Table 2. Frequencies of chromosomal arrangements (%) in selection lines.

	R		P2		N2	C1	C2
	n=157	n=128	3 n=89		n=145		n=149
Ast	45.65	1.3	-	-	98.75	52.1	-
<sup>A</sup> 1	7.52	-	-	-	-	-	-
$A_2$	47.83	98.7	100.0	100.0	1.25	47.9	100.0
J st	24.34	0.8	-	-	33.79	9.5	-
$J_1$	75.66	99.2	100.0	100.0	66.21	90.5	100.0
U <sub>st</sub>	0.66	-	-	-	-	-	_
U <sub>1+2</sub>	47.37	100.0	100.0	-	72.73	60.0	48.30
U <sub>1+2+8</sub>	51.97	-	-	100.0	27.27	40.0	51.70
Est	46.05	16.4	51.69	-	43.75	46.0	23.81
E <sub>1+2</sub>	11.19	-	1.12	73.3	24.31	2.4	12.93
E <sub>1+2+9</sub>	9.21	-	-	-	-	-	-
E <sub>1+2+9+12</sub>	27.63	83.6	47.19	26.7	31.94	51.6	63.27
E <sub>1+2+9+3</sub>	3.95	-		-	-	-	-
E <sub>8</sub>	1.97	-	_	-	_	-	-
0 <sub>st</sub>	21.79	-	56.82	-	-	58.1	32.21
03+4	24.36	-	43.18	100.0	100.0	17.7	67.11
03+4+7	25.64	100.0	_	-	-	24.2	
03+4+8	19.87	-		-	-	-	-
03+4+2	4.49	-	-	-	-	-	0.67
03+4+22	0.64	-	-	-		-	-
07	3.21	-	-	-	-	-	-
°7	3.21			<u>-</u>		<del>-</del>	

A<sub>st</sub> is fixed in N<sub>2</sub>. In chromosome J, frequency of the J, arrangement reaches values close to 100% in all selection lines, except in  $\mathbf{N}_2$  where  $\mathbf{J}_1$  arrangement shows samller  $^2\mathrm{values}$ than in the initial population.

As in A and J chromosomes, in U chromosome the same arrangement  $(U_{1+2})$  is fixed in the two high selection lines and N<sub>2</sub> shows a different behaviour to N<sub>1</sub>. In control lines the two most frequent arrangements ( $U_{1+2}$  and  $U_{1+2+8}$ ) reach values close to 50%.

In chromosome E, the most polymorphic, in no case was homozygosity reached. A different arrangement tends to be increased in each line.

In chromosome 0 which shows a great number of gene arrangements, homozygosity was reached in several selection lines. The two low selection lines are homozygotic for the  $0_{3+4}$  arrangement and  $P_1$  for  $0_{3+4+7}$  arrangement. The rest of the lines remained polymorphic.

On comparing the chromosomal frequencies in the initial population with the frequencies in the selection lines and control lines after 24 generations of artifical selection, it can be seen that the two high selection lines and one low selection line (N<sub>1</sub>) tend to reach homozygosity while the control lines and No low line tend to remain polymorphíc.

In some chromosomes the same arrangement was fixed in the two high selection lines or in the two low selection lines, as happens with the  $A_2$  arrangement in the two high selection lines and with the  $0_{3+4}$  arrangement in the two low selection lines. These two arrangements tend to be increased in the laboratory population. Also the  $J_1$  and  $U_{1+2}$  arrangements were fixed in high selection lines. In the rest of the chromosomes the behaviour of high and low selection lines is similar.

Martinez-Sebastian, M.J. & J.L. Mensua. University of Valencia, Spain. Variations of wing dimensions in Drosophila subobscura populations selected for

In a laboratory population (R) od D. subobscura, the characters of wing length and wing width were measued.

Two replicate selection lines for abdominal abdominal bristle number. bristle number in both, high ( $P_1$  and  $P_2$ ) and low ( $N_1$  and  $N_2$ ), directions and two control lines ( $C_1$  and  $C_2$ ) were established from the laboratory population. At 17th generation of selection, wing length and wing width were

measured.

Table shows the means of wing dimensions in the laboratory ppulation, control lines and selection lines.

Significant differences exist between the laboratory population and the selection and control lines (except wing length males R versus males  $C_1$ ). Also there are significant

Table 1. Wing dimensions in laboratory population and in lines selected for abdominal bristles number (in micrometer units, one unit=0.03 mm).

MALES				FEMALES			
	ving length	wing width		wing length	wing width		
	67.95±0.17 65.24±0.17	31.11±0.10 30.17±0.07	R P <sub>1</sub>	74.42±0.17 72.75±0.20	34.01±0.08 33.18±0.09		
$\mathbf{P}_{2}$	71.36±0.14	32.42±0.08	$P_2$	78.73±0.16	35.84±0.07		
N <sub>1</sub>	64.92±0.14	29.84±0.07	$^{\mathrm{N}}_{1}$	71.60±0.13	32.73±0.07		
$N_2$	64.45±0.17	30.65±0.08	N <sub>2</sub>	70.68±0.20	33.71±0.08		
$c_1$	67.45±0.22	30.70±0.11	$^{\rm c}_{\rm 1}$	73.55±0.26	33.40±0.13		
$c_2$	68.88±0.13	31.90±0.07	c <sub>2</sub>	76.51±0.18	35.40±0.07		

differences between the two replications of each selection and control line.

On the other hand, it could be pointed out that in all cases the wing width and wing length means of low selection lines are smaller than the means of high and control lines. In replication 1, the mean of the control line is bigger than the mean of the high selection line.

The correlation between wing length or wing width and number of abdominal bristles of the 4th and 5th sternites in the laboratory population, control lines and selection lines were estimated.

No significant correlation exists between wing dimensions

and number of abdominal bristles in the laboratory population (). In the control lines, one of them  $(C_1)$  shows a significant correlation between wing dimensions and number of abdominal bristles, but no correlation exists in the other control line  $(C_2)$ .

High and low selection lines show different behaviour. In the low selection lines no significant correlation exists in any case, while in high selection lines a significant correlation exists between wing dimensions and nr. of abdominal bristles in males from P<sub>1</sub> line, and also between wing width and number of abdominal bristles in females from P<sub>1</sub> and P<sub>2</sub> lines. In spite of the lack of correlation between wing dimensions and number of abdominal

In spite of the lack of correlation between wing dimensions and number of abdominal bristles, in the majority of lines, there are differences in wing dimensions between the laboratory population and the selection lines. There are differences particularly between the high and low selection lines. Body size (as estimated by wing dimensions) is slightly modified by selection for abdominal bristle number. This could be due to the increase in the number of abdominal bristles in the high lines was accompanied by an increase in the area of the sternital and the reverse apparently occurs in the low lines.

Mason, J.M. National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. Nitrogen mustard induced translocations in mutagen-sensitive mutants.

Graf et al. (1979) tested the effects of a number of DNA repair-defective mutants on mutation frequencies induced by radiation or chemical mutagens. Their most striking observation was that when sperm treated with 0.2% nitrogen mustard (HN2) fertilize eggs from musl01 homozygous females, no chemically induced recessive

lethal mutations were found. In contrast, when such sperm fertilize eggs from the control or from mei-45<sup>D5</sup>, musl04<sup>D1</sup> or mei-9<sup>L1</sup> females the recessive lethal frequency is on the order of 4-6%. Similar results are reported here using reciprocal translocations as an endpoint (Table 1). In these experiments SM1, Cy; TM2, Ubx <sup>1</sup> e /T(2;3)ap males were fed 0.2% HN2 for 24 hours and mated with females homozygous for either mei-41<sup>D5</sup>, musl01<sup>D1</sup>, musl04<sup>D1</sup> or w so that lesions induced in mature sperm could be repaired in eggs produced by these females. Cy Ubx sons were crossed with e<sup>11</sup> females and the resulting F2 progeny screened for the segregation of Cy and e<sup>S</sup>. Females bearing musl04<sup>D1</sup> or mei-41<sup>D5</sup> produced about 0.5% translocations, similar to the control. However, musl01<sup>D1</sup> females produced no translocations in 1291 progeny. Thus musl01 has the same effect on the recovery of HN2 induced translocations as recessive lethals.

Wurgler and Graf (1980) suggested that, because DNA-DNA crosslinks are induced by HN2 and are normally resolved with a loss of information, recessive lethals are a by-product of normal repair of these lesions. They further proposed that musl01 mutants are defective in the ability to resolve DNA crosslinks and that such unresolved lesions cannot replicate and are dominantly lethal. Similar arguments can be made for the absence of HN2 induced translocations from musl01 females.