within populations at 31°C. This increase in variance is most pronounced in the Spanish population where the length of the sterility period was most severely affected by the high temperature stress.

In summary, the results show that the thermal environment affects the duration of heat-induced sterility in *D. buzzatii* and furthermore that genetic variation for this trait is present among populations.

References: Bundgaard, J., and J.S.F. Barker 2000, Biol. J. Linn. Soc. (in press); David, J.R., R. Allemand, J. Van Herrewege, and Y. Cohet 1983, In: *The Genetics and Biology of* Drosophila, (M. Ashburner, H.L. Carson and J.N. Thompson, jr., eds.), Academic Press, London, vol. 3, pp. 105-170.

The synthesis of a double fourth chromosome marked with y^+.

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Here I report the construction of C(4)DRA, a compound of the fourth chromosome that bears two markers, the recessive ci^l and y^+. This chromosome was built by a recombination between C(4)RM, ci^l ey^R and T(1;4)*sc H,* y^+.

I have previously noticed by complementation tests that T(1;4)*sc H,* y^+ is a deficient translocation that deletes at least 11 terminal genes of the fourth (Table 1). Since the fourth chromosome does not recombine normally, to induce recombination between C(4)RM and T(1;4)*sc H*, I made heterozygous animals *y w mit/ y w mit*; C(4)RM/T(1;4)*sc H,* y^+ and heat shocked larvae (1^{st} to 3^{rd}) for 14 hours at 37°C. This treatment was extremely severe and from about 100 larvae, I recovered 1 y^+ adult female. This single female was then crossed to *y w mit*/Y; *spa*[rgo]/*spa*[rugoso-nu] flies to detect the recombination event. *spa*[rugoso-nu] is a new homozygous viable allele of *spa*, that presents *shaven* and *sparkling* mutant phenotypes and is lethal over T(1;4)*sc H,* y^+(see scheme). T(1;4)*sc H,* y^+/*spa*[rgo] produce flies with extensive loss of macrochaeta that die as pupae or soon after hatching.

Any survivor of this cross presenting y^+ and normal macrochaetae may correspond to a recombination between T(1;4)*sc H* and C(4)RM. One female in a progeny of 38 presented was y^+ spa^+ and it was crossed again with *y w mit*/Y ; *spa*[rugoso-nu]/*spa*[rugoso-nu] males. From this cross only emerged animals *y spa*[rugoso-nu] and y^+ spa^+ flies. Some of y^+ spa^+ flies were crossed to the parental chromosome C(4)RM. With this combination I have observed the appearance of ci^l y^+ tetra-4 flies, that indicates a recombination between *ci* and *ey* that replaced ey^R by ey^+. This chromosome was named C(4)DRA-1 (Deleted on the Right Arm) and further tests showed that it is hemizygous viable in males and females, homozygous viable in females but lethal in homozygous males. Since this chromosome is completely viable over C(4)RM and in hemizygous males, I presumed that the lethality observed in homozygous males is due to an excess of a gene located in the X component of this chromosome. This element received the provisory name *male lethal dosage dependent* or *mald.* This chromosome has been quite stable after 6 months of its synthesis, and until now there are no signs that it can become homozygous for ey^R .

References: Hochman, B., H. Gloor, and M.M. Green 1964, Genetica 35:109-126; Hochman, B., 1971, Genetics 67: 235-252; Hochman, B., 1974, Cold Spring Harbor Symp. Quant. Biol. 38: 581-589; Hochman, B., 1976, In: *The Genetics and Biology of* Drosophila. (Ashburner,

Table 1. Genes complemented and not complemented by T(1;4)sc H. Bars indicate the position of these genes on the fourth chromosome.

T(1;4)sc H		101						102					
Fails to complement		A	B	C	D	E	F	A	B	C	D	E	F
spa-sv	*(8)*												
l(4)pho	*(1)*												
l(4)102EFd	*(2)*												
l(4)ABi	*(2,3)*												
l(4)1	*(9)*												
l(4)102ABf	*(2)*												
l(4)ABh	*(1)*												
l(4)CDe	*(4)*												
l(4)CDj	*(4,5)*												
l(4)CDo	*(4,5)*												
l(4)K-2													
Complements													
l(4)Abd	*(1)*												
l(4)CDf	*(4,5)*												
l(4)CDi	*(4,5)*												
l(4)CDm	*(4,5)*												
l(4)CDn	*(4,5)*												
l(4)CDp	*(4,5)*												
l(4)CDq	*(4,5)*												
ey	*(1, 8)*												
pan	*(6)*												
ci	*(7)*												

1-Hochman *et al.*, 1964
2-Hochman, 1971
3-Hochman, 1974
4-Hochman, 1976
5-Lindsley and Zimm, 1992
6-Brunner *et al.*, 1998
7-Stern and Kodani, 1955
8-FlyBase
9-Stronach, personal communication to FlyBase

These data suggest that T(1;4) carries deficiency, called here Df(4)rgo or Df(4)rugoso-nu. The break points were tentatively mapped to 102D1-D6 (exclusion of eyeless) and 102F2-F8 (inclusion of sparkling, *pho* and *l(4)102EFf*). The complementation tests above places *l(4)CDj* and *l(4)CDo* to the right of *l(4)CDf*, *l(4)CDi*, *l(4)CDm*, *l(4)CDn*, *l(4)CDp* and *l(4)CDq*. The latter genes were tentatively placed at the position 102D1-D6 (the previous left break point and the tentative left limit of the deficiency).

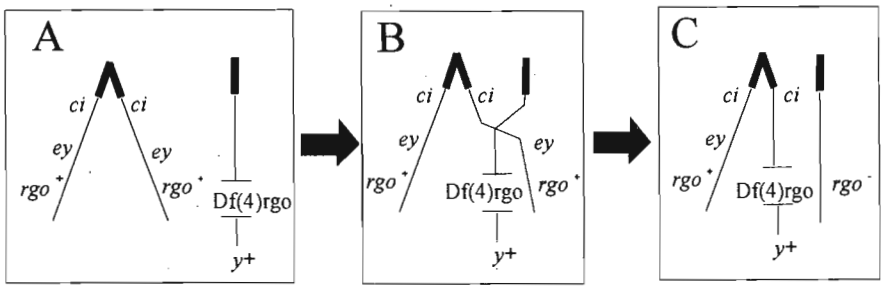

Figure 1. Scheme of heat shock induced recombination and selection of the recombinants.

M., and E. Novitski, eds.), volume 1b, pages 903-928, Academic Press, New York; Lindsley, D.L., and G.G. Zimm 1992, *The Genome of* Drosophila melanogaster. Academic Press, New York; Stern, C., and M. Kodani 1955, Genetics 40(3): 343-373; Flybase- http://flybase.bio.indiana.edu; Stronach, personal communication to Flybase http://flybase.bio.indiana.edu/.bin/fbidq.html?FBrf0093854.

Can *D. simulans* breed on *Morinda citrifolia*, the host plant of *D. sechellia*?

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Introduction

D. sechellia is a member of the *melanogaster* species-subgroup and endemic to Seychelles archipelago. Unlike most of the other members of the subgroup, it is monophagous and breeds exclusively on the ripe fruit of an arboreal rubiaceae, *Morinda citrifolia* (Tsacas and Bachli, 1981). The morinda fruit is known to contain several fatty acids such as *n*-caproic acid (hexanoic acid) and *n*-caprylic acid (octanoic acid) which are extremely toxic to the sibling species of *D. sechellia* (Amlou *et al.*, 1997, 1998a, b). Genetic analyses have shown that the tolerance of *D. sechellia* to the toxic substances of the fruit is mainly controlled by genes located on the third chromosome, suggesting that relatively small number of genes are involved in the adaptation to the host plant (Jones, 1998). It may be expected, therefore, that some degree of tolerance to the toxic substances can evolve within the local populations of sibling species where *M. citrifolia* is available for a potential food resource. In the fall of 1998, we happened to find a considerable number of *D. simulans*, one of the sibling species of *D. sechellia*, were gathering on the fallen fruit of *M. citrifolia* in Hahajima Island of the Ogaswara Islands (the Bonin Islands), Tokyo. Surprisingly, a number of adult flies of *D. simulans* emerged from the fruit collected in the field and brought back to the laboratory. The Ogasawara Islands are located about 1000Km south of Tokyo. On these islands, *D. simulans* is known to have been a dominant species for more than 60 years since its first discovery (Kikkawa and Peng, 1938; Okada, 1971). *M. citrifolia* is abundant in some of the islands. We examined for the possibility that some of *D. simulans* on Hahajima Is. might have acquired a