homomorphic (subtelocentric). This seems to favour the hypothesis that the $2n = 38$ 'populations are interconnected by the same system that is responsible for the ubiquitous diffusion of the species, which is that associated with the black rat's condition as man's commensal' (CAPANNA et al.[7]). Moreover, the probability that 'centric fusion has occurred between the same pairs of autosomes independently' (CAPANNA et al.[7]) in 3 widely separated $2n = 38$ populations is rather remote.

Another interesting point is that the longest autosome in the Asian ($2n = 42$) populations of *Rattus rattus* seems to possess a readiness for aberration. Heteromorphism has been reported for the Japan and Korean taxa (YOSIDA et al.[9,10]), Malayan taxon (YONG and DHALIWAL[4]) and also the Vietnamese taxon (T. C. HSU, personal communication). In the present report, the longest autosome is involved in centric fusion. On the other hand, the longest autosome in the $2n = 38$ populations is homomorphic.

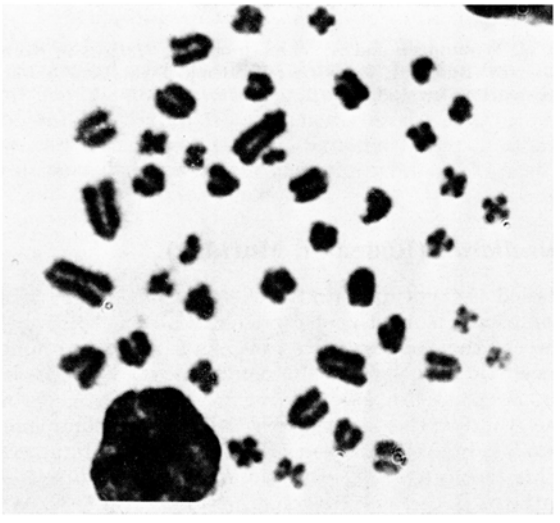


Fig. 2. Normal metaphase of *Rattus rattus diardii* with 42 chromosomes.

It is noteworthy that these 2 major populations exhibit different chromosomal polymorphism. This is probably not a result of environmental conditions as chromosomal polymorphism has been found in the Malayan house shrew, *Suncus murinus*, in which Robertsonian-type translocation is responsible for the existence of $2n = 38$, 39, and 40 individuals within the same population (YONG, Experientia, in press).

As stated by CAPANNA et al.[7] 'the study of the polymorphism of *Rattus rattus* appears highly promising'. It would be exciting if there should exist other populations in addition to the existing $2n = 42$ (Asian) and $2n = 38$ (European, S. American, Oceanian) populations.

Zusammenfassung. Bei einem Männchen von *Rattus rattus diardii* aus Kuala Lumpur (Malaysia) wurden 41 Chromosomen (normal $2n = 42$) gefunden, was mit der Robertson'schen Translokation in Zusammenhang stehen dürfte, welche die Fusion 2 einarmiger Autosomen in ein grosses, zweiarmiges Chromosom vollzieht.

H. S. YONG

Division of Genetics, School of Biological Sciences, University of Malaya, Kuala Lumpur (Malaysia), 23 September 1970.

[9] T. H. YOSIDA, A. NAKAMURA and T. FUKAYA, Chromosoma *16*, 70 (1965).

[10] T. H. YOSIDA, Y. MORIGUCHI, Y. S. KANG and K. SHIMAKURA, A. Rep. natn. Inst. Genet., Japan *17*, 61 (1967).

Studies on rII Region of T2L Phage

GANDHI, MEHTA and MODI[1] reported that rII region of phage T2L consists of one cistron and that in crosses between the mutants studied the maximal frequency of recombinants does not exceed 0.13%. In the course of the comparative genetic research of r-mutants of phages T2L and T4B, we obtained results contrary to that of GANDHI et al.[1]. It is evident from our results that rII region of phage T2L consists of two cistrons, the length of which corresponds to that of rII region of phage T4.

Materials and methods. Bacteria. Strain of *Escherichia coli* B was used as a host for the titrations of phages and for plating the progeny of crosses between rII mutants; *E. coli* BB was used to obtain rII mutants in high titer as on B cells the titres of r mutants are not as rule high. *E. coli* K12 (λ) and *E. coli* B (λ) were used as a plating indicator for assaying the revertants and recombinants and also in the experiments on complementation. Strain B (λ) was kindly supplied by Dr. ARBER.

Bacteriophages. Phage T2L of the wild type was obtained from laboratory of Dr. GOLDFARB (Institute of General Genetics, Moscow). Deletion mutants of rII region of phage T4B rII638, rII168, rIIW8-33 were obtained from Dr. BENZER. Mutant rII638 carries the deletion of the whole rIIB cistron, rII168 and rIIW8-33 contain the small deletions in rIIA and rIIB cistrons of phage T4B, respectively.

The phage T2L r^+ was treated with 0.2 M solution of HCl-hydroxylamine in the $^1/_{15}M$ Na-phosphate buffer pH = 6.0 at 4–10 h. Mutagenic reaction was stopped by dilution of sample in 2% solution of acetone in broth. Mutagenized phage was plated on Petri dishes and the selection of r-mutants from the pure and mottled plaques was carried out. The sample was not eliminated from mutational heterozygotes and thus the independent occurrence of every selected mutant was provided[2]. The media used in our experiments is tryptic digest of meat ('broth'), and broth with agar (1.2% agar-bottom layer, 0.7% agar-top layer).

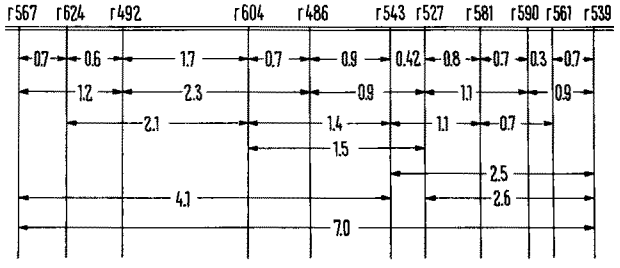
Complementation methods. a) Qualitative complementation. The drops of suspension of the investigated mutants, taken with titre of 1×10^7 phages/ml were placed on the newly plated lawn of B (λ) bacteria (approximately 1×10^8 cells) in top layer with tester phage (multiplicity of infection is 0.1). After incubation for 18–20 h the occurrence of spot of lysis was estimated as a complementation. b) Quantitative complementation. Bacteria

[1] N. R. GANDHI, R. J. MEHTA and V. V. MODI, Experientia *24*, 279 (1968).

[2] E. H. SIMON and I. TESSMAN, Proc. natn. Acad. Sci., USA *50*, 526 (1963).

of *E. coli* B (λ), 2×10^8 cells, were infected with the mixture of phages under investigation with the multiplicity of infection with each phage equal to 7.0 and after 10 min adsorption the infected bacteria were sedimented by centrifuging, resuspended in a fresh media and incubated for 1 h at 37 °C. The yields of each mutant were analyzed as controls. Crosses were carried out according to the method described in work of STEINBERG and EDGAR[3]. The general methods of work with phages corresponded to those described by ADAMS[4].

Results and discussion. On strain of *E. coli* K12 (λ), phage T2L of the wild type produces small turbid plaques with decreased efficiency of plating. In order to avoid inadequate estimation of qualitative complementation (as a result of poor development of phage) a strain of *E. coli* B (λ) was used in this experiment. On cells of B (λ) wild phage T2L forms specific r^+ plaques with the same efficiency of plating as that of on *E. coli* B, and rII mutants of phage T2L do not grow on this lawn. For the complementation we used rII mutants of T2L, previously localized on the genetic map (see Figure) and deletion mutants of phage T4B. The crossing of end markers showed that the maximum length of rII region of T2L is equal at least to 7.0 map units.



The genetic map of rII region of bacteriophage T2L (map units).

Table I. Qualitative complementation between the mutants of T2L and rII638

	Mutants										
	567	624	492	604	486	543	527	581	590	561	539
567	0	0	0	0	0	0	+	+	+	+	+
624		0	0	0	0	0	+	+	+	+	+
492			0	0	0	0	+	+	+	+	+
604				0	0	0	+	+	+	+	+
486					0	0	+	+	+	+	+
543						0	+	+	+	+	+
527							0	0	0	0	0
581								0	0	0	0
590									0	0	0
561										0	0
539											0
rII638	+	+	+	+	+	+	0	0	0	0	0

Table II. The results of quantitative complementation with rII T2L mutants

Phage or mixture of phages under experiment			Yield of phage per 1 infected cell
rW8-33		T4B-cistron	0
r168		T4A-cistron	0
r442		T2L	0
r527		T2L	0
r543		T2L	0
r168	+	rW8-33	38.4
r442	+	r527	29.1
r442	+	r543	0
r527	+	r543	30.0
rW8-33	+	r543	27.8
rW8-33	+	r527	0
r168	+	r543	0
r168	+	r527	24.3
r168	+	r442	0
rW8-33	+	r442	29.3

Studies of 11 mutants taken in pairs and mapped (Table I) and, besides the experiments with 23 mutants not as yet mapped made it possible to classify them into 2 groups of complementation. Among 34 rII T2L mutants under experiment, 18 mutants belong to A-cistron, i.e. those complemented with rII638, all the rest belong to B-cistron. On the genetic map, the boundary line between the mutants of these 2 groups of complementation is situated between r543 and r527.

Further evidence in favour of detection of these 2 groups is found in the results of the quantitative complementation with the mutants adjacent to the boundary line between the cistrons presupposed. The results of the experiment are given in Table II. Thus, mutants r527 and r543 belong to different groups (cistrons) and the functions of rII cistrons of phage T2L may be compensated by the functions of T4 phage rII cistrons. The minimal distance between the mutants, belonging to different cistrons, makes 0.42 map units for T2L phage, which is practically equal to the corresponding distance for rII region of phage T4B[5].

The genetic differences between the closely related species may be the consequence of various numbers of genes, different fine structure of genes or different localization of homologous genes in the chromosomes of closely related species. According to our data, rII region of T2L does not differ in size from that of phage T4B and consists of 2 cistrons that are functionally homologous to rIIA and rIIB cistrons of phage T4B.

The results of GANDHI et al.[1] may be explained by the insufficient volume of the material under experiment, by the fact that cells of K12 (λ) were used as indicator. Finally there is a possibility that some of these mutants were somehow repeated, as these authors only studied the mutants induced in vivo with acridine, so that it looks quite possible that a clone of mutants was selected. This last assumption may explain the result of GANDHI, as the frequencies of recombination in crosses of 4 mutants are low and do not differ greatly from the frequencies of revertants.

Выводы. rII область фага T2L состоит из двух цистронов, функционально гомологичных цистронам A и B rII области фага T4B. Общая длина rII области фага T2L соответствует длине rII области фага T4B. Расстояние между ближайшими мутациями в соседних цистронах фага T2L соответствует максимальному межгенному расстоянию в rII области фага T4B.

S. I. ALIKHANIAN, V. N. KRYLOV and I. ZHAZYKOV

Institute of Genetics of Microorganisms, Box 379, Moskwa D-182 (USSR), 23 July 1970.

[3] C. M. STEINBERG and R. H. EDGAR, Genetics *47*, 187 (1962).
[4] M. ADAMS, *Bacteriophages* (Intern. Publishers, New York, London 1959).
[5] L. H. HARTWELL, Virology *15*, 510 (1961).