases tended to be biphasic (Fig. 6). With the 25%- and 50%-semisterile T-strains, the initial decline was almost entirely due to reduction in the frequency of homozygosis of mutations, but with the 75%-semisterile T-strain the translocation frequency dropped much more rapidly, especially at higher R_{max} values. Death from homozygosis of mutations tends to vary as a function of the product of the frequencies of heterozygotes, while death from semisterility is directly proportional to the translocation frequency. The decline in heterozygote frequency was virtually independent of strain semisterility, whereas the decline in translocation frequency was inversely related to semisterility.

Female heterozygote frequencies were initially higher than in males, but as the translocation frequencies approached zero, mutation frequencies in the two sexes approached equality. This is because translocation males generally inherit only the wild-type alleles from their fathers, whereas females and non-translocation males can inherit mutations from both parents (Foster et al. 1985).

Effect of releasing in alternate generations

The simulations summarized in Fig. 7 provide further examples of the effects of alteration of release tactics on the relative effectiveness of sterile males and T(Y;3;5)/w yw to strains. Males were released every second generation into a density-dependent population ($R_{max} = 10$) with IRR = 1:1 (Fig. 7a) or 2:1 (Fig. 7b). The genetic death rate fluctuated markedly each generation, particularly with the sterile male releases. The fluctuations in genetic death rate were dampened with T-strain releases, but the relative effectiveness of the T-strains with respect to both the amplitude of the fluctuations and the average genetic death rate differed between the two release rates.

At IRR = 1:1 (Fig. 7a), the 50%-semisterile T-strain produced the greatest population suppression, the 25%-semisterile T-strain was marginally better (in terms of average population density) than the 75%-semisterile T-strain and sterile males were least effective in popula-

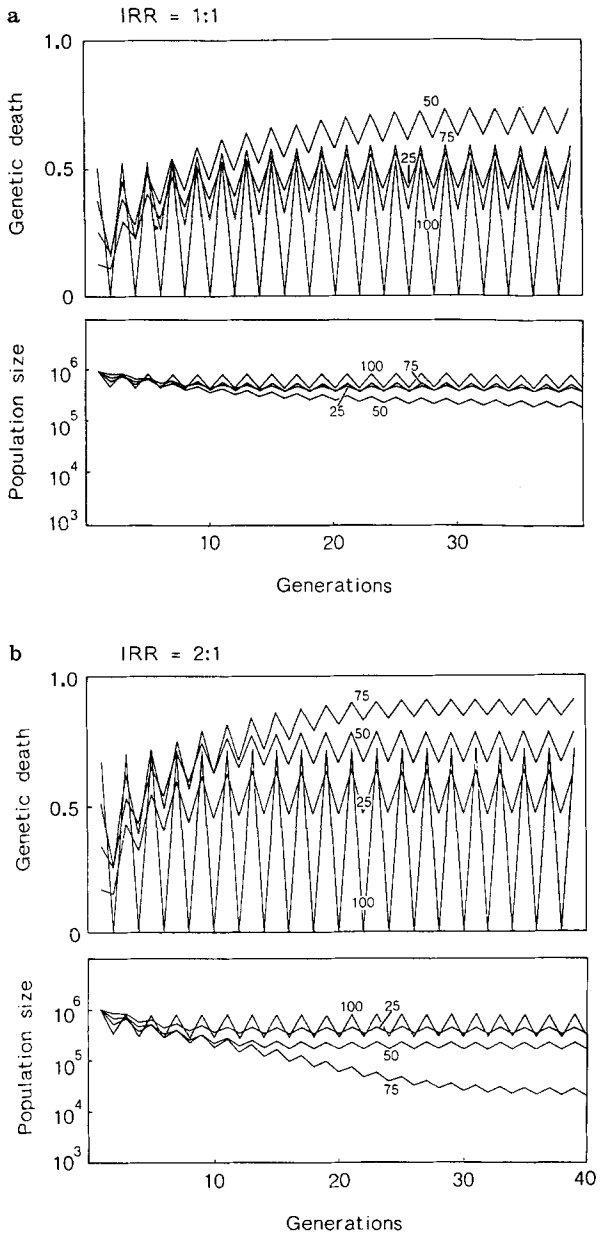

Fig. 7 a and b. Effects on genetic death and population size of releases in alternate generations of translocation males heterozygous for three mutations (25%, 50%, 75% semisterility) or sterile males (100%)

tion suppression. None of these trials resulted in eradication. This simulation can be compared directly to continuous releases with IRR = 0.5:1 (Fig. 5d), in which the same total number of males was released. With continuous releases the population suppression was much greater than with releases in alternate generations. In that simulation the 75%-semisterile T-strain was the only one which caused eradication, followed in order of effectiveness by the 50%-semisterile T-strain, fully sterile males, and the 25%-semisterile T-strain.

With IRR = 2:1 (Fig. 7b), the 75%-semisterile strain ranked first in terms of population suppression, followed by the 50%-semisterile strain and the 25%-semisterile strain, with fully sterile males again being least effective. None of these release regimes led to eradication of the population, however. In the comparable continuous release (IRR = 1:1) simulation (Fig. 5b), all male types except the 25%-semisterile T-strain achieved eradication.

Discussion

The simulations presented here demonstrate that the effectiveness of release of fully sterile and semisterile T-strain males in controlling natural populations is clearly dependent on release rates and ecological constraints, as well as on the genetic properties of the strain. In density-dependent populations, low release rates of T-strain males heterozygous for several mutations can be more effective than identical release rates of sterile males.

When eradication is the goal and overkill (i.e. a large overflood ratio) is feasible, the use of sterile males will achieve this result more rapidly than genetically altered strains. In certain circumstances, however, a low release rate into a density-dependent population can lead to eradication with translocation strains but not with sterile males (Fig. 5d). Thus, in closed systems (e.g. islands), eradication using T-strains may be more cost-effective (both in terms of capital and operating costs) than with sterile males, particularly if rapid eradication is not essential.

Where suppression rather than eradication is the aim of a control program, the simulations described in the present paper suggest that field female-killing systems (T-strains) constructed from sex-linked translocations and mutations which are lethal in field conditions (Whitten et al. 1977), may be more cost-effective than sterile males. Initially the rate of genetic death caused by release of fully sterile males is greater than that from T-strains. However, the build-up in the population of genetic death from the field-reared descendents of released T-strain males can in some circumstances lead to a greater total suppression than with sterile males.

In density-independent populations with RATINC = 1.0 (which probably do not exist), sustained release of males (with either inherited or induced sterility) leads to eradication, whatever the released to wild ratio. Under these circumstances, suppression using fully sterile males occurs much more rapidly than that using translocations. This is partly because of the immediate effects of the differences in sterility, and partly because with each successive generation, the released to wild ratio increases more rapidly with sterile males than with translocation males. In the kind of model examined here, populations

regulated in a density-dependent fashion return to the equilibrium level if releases cease before eradication. If releases continue, eradication can occur in such populations, but it is not inevitable, depending essentially on whether the genetic death introduced by the released males can overcome the greater rates of population increase encountered at low densities. This becomes increasingly difficult with higher density reactive effects (i.e. with higher R_{max}), and is dependent on other factors such as release strategy, sterility of the released males, and (in the case of T-strains) the mutational load generated by the released males.

During a release program, increased sterility increases the rate of suppression. However, if releases are deliberately or accidentally stopped, the rate of population recovery is inversely related to T-strain semisterility, and is most rapid with 100% sterile males.

As the number of deleterious mutations which can be pseudo-linked to sex increases, the suppression caused by T-strains increases, particularly if the mutations are on different linkage groups or are far apart on the same linkage group. Limiting factors in this aspect of strain design include the number and genetic size of the linkage groups, the number and linkage relationships of available mutations, and whether there are likely to be adverse effects of particular mutations or rearrangements on strain fitness or stability.

In designing field-lethal systems for inundative release, both the practicability of mass-rearing such strains and their suppressive potential must also be taken into account. Strains with higher semisterility levels are more costly to rear because of the increased adult colony sizes necessary to obtain a given number of insects for release. Similarly, strains whose females are homozygous for several mutations may have severely reduced fecundity. The simulations in the present paper suggest that T-strains with a semisterility of approximately 50% afford a reasonable compromise between suppressive potential and ease of mass-rearing. Indeed, under some circumstances, such strains cause greater suppression than ones with higher semisterilities. Although most three-chromosome translocations have semisterilities of approximately 75%, strains can be selected in which egg-to-adult survival is considerably higher. For example, two T(Y;3;5) translocations used in a series of field trials of genetic control of *L. cuprina* (Foster et al. 1985; R. J. Mahon, T. L. Woodburn and G. G. Foster, unpublished) have semisterilities of approximately 60% and 56%. Both rearrangements were derived from the same T(Y;5) translocation, whose semisterility is less than 10% (Foster 1982). The low semisterility of this rearrangement results from the position of the break-points with respect to the meiotic pairing sites of both chromosomes involved in the rearrangement (Foster and Maddern 1985; Bedo 1987).

Release tactics may be as important as strain design in optimising the results of a control program where long-term suppression is the aim. Lower release ratios for longer periods could be more cost-effective than higher release ratios for shorter periods, provided that the level of suppression achieved produces acceptable economic gains. Cost-benefit analyses must consider all these factors.

The density-dependent regulation of *L. cuprina* populations in Australia is probably related more to the activities of man than to increased larval competition, at least in areas where livestock densities are high enough to make regular inspection and treatment feasible (Foster et al. 1975; Dallwitz 1987). It may be possible to use insecticides at times of low population density (Hughes and McKenzie 1987) as a useful supplement to genetic suppression measures.

It is clear from both field and theoretical studies that autocidal control systems are extremely sensitive to immigration (Fig. 4; Dietz 1976; McKenzie 1976; 1977; Prout 1978; R. J. Mahon T. L. Woodburn and G. G. Foster, unpublished). In Australia, Bass Strait, which separates Tasmania from the mainland, was apparently effective in excluding *L. cuprina* from that state until its introduction in the late 1940s on infested sheep (Ryan 1954). Within the two major sheep-growing regions of mainland Australia, there are few if any absolute natural barriers to fly migration other than distance. Fortunately, *L. cuprina* does not appear to be behaviourally predisposed to long-distance migration with weather systems, as is the case with some Australian insects (Hughes and Nicholas 1974; Helm 1975; Drake and Farrow 1985). Thus if autocidal control programs are conducted over sufficiently large areas, fly migration may only pose problems at the edges of release areas.

The success of the campaign against the screwworm fly in North America has demonstrated that with a determined effort a pest can be eradicated from large land areas (Krafsur et al. 1987). However, this campaign benefitted from biological and geographical factors which would not apply to sheep blowfly populations in Australia. First, the inability of screwworms to survive the winter in most of the United States confined overwintering populations in that country to southern Florida (from 1933 until eradication there in 1959), the southern end of the Gulf coast of Texas and some areas along the southern border with Mexico. *L. cuprina,* on the other hand, survives the winter throughout its range in Australia (three million km^2 of sheep grazing country plus the coastal regions of eastern and northern Australia, which are largely free of sheep). Secondly, the geography of North America permitted confinement of screwworm populations to an ever-narrowing region as the eradication campaign progressed southward through Mexico. There are no geographical corners in Australia to which

the fly can be confined. Thus, while eradication in Tasmania should be achievable, it is probably not feasible on the mainland. It would appear to be more prudent to attempt a suppression strategy using a sex-linked translocation system with deleterious mutants, as modelled here, concentrating initially on the more intensively farmed regions (i.e. less than half of the total sheep grazing area). This should produce the maximum benefits for the least outlay, using considerably less capital and operating resources than sterile male releases.

Acknowledgements. We thank Drs. D. G. Bedo and T. Prout for their constructive criticisms of the manuscript. Supported by funds from the Australian Meat and Livestock Research & Development Corporation and the Australian Wool Corporation.

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