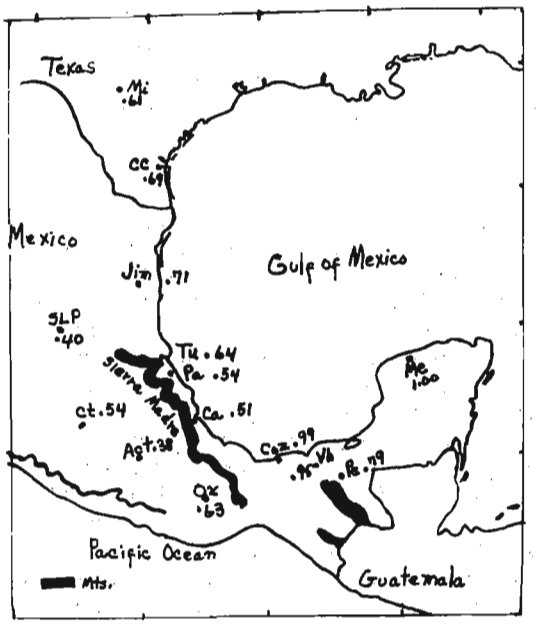
Pipkin, S.B. and C. Rhodes. Howard University, Washington, D.C. Frequency of the D. melanogaster allele, Adh^{II}, in natural populations of Mexico.

Drosophila collections were made at 13 stations in Mexico and two in South Texas, using fruit-baited traps during the period June 15 - July 13, 1972. Species other than D. melanogaster and D. simulans, females of which are indistinguishable, were separated from one another immediately. Upon return from the collection trip, single fly assays of alcohol dehydrogenase were made using electrophoresis at 250 V for 25 min. on noble agar gels (method of Ursprung and Leone, 1965), followed by formazan staining with 2-butanol as substrate. In cultures where both D. melanogaster and D. simulans were present, only D. melanogaster males were assayed. An attempt was made to score at least 50 flies for each station in the first 3 generations after collection. However, enzyme level was so low in certain cultures that 10 cultures consisting of 3 females each were prepared and assays made of the pooled progeny. The Adh^{II} allele specifies subunits with from 1/2 to 1/4 or less the specific activity of those coded by Adh^{I} (Pipkin and Hewitt, 1971). For the most part the frequency of the Adh^{II}, allele in the populations studied can be related to temperature. Thus Fig. 1 shows that along the gulf coast region Adh^{II} increases from .61 (Mico, Texas) to .69 (Corpus Christi, Texas) to .71 at Jiminez (near Cuidad Victoria) and .99 at Coatzocoalcos. Similarly in the interior high plateau region of central Mexico, Adh^{II} increases from .40 at San Luis Potosi to .54 at Cuatla (near Mexico City) to .63 at Oaxaca City in the mountains. A small collection at Acatlán (southern tip of state of Puebla) gave a lower frequency of .38 for Adh^{II}. The frequencies of Adh^{II} at 3 stations located beside the Sierra Madre Oriental (mountains) approximated corresponding frequencies in stations of about the same latitude in central Mexico, possibly because of gene flow from the cooler regions. (Tuxpan, .64; Paplantla, .54; and Cardel, near Vera Cruz, .51). Adh^{II} frequencies varied little in the coastal region from Coatzococalcos (.99) to Villa Hermosa (.95) to Merida where Adh^{II} reached fixation in the population sampled. However at Palenque, located in the edge of the mountains of Chiapas, the frequency of Adh^{II} dropped to .79, reflecting apparently gene flow from mountain populations.



References: Pipkin, S.B. and N.E. Hewitt 1971, DIS 46:66-67; Ursprung, H. and J. Leone 1965, J. Exp. Zool. 160:147-154. (Laboratory assays were supported by NIH grant 18409-02; collection trip, courtesy of Alan C. Pipkin, Sr.)

Minamori, S. Hiroshima University, Japan. Extrachromosomal element delta correlates with "segregation distortion" phenomenon.

It has been known that Segregation-distorter gene (SD, II-55.0) in D. melanogaster is recovered in functional sperm much more often than the expected 50% when heterozygous for an SD^{+} gene (Sandler, Hiraizumi and Sandler 1959). On the other hand, an SD-bearing second chromosome SD-5 was observed to retain an extrachromosomal element denoted by delta r which was assumed to be a copy of a gene locating on that chromosome (Da^{r}, 24.9; Minamori 1971 ; Minamori and Sugimoto, in press). The correlation of delta r to the distortion phenomenon was examined with SD-5 chromosomes. Second chromosomes recovered from SD-5/I-521($SD\ Da^{r}/SD^{+}\ Da^{+}$) females were individually tested for their sensitivity to the killing action of delta r (sensitive chromosome retains delta r; the original SD-5 is sensitive) and for their recovery from heterozygous males for the chromosome and a cn bw chromosome. The distribution of k values (the ratio of the chromosome among the total chromosomes recovered) of sensitive and insensitive chromosomes are separately shown in the follow-

ing table. It is evident that a sensitive chromosome showing no distortion (k < 0.60; presum-

Chromosome	k value 0.46 - 0.60	0.61 - 0.90	0.91 - 100	Total
Sensitive	85	13	111	209
Insensitive	190	20	3*	213

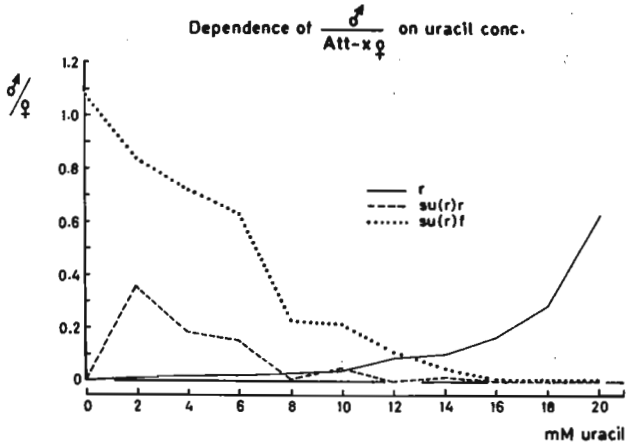
* showed no distortion in tests made in the subsequent generation

ably $SD^+ Da^r$) was produced by recombination from SD-5. The percentage was 40.7%. This finding suggests that the distortion cannot be induced by delta r alone. The percentage of insensitives which showed distorted segregation (k > 0.61) was 10.8%. Since this percentage is far lower for the expected 30%, the action of SD gene appears to be not or not fully expressed in the absence of delta r (SD Da^+ constitution). It is interpreted that the distortion phenomenon may be induced by a complemental interaction between SD gene and delta r.

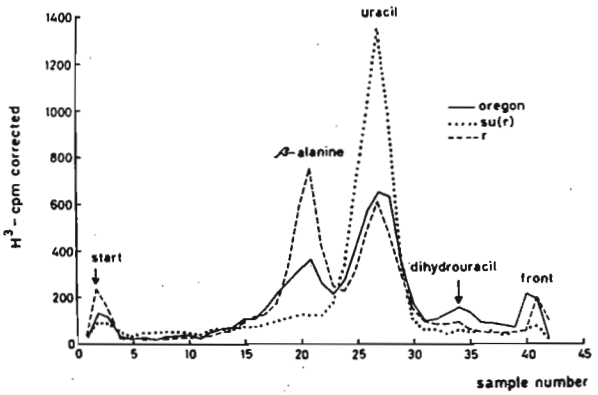
References: Minamori, S. 1971, Japan. J. Genetics 46:169-180; Sandler, L., Y. Hiraizumi and I. Sandler 1959, Genetics 44:233-250.

Bahn, E. University of Copenhagen, Denmark. A suppressor locus for the pyrimidine requiring mutant: rudimentary.

Studies have shown that an EMS induced recessive mutation situated at 1-27.7 is a suppressor of all rudimentary alleles. In contrast to the pyrimidine requiring rudimentary alleles (Nørby, 1970 Hereditas 66:205-214) this mutant is very sensitive to an exogenous supply of pyrimidines. The curves in Figure 1 show that in the cross su(r) f♂ x Att-X♀ on the minimal medium Eledon with increasing concentrations of uracil the suppressor mutant will not survive at concentrations higher than 16 mMolar of uracil. The rudimentary mutant on the contrary will hardly appear at concentrations lower than 12 mM uracil whereas the double mutant being rudimentary as well as suppressor of rudimentary has an optimum of survival at a concentration of 2 mM uracil. Thus, the r and the su(r) mutants clearly act as antagonists with respect to development of the flies on media with different concentrations of uracil. Together with the fact that dihydrouracil and dihydrothymine has no poisonous effect on the suppressor mutant the results suggested that the suppressing effect of the mutant was due to a block in the first step of pyrimidine catabolism.



Chromatography of extract of H^3-uracil fed whole larvae.



In an experiment using paper chromatography of the extract of H^3-uracil fed larvae the results depicted in Figure 2 showed no degradation of uracil in the su(r) larvae whereas the wild type and r larvae had been able to degrade uracil into dihydrouracil and further down to β-alanine. Other experiments based on in vitro degradation of H^3-uracil have given similar results. It is concluded that the suppressor of rudimentary mutant is a metabolic suppressor and that it causes a block in the first step of pyrimidine catabolism by greatly reducing dihydrouracil dehydrogenase activity and that the suppressing effect is due to a consequent sparesome degradation of special importance to a pyrimidine requiring mutant as rudimentary.