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Artificial Selection for Short and Long Attack Latencies in Wild *Mus musculus domesticus*

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Artificial selection for short and long attack latency levels in wild male Mus musculus *over 11 generations was successful for short latencies. The realized heritability of 0.30 is comparable to those found in other selection studies on aggression. In part selection may have been for faster ontogenetic development of short attack latencies. Four attempts to select for longer attack latencies failed because the lines died out immediately or within two generations for unknown reasons. But neither the physical condition of the animals nor their behavior appeared to have been the cause. Female aggressiveness as measured in female–female encounters was not affected by the selection exerted on the males. This suggests that no genetic correlation exists between aggressiveness of males and females, confirming results of P. D. Ebert and J. S. Hyde [(1976).* Behav. Genet. *6:291–304] obtained in a selection experiment on aggression using females.*

KEY WORDS: artificial selection; *Mus musculus*; aggression; attack latency; mice.

INTRODUCTION

Although aggressive behavior has been a favorite topic in behavior studies during recent years, only little is known about the genetics of this behavior. One of the means to study the genetics of a character is artificial selection. Apart from ascertaining whether observed individual differences stem from genetic differences or not, it often provides information about the amount of genetic variation present, the limits to selection, and

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the number of loci involved. Furthermore, it may create strains differing genetically for the selected trait and possibly for correlated traits. Such strains are very useful for further behavior-genetic research (DeFries, 1967).

Selection experiments on aggressive behavior in house mice have been carried out by Lagerspetz (1964) on aggressiveness in males of laboratory strains and by Ebert and Hyde (1976) on aggressiveness in wild females. In both studies a complex rating scale has been used to measure aggressiveness. The scale of Lagerspetz included attack behavior and flight behavior, so that it may be regarded as a measure of agonistic behavior (Manning, 1972, p. 100) instead of aggressive behavior. Although clear flight elements were not included in the Ebert and Hyde scale, some elements of avoidance behavior seem to have been included, according to the description of the scale. If so, experiments using either scale select for two opposing tendencies, a tendency to attack and a tendency to flee. If flight and attack are extreme manifestations of one causal mechanism, nothing is wrong in doing so. However, the existence of two separate mechanisms, one for aggression and one for flight, cannot be excluded. Some studies indeed showed that a tendency to flee may vary independently from a tendency to attack (for examples, see Manning, 1972, p. 101), suggesting that selection using bidirectional rating scales may not provide adequate estimates of the genetic influences on either aggression or flight. These considerations led to the present selection experiment in which attack behavior, a pure measure of aggression by definition, served as the criterion.

MATERIALS AND METHODS

The mice came from a colony of wild mice maintained in our laboratory since 1971. This colony descended from 4 males and 3 females caught in a mansion situated near the town of Groningen (The Netherlands) at latitude 53°11′ N and longitude 6°36′ E. The colony was bred at random. Every generation 15 to 20 pairs were taken to be parents for the next generation. In the summer of 1973 selection was started with 21 males and 21 females. The original colony was used as a control. It was tested as such at the 4th, 9th, 10th, and 11th generations of selection.

All animals were housed in small Plexiglas cages ($17 \times 11 \times 13$ cm^3) in a room with a reversed day–night cycle (darkness from 11 AM to 11 PM). Temperature varied between 18 and 21°C; humidity, from 45 to 60%. At weaning age (3–4 weeks), the litters were transferred to larger cages ($32 \times 17 \times 13$ cm^3). At the age of sexual maturity (7–9 weeks), the animals were set up in pairs in the smaller cages. Cages were cleaned at

least once a fortnight. Water and food (standard pellets, Hope Farms AM 11) were present ad libitum. During observations the room was lit by three 15-W bulbs.

Dummies do not elicit aggressive behavior in house mice (Lagerspetz, 1964), therefore, young males of an inbred albino strain (MAS-Gro) were used as opponents. These mice only very seldom attacked the experimental animals. They were used a couple of times but discarded as soon as they attacked by themselves.

Female mice do not readily attack other mice. However, Edwards (according to White *et al.*, 1969) showed that a number of them will attack young female opponents, but only if the experimental females have been isolated from the time of weaning. Therefore, all experimental females tested in this study were treated this way and tested against young MAS-Gro females that were smaller than they.

Attack behavior may be measured by frequency, latency, and/or duration. Catlett (1961), who evaluated the use of these criteria, found a high degree of association among them. Wishing to minimize the influence of experience in fighting (Ginsburg and Allee, 1942; Lagerspetz, 1964), we chose latency of attack as the measure. For as soon as an attack starts, the fighters can be separated, whereas when frequency or duration is used the animals get much more experience in fighting.

Male house mice regularly patrol the borders of their territories, and most agonistic confrontations occur there (Crowcroft, 1966). In our experiments we tried to create such a border situation. We, therefore, measured attack latency in cages of 80 × 30 × 30 cm^3. The cages (Fig. 1) were divided into four compartments (A, B, C, and D) by Plexiglas slides 1, 2, and 3, the last one being perforated. Compartments A and B functioned as the home cage for the experimental animals, C as the border area with which the experimental animal was only superficially acquainted, and D as an introduction chamber for the opponents. The floor was covered with sawdust and shavings. The test cage was cleaned before an experimental animal was introduced.

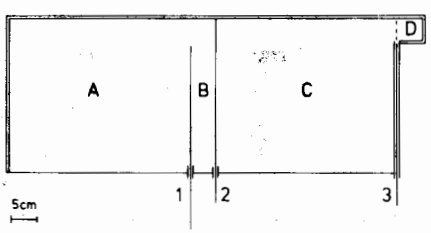

Fig. 1. Ground plan of the test cage. For explanation, see text.

An experimental animal lived in the test cage for 4 days. On Monday it was introduced and got the opportunity to explore compartments A, B, and C for 1 hr. Then slide 2 was closed, confining the animal to A and B. Tuesday afternoon, the mouse was allowed to explore C for another hour, to make it familiar with the border area. An attack latency test took place each afternoon on the following 3 days. For a test, the experimental mouse was locked up in B and the opponent in D. Then slide 2 was opened. The time from the moment the experimental animal entered C until it nosed at compartment D was measured and called the meet latency. Then slide 3 was opened and the actual attack latency measurement started, ending at the first sign of attack by the experimental animal.

Pilot tests showed that if an animal did not attack within 10 min, it was not likely to attack at all; thus 600 sec was taken as the maximum latency. Only a slight association was found between meet latency and attack latency (Spearman's rank correlation coefficient $r_s = 0.143$; $t = 1.543$; $N = 116$; $P < 0.01$; two tailed) (Siegel, 1956). This confirmed our impression that the experimental animals detected the opponent in compartment D only at very short distances. The few tests in which the opponent attacked first were discarded and repeated the next day, unless the experimental animal had counterattacked immediately. An experimental animal was never tested more than once a day and never met the same opponent in more than one test.

The mean of three tests of a given animal was its attack latency score (ALS). In this way animals that failed to attack at least once obtained a minimum ALS of 200 sec. All animals with an ALS shorter than 200 sec were called fast attacking (Fast); the others, slow attacking (Slow).

Only males were selected because females normally do not show attack behavior. A male chosen for breeding was paired to other females if breeding with his first mate failed, but never to more than two females simultaneously. Females were chosen for breeding, using the scores of their brothers.

In selecting for long latencies (see Table II), all males that failed to attack at least once were chosen to sire the next generation. In selecting for short latencies (see Table I), those males were chosen which had the shortest latencies and good breeding results. The number of breeder pairs and the number of animals bred each generation varied depending on the number of mice needed for other purposes. Only healthy-looking animals were chosen as breeders.

During the first three generations selection was combined with inbreeding (brother × sister) because it seems that in nature strong inbreeding occurs within demes (Selander, 1970). Generation 4 was bred by inbreeding and outbreeding. No differences between the two groups

of descendants could be detected in this generation. Therefore from then on, to avoid genetic drift, only outbreeding was practiced.

RESULTS AND DISCUSSION

Selection for Short Attack Latency

Results of the selection for short attack latency are given in Table I and Fig. 2. Data on generation 8 of the short attack latency line (SAL line) are lacking. At this generation a disease, probably pleuropneumonia, broke out among the mice of this line, forcing us to use without selection all remaining healthy animals to produce the ninth generation. Selection for short latency was successful as indicated by the regression of cumulated weighted selection differential on mean ALS (attack latency score) per generation: $\hat{y}_i = 204.1 + 0.148\, x_i$; df = 9; $t = 3.55$; $P < 0.01$, with a realized heritability of 0.30 ± 0.19 (95% confidence interval).

The heritability found is slightly lower than those found in the other two selection studies on aggression in house mice: for males, 0.36, as estimated by McClearn and DeFries (1973) from data of Lagerspetz; for females, 0.38 to 0.49, as found by Ebert and Hyde (1976). The difference might be due to factors known to reduce genetic variability such as the small number of animals that started the base population prior to selection or to inbreeding practiced during the initial generations of selection. Nevertheless, the realized heritability observed is still rather large.

The control line (C line) was tested at the same time as the 4th, 8th, 9th, 10th, and 11th generations of the SAL line (Table I and Fig. 2). Comparison of the results of both lines shows that at comparable generations the SAL line always obtained lower mean ALS values than the C line (Kruskal–Wallis H test; H varying between 10.42 and 30.25; df = 1; P always less than 0.01). This is also expressed by the fact that the percentages of Fast males in the SAL line increased from 61.9 to 96.5%, which is a significant increase ($\hat{y}_i = 72.4 + 2.1\, x_i$; $t = 2.75$; df = 9; $P < 0.03$), whereas in the C line such an increase could not be demonstrated ($\hat{y}_i = 59.8 - 0.3\, x_i$; $t = 0.31$; df = 4). When we compare the groups of Fast animals in the SAL$^-$ and the C lines using the Kruskal–Wallis H test, it again becomes clear that in all comparable generations the samples were taken from different populations (H varying between 4.92 and 18.62; df = 1; P always less than 0.05). This means not only that selection acted against Slow types but that among the Fast animals a shift to shorter attack latencies had taken place as well. In generation 11 of the SAL line most animals obtained very low ALS scores. In 75.9% of all cases (N = 261) attacks took place within 10 sec; for the C line this percentage

Table I. Results of Selection for Short Attack Latency (SAL Line) and of the Control Line

	Short attack latency line										Control line			
	Tested males				Selected males									
Generation	*N*	$\overline{ALS}$	% Fast males	$\overline{ALS}$ of Fast males	*N*	$\overline{ALS}$	Selection differential (SD)	Weighted SD[a]	Cumulated weighted SD	Response	*N*	$\overline{ALS}$	% Fast males	$\overline{ALS}$ of Fast males
0	21	220.4	61.9	59.3	9	44.8	−175.6	−176.0	0.0	0.0	21	220.4	61.9	59.3
1	30	166.4	73.3	53.4	8	33.0	−133.4	−140.7	−176.0	−54.0	—	—	—	—
2	37	71.1	91.9	29.6	5	4.5	−66.6	−66.7	−316.7	−95.3	—	—	—	—
3	37	231.3	67.6	88.1	7	46.5	−184.8	−189.6	−383.4	+160.2	—	—	—	—
4	45	104.2	88.9	60.6	11	8.1	−96.1	−97.3	−573.0	−127.1	12	328.5	54.5	120.7
5	47	129.7	83.0	44.5	20	22.9	−106.8	−115.1	−670.3	+25.5	—	—	—	—
6	84	53.2	92.9	34.0	15	2.1	−51.1	−51.5	−785.4	−76.5	—	—	—	—
7	45	112.9	82.2	45.6	8	3.6	−109.3	−110.9	−836.9	−59.7	—	—	—	—
8	—	—	—	—	—	—	—	—	−947.8	—	12	273.0	66.7	91.4
9	12	43.2	91.7	20.6	6	4.3	−38.9	−38.2	−947.8	−69.7	25	309.6	44.0	69.5
10	14	83.6	85.7	50.0	8	38.2	−45.4	−62.2	−986.0	+40.4	56	310.2	48.2	74.3
11	87	31.1	96.5	15.1	—	—	—	—	−1048.2	−59.2	24	180.5	66.7	67.7

[a] Weighted selection differential $= (1/N) \sum f_i/\bar{f}\ (X_i - \bar{X})$, where N is the number of selected males, f_i is the number of tested progeny produced by the ith parental male, $\bar{f}$ is the average contribution of all parental males, X_i is the ALS of the ith parental male, and $\bar{X}$ is the mean ALS of all tested males.

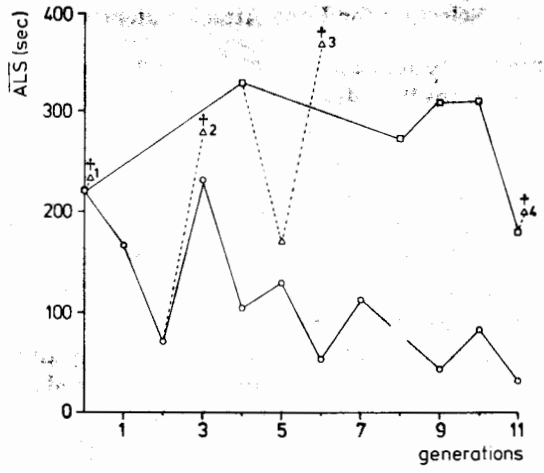

Fig. 2. Mean attack latency scores ($\overline{ALS}$) of six lines of mice: one selected for short attack latency scores (SAL line, circles), four selected for long attack latency scores (LAL lines, triangles 1 to 4), and one control line (C line, squares). †, Extinction.

was 22.2% (N = 72). In 24.9% of the cases SAL animals even attacked sliding door 3, behind which the opponent was staying (see Fig. 1), before it could be opened; this behavior was never seen in the C line.

In each generation a number of animals seemed terrified by the opening of slide 3, causing them to run away from the opponent. This resulted in some high attack latency measures in generations in which also very low attack latencies were found, e.g., in generation 11 of the SAL line. Consequently, as selection continued the experimental procedure seemed to preclude further reduction of ALS values. Therefore the maximum response in this selection was almost reached in 11 generations, a fairly short time. This makes it another example of the finding that in selection experiments using a behavioral character the maximum response is often reached much more quickly than in selection experiments using a nonbehavioral character (Roberts, 1967).

Breeding pairs stayed together during pregnancy and the rearing of young. Thus the behavior of both parents may have influenced their behavior. For instance, just after cleaning of the cages, males tend to be aggressive toward their females and progeny. Males of the SAL line showed this behavior more frequently than males of the C line. In a few cases, SAL males even killed their young. However, cross-fostering experiments between long- and short-latency animals of the C line and between the C and the Sal lines did not result in detectable significant influences of rearing on ALS values.

Selection for Long Attack Latency

Four attempts were made to select for long attack latency simultaneously with generations 0, 2, 4, and 11 of the SAL line. Results are given in Table II and Fig. 2. The first and the last two attempts started from the original or control population (at generations 0, 4, and 11); the second attempt started from the second generation of the SAL line. All attempts failed because the lines died out immediately or within two generations, as no progeny could be obtained. In the first and second attempts, inbreeding was practiced; in the other two, outbreeding. In the third attempt selection was more relaxed than in the other three, in that *all* Slow males were used to sire a next generation rather than only those males that failed to attack at least once during testing (see Materials and Methods). This may be why this attempt lasted for two generations before the line died out.

Selection for long attack latency (LAL) failed because, sooner or later, no progeny could be obtained. All males used to breed an LAL line were of the Slow type. In the SAL line all sires were Fast males. Differences in reproduction between Fast and Slow males of inbred as well as feral mice have been presented by Busser *et al.* (1974). In that study a high number of Slow males failed to breed, and those who did produced fewer progeny than Fast males. In this study the mean number of young per reproducing pair per 3 weeks was 4.8 ± 0.1 (SEM) for Fast males and 2.9 ± 0.6 for Slow males, a difference that could not be ascribed specifically to litter size, litter interval, or death rate of young. Since only healthy-looking animals were used for breeding, inferior physical con-

Table II. Selection for Long Attack Latency (LAL Lines)

Attempt	Generation (origin)[a]	Tested males		Selected males		Selection differential (SD)	Weighted SD	Cumulated weighted SD	Response
		N	$\overline{ALS}$	*N*	$\overline{ALS}$				
I	0 (original population)	21	220.4	8	482.1	+261.7	—	—	—
II	0 (SAL 2)	37	71.1	2	554.2	+483.1	456.9	0.0	—
	1	14	277.5	7	443.9	+166.4	—	+456.9	+206.4
III	0 (C 4)	12	328.5	6	536.3	+207.8	+78.4	0.0	—
	1	11	170.5	5	243.9	+73.4	+104.4	+78.4	−158.0
	2	10	369.5	6	570.6	+201.1	—	+182.8	+194.0
IV	0 (C 11)	24	180.5	8	406.0	+225.5	—	—	—

[a] SAL, short attack latency line; C, control line.

dition cannot have been the main cause for the infertility of animals with long attack latencies, unless we dealt with a health problem we were unable to detect. Inbreeding is known to affect fertility after a number of generations. The SAL line, the C line, and the LAL lines were inbred by the same amount. As we had no problem breeding the SAL^- and the C lines, it is not likely that inbreeding was a major factor causing the infertility problems of the LAL lines. Variation in sexual behavior cannot be involved, for males with long attack latencies elicited the proper courting behavior, seducing females into copulation as easily as males with short attack latencies did.

Attack latency matures. All animals tend to show shorter attack latencies as they become older. This change develops more quickly in males with short attack latencies than in those with long attack latencies. The development of other characters, essential for a male to become fertile, might be influenced by the delay in the same maturation process.

In selection studies on aggression in house mice, whether exerted on males or on females, selection for low aggressiveness always appears less effective than selection for high aggressiveness. This is also confirmed by this study, in which selection for low aggressiveness could not even be practiced because the lines died out. These findings suggest that a minimum amount of aggression in the animals is protected by the genotype. If such a protection occurs by means of a balancing system, it presents another explanation for the fertility problems in selecting for long attack latencies (Falconer, 1964; van Oortmerssen, 1971).

Attack Latency Scores in Females

Using Edwards' method (see Materials and Methods), we tested females of the 6th, 7th, 10th, 11th, and 12th generations of the SAL line and females of the 9th, 10th, 11th, and 12th generations of the C line. Results are summarized in Table III. About 80% of the females of the 6th, 7th, and 10th generations of the SAL line attacked at least once in one of the three attack latency tests; 30 to 40% even showed ALS values comparable to those of Fast males.

Females of generation SAL 10 showed significantly shorter ALS values than females of generation C 10 (Kruskal–Wallis test; $H = 4.81$; $df = 1$; $P < 0.05$). Up to generation 10, the results suggest that the selection of males affected also the ALS values of females. However, in generations 11 and 12 no significant differences in ALS values between the SAL^- and C lines could be detected any longer (Table IV) because the ALS values of generations 11 and 12 of the SAL line had gone up to the level of those of the C line. This becomes especially clear when we

Table III. Attack Latency in Females of the Short Attack Latency Line and of the Control Line

	Short attack latency line						Control line					
	All females		Females that attacked at least once		Fast females		All females		Females that attacked at least once		Fast females	
Generation	*N*	$\overline{ALS}$	%	$\overline{ALS}$	%	$\overline{ALS}$	*N*	$\overline{ALS}$	%	$\overline{ALS}$	%	$\overline{ALS}$
6	75	277.7	88.0	233.7	41.3	67.5	—	—	—	—	—	—
7	49	260.1	81.6	183.7	44.9	68.0	—	—	—	—	—	—
9	—	—	—	—	—	—	13	524.6	30.8	355.0	0	—
10	10	318.2	80.0	130.3	30.0	67.6	24	491.4	29.2	227.8	12.5	52.0
11	19	532.7	31.6	386.8	5.2	136.5	25	594.6	4.0	463.9	0	—
12	48	553.2	14.5	279.2	4.2	89.7	34	596.4	5.8	538.8	0	—

compare the ALS values of generations SAL 10 and SAL 11, which differ significantly (Table IV). We could not detect a cause for the rise in ALS values of the SAL line. A similar but statistically nonsignificant change occurred in the C line. In this line the change is most clear when generations are compared for percentages of females attacking at least once (see Table III). The results suggest an environmental influence, which affects SAL females more than C line females. It is not clear what kind of influence that may have been. It is clear, however, that the change in ALS values of the females of the SAL line is opposite to that of the males, suggesting that aggression, as measured here, has a different genetic back-

Table IV. Comparison of the Distribution of ALS Values of the Females of the Short Attack Latency Line and of the Control Line for a Number of Generations Between and Within Lines with the Kruskal–Wallis *H* Test

Comparison	*H*	df	*P*[a]	Comparison	*H*	df	*P*[a]	Comparison	*H*	df	*P*[a]
SAL 10–C 10	4.81	1	a	SAL 6–SAL 7	0.15	1	n	C 9–C 10	0.02	1	n
SAL 11–C 11	2.48	1	n	SAL 6–SAL 10	0.39	1	n	C 9–C 11	1.87	1	n
SAL 12–C 12	0.54	1	n	SAL 6–SAL 11	21.37	1	b	C 9–C 12	1.97	1	n
				SAL 6–SAL 12	50.18	1	b	C 10–C 11	2.43	1	n
				SAL 7–SAL 10	0.88	1	n	C 10–C 12	2.59	1	n
				SAL 7–SAL 11	17.01	1	b	C 11–C 12	0.01	1	n
				SAL 7–SAL 12	36.34	1	b				
				SAL 10–SAL 11	5.34	1	a				
				SAL 10–SAL 12	4.42	1	a				
				SAL 11–SAL 12	0.94	1	n				

[a] n, nonsignificant; a, $P < 0.05$; b, $P < 0.01$.

ground in males than in females. This agrees with the findings of Hyde and Ebert (1976), who arrived at the same conclusion when selecting females for aggression.

Nongenetic Factors Influencing Attack Latency

Age. In some generations of the SAL$^-$ and the C lines, groups of males were tested for attack latency a second time when older. The distributions of ALS values of nine SAL$^-$ and three C groups were compared within groups. In all groups ALS values tended to decrease with increasing age; in five SAL$^-$ and two C groups this change was significant at the 5% level (Wilcoxon signed rank test), depending on the test age and the time between tests. The decrease in ALS values was most clear in C animals, because ALS values of SAL animals were already small at the first test. This difference is expressed by the average decreases in seconds per day over 150 days shown by all SAL animals ($N = 102$) and by all C animals ($N = 31$), which were 0.2 ± 0.03 (SEM) and 2.1 ± 0.38, respectively. This suggests that selection for short ALS values might in part have been selection for a quicker maturation of attack readiness. If this were so, one would want to know whether this accelerated development affects only attack behavior or whether it is a general developmental feature of SAL animals.

Social Isolation. Some ($N = 25$) of the males of generation 11 of the SAL line were isolated at weaning, others ($N = 35$) were isolated about a month before testing, while the rest ($N = 26$) grew up with their littermates up to sexual maturity, living thereafter in pairs with a female. No significant difference in mean ALS between groups could be detected (Kruskal–Wallis H test; $H = 2.38$; df $= 2$; $P = 0.3$). Thus it seems that isolation does not have a profound influence on ALS.

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