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	
	wing length	wing width		wing length	wing width
R	67.95±0.17	31.11±0.10	R	74.42±0.17	34.01±0.08
P_1	65.24±0.17	30.17±0.07	P_1	72.75±0.20	33.18±0.09
P_2	71.36±0.14	32.42±0.08	P_2	78.73±0.16	35.84±0.07
N_1	64.92±0.14	29.84±0.07	N_1	71.60±0.13	32.73±0.07
N_2	64.45±0.17	30.65±0.08	N_2	70.68±0.20	33.71±0.08
C_1	67.45±0.22	30.70±0.11	C_1	73.55±0.26	33.40±0.13
C_2	68.88±0.13	31.90±0.07	C_2	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_1 line, and also between wing width and number of abdominal bristles in females from P_1 and P_2 lines.

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 mus101 homozygous females, no chemically induced recessive lethal mutations were found. In contrast, when such sperm fertilize eggs from the control or from mei-45^{D5}, mus104^{DI} or mei-9^{LI} 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^{130} e^s/T(2;3)ap^{Xa} males were fed 0.2% HN2 for 24 hours and mated with females homozygous for either mei-41^{D5}, mus101^{D1}, mus104^{D1} 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^{11} females and the resulting F2 progeny screened for the segregation of Cy and e^s. Females bearing mus104^{D1} or mei-41^{D5} produced about 0.5% translocations, similar to the control. However, mus101^{DI} females produced no translocations in 1291 progeny. Thus mus101 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 mus101 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 mus101 females.

Table 1. Reciprocal translocations from mutagen-sensitive mutants.

Maternal genotype	Dose	Genotypes of progeny recovered +	T(Y;2)	T(Y;3)	T(2;3)	% trans
w	-	1366	0	0	0	-
$mus101^{D1}$	-	1314	0	0	0	-
$mus104^{D1}$	-	1458	0	0	0	-
$mei\text{-}41^{D5}$	-	1259	0	0	2	0.16
w	0.2%	1106	0	1	7	0.72
$mus101^{D1}$	0.2%	1291	0	0	0	-
$mus104^{D1}$	0.2%	1553	1	1	5	0.45
$mei\text{-}41^{D5}$	0.2%	707	1	1	3	0.71

The untreated controls produced a surprising result; two independent translocations were recovered from $mei\text{-}41^{D5}$ females, while no spontaneous translocations were recovered from the other crosses. A recent large scale study suggests that the spontaneous translocation frequency is about 10^{-5} (Mason et al. in prep.). In previous studies $mei\text{-}41^{D5}$ females did not show an increase in the spontaneous recessive lethal frequency (Graf et al. 1979). Smith (1973) reported an increase in the spontaneous recessive lethal frequency from $emi\text{-}41^{A1}$ males, although Mason (1980) could not find an increase from $mei\text{-}41^{D3}$ or $mei\text{-}41^{D5}$ males. It is not entirely clear why $mei\text{-}41^{D5}$ should increase the frequency of spontaneous translocations but not recessive lethals, although the extremely low spontaneous translocation frequency in the control makes any induced translocations much more noticeable.

References: Graf, Green & Wurgler 1979, Mutation Res. 63:101-112; Mason 1980, Mutation Res. 72:323-326; Smith 1973, Mutation Res. 20:215-220; Wurgler & Graf 1980 in "DNA Repair and Mutagenesis in Eukaryotes" pp. 223-240.

Mather, W.G. & A.K. Pope. University of Queensland, Brisbane, Australia. Inversions from Chiang Mai, Thailand.

In July 1982 thirty-six isolines of D.s. albostrigata and six isolines of D. albomicans were established from Chiang Mai, Thailand.

Inversions in these species were last reported on from a collection made at Phuket in February 1982 (Mather & Pope DIS 59:-).

(a) D.s. albostrigata: Seven simple inversions were detected. All had previously been recorded from East and South East Asia (Table 1).

(b) D. albomicans: Four simple and one complex inversion were detected. All had been previously recorded elsewhere (Table 2).

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by A.K.P.

Table 1. D.s.albostrigata, Chiang Mai.

Inversion	Chromosome	Het. Freq. %
A_5	IIL	27.7
C_1	III	2.7
I_2	IIL	22.2
B_5	III	13.8
N_5	III	2.7
C_5	IIR	2.7
P_5	III	2.7

Table 2. D. albomincans, Chiang Mai.

Inversion	Chromosome	Simple	Complex
S_5	IIL	X	
C_1	III	X	
I_2	IIL	X	
E_6	III		X
L_3	III	X	