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Effective population number estimates of laboratory populations of *Drosophila melanogaster*

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Summary. A sizeable fraction of the current information in population genetics comes from experiments with population cages of *Drosophila melanogaster* in which a high census (circa 5000 adults) is kept in order to obviate the influence of drift. We have estimated the effective population number in such cages by lethal complementation and our estimates are lower than the census by an order of magnitude.

The estimation of the effective population number *(Ne)* by lethal complementation has a well established theoretical basis[2–4], has been tested experimentally[5], and is of common use in population genetics[5–7]. The expression for the estimate is

$$\hat{N}e = \frac{1 - Ig}{4(Ig\ U - u)} \qquad \text{(Nei}^2\text{)},$$

where U and u are the per chromosome and per locus mutation rates and Ig is the allelic rate of lethal genes, which can be estimated by

$$\hat{Ig} = -\log(1 - Ic\ Q^2)/\{\log(1 - Q)\}^2$$

where Q is the observed frequency of lethal chromosomes in the population and Ic is the proportion of inviable, random heterozygous combinations of such chromosomes.

The estimation was performed in populations kept for different times under cage conditions that maintain a steady census of about 5000 adults[8] (table). The populations were founded with wild caught individuals. Foundation stocks were over 500 inseminated females. Kaduna is known to be free of inversions, as seems to happen with populations kept under laboratory conditions[19]. Riudevella, Amherst and Stellenbosch samples were checked for inversions at the 3rd chromosome, the frequency of inversion-carrying 3rd chromosomes was below 6% in all of them. Values of Ic and Q were obtained for the 3rd chromosome and the usual values of $U = 0.005$ and $u = 10^{-5}$ calculated for the 2nd chromosome were used[9–11], as the available data indicate that the lethal structure for the 2nd and 3rd chromosomes is very similar[12–15] (J.M. Vassallo and J.M.M., unpublished results). The effective population numbers for the investigated populations were in the range 190–1235 (table).

Alternative values for u proposed in the literature[6] would yield lower estimates for the effective population number. The estimation method presupposes that the elimination of lethals occurs mainly as heterozygous combinations. The above population numbers are close to the limit with respect to such a condition[4,16], but if the observed frequencies of lethal chromosomes in our populations were to be explained in terms of elimination of lethals as homozygous combinations, the population numbers would be even lower (range 50–380, estimated as in Wright[17]).

Possible mechanisms for this difference between census and effective population size can be found[18]. Although estimations of the effective population number in line with those reported here have occasionally been used[5,15], the interpretation of a considerable number of experiments in which the number of adults has been used as an estimate of the effective population number should be revised, especially those in which this parameter is used a) to reject drift as an explanation for changes in gene frequencies, b) to predict variability, or c) to calculate expectations of associations among loci.

Estimates of effective population numbers

Populations	Riudevella	Amherst	Stellenbosch	Canberra	Pacific	Kaduna
Months under cage conditions	8	24	36	156	204	276
$Q^a \pm$ SE	0.13 ± 0.04	0.21 ± 0.04	0.24 ± 0.04	0.10 ± 0.03	0.09 ± 0.03	0.13 ± 0.03
$I_c{}^b \pm$ SE	0.05 ± 0.03	0.15 ± 0.04	0.14 ± 0.06	0.13 ± 0.04	0.04 ± 0.03	0.24 ± 0.09
$\hat{N}_e{}^c$	1,123	383	427	395	1,252	190

[a] Q, frequency of lethal 3rd chromosomes. A TM3, Sb, Ser[20] balancer chromosome was used. Males direct drawn from the cage were individually mated to TM3, Sb, Sr/Pr females, only one line from each of the successful matings was subsequently used.
[b] I_c, proportion of inviable, random heterozygous combinations of lethal chromosomes.
[c] $\hat{N}_e{}^c$ estimate of effective population number, for estimation method see text.

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Dominant lethal mutations induced by ^{14}C in mice[1]

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Summary. The mutagenic potential of 1.0 μCi ^{14}C was evaluated in Swiss albino male mice by the dominant lethal assay. A significant increase in post-implantation loss was seen, the maximum being in the 3rd week after treatment.

^{14}C is a naturally occurring radionuclide that is produced in a nuclear reaction between cosmic ray neutrons and the nitrogen atoms of the air. It is also a by-product of nuclear fission. ^{14}C is widely used as a tracer in different biochemical studies and in the diagnosis of some human disorders[2-4]. Earlier investigations on the genetic effects of ^{14}C were confined to onion[5,6], *E. coli*[7], fish eggs[8], Chinese hamster cells[9] and *Drosophila*[10-13]. Its mutagenic potential has not been studied in mice. Hence the present investigation was taken up.

Materials and methods. 25 male mice of the Swiss albino strain weighing 20-25 g (8 weeks old) were injected i.p. with 1.0 μCi/0.5 ml of of ^{14}C in the form of glucose-C-14 (sp. act. 215 mCi/mM supplied by Isotope Group, Bhabha Atomic Research Centre, Bombay). The control mice received 0.5 ml of physiological saline (0.9%). Immediately after treatment each male was caged with 2 virgin females (8 weeks old) which were replaced at weekly intervals for 6 consecutive weeks. Females were autopsied on 17th day of gestation and the uterus was checked for live and dead implantations. Dominant lethals were estimated in terms of pre-, post- and total-implantation losses[14].

Results. The results on the data on uterine contents are presented in the table. Their significance was tested using the chi-square and t-tests. There was a significant ($p < 0.05$) increase in the dead implantations per female in the ^{14}C-treated group during the 1st, 3rd and 6th weeks of mating. However, there was no significant ($p > 0.05$) change in the live and total embryos when compared to controls. The difference in pregnant females also remained statistically insignificant ($p > 0.05$) between control and treated groups.

Discussion and conclusions. The test for dominant lethal mutations is one of the few methods available for evaluating the in vivo mutagenic potential of chemicals and other environmental pollutants in the germ cells of mouse and rat. The genetic basis of dominant lethality can be due to structural and numerical chromosomal aberrations and point mutations. It is evident from the results, that 1.0 μCi of ^{14}C induces damage in post-meiotic and in pre-meiotic stages of spermatogenesis, causing a significant increase in post-implantation loss.

Chromosomal aberrations produced by C-14 were first reported by McQuade et al.[5] in onion root tips. In their subsequent study they found that ^{14}C is more effective than

Results on the uterine contents in treated and control mice

	Mating week	Pregnant females	Total implants	Dead implants	Live implants	Pre-implantation loss Total implants / Female	Post-implantation loss Dead implants / Female	%	Total loss Live implants / Female
^{14}C-treated (1.0 μCi)	1	39	343	37	306	8.79	0.95*	10.79*	7.85
	2	37	355	20	335	9.59	0.54	5.63	9.05
	3	41	353	58	295	8.60	1.41*	19.18*	7.20
	4	42	381	30	351	9.07	0.71	7.87	8.35
	5	39	316	25	291	8.10	0.64	7.91	7.46
	6	35	310	29	281	8.86	0.83*	9.35*	8.02
Control (saline)	1	40	336	16	320	8.40	0.40	4.76	8.00
	2	38	342	18	324	9.00	0.47	5.26	8.53
	3	40	347	21	326	8.65	0.53	6.05	8.15
	4	37	311	16	295	8.40	0.43	5.14	7.97
	5	35	339	15	324	9.69	0.43	4.42	9.26
	6	39	306	14	292	7.85	0.36	4.58	7.49

* Significant in comparison to controls at 0.05 level.