Hall, L.* University of British Columbia Vancouver, Canada. Conditional dominance of temperature-sensitive locomotor mutants.

The use of an attached X mating scheme has been very successful for the isolation of temperature-sensitive (ts) mutations affecting adult locomotor ability (Grigliatti et al., 1973). When 22°C was used as the permissive temperature and 29°C as the nonpermissive temperature, all ten of the temperature-sensitive locomotor mutants recovered from this screen of 1.1 million progeny were recessives on the X chromosome. This suggests that autosomal dominant mutations of this type either do not occur or are too rare to merit consideration. It would be very interesting to begin to isolate and characterize locomotor mutations on the autosomes but such studies have been hindered to date by the laborious procedure necessary to make an autosome homozygous in order to detect the relatively rare recessive temperature-sensitive mutants. However, recent studies on the ts X-linked locomotor mutants have suggested a procedure which would greatly simplify the task of screening for similar mutants on the autosomes.

The X-linked mutants that show temperature-dependent effects on adult locomotor ability fall into three complementation groups: shibire^{ts} (shi^{ts}), paralytic^{ts} (para^{ts}), and stoned^{ts} (stn^{ts}). All of these mutants when homozygous or hemizygous show wild type behavior at 22°C and are severely but reversibly crippled at 29°C. As heterozygotes these mutants all show wild type locomotor ability at 29°C acting as recessive mutations at this temperature. However, at 40°C as shown in Table 1 two out of the three complementation groups are reversibly

Table 1

Stock	29°C	40°C
Oregon R	+ (wild type)	+ for first ten minutes after which flies show signs of debilitation
Oregon R/FM6	+	Same as Oregon R
shi^{ts1}/FM6	+	Debilitated within 1 minute
shi^{ts2}/FM6	+	Debilitated within 1 minute
shi^{ts3}/FM6	+	Debilitated within 1 minute
shi^{ts4}/FM6	+	Debilitated within 1 minute
shi^{ts5}/FM6	+	Debilitated within 2 minutes
shi^{ts6}/FM6	+	Debilitated within 1 minute
para^{ts1}/FM6	+	Debilitated within 1 minute
para^{ts2}/FM6	+	Debilitated within 1 minute
para^{ts3}/FM6	+	Debilitated within 1 minute
stn^{ts1}/FM6	+	Behavior same as Oregon R
stn^{ts2}/FM6	+	Behavior same as Oregon R

paralyzed under conditions in which the locomotor ability of wild type flies (Oregon R) is unaffected. All of the alleles of both the shibire^{ts} and paralytic^{ts} complementation groups can be distinguished as heterozygotes from wild type flies after a short incubation at 40°C. This suggests that temperature-sensitive autosomal locomotor mutants could be isolated as heterozygotes from the progeny of mutagenized parents using the standard screening device (Williamson, 1971) but modifying the screening conditions to use short incubation times (less than 10 minutes at 40°C. Experiments to test the fertility of flies exposed to 40°C showed that such short exposures have no effect on the fertility or viability of either the wild type or the heterozygous mutant stocks tested.

Table 2

Temperature	Time required for crippling of shi^{ts1}/Oregon R hybrids
36°C	25-35 minutes
37°C	10 minutes
38°C	6 minutes
40°C	1 minute

The conditionally expressed dominant effects of the shibire^{ts} and paralytic^{ts} alleles should be very useful in future genetic manipulations of these stocks since it allows the experimenter to fractionate a population of +/+, +/mutant, and mutant/mutant individuals very rapidly simply by varying the temperature.

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Milkman, R., R. Zeitler and L. Layfer. University of Iowa, Iowa City. Multiple paternity in cage populations.

Individual D.m. females were removed from a polymorphic cage population and immediately allowed to lay eggs in vials, then genotyped with respect to α-glycerophosphate dehydrogenase, malate dehydrogenase, and alcohol dehydrogenase. For each double or triple homozygote, 20 F_1 progeny flies were genotyped. All 3 loci are on one chromosome, so the occurrence of more than two genotypes in a progeny sample indicates multiple paternity. Frequencies of the fast allele at each of the loci are 0.30, 0.38, and 0.22, respectively.

Nine of forty-five double homozygotes produced progeny with more than two genotypes; five of thirteen triple homozygotes did also. Additional double inseminations can be inferred: the observed certain cases are those where at least one male parent was heterozygous and the other male parent had a genotype differing from that of the first. The observed frequency, then, is equal to the true frequency of multiple mating multiplied by the frequency

Table 1. Number of progenies in each class.

Number of genotypes	Number of loci at which mother is homozygous: 2	3
1	7	1
2s*	27	6
2A*	2	1
3	7	3
4	2	2
Total	45	13

*2s - more common genotype ≤14; 2A - more common genotype >14 of the sample of 20. The 2A progenies may have resulted from sampling, differential viability of chromosomes, meiotic drive, or the participation of two different homozygous male parents.

of fulfillment of the above conditions. For doubly homozygous females, the value of 0.20 (9/45) is corrected to 0.38, and for triple homozygotes, 0.38 (5/13) is corrected to 0.44, the correction being less when more loci are involved. Smaller previous experiments using two loci and flies from different cages suggested that multiple paternity was rare in the populations studied. For this reason, and because culture conditions are quite diverse, the present estimated frequency, about 0.4, is not intended as a generalization. Also, when one of two male parents makes the larger contribution by far in an egg sample, the multiplicity may go undetected.

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The effect of varying the temperature on flies heterozygous for the shibire^{ts-1} allele are summarized in Table 2. This is one of the most extreme of the shibire^{ts} alleles. It may be seen that the dominant effect is evident at lower temperatures but the time required for crippling is increased at lower temperatures.

References: Grigliatti, T.A., L. Hall R. Rosenbluth and D.T. Suzuki 1973, Molec. Gen. Genet. 120:107; Williamson, R. 1971, DIS 46:148.

This work was supported by a Medical Research Council of Canada Fellowship to L.H. and by NRC grant A1764 and NCI grant 6051 to Dr. David T. Suzuki.

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