Table 2. Number of baited and unbaited traps containing *Drosophila*, hymenopteran parasitoids and spiders at each of six sites near Christchurch, New Zealand (N = 30).

Site	Date	*Drosophila*		Parasitoids		Spiders	
		baited	unbaited	baited	unbaited	baited	unbaited
Organic orchard	30/11/98	29	0*	10	0*	5	3
Sub-urban garden	22/12/98	19	0*	11	0*	4	0
Restoration site	29/3/99	25	0*	10	2*	2	3
Pine plantation	21/4/99	10	0*	7	10	2	2
Remnant forest	20/5/99	19	0*	0	1	5	1
Farm out-buildings	1/6/99	20	1*	1	0	1	2
Total (N = 180)		122	1*	39	13	19	11

* - significant preference for baited traps identified using χ^2 test; $P<0.05$

Our casual observation that spiders were frequent visitors to *Drosophila* traps did not withstand systematic examination. We found fewer spiders than expected, and those that were collected showed no significant preference towards baited traps. However, as almost two thirds of spiders collected were caught in the baited traps, this issue may warrant further investigation. Separating spiders into different families or species may reveal more detailed patterns in preference of foraging site. Also, the use of olfactometer experiments in the laboratory may clarify whether certain species of spider are attracted to *Drosophila* resources.

Acknowledgments: Thanks to Jo Berry of Landcare Research, Auckland, for identifying the Hymenoptera.

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Factors of spontaneous mutations, mutability in large chromosomes and mortality from dominant lethals in *Drosophila melanogaster*.

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The ideas are widespread that changes of the natural radiation background increase the rate of mutability and are responsible for such prominent events in life on the Earth as extinction of fossil faunas (dinosaurs are mentioned most frequently), outbreaks of pest reproduction, epizooites and other phenomena believed to be associated with emergence of mutations causing resistance to pesticides and of more virulent or antibiotic-resistant microbial strains. However, 1) mutations are destructive and have nothing to do with biogenesis (Ivanov and Ivannikov, 1997), that is why the above ideas about the emergence of new forms with a heightened fitness are extremely doubtful. It is not new mutant forms that arise, it is that tolerable hereditary types preadapted to new conditions which are inherent in the given species from its very origin, but rare under the usual conditions, begin to reproduce more intensely. The species becomes adapted to various possible

conditions of its ecological niche not by means of emergence of outstanding mutant individuals with universal adaptation, but due to the enormous diversity of its tolerable hereditary types each of which is adapted in the best way to a very limited assortment of environmental conditions. 2) Extinction of faunas in the history of the Earth finds its explanation in the fact that CO_2 reserve in the atmosphere, which is replenished by mantle degasation during paroxysms of volcanic activity (geological revolutions) (Ronov, 1978; Ronov, 1980), is periodically exhausted, and this extinction may not be attributed to catastrophical radiation outbreaks, because these are extremely doubtful. In our recent work (Ivanov, 1998a) based on data known since long ago (Stern, 1960), it was demonstrated that the natural radiation background practically did not influence the spontaneous mutability, since it brings about less than 10^{-4} of all the spontaneous mutations, that is why even its repeated changes could not produce the observed changes of mutability in the populations. This and other considerations make us conclude that the mutability in nature is determined by biotic factors.

Table 1. Decomposition of the spontaneous mutation process into the main mutation types in *Drosophila melanogaster*.

Mutation type	Percent among all mutations	Percent among all DLM	Lethality of mutations	Percent among all mutations	Lethal type	Percent among all mutations	Percent among all genic mutations	Type of genic lethals	Percent among all genic lethals
CDLM	64	94			Chromosomal	64	-	-	-
GDLM	4	6	Lethals	97	Genic	33	92	GDLM	12
RLM	29	-						RLM	88
VM	3		Non-lethals	3	Non-lethals	3	8	-	-

CDLM and GDLM and sets of gametes carrying chromosomal and genic DLM, respectively. RLM and VM are sets of respective gametes

A very important result of studies of spontaneous mutagenesis in *D. melanogaster* is its division into basic mutation types (Ivanov, 1998b). It permits associating the mutation rates in separate chromosomes with the mortality of zygotes from dominant lethal mutations (DLM) in the whole genome. Therein it is assumed that constant proportions between the mutability in single chromosomes and that in the whole genome, and between rates of various mutation types, are maintained.

Under usual conditions, the fraction of mutant gametes among all gametes of *D. melanogaster* is about 3.25%. Decomposition of the spontaneous mutation process into basic mutation types in *D. melanogaster* genome is division of the set of mutant gametes into subsets that carry DLM (68%), recessive lethals (RLM) (29%), and visible mutations (VM) (3%). Therein, if VM and RLM coincide with DLM, they are recorded as DLM, and if VM coincide with RLM, they are recorded as RLM due to lethality of respective genotypes. In their turn, DLM are subdivided into chromosomal and genic ones, wherein if the genic DLM coincide with chromosomal ones, they are recorded as chromosomal (Ivanov, 1998c). All possible divisions of the set of mutant gametes are presented in Table 1. Having the decomposition of the mutation process into mutation types, the rate u_i of RLM and VM emergence in the i-th chromosome and its fraction s_i in the whole *D. melanogaster* genome, one can estimate the frequency U of DLM emergence in the whole genome, and through it the zygotes' lethality from DLM as a function $S(U)$ or $S(u_i)$.

Let, as already indicated, U be the frequency of DLM emergence in the whole genome (autosomes + X chromosome). The fraction of X chromosome in the whole (complete) genome is $s_1 = 0.19$ (Ivanov, 1998b). That is why the incomplete genome in which the X chromosome is genetically substituted by the empty Y chromosome makes up 0.81 of the complete one, and the frequency of DLM emergence in it is $0.81U$.

The probability of a female not dying from DLM is the probability of her two complete genomes turning out to be free from DLM, *i.e.* to be equal to $(1-U)^2$. The probability of her death from DLM is

$$S_f(U) = 1 - (1-U)^2 = 2U - U^2. \qquad (1)$$

The probability of a male not dying from DLM is the probability of his two genomes, the whole (complete) and the incomplete ones, turning out to be free from DLM, *i.e.* to be equal to the product $(1-U)(1-0.81U)$. The probability of his death from DLM is

$$S_m(U) = 1 - (1-U)(1-0.81U) = 1.81U - 0.81U^2. \qquad (2)$$

At the primary sex ratio of 1:1, the zygotes' death from DLM is the arithmetic mean of these quantities (1) and (2) $S(U) = 0.5[S_f(U) + S_m(U)]$, *i.e.*

$$S(U) = 1.905U - 0.905U^2. \qquad (3)$$

Now, to calculate the sought mortality, one has to estimate the value for U. Let u and u_i be the frequencies of RLM and VM in the whole genome and in the X chromosome, respectively, and s_i be the fraction of the *i*-th chromosome in the whole genome. From the proportion $u/u_i = 1/s_i$ we have: $u = u_i/s_i$, and from the proportion $U/u = 68/32$ which follows from the decomposition we have

$$U = 2.125u = 2.125u_i/s_i.$$

By means of substituting the found expression for U into equalities (1) - (3), we obtain expressions for the mortality of sexes and zygotes in general through the mutability u_i in the *i*-th chromosome:

$$\begin{aligned} S_f(u_i) &= 4.250u_i/s_i - 4.516u_i^2/s_i^2; \\ S_m(u_i) &= 3.846u_i/s_i - 3.658u_i^2/s_i^2; \qquad (4) \\ S(u_i) &= 4.048u_i/s_i - 4.087u_i^2/s_i^2. \end{aligned}$$

In particular, the expressions for zygotes' mortality from DLM through the mutability u_1 in the X chromosome and u_2 in chromosome 2 at $s_1 = 0.19$ and $s_2 = 0.36$, becomes as follows:

$$S(u_1) = 21.3u_1 - 113.2u_1^2; \qquad (5)$$

$$S(u_2) = 11.2u_2 - 31.5u_2^2. \qquad (6)$$

These formulae, unlike the approximated formulae of our previous works (Ivanov and Ivannikov,1997; Ivanov 1998a) are more precise.

From the maximal rates of spontaneous mutability known to us in large chromosomes $\hat{u}_1$ = 1.3% (Ivanov, 1998a) and $\hat{u}_2$ = 1.27% (Dubinin, 1966), by means substituting them into formulae (5) and (6), it is possible to estimate the maximal values for zygotes' mortality from DLM in populations: $S(\hat{u}_1)$ = 25.8% and $S(\hat{u}_2)$ = 13.7%. These values are rather large, from which it follows that the most important consequence of mutability increase in the population is a considerable heightening of zygotes' mortality. As it has been found (Ivanov and Ivannikov, 1997), mutability depends on the population density as an increasing function; that is why, as the species abundance increases, so does the zygotes' mortality from DLM, due to which the mutation process is one of regulators of the population numbers. This is confirmed by the fact that the basic factors of spontaneous mutagenesis have a biotic nature (paramutations, insertional mutagenesis, viruses, transduction, mutator genes, MR-factors, etc.).

Acknowledgments: The author is sincerely grateful to Dr. V.G. Kolpakov for his help in translation of this paper into English and Drs. D.A. Afonnikov and T.M. Mischenko for preparation of the manuscript for publication.

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