Genet. Res., Camb. (1990), **56**, *pp.* 35–42 *With* 1 *text-figure Printed in Great Britain*

Are responses to artificial selection for reproductive fitness characters consistently asymmetrical*?

R. FRANKHAM
School of Biological Sciences, Macquarie University, Sydney, NSW 2109, Australia

(Received 23 February 1990)

Summary

Non-linear offspring-parent regressions and heritabilities are expected for characters showing genetic asymmetry due to directional dominance and/or asymmetrical gene frequencies. Since reproductive fitness characters exhibit these characteristics, they should show consistently non-linear heritabilities, with greater heritabilities in the direction of lower fitness. As a consequence, responses to bi-directional selection on fitness traits should be asymmetrical in the same direction. This prediction has been tested by an analysis of published bi-directional selection experiments for reproductive fitness traits. Significant asymmetry (24 of 30 studies) in the predicted direction was found. For studies reporting realized heritabilities, the means were 0·173 and 0·259 for lines selected for higher and lower reproductive fitness, respectively, the high lines being 33% less than the low lines. Asymmetry was evident for studies reporting realized heritabilities and for those with random mating controls of the same size as the selection lines. Consequently, it is argued that the asymmetry results from genetic asymmetries. This asymmetry has important implications in the improvement of reproductive fitness traits in plant and animal breeding.

1. Introduction

Reproductive fitness traits represent the most difficult and least understood traits in quantitative genetics, yet they are among the most important in plant and animal production (Frankham, 1982).

The starting point for discussions of the evolution of reproductive fitness must be Fisher's Fundamental Theorem of Natural Selection (Fisher, 1930). This states that 'the rate of increase in fitness of any organism at any time is equal to its genetic variation in fitness at that time'. It has since been recognized that this theorem depends on several assumptions that Fisher did not make explicit, most notably weak selection, constancy of genotypic fitnesses over time and linkage equilibrium (Kimura, 1958; Kojima & Kelleher, 1961; Crow & Kimura, 1970; Turner, 1970; Naglaki, 1979; Charlesworth, 1980; see Charlesworth, 1987 for review). A more general result is that a population under continued selection exhibits no additive genetic variation in fitness. This follows from Kimura's generalization of Fisher's theorem (Kimura, 1958). Any genetic component of variance in fitness must therefore be due to dominance or epistasis (Charlesworth, 1987). Models that include mutation and finite size do, however, result in small amounts of additive genetic variation in fitness (Latter, 1970), but do not affect the overall thrust of the above predictions.

When applied to components of fitness, these arguments are valid only for those subject to directional natural selection. Further, negative genetic correlations between fitness components can result in the presence of additive genetic variation for fitness components, while fitness itself exhibits none (Robertson, 1955). While it is clear that additive genetic variation is present for fitness components (see Falconer, 1989; Table 1 herein), such characters exhibit lower heritabilities on average than other characters (see Gustafsson, 1986; Mousseau & Roff, 1987; Roff & Mousseau, 1987; Falconer, 1989). Fitness characters exhibit non-additive genetic variation, as demonstrated by inbreeding decline and heterosis (see Falconer, 1989).

Gowe (1983 and *pers. comm.*) predicted that heritabilities of reproductive fitness traits were non-linear, being close to zero in the upper 80–90% of the phenotypic range, and moderate in the lower 10–20% of the range. He attributed this to the segregation of deleterious recessive genes in mutation-selection balance. Consequently, he predicted that moderate

* Dedicated to the memory of Alan Robertson.

culling on reproductive traits in artificial selection lines would be effective in preventing the usual declines in fitness. Gowe (1983) applied such culling against low fertility and hatchability in lines of chickens selected for increased egg production and apparently was successful in preventing the decline of fertility and hatchability. A systematic evaluation of this approach, involving selection for a trait with and without culling on reproductive fitness, was done by Frankham, Yoo & Sheldon (1988). They showed that culling against low fitness was effective in preventing the usual declines in fitness in lines selected for another character.

Non-linear parent-offspring regressions have been found for various traits by Nishida (1972), Meyer & Enfield (1975), Robertson (1977) and Maki-Tanila (1982) and have been discussed by them and by Curnow (1960), Kempthorne (1960), Abplanalp (1961) Nishida & Abe (1974), Bulmer (1980) and Gimelfarb (1986). Notable among the factors expected to lead to non-linear heritabilities are directional dominance for alleles affecting a trait, and asymmetrical allele frequencies.

There is substantial evidence for directional dominance for alleles with favourable effects on reproductive fitness from the inbreeding depression and heterosis shown by such traits (see Falconer, 1989). Directional selection on reproductive fitness as proposed by Fisher is expected to lead to asymmetrical gene frequencies, with alleles increasing fitness at high frequencies and deleterious alleles at low frequencies. Further, deleterious mutations are partially to completely recessive (Charlesworth & Charlesworth, 1987) and are found at low frequencies in approximate mutation-selection balance. Consequently, reproductive fitness characters should show consistently non-linear heritabilities, of the type proposed by Gowe. In spite of the above information there is no general appreciation that heritabilities for reproductive traits might be non-linear.

Non-linear heritabilities would be expected to cause asymmetrical short-term response to selection. Falconer (1989) predicted such asymmetry, commenting that 'if the character selected is a component of natural fitness, asymmetry should be expected, with selection towards increased fitness giving a slower response than selection towards decreased fitness. The reasons are, first, that these characters usually show inbreeding depression, which is itself a cause of asymmetry, and, second, that if the character has been subject to natural selection the gene frequencies are likely to be above the symmetrical point, i.e. nearer the upper limit, thus giving rise to genetic asymmetry.' Such asymmetry is illustrated in Fig. 1 where theoretical response curves for two way selection are plotted for a trait determined by four dominant genes with initial frequencies of 0·1, 0·25, 0·25 and 0·4 (from Falconer, 1954). Kojima (1961) has shown that alleles showing directional dominance will lead to asymmetrical responses to selection. The existence of such general fitness asymmetry has received scant attention in spite of its important practical implications.

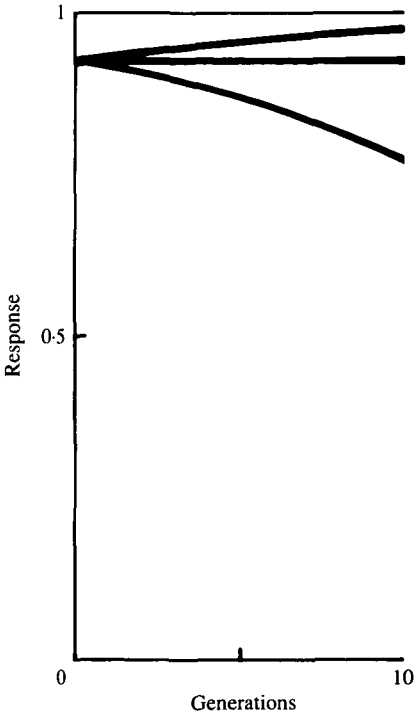

Fig. 1. Theoretical response curves for two way selection for a trait determined by four dominant genes with initial frequencies of 0·1, 0·25, 0·25 and 0·4, and $s = 0·2$ (after Falconer, 1954).

The aim of the present paper is to evaluate the prediction that selection responses for reproductive fitness traits will be consistently asymmetrical, with response greater in the direction of low fitness. Data from published studies were compiled and analysed. Significant asymmetry in the predicted direction was found.

2. Materials and methods

A literature search was undertaken to collect all studies where short-term bi-directional selection was performed on a reproductive fitness character, using an outbred base population, and with maintenance of a contemporary control.

The studies are listed in Table 1, along with details of the duration of the experiment, the parent population sizes, the intensities of selection, type of selection applied (individual, family or within family), the type of base population and the type of control population used. Where selection was in only one sex, the selection differential has been converted to the overall intensity in both sexes. Where different numbers of male and female parents were used, the effective population size is reported. Where lines were maintained under minimum inbreeding regimes (sampling of one male per male parent and one female per female parent) the effective size is double the number of parents and this doubled value is reported.

Wherever possible the results are presented as realized heritabilities as this measure avoids asymmetry due to different selection differentials and is less subject to scale effects.

In the cases of Falconer (1955), Manning (1961, 1963), Land & Falconer (1969) and Sherwin (1975), the data points have been read from enlargements of their Figures and regressions carried out. Where the studies were long term, only the data through to generation 10 were used, so that short-term responses to selection were considered. For Falconer's litter size selection experiment, responses to generation 9 (Falconer, 1955) and to generation 20 (Falconer, 1971) are both presented, as realized heritabilities were not available for the former study. In the case of the study by Bakker (1969) the absolute responses are reported as the duration of the study is unclear (greater than 10 generations).

The traits regarded as fitness traits are: egg production, mating ability, time to sexual maturity, duration of development and larval feeding rate in *Drosophila*; litter size, ovulation rate and age at sexual maturity in mice; egg production and male mating ability in Japanese quail; male mating ability and hatching time in chickens; and developmental time in *Tribolium*. Size and growth characters were excluded as was ovary size in *Drosophila*.

The study by Marien (1958) on developmental time deserves special mention as it is the most extensively replicated study (10 replicates each of Fast, Slow and Control). It has been interpreted as showing a greater selection response in the direction of *increased* fitness, namely responses of 0·175 days per generation in Fast (27 generations) and 0·145 days per generation for Slow lines (17 generations). However, this study is flawed because generations of Fast, Slow and Control lines were not maintained contemporaneously, density was uncontrolled and there were large fluctuations in control means over generations. The durations mentioned above represent relatively long-term responses. A contemporary comparison of Fast, Control and Slow lines, carried out when these lines were at generations 17, 12 and 11, yielded mean development times of 18·5, 21·0 and 24·0 days, respectively. When they are converted into response per generation (0·147 days for Fast and 0·273 days for Slow), these results show asymmetry in the direction of slow development.

The study of Spiess & Spiess (1966) was omitted since their synthetic control population was resynthesized four years after the selection lines were founded and it had a different frequency of inversions than most of the selection lines. Further, the study by Sang (1962) was excluded as he used drastically altered environments (nutrient-deficient diets) where the past history of natural selection may be largely irrelevant due to genotype × environment interactions.

Each study was scored as to whether selection response was greater in the high fitness than the low fitness direction or vice versa. In some cases replicate lines were maintained, but these have been counted only once using the mean, except where lines were derived from different base populations.

On the null hypothesis that there is symmetry of response one would expect equal numbers in the two classes. Chi-square tests were carried out to determine whether the numbers in the two classes were equal. These each have one degree of freedom. Since the hypothesis being tested is that there will be more studies showing greater response for reduced fitness, a one tailed test is appropriate.

3. Results

Twenty-eight relevant publications involving 30 studies were found. Of these, 24 (80%) showed asymmetry of response towards lower fitness and 6 showed asymmetry towards higher fitness. This represents a highly significant deviation from equality (chi-square = 10·8, $P = 0{\cdot}0005$). One study (Dawson, 1965) changed from very weak asymmetry in the high fitness direction for generations 0–7 to strong asymmetry in the direction of low fitness for generations 0–13. If the generation 1–7 results are used, the ratio of studies becomes 23:7, but this is still a highly significant deviation from equality (chi-square = 8·5, $P = 0{\cdot}0018$).

Nineteen studies reported realized heritabilities, 16 (84%) showing asymmetry in the direction of lowered fitness. This represents a highly significant deviation from equality (chi-square = 8·9, $P = 0{\cdot}0015$).

Eighteen studies used random mating control populations of the same size as the selection lines, 15 (83%) showing asymmetry in the direction of lowered fitness. This represents a highly significant deviation from equality (chi-square = 8·0, $P = 0{\cdot}0024$).

4. Discussion

Significant asymmetry in response to short-term selection for reproductive fitness characters was detected, there being more response in the direction of lowered fitness as predicted. This seems consistent over a range of fitness characters in mice, rats, Japanese quail, chickens, *Tribolium*, and several species of *Drosophila*

How great were the differences in response in the two directions? The most appropriate way to compare responses is to take those studies reporting realized heritabilities since they are independent of scale for the characters. Mean realized heritabilities were 0·160 for the lines selected for high fitness and 0·267 for those selected for low fitness, based on the means of replicate lines within a study. Alternatively, mean realized heritabilities were 0·173 and 0·259, respectively, for high and low fitness selection lines, when all replicate lines were used. Thus the realized heritabilities were 33%–40% lower in lines selected for high fitness than in lines selected for low fitness.

https://doi.org/10.1017/S0016672300028858 Published online by Cambridge University Press

Table 1. *Responses to bi-directional selection for reproductive fitness characters (column headings and codes are explained in the key)*

Organism and character	M	Selection High	Selection Low	Asym	G	N	i	S	Base	Control	Ref:
Drosophila											
Fecundity	b	0·089	0·238	L > H	10	40	59	IS	OL	RMC	**1**
	b	0·495	0·456	H > L	7	40	59	IS	OL	RMC	**1**
	h	0·010	0·495	L > H	8	15	17	FS	WX	ILX	**2**
Mating speed	r	0·14	4·57	L > H	9·5	20	20	IS	OL	BP	**3**
	b	0·0	0·064	L > H	10	20	57	IS	OL	BP	**4**
		0·0	0·022								
	h	0·193	0·062	H > L	5	20	20	IS	OX	RMC	**5**
	b	−5·677	8·577	L > H	10	10	6	IS	OX	RMC	**6**
	b	−4·732	12·859	L > H	10	10	6	IS	OX	RMC	**6**
	h	0·016	0·029	L > H	19	20	20	IS	OX	RMC	**7**
		0·007	0·014								
Rate of development	r	1·40	1·13	H > L	3	100	23	IS	O	RMC	**8**
	r	0·147	0·273	L > H	*	20	?	IS	OS	RMC	**9**
	h	0·17	0·70	L > H	#	42	21	IS	SX	RMC	**10**
	h	0·063	0·186	L > H	11	36	14	IS	SX	ILX	**11**
	h	0·19	0·20	L > H	9	?	20	IS	WX	RMC	**12**
	r	13·9	19·4	L > H	?	V	V	IS	SL	BP	**13**
Time to sexual maturity	h	0·12	0·15	L > H	8	20	20	IS	W	BP	**14**
		0·04	0·19								
Larval feeding rate	h	0·112	0·209	L > H	16	30	25	IS	SX	RMC	**15**
		0·205	0·197								
Mice											
Litter size	h	0·076	0·250	L > H	20	40	75	WFS	SX	RMC	**16a**
	b	0·054	0·133	L > H	9	40	75	WFS	SX	RMC	**16b**
	h	0·22	0·26	L > H	7	90	30	IS	SX	RMC	**17**
	h	0·28	0·21	H > L	8	30	65	IS	OL	AIC	**18**
Ovulation rate	b	0·09	0·13	L > H	10	32	69	WFS	SX	RMC	**19**
Age of sexual maturity	h	0·32	0·49	L > H	6	40	75	WFS	L	RMC	**20**
		0·58	0·55								
Japanese Quail											
Egg Production	h	0·06	0·11	L > H	3	72	?	IFS	L	RMC	**21**
Male mating	h	0·03	0·28	L > H	6	38	61	IS	L	BP	**22**
		−0·08	0·17								
Chickens											
Male mating	b	2·53	1·50	H > L	2	?	?	IS	SL	BP	**23**
	h	0·18	0·31	L > H	6	32	58	IS	SL	BP	**24**
Hatching time	h	0·19	0·25	L > H	4	38	7	IS	OL	RMC	**25**
Tribolium											
Development time	h	0·11	0·32	L > H	13	80	10	IS	SL	BP	**26**
		0·14	0·14		7						
	h	0·49	0·22	H > L	6	16	18	IS	L	RMC	**27**
		0·35	0·27								
		0·35	0·29								
	h	-0·219	0·324	L > H	7	30	20	IS	L	RMC	**28**

Selection: A positive sign indicates response in the direction of selection and a negative sign response in the opposite direction

Key: M = method of estimation
- h = realized heritability (h^2)
- b = regression of response (as deviation from controls) on generations
- r = response to selection in the terminal generation(s) (as deviation from controls) divided by number of generations

G = duration in generations
N = parent population size per generation
i = selection differential as percentage selected
S = selection regime
- FS = family selection
- IFS = combination of individual selection in females and family selection in males
- IS = individual selection
- WFS = within-family selection

https://doi.org/10.1017/S0016672300028858 Published online by Cambridge University Press

Why was the predicted asymmetry not detected in all studies? Four possible reasons may be given without affecting the generality of the conclusion, namely: (a) sampling variation, (b) genetic drift, (c) differential forces of natural selection in laboratory and field populations, and (d) asymmetrical environmental variances such that selection in the two directions has differential efficiency.

It is instructive to examine the seven exceptions. Asymmetry changed direction from generation to generation in the second study reported by Narain *et al.* (1962), so sampling variation is an important consideration in that study.

In the study by Kessler (1969) the selection procedure was less satisfactory for S than F. The F parents were the first to mate, while S parents were chosen randomly from the unmated flies, such that (d) above appears to apply. Further, there were scale problems in the analyses.

De la Fuente & San Primitivo (1985) reported realized heritabilities of 0·28 and 0·21 for their high and low litter size lines, computed from regressions of deviations from controls on cumulative selection differentials. This response is asymmetrical in the opposite direction to that predicted. This direction is reversed when total responses (obtained from the means in the final generation) are divided by total selection differentials, yielding realized heritabilities of 0·192 and 0·214 for increased and decreased litter size. The early generations showed an anomalous decline in the high line (presumably due to a negative maternal effect of the type described by Falconer (1965)), that affected the regression and so has contributed to the reversed asymmetry.

Sang & Clayton (1957) reported reversed asymmetry of response over three generations for larval development time. Neither intermediate generation means nor selection differentials were reported, so it is not possible to calculate realized heritabilities or regressions.

The study by Tindell & Arze (1965) was a small scale study over two generations where the asymmetry in response reversed between generations one and two. They presented results only as responses. In a more extensive study, Siegel (1965) also reported asymmetrical responses for mating ability in chickens favouring the high direction, but he showed that there was marked asymmetry in the selection differentials, such that the realized heritabilities were asymmetrical in the predicted direction.

In the study by Dawson (1965) response changed from very weakly asymmetrical with slightly more response for higher fitness for generations 0–7, to strongly asymmetrical with more response for low fitness for generations 0–13.

The studies on developmental time in *Tribolium* are particularly instructive. Englert & Bell (1970), using a long-established base population, found asymmetrical response to selection, with greater response in the slow direction as predicted. However, this was reversed when realized heritabilities were taken, as the variance rose markedly in the S line (also observed by Soliman, 1982). Dawson (1975) has shown that selection for fast development is successful in long-established laboratory populations of *Tribolium*, but not in recently derived natural populations. In dense laboratory populations, selection against fast development occurs through cannibalism of early pupae by larvae, such that natural selection changes from directional selection for rapid development in the wild to stabilising selection in the laboratory. Consequently, the lack of asymmetry in the direction of lowered fitness in the result of Englert & Bell (1970) can be accounted for as being due to changed forces

---

B = Base population code
- OX = outbred cross
- W = recently caught wild strain
- L = wild laboratory strain
- OL = old laboratory strain
- WX = cross of wild strains
- SX = synthetic cross

C = Control code
- RMC = random mating control maintained with the same size as the selection lines
- BP = base population sample
- ILX = inbred line cross
- AIC = outbred control of same size, but with maximum avoidance of inbreeding mating system

? Information not reported
V Variable
* 17 generations for Fast and 11 for Slow
\# 11 generations for Short (High) and 6 for Long (Low)

References

1 Narain, Joshi & Prabhu (1962); **2** Richardson, Kojima & Lucas (1968); **3** Manning (1961); **4** Manning (1963); **5** Kessler (1969); **6** Sherwin (1975); **7** Spuhler *et al.* (1978); **8** Sang & Clayton (1957); **9** Marien (1958); **10** Hunter (1959); **11** Clarke, Maynard Smith & Sondhi (1961); **12** Moriwaki & Fuyama (1963); **13** Bakker (1969); **14** Hudak & Gromko (1989); **15** Sewell, Burnett & Connolly (1975); **16a** Falconer (1971); **16b** Falconer (1955); **17** Joakimsen & Baker (1977); **18** de la Fuente & San Primitivo (1985); **19** Land & Falconer (1969); **20** Drickamer (1981); **21** Lambio (1981); **22** Siegel (1980); **23** Tindell & Arze (1965); **24** Siegel (1965); **25** Smith & Bohren (1974); **26** Dawson (1965); **27** Englert & Bell (1970); **28** Soliman (1982)

https://doi.org/10.1017/S0016672300028858 Published online by Cambridge University Press

of natural selection in the laboratory versus field populations. In essence this is the exception that 'proves' the rule.

What can cause asymmetrical responses to selection? Falconer (1989) listed the following potential causes: (1) random drift, (2) different selection differentials, (3) inbreeding depression, (4) maternal-effects, (5) scalar asymmetry, (6) indirect selection, (7) genetic asymmetry, and (8) genes of large effect. In addition, asymmetrical natural selection opposing artificial selection can cause asymmetrical responses to selection. However, that cannot be the reason for the asymmetry observed here as natural selection is operating in the direction of increased fitness, while the observed asymmetry is in the direction of greater response for decreased fitness.

Each of these possible causes of asymmetrical responses to selection is discussed in turn below, in relation to the observed asymmetry of responses to selection for fitness traits.

(1) Genetic drift is a dispersive process that is random in direction. It may account for greater response in the direction of higher fitness in individual studies and replicates, but it cannot account for the asymmetry evident over the totality of studies.

(2) Different selection differentials in the two directions can be eliminated as the cause of the overall asymmetry, as significant asymmetry in the predicted direction (84%) is found when the comparison is limited to those studies reporting realized heritabilities.

(3) Inbreeding depression leads to asymmetry in the observed direction. This may well be the explanation in the Richardson *et al.* (1968) study as the inbreeding coefficient reached 35% at the end and the control population was not a random mating control of the same size and inbreeding level. However, it is not the main explanation in the cases reported here as the 18 studies using random mating controls of the same size reported significant asymmetry (83%) in the direction of reduced fitness. Inbreeding levels in these controls will be similar to those in the selection lines and so will act as a control for this effect. It might be argued that the inbreeding effect of selection results in higher inbreeding levels in the selection lines than the controls. While this is true, the inbreeding effect of selection is proportional to the heritability (Robertson, 1961). Since heritabilities of fitness characters are relatively low (see Mousseau & Roff, 1987; Roff & Mousseau, 1987; Falconer, 1989) this bias will be small.

(4) Maternal effects are an unlikely explanation for the observed asymmetry as many of the traits are unlikely to be subject to maternal effects (particularly those in *Drosophila*) and care has been taken in most cases where this is a potential problem to control it. As Falconer (1989) pointed out 'To attribute asymmetry of response to a maternal effect, however, only transfers the problem from the character selected to another and does not explain the asymmetry'.

(5) Scalar asymmetry is unlikely to explain the overall asymmetry as there is no reason to believe that it is operating in a consistent direction in all the studies. Further, it is largely eliminated by taking realized heritabilities. In the case of Kessler (1969), scale effects may have been partially responsible for asymmetrical response in the opposite direction to the majority of studies.

(6) Indirect selection is not relevant to the studies discussed here.

(7) Genetic asymmetry is the most probable cause of the observed asymmetry. As described in the Introduction, reproductive fitness characters exhibit directional dominance as indicated by inbreeding depression. Asymmetrical gene frequencies are to be expected from the directional forces of natural selection operating on these characters, though direct evidence for this is scanty and difficult to obtain. The component of the genetic variation for fitness that is due to mutation-selection balance for deleterious mutations certainly results in asymmetrical gene frequencies with low frequencies of deleterious alleles (see Lewontin, 1974).

(8) Genes of large effect at asymmetrical initial frequencies may also lead to asymmetrical response in the early generations, especially if selection is intense (Latter, 1965). Even with directional dominance and asymmetrical gene frequencies, gene effects must be of a reasonable size for asymmetry to be detectable in short-term response. Asymmetrical responses to selection due to rare essentially recessive alleles of large effect have been observed by Costantino *et al.* (1967) and Frankham & Nurthen (1981). Rare deleterious recessive mutations in mutation-selection balance could be contributing to the observed asymmetry. Such genes will lead to immediate asymmetry in response to selection. Studies vary somewhat in the onset of detectable asymmetry. However, the time of onset is difficult to detect in most cases due to the large environmental variances for fitness characters.

Overall, it is most probable that the observed asymmetry is due to genetic asymmetry resulting from directional dominance, asymmetrical gene frequencies and genes of large effect.

Does the potential for asymmetry exist initially (reflected in non-linear offspring-parent regressions and initial heritabilities), or does it manifest itself later as selection changes gene frequencies? It is not possible to answer this question definitively on the basis of information that is available. Direct tests for non-linear offspring-parent regressions for fitness traits need to be performed.

The detection of overall asymmetry of selection responses for reproductive fitness characters has important practical implications. Firstly, the effectiveness of low intensity culling on fitness in lines selected for other characters is likely to be greater than the average heritability of fitness indicates. Secondly, response to selection for increased fitness is likely to

be less effective than predicted from average heritabilities, so cost benefit analyses are likely to be over optimistic. It may be possible to predict the asymmetry using Abplanalp's linear heritability procedures (Abplanalp, 1961; Arthur & Abplanalp, 1975; Hill, 1978; Medrano & Abplanalp, 1989). Further, asymmetry has been predicted from the directional nature of natural selection on reproductive fitness. This also predicts low additive genetic variation for fitness characters. Consequently, the use of line or population crosses as base populations for selection studies involving fitness characters can be recommended to supply genetic variation.

In conclusion, it has been hypothesized that heritabilities for reproductive fitness traits are non-linear, and higher in the low fitness direction. Two predictions of this hypothesis have been validated. Firstly, culling on fitness has been shown to be effective in alleviating fitness declines in lines selected for another trait (Frankham *et al.* 1988). Secondly, responses to selection for fitness traits have been shown to be consistently asymmetrical, with greater response in the direction of lowered fitness.

I am grateful to M. Graham for technical assistance, to B. Duckworth for drawing Figure 1, to K. E. Nestor for information on the Lambio Study, to A. K. Sheridan for supplying references, and to H. Abplanalp, J. S. F. Barker, B. Charlesworth, F. D. Enfield, D. S. Falconer, D. Haig, W. G. Hill, B. D. H. Latter, A. Maki-Tanila, T. A. Mousseau, F. W. Nicholas, D. A. Roff, P. B. Siegel, K. E. Weber and A. Wilton for comments on manuscripts. This work was supported by Australian Research Council and Macquarie University Research grants.

References

Abplanalp, H. (1961). Linear heritability estimates. *Genetical Research* **21**, 439–448.

Arthur, J. A. & Abplanalp, H. (1975). Linear estimates of heritability and genetic correlation for egg production, body weight, conformation and egg weight of turkeys. *Poultry Science* **54**, 11–23

Bakker, K. (1969). Selection for rate of growth and its influence on competitive ability of larvae of *Drosophila melanogaster*. *Netherlands Journal of Zoology* **19**, 541–595.

Bulmer, M. G. (1980). *The Mathematical Theory of Quantitative Genetics*. Oxford University Press.

Charlesworth, B. (1980). *Evolution in Age-Structured Populations*. Cambridge University Press.

Charlesworth, B. (1987). The heritability of fitness. In *Sexual Selection: Testing the Alternatives* (ed. J. W. Bradbury and M. B. Andersson), pp. 21–40. New York: John Wiley.

Charlesworth, D. & Charlesworth, B. (1987). Inbreeding depression and its evolutionary consequences. *Annual Review of Ecology and Systematics* **18**, 237–268.

Clarke, J. M., Maynard Smith J. & Sondhi, K. C. (1961). Asymmetrical response to selection for rate of development in *Drosophila subobscura*. *Genetical Research* **2**, 70–81.

Costantino, R. F., Bell, A. E. & Rogler, J. C. (1967). Genetic analysis of a population of *Tribolium*. *Heredity* **22**, 529–539.

Crow, J. F. & Kimura, M. (1970). *An Introduction to Population Genetics Theory*. New York: Harper and Row.

Curnow, R. N. (1960). The regression of true value on estimated value. *Biometrika* **47**, 457–460.

Dawson, P. S. (1965). Genetic homeostasis and developmental rate in *Tribolium*. *Genetics* **51**, 873–885.

Dawson, P. S. (1975). Directional versus stabilising selection for developmental time in natural and laboratory populations of flour beetles. *Genetics* **80**, 773–783.

de la Fuente, L. F. & San Primitivo, F. (1985). Selection for large and small litter size of the first three litters of mice. *Génétique, Sélection, Evolution* **17**, 251–264.

Drickamer, L. C. (1981). Selection for age of sexual maturation in mice and the consequences for population regulation. *Behavioral and Neural Biology* **31**, 82–89.

Englert, D. C. & Bell, A. E. (1970). Selection for time of pupation in *Tribolium castaneum*. *Genetics* **64**, 541–552.

Falconer, D. S. (1954). Asymmetrical responses in selection experiments. In *Symposium on the Genetics of Population Structure*, pp. 16–41. Naples: International Union of Biological Sciences, Series B, no. 15.

Falconer, D. S. (1955). Patterns of response in selection experiments with mice. *Cold Spring Harbor Symposia on Quantitative Biology* **20**, 178–196.

Falconer, D. S. (1965). Maternal effects and selection response. In *Genetics Today*, Vol. 3 (ed. S. J. Geerts), pp. 767–773. Oxford: Pergamon Press.

Falconer, D. S. (1971). Improvement of litter size in a strain of mice at a selection limit. *Genetical Research* **17**, 215–235.

Falconer, D. S. (1989). *Introduction to Quantitative Genetics*, 3rd edn. London: Longman.

Fisher, R. A. (1930). *The Genetical Theory of Natural Selection*. Oxford: Clarendon.

Frankham, R. (1982). Contribution of *Drosophila* research to quantitative genetics and animal breeding. *Proceedings of the 2nd World Congress on Genetics Applied to Livestock Production* V, 43–56.

Frankham, R. & Nurthen, R. K. (1981). Forging links between population and quantitative genetics. *Theoretical and Applied Genetics* **59**, 251–263.

Frankham, R., Yoo, B. H. & Sheldon, B. L. (1988). Reproductive fitness and artificial selection in animal breeding: culling on fitness prevents a decline in reproductive fitness in lines of *Drosophila melanogaster* selected for increased inebriation time. *Theoretical and Applied Genetics* **76**, 909–914.

Gimelfarb, A. (1986). Offspring-parent genotypic regression: how linear is it. *Biometrics* **42**, 67–71.

Gowe, R. S. (1983). Lessons from selection studies in poultry for animal breeders. *Proceedings of the 32nd Annual Breeders' Roundtable*, pp. 22–50.

Gustafsson, L. (1986). Lifetime reproductive success and heritability: empirical support for Fisher's fundamental theorem. *American Naturalist* **128**, 761–764.

Hill, W. G. (1978). Estimation of heritability by regression using collateral relatives: linear heritability estimation. *Genetical Research* **32**, 265–274.

Hudak, M. J. & Gromko, M. H. (1989). Responses to selection for early and late development of sexual maturity in *Drosophila melanogaster*. *Animal Behaviour* **38**, 344–351.

Hunter, P. E. (1959). Selection of *Drosophila melanogaster* for length of larval period. *Zeitschrift für Vererbungslehre* **90**, 7–28.

Joakimsen, O. & Baker, R. L. (1977). Selection for litter size in mice. *Acta Agriculturae Scandinavica* **27**, 301–318.

Kempthorne, O. (1960). Biometrical relationships between relatives and selection theory. In *Biometrical Genetics* (ed. O. Kempthorne), pp. 12–23. London: Pergamon.

Kessler, S. (1969). The genetics of Drosophila mating

behaviour. II. The genetic architecture of mating speed in *Drosophila pseudoobscura*. *Genetics* **62**, 421–433.
Kimura, M. (1958). On the change of population fitness by natural selection. *Heredity* **12**, 145–167.
Kojima, K. (1961). Effects of dominance and size of population on response to mass selection. *Genetical Research* **2**, 177–188.
Kojima, K. & Kelleher, T. M. (1961). Changes in mean fitness in random mating populations when linkage and epistasis are present. *Genetics* **46**, 527–540.
Lambio, A. L. (1981). Response to divergent selection for 4-week body weight, egg production and total plasma phosphorus in Japanese quail. *Dissertation Abstracts International* B **42**, 2694.
Land, R. B. & Falconer, D. S. (1969). Genetic studies of ovulation rate in the mouse. *Genetical Research* **13**, 25–46.
Latter, B. D. H. (1965). The response to artificial selection due to autosomal genes of large effect. I. Changes in gene frequency at an additive locus. *Australian Journal of Biological Sciences* **18**, 585–598.
Latter, B. D. H. (1970). Selection in finite populations with multiple alleles. II. Centripetal selection, mutation, and isoallelic variation. *Genetics* **66**, 165–186.
Lewontin, R. C. (1974). *The Genetic Basis of Evolutionary Change*. New York: Columbia University Press.
Maki-Tanila A. (1982). The validity of the heritability concept in quantitative genetics. PhD Thesis, University of Edinburgh.
Manning, A. (1961). The effects of artificial selection for mating speed in *Drosophila melanogaster*. *Animal Behaviour* **11**, 116–120.
Manning, A. (1963). Selection for mating speed in *Drosophila melanogaster* based on the behaviour of one sex. *Animal Behaviour* **11**, 116–120.
Marien, D. (1958). Selection for developmental rate in *Drosophila pseudoobscura*. *Genetics* **50**, 3–15.
Medrano, J. F. & Abplanalp, H. (1989). Prediction of selection response in asymmetrically distributed traits from selection components. *Journal of Animal Breeding and Genetics* **106**, 110–119.
Meyer, H. H. & Enfield, F. D. (1975). Experimental evidence on limitations of the heritability parameter. *Theoretical and Applied Genetics* **45**, 268–273.
Moriwaki, D. & Fuyama, Y. (1963). Responses to selection for rate of development in *D. melanogaster*. *Drosophila Information Service* **38**, 74.
Mousseau, T. A. & Roff, D. A. (1987). Natural selection and the heritability of fitness components. *Heredity* **59**, 181–197.
Naglaki, T. (1979). Selection in dioecious populations. *Annals of Human Genetics* **43**, 143–150.
Narain, P., Joshi, C. & Prabhu, S. S. (1962). Response to selection for fecundity in *D. melanogaster*. *Drosophila Information Service* **36**, 96–99.
Nishida, A. (1972). Some characteristics of parent-offspring regression in body weight of *Mus musculus* at different ages. *Canadian Journal of Genetics and Cytology* **14**, 292–303.
Nishida, A. & Abe, T. (1974). The distribution of genetic and environmental effects and the linearity of heritability. *Canadian Journal of Genetics and Cytology* **16**, 3–10.
Richardson, R. H., Kojima, K. & Lucas, H. L. (1968). An analysis of short-term selection experiments. *Heredity* **23**, 493–506.
Robertson, A. (1955). Selection in animals: synthesis. *Cold Spring Harbor Symposia on Quantitative Biology* **20**, 225–229.
Robertson, A. (1961). Inbreeding in artificial selection programmes. *Genetical Research* **2**, 189–194.
Robertson, A. (1977). The non-linearity of offspring-parent regression. In *Proceedings of the International Conference on Quantitative Genetics* (ed. E. Pollak, O. Kempthorne and T. B. Bailey), pp. 297–304. Ames: Iowa State University Press.
Roff, D. A. & Mousseau, T. A. (1987). Quantitative genetics and fitness: lessons from *Drosophila*. *Heredity* **58**, 103–118.
Sang, J. H. (1962). Selection for rate of development using *Drosophila melanogaster* cultured axenically on deficient diets. *Genetical Research* **3**, 90–109.
Sang, J. H. & Clayton, G. A. (1957). Selection for larval development time in *Drosophila*. *Journal of Heredity* **48**, 265–270.
Sewell, D., Burnett, B. & Connolly, K. (1975). Genetic analysis of larval feeding behaviour in *Drosophila melanogaster*. *Genetical Research* **24**, 163–173.
Sherwin, R. N. (1975). Selection for mating activity in two chromosomal arrangements of *Drosophila pseudoobscura*. *Evolution* **29**, 519–530.
Siegel, P. B. (1965). Genetics of behavior: selection for mating ability in chickens. *Genetics* **52**, 1269–1277.
Siegel, P. B. (1980). Behavior genetic analysis in chickens and quail. *Proceedings of the 29th National Breeders' Roundtable*, pp. 1–13.
Smith, K. P. & Bohren, B. B. (1974). Direct and correlated responses to selection for hatching time in the fowl. *British Poultry Science* **15**, 597–604.
Soliman, M. H. (1982). Directional and stabilising selection for developmental time and correlated response in reproductive fitness in *Tribolium castaneum*. *Theoretical and Applied Genetics* **63**, 111–116.
Spiess, E. B. & Spiess, L. B. (1966). Selection for rate of development and gene arrangement frequencies in *Drosophila persimilis*. II. Fitness properties at equilibrium. *Genetics* **53**, 695–708.
Spuhler, K. P., Crumpacker, D. W., Williams, J. S. & Bradley, B. P. (1978). Response to selection for mating speed and changes in gene arrangement frequencies in descendants from a single population of *Drosophila pseudoobscura*. *Genetics* **89**, 729–749.
Tindell, D. & Arze, C. G. (1965). Sexual maturity of male chickens selected for mating ability. *Poultry Science* **44**, 70–72.
Turner, J. R. G. (1970). Changes in mean fitness under natural selection. In *Mathematical Topics in Population Genetics* (ed. K. Kojima), pp. 32–78. Berlin: Springer-Verlag.