oviparous and viviparous animals with ZW sex chromosomes such as snakes and reptiles are expected to have the H-W antigen expressed on their cell surface.

In viviparous animals, many reproductive tissues, such as early embryos and most placental trophoblast cells, do not express major histocompatibility complex (MHC) antigens probably because foreign paternal MHC antigens are potential elicitors of the immunologic rejection of embryonic tissues in the maternal organism[11]. Similarly, lack of expression of such antigens by sperm may also help sperm to escape immunologic destruction in genetically disparate hosts[12].

In contrast, sperm from heterogametic males (XY) express H-Y antigen and are killed by H-Y antibodies produced by male-sensitized female mice[13,14]. This potential vulnerability is reflected in part by the fact that fertilization takes place only within days (6–144 h[15]) of mating in mammals. This compromise may be a weighted advantage if we consider that males are generally more accessible in social animals, such as mammals, in general.

In lower vertebrates, the lack of expression of H-W antigen in homogametic (ZZ) males and the presence of the antigen in heterogametic females (ZW) may be one of the means which facilitate sperm storage thus rendering the female partially independent of the male sexual cycle or availability. For example, females of numerous birds and reptiles, all of which are of ZW heterogametic sex chromosome type, and of snakes, many of which are ZW, are known to retain viable sperm within the oviduct for a considerable time before actual fertilization of the egg. This delay in actual fertilization of eggs, as distinct from the mating process, was termed 'amphigonia retardata' by Kopstein in 1938[16]. Mating occurs in snakes several months before ovulation and live sperm have been recovered from the uterus after 3–8 months[16,17] and young were reported to be born from isolated females of Pope's pit viper, *Trimeresurus popeorum*, at least a year after mating[18]. In turtles, a high percentage of fertile eggs have been shown to be obtained from female *Malaclemys* for 2 years without annual copulation, although fertility levels tend to fall later[19]. In birds, live sperm were found in oviducts for many weeks and fertile eggs were obtained for 30–32 days after removal of the male[20,21]. Rahn[22] suggested that the survival of sperm in the uterus might be a safety factor enabling eggs to be fertilized in the absence of males at the time of ovulation or when ecologic conditions in some way prevent preovulatory mating[23].

The presence of H-W antigen in females rather than males of heterogametic ZW animals such as snakes, reptiles, and birds suggests an immunologic basis of reproduction without constant availability of males in these desert dwelling, 'non-social', or polygamic animals.

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Dominance for enzyme activity in *Drosophila melanogaster*

J.B. Gibson, A.V. Wilks, A. Cao and A.L. Freeth

Department of Population Biology, Research School of Biological Sciences, Australian National University, P.O. Box 475, Canberra City, A.C.T. 2601 (Australia), 9 April 1985

Summary. A regulatory element tightly linked to the *Gpdh* locus in *Drosophila melanogaster* has been isolated from a natural population. Flies homozygous for second chromosomes bearing the element, *H31*, have half the GPDH activity of normal homozygotes. Heterozygotes between *H31* and *F* or *S* alleles exhibit dominance in GPDH activity. Heterozygotes between *H31*, *F* or *S* and *Df(2L)GdhA* have half the diploid level. The contribution of the *S* allele to the activity in *S/H31* heterozygotes is more than four times that of *H31*. The regulatory element distinguishing *H31* is tightly linked to the *Gpdh*$^+$ locus.
Key words. Drosophila; enzyme; sn-glycerol-3-phosphate; dehydrogenase; dominance; *trans*, regulation.

Heterozygotes for allozyme variants that differ in the amount of enzyme protein they produce generally have an intermediate level of enzyme activity. The activity variation is additive and the expression of the alleles is co-dominant[1,2]. Preferential expression of one allele over another, giving rise to non-additivity in enzyme levels, has been reported in a number of eukaryotes, usually in interspecific hybrids[3–5], but also within species[6–8]. In *D. melanogaster* examples of differential gene expression have been found to be due to *cis* acting regulatory elements[8,9], although both *cis* and *trans* acting modifiers of enzyme levels have been described[10–12]. We describe here a *trans* acting element affecting the steady state level of soluble sn-glycerol-3-phosphate dehydrogenase (GPDH; NAD$^+$ oxidoreductase; EC 1.1.1.8) in *D. melanogaster* which, in heterozygotes, results in dominance in GPDH activity.

The second chromosome bearing the variant was discovered in a survey of GPDH activity in a Huonville, Tasmanian population of *D. melanogaster*. This chromosome, designated *H31*, was

Table 1. GPDH activity in extracts of adult flies between four and eight days old

Gpdh genotype	Number of samples	GPDH activity (± SE)
F/F	5	408.4 ± 9.5
S/S	4	434.5 ± 10.6
H31/H31	5	188.8 ± 11.8
F/H31	4	373.0 ± 4.4
H31/F	5	384.1 ± 10.4
S/H31	5	394.6 ± 13.3
H31/S	6	398.7 ± 11.5

Table 2. Mean percentage relative staining intensity (± SE) in each band of activity on cellulose acetate membranes kept for 20 min at 50 °C prior to staining for GPDH

Gpdh genotype	Number of samples	Band of GPDH-1 activity S1	Heterodimer	FI
S/H31	6	68.1 ± 2.9	31.9 ± 1.5	None detected
S/F	6	29.7 ± 1.5	53.2 ± 2.3	17.1 ± 0.9

made homozygous (using *CyO* as a balancer) and found to produce relatively low GPDH activity in 3rd instar larvae and adults. It was shown by electrophoresis of single fly homogenates on cellulose acetate membranes that the chromosome carried the electrophoretic *F* allele at the sn-glycerol-3-phosphate dehydrogenase locus *(Gpdh)*[13]. Reciprocal crosses were set up between flies homozygous for the *H31* chromosome and those homozygous for chromosomes bearing the standard *S* or *F* electrophoretic variants at the *Gpdh* locus[14]. On emergence, the male progeny were aged for between four and eight days and assayed for GPDH activity in a Gilford spectrophotometer[15].

The results (table 1) show that the GPDH activity of flies homozygous for the *H31* chromosome is about half that of flies homozygous for chromosomes bearing standard $Gpdh^F$ or $Gpdh^S$ alleles. However, heterozygotes between *H31* and either $Gpdh^F$ or $Gpdh^S$ have significantly higher GPDH activity than the midparental value. The reciprocal crosses give consonant results and show that the inheritance of GPDH activity is non-additive. Heterozygotes between either *H31, F* or *S* and a second chromosome with a deficiency for the $Gpdh^+$ structural gene, *Df(2L)GdhA*[13], have half the diploid level of GPDH activity. In all these experiments there was a correlation of 0.96 between GPDH activity and the amount of GPDH protein assayed immunologically.

To test whether both alleles contribute similarly to the increased activity in heterozygotes, we compared the electrophoretic phenotypes of the *S/H31* and *S/F* heterozygotes on cellulose acetate membranes. In order to investigate the main band of activity, GPDH-1, the membranes were held in a water bath at 50 °C for 20 min to remove the GPDH-2 and GPDH-3 isozymes which are heat sensitive[15] and known to be derived from GPDH-1 by post-translational modification[16]. The enzyme is a dimer so that after the heat treatment heterozygotes are expected to exhibit three bands of activity. The membranes were scanned with an integrating densitometer to quantify the amount of enzyme in each band. The results (table 2) show that *S/H31* heterozygotes differ from *S/F* heterozygotes. In *S/H31* the band of activity contributed by the *S* allele is about twice as intense as the heterodimer band, and GPDH-1 produced by *H31* is barely visible and too faint to quantify on the densitometer. These proportions suggest that in *S/H31* heterozygotes the GPDH-1 produced by the *S* allele is present at more than five times the level of GPDH-1 produced by the *H31* allele, whereas *H31/H31* homozygotes have about half the activity of *S/S* homozygotes. The extra activity produced by the *S* allele is also clearly evident in extracts of *S/H31* after electrophoresis and staining of untreated membranes but the patterns are complicated by the presence of isozymes GPDH-2, GPDH-3 and heterodimers.

We have tested the possibility that the product of *H31* interacts with the product of the standard allele by mixing extracts of the two genotypes for up to 1 h and then assaying for GPDH activity. The activity levels were intermediate.

To test whether the properties of *H31* were due to a loosely linked modifier of the $Gpdh^+$ locus we have investigated the electrophoretic phenotypes of 140 recombinants between the second chromosome markers clot (2-18.3) and spade-like (2-25.15) on either side of the $Gpdh^+$ locus (2-20.5)[17]. The recombinants were obtained in the cross $H31/cl \cdot Gpdh^S spd$ ♀ × $cl \cdot Gpdh^S spd/cl\ Gpdh^S spd$ ♂. All of the recombinants in the *cl-spd* region had electrophoretic phenotypes like $Gpdh^{S/S}$ or $H31/Gpdh^S$ and there were none like *F/S*. These data suggest that if the effect is due to a modifier, then it must be located closer than 0.05 cM to the $Gpdh^+$ locus.

It is not yet known whether the factor giving dominance in GPDH activity in *S/H31* heterozygotes via preferential expression of *S* affects transcription or acts post-translationally to increase the stability of GPDH. A dominant *trans* acting modifier located on the third chromosome is known to affect the in vivo stability of GPDH by lowering degradation rates[18], and there is also evidence for a number of *cis* acting elements, tightly linked to the *Gpdh* gene, which alter GPDH activity by changing the rate of synthesis of the enzyme[10,11]. It is possible that *H31* is a structural variant at the $Gpdh^+$ locus itself as some variants are known to modify the rate of synthesis of GPDH[19].

Comparisons of the synthesis and degradation of GPDH-1 in euploid and aneuploid flies have usually shown that changes in gene dosage have an additive effect on GPDH levels[20], although there is evidence for dosage compensation in trisomics[21]. It is possible that the dominance we have observed derives from a compensatory effect in diploids, mediated via a metabolic feedback mechanism to which *S*, but not *H31*, responds.

It is worth noting that we have found variants with similar properties to *H31* in a number of Tasmanian populations and, in heterozygotes with normal alleles, the 'dominance' effect will mask any selective disadvantage known to be associated with low GPDH activity[14].

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