

Resource subdivision and the advantage of genotypic diversity in *Drosophila*

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It is noted that maintenance of genetic heterogeneity in natural populations of *Drosophila*, through the effect that variation in genotypic diversity across breeding sites may have on their productivity, depends not only on the effective number of parents contributing gametes to a site, as previously shown by other authors, but also on the number of loci underlying the variation in fitness. Using Monte Carlo simulation, it is found that as the number of loci increases, the effect of resource subdivision on the establishment of an initially rare allele introduced into the population becomes virtually indistinguishable from the pure drift case. It seems unlikely that this mechanism can explain the maintenance of a significant proportion of genetic variation in natural populations of *Drosophila*, although it may still be important in preserving linked gene complexes such as inversions.

Keywords: *Drosophila*, genetic heterogeneity, genetic variation, microgeographical structure, resource subdivision.

Introduction

In patchy environments, where the number of contributing parental pairs is limited within the patch size, environmental heterogeneity may arise simply because (i) genetic differences across patches exist from the sampling process, and (ii) the performance of an individual is often affected by its competing neighbours (Ayala & Campbell, 1974; Lewontin, 1974; Maynard Smith, 1989). Even though competition is a complex phenomenon whose outcome will depend on the parameters that determine the intra- and intergenotype competitive interactions (Mather & Caligari, 1981, 1983), experimental evidence suggests that genotypic mixtures may outyield single genotype populations (Kearsey, 1965; Caligari, 1980; Pérez-Tomé & Toro, 1982; Ellstrand & Antonovics, 1985; Martin *et al.*, 1988; Kelley, 1989a; López-Suárez *et al.*, 1993). The empirical results are not, however, always compatible with the idea that there are complementary interactions among genotypes, and in some cases no relationship has been detected between genetic heterogeneity and total production (Fowler & Partridge, 1986; Kelley, 1989b; Bell, 1991; Burt and Bell, 1992; Garcia & Toro, 1992). Models of competition among interbreeding genotypes show

that competitive interactions can maintain genetic polymorphism (Schutz & Usanis, 1969; Cockerham *et al.*, 1972; Antonovics, 1978), and it would be most interesting to know if there is indeed a positive correlation between fitness and genetically variable progeny in natural populations.

Flies of the genus *Drosophila* are primarily consumers of the microorganisms associated with patchy and ephemeral rotting plant materials that usually support one or a few generations of flies coming from a limited number of parents (Heed, 1968; Jaenike & Selander, 1979; Shorrocks, 1982; Lacy, 1983; Hoffmann *et al.*, 1984; Santos *et al.*, 1989). The general view that emerges from this resource subdivision is that of a few sibships growing together in the same habitat patch, so there will be an appreciable chance (F_{ST}) that two genes in the same patch will be identical by descent, relative to the whole, presumably panmictic, population. Based on the frequency-dependent selection model of Cockerham *et al.* (1972) and Templeton (1979), Hoffmann & Nielsen (1985) proposed that differences in genotype fitnesses arising from the effects that genetic heterogeneity within breeding sites may have on the number of progeny emerging from each site, makes it likely that genetic polymorphisms can be maintained in natural populations. In essence, they are

considering a patchy and physically uniform environment where genetic diversity is being maintained through interactions among genotypes, with heterogeneous groups having a higher fitness than homogeneous ones. Within-patch genetic variation for fitness in their basic model is, however, based on single gene (additive) effects. I feel that it may be more realistic to consider that many loci can be acting independently (i.e. additively) to determine the fitness of a patch. Under these circumstances, their conclusion critically depends on the number of loci involved, and maintenance of allelic diversity through interactions among competing genotypes seems unlikely with many loci. This is simply because genotypic diversity at one locus is not correlated with that at other independently segregating loci, and the level of genetic heterogeneity within breeding sites would tend to be the same as the number of loci increases.

In what follows, I first outline the Hoffmann & Nielsen (1985) model and summarize the literature on electrophoretic data for *Drosophila* collected from individual breeding sites. This allows us to obtain a crude estimate of the number of flies contributing progeny to a single patch. Using Monte Carlo simulations (Jacquard, 1974), I then follow the establishment of an initially rare allele when fitness differences across breeding sites depend on various numbers of loci.

The model

Hoffmann & Nielsen consider a random mating population subdivided into a number of ephemeral breeding sites. Within a site, the relative fitnesses of genotypes are the same, but the number of offspring

produced from any one site is proportional to the variance of the genotypic values among the offspring at the site. The genetic outcome of this selection depends on the matrix of brood comparisons in Table 1 (in analogy with the competitive comparisons of Cockerham *et al.*, 1972):

$$\begin{bmatrix} 0 & W_{22} - \frac{(W_{13} + W_{14})}{2} & W_{24} - W_{06} \\ W_{12} - W_{21} & 0 & W_{15} - W_{06} \\ W_{04} - W_{21} & W_{05} - \frac{(W_{13} + W_{14})}{2} & 0 \end{bmatrix} = \begin{bmatrix} 0 & d_{21} & d_{20} \\ d_{12} & 0 & d_{10} \\ d_{02} & d_{01} & 0 \end{bmatrix}$$

For one locus with additive gene effects, we have the matrix:

$$\begin{bmatrix} 0 & 0 & \frac{1}{2} \\ \frac{1}{4} & 0 & \frac{1}{4} \\ \frac{1}{2} & 0 & 0 \end{bmatrix}$$

This matrix satisfies condition 1 of Cockerham *et al.* (1972) for a protected polymorphism: $d_{10} > 0$; $d_{12} > 0$. The value for Δp in this case is

$$\Delta p = \frac{\frac{1}{4} pq (q^2 - p^2)}{K + pq}$$

Table 1 Fitness of offspring (W_{ij}) when genotype i is in brood type j and gene effects are additive

Mating	Frequency	Fitness of offspring in brood		
		<i>AA</i>	<i>Aa</i>	<i>aa</i>
<i>AA</i> × <i>AA</i>	p^4	$W_{21} = K$		
<i>AA</i> × <i>Aa</i>	$4p^3q$	$W_{22} = K + 0.25$	$W_{12} = K + 0.25$	
<i>AA</i> × <i>aa</i>	$2p^2q^2$		$W_{13} = K$	
<i>Aa</i> × <i>Aa</i>	$4p^2q^2$	$W_{24} = K + 0.5$	$W_{14} = K + 0.5$	$W_{04} = K + 0.5$
<i>Aa</i> × <i>aa</i>	$4pq^3$		$W_{15} = K + 0.25$	$W_{05} = K + 0.25$
<i>aa</i> × <i>aa</i>	q^4			$W_{06} = K$

Number of offspring produced from any one brood (breeding site) is proportional to the constant K plus the variance of the genotypic values among the offspring.

which gives a stable equilibrium at $p = 0.5$. For the dominance case (genotypic value of $Aa = AA = 2$), the matrix of brood comparisons:

$$\begin{bmatrix} 0 & -\frac{3}{8} & \frac{3}{4} \\ 0 & 0 & 1 \\ \frac{3}{4} & \frac{5}{8} & 0 \end{bmatrix}$$

also leads to a protected polymorphism under condition 3 of Cockerham *et al.* (1972): $d_{10} > 0$; $d_{12} = 0$, $d_{02} > 2d_{21}$. A stable equilibrium is attained at $p = 0.314$.

A considerable change in gene frequency towards the equilibrium value in the additive model is evident in Hoffmann & Nielsen simulations when the establishment of an initially rare allele,

$p_0(A) = 0.025$, is followed and five or fewer mating pairs contribute progeny to a breeding site.

Genetic microdifferentiation in *Drosophila*

Genetic variation at the level of the between-breeding sites component can be estimated using Wright's (1951) measure of genetic differentiation, F_{ST} . Table 2 presents some estimates of this parameter calculated for different *Drosophila* species. The list is not intended to be an exhaustive survey but should be representative of recent work. Computations of F_{ST} in *Drosophila buzzatii* were performed using the methods of Weir & Cockerham (1984, refs. 1, 3, 4), and Nei & Chesser (1983, ref. 2). The use of either method provides almost identical large sample estimates of this parameter (Chakraborty & Danker-Hopfe, 1991). For the mycophagous *Drosophila*, F_{ST}

Table 2 Estimates of the standardised variance (F_{ST}) among breeding sites from several species of *Drosophila* based on allozyme variation

Species and population	Breeding sites	Loci	F_{ST}
<i>Drosophila buzzatii</i>			
Trinkey (Australia) ¹	<i>Opuntia cladodes</i>	6	0.059
Trinkey (Australia) ¹	<i>Opuntia cladodes</i>	6	0.025
O'Hara (Australia) ¹	<i>Opuntia cladodes</i>	6	0.002
Grandchester (Australia) ¹	<i>Opuntia cladodes</i>	6	0.038
Grandchester Hill (Australia) ¹	<i>Opuntia cladodes</i>	6	0.056
Borallon (Australia) ¹	<i>Opuntia cladodes</i>	6	0.048
Hemmant (Australia) ¹	<i>Opuntia cladodes</i>	6	0.037
Carboneras (Spain) ²	<i>Opuntia cladodes</i>	4	0.012
Trinkey (Australia) ³	<i>Opuntia cladodes</i>	6	0.032
Carboneras (Spain) ⁴	<i>Opuntia</i> fruits (prickly pears)	4	0.046
Carboneras (Spain) ⁴	<i>Opuntia</i> fruits	4	0.013
<i>Drosophila falleni</i>			
New York and Tennessee ⁵	Mushrooms	3	0.013
<i>Drosophila melanogaster</i>			
Wandin North (Australia) ⁶	Apples and peaches	3	0.038
Carboneras (Spain) ⁷	<i>Opuntia</i> fruits	3	0.017
<i>Drosophila ordinaria</i>			
New York and Tennessee ⁵	Mushrooms	2	0.019
<i>Drosophila putrida</i>			
New York and Tennessee ⁵	Mushrooms	3	0.014
<i>Drosophila testacea</i>			
New York and Tennessee ⁵	Mushrooms	3	0.011
<i>Drosophila tripunctata</i>			
New York and Tennessee ⁵	Mushrooms	4	0.028

¹Thomas & Barker (1990); ²Quezada-Díaz (1993); ³Prout & Barker (1993);

⁴Quezada-Díaz *et al.* (1995); ⁵Lacy (1983); ⁶Hoffmann *et al.* (1984); ⁷M. Santos & K. Th. Eisses (unpublished data).

estimates are the average of the values for each locus and are defined as the divergence observed between populations of flies collected from various species of mushrooms, within small study sites, within years and within months (Lacy, 1983). These values are to be compared with those for *Drosophila buzzatii* because they make reference to the genetic differentiation among *Opuntia* substrates collected at the same time. For *Drosophila melanogaster*, F_{ST} was calculated as the average standardized variance across breeding sites and loci (ref. 6 in Table 2), or following Weir & Cockerham (1984, ref. 7).

We can think of *Drosophila* population structure as consisting in an array of local breeding populations with high extinction and recolonization rates, where the fraction of genetic variance owing to the sampling effect of colonization among the newly founded populations is

$$F_{ST}(0) = \frac{1}{2N_0},$$

N_0 being the effective number of locally breeding adults (Wade & McCauley, 1988). In the model we are considering (Table 1), the fraction of these populations that survive for a generation, grow to size N_1 , breed and receive a fraction m of migrants from other sites is equal to 0, which amounts to saying that breeding sites allow only one generation of flies before drying out and/or that newly emerging

adults tend to disperse rather than to remain in their natal patch. This may not be true in some cases (see Thomas & Barker, 1990), and the estimate of the effective size of locally breeding adults (N_e) from the F_{ST} values would be approximately the harmonic mean size over generations (Prout & Barker, 1993). The outcome in such cases will depend on the details of the breeding structure, but the qualitative conclusions would be the same as far as higher fitnesses are associated with those sites where both alleles are segregating at intermediate frequencies.

Using the F_{ST} values in Table 2, crude estimates of N_e under the assumptions of selectively neutral loci and only one round of genetic drift suggest that fewer than 20 individuals usually contribute gametes to each breeding site (the estimates range between eight and 250, with an average of 17). On empirical grounds it seems, therefore, that Hoffmann & Nielsen's model is a plausible possibility in *Drosophila* natural populations, but the effect may not be very strong in most cases.

Simulations

As in Hoffmann & Nielsen, I followed the establishment of an initially rare allele introduced at a frequency $p_0(A) = 0.025$. From the empirical information available, five mating pairs taken at random from the whole population and contributing progeny to each breeding site were used in the simulations as

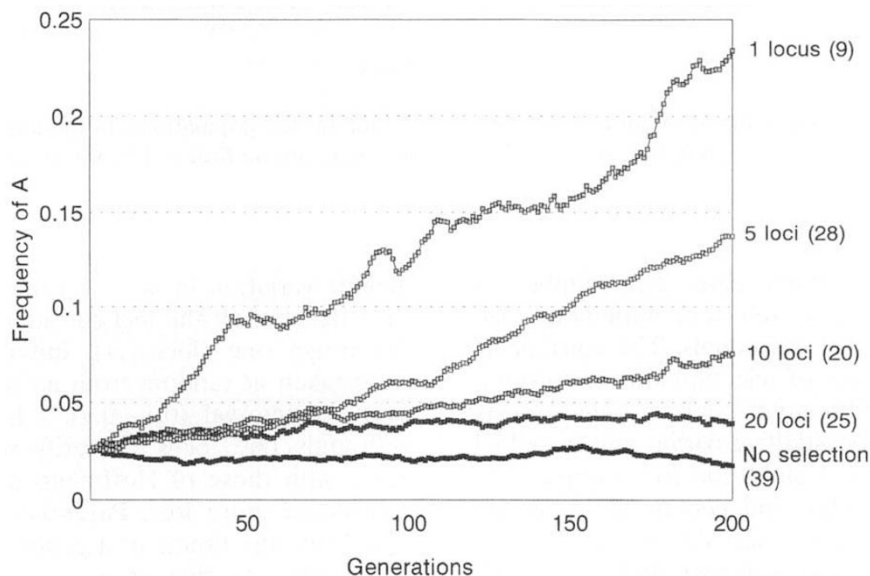


Fig. 1 Computer simulations of the establishment of an initially rare allele ($p_0(A) = 0.025$) in a random mating population subdivided into 100 breeding sites and five mating pairs contributing progeny to each site. Variation in fitness across breeding sites results from the segregation of 1, 5, 10 and 20 autosomal loci with two alleles each. Lines are the averages over all independent runs (numbers in parentheses).

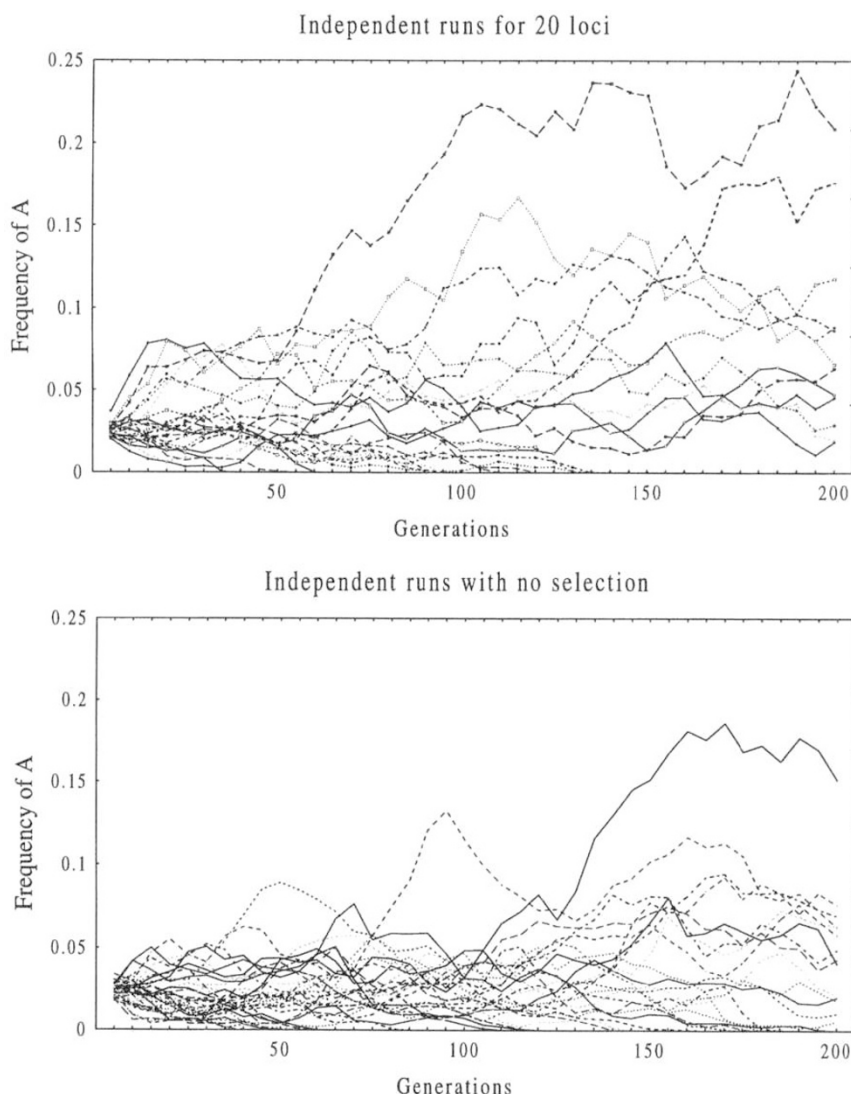


Fig. 2 Simulations of the establishment of an initially rare allele in subdivided populations. In the upper graph 20 autosomal loci contribute to variation in fitness. In the lower graph there are no fitness differences across patches.

a believable and favourable case. The number of breeding sites was set to 100, and population size was maintained at 1000 individuals. The constant K determines the intensity of selection and was given a value of 5, as in Hoffmann & Nielsen. Gene effects were considered to be additive within and over loci (genotypic values of 2, 1 and 0 for, for example, AA , Aa and aa , respectively), and genetic variation for fitness across breeding sites was determined by 1, 5, 10 and 20 autosomal loci with two alleles each. All the loci are assumed to assort independently, i.e. they are unlinked. In such an additive model, linkage disequilibrium generated by chance because of the small number of founders is ignored, and

genetic variation in any breeding site is the sum of the effects of all the loci considered. For all loci but the target one (locus A), initial gene frequencies were taken at random from an uniform distribution over the interval (0.1–0.9). I first ran simulations with only one locus to verify whether my results agree with those of Hoffmann & Nielsen and then considered more loci. Parenthetically, it should be noted that the fitness of a genetically heterogeneous site relative to that of a genetically homogeneous site is

$$\frac{K + V_a}{K},$$

where V_a is the variance of genotypic values among the offspring at the former site. A value of 5 for K yields, therefore, a very high difference in fitness between a pure and a mixed culture when several loci are contributing to the variation in fitness.

The interactive matrix algebra program MATLAB® (V. 4.0 for Windows) was used for computations, and the pseudorandom number generator was seeded before each run to make the pseudorandom numbers as 'random' as possible. The model was run on a 486 (66 Mhz) PC-compatible.

The simulations (Fig. 1) show that there is a considerable effect when only one locus is involved, in agreement with the previous results obtained by Hoffmann & Nielsen. As the number of loci increases, however, the effect of resource subdivision on the establishment of an initially rare allele introduced into the population decreases, and there is little effect when ten or more loci are acting independently to produce the variation in fitnesses across breeding substrates. Therefore, the advantage of increasing genetic variability within breeding sites clearly depends on the number of loci involved. Figure 2 shows the independent runs when 20 loci contribute to variation in fitness, and when no fitness differences exist across patches (pure drift case). Note that part of the variation between runs in the case of 20 loci is attributable to the initially different gene frequencies at all loci but locus *A*.

Discussion

Maintenance of genetic variation through environmental heterogeneity has been suggested by assuming either genotype–environment interaction (Levene, 1953; Hedrick, 1986; Gillespie & Turelli, 1989), or complementary interactions among genotypes (Schutz & Usanis, 1969; Cockerham *et al.*, 1972; Antonovics, 1978). The second mechanism proposes that if a significant proportion of genetic variation is maintained by balancing selection, that selection must be frequency-dependent.

Frequency-dependent selection has been studied in many laboratory populations of *Drosophila* (Curtis, 1990 and references therein), and maintenance of genetic variation in the use of different pupation sites in population cages of *Drosophila willistoni*, where polymorphic populations reach larger sizes and greater biomass than monomorphic ones, can be taken as a good example (De Souza *et al.*, 1970). We do not know, however, whether the laboratory examples are representatives of a widespread phenomenon.

In the Hoffmann & Nielsen model there seems to be a trade-off between founder events producing variability among breeding sites, and a large number of offspring. Martin *et al.* (1988), working with *Drosophila melanogaster*, showed that productivity is not related to genotypic variability unless larval competition is strong and viability is greatly reduced. These results suggest that strong density-dependent mortality, in addition to subdivision into discrete breeding sites, is also a basic ingredient of the model. As far as I know, density-dependent mortality in nature has only been shown to occur in *Drosophila falleni*, *Drosophila putrida* (Grimaldi & Jaenike, 1984) and *Drosophila buzzatii* (Quezada-Díaz *et al.*, 1995). In the latter species, larval mortality in *Opuntia* fruits is very high (approximately 78 per cent), but the fraction of genetic variance owing to the sampling effect provided an estimate of 30 individuals contributing progeny to a fruit (Quezada-Díaz *et al.*, 1995). Therefore, very little subdivision, and hence very little selection, probably takes place to overcome the pure drift case (see Hoffmann & Nielsen, 1985).

In addition to the density effect, the genetic basis of the variance in fitness across breeding sites is also relevant. The advantage of resource subdivision decreases with the number of loci. It seems, therefore, that maintenance of genetic variation by means of resource subdivision in natural populations of *Drosophila* would require: (i) that the fraction of genetic variance owing to the sampling effect of colonization should be not lower than approximately 0.025 (i.e. fewer than ten mating pairs should contribute progeny to a breeding site (see Hoffmann & Nielsen, 1985); (ii) relatively strong density-dependent mortality within breeding sites; and (iii) single gene effects strong enough to produce 'overcompensatory' effects, defined as the differential exploitation of the available resources by alternative genotypes (Tosic & Ayala, 1980; Milosevic *et al.*, 1990; Peng *et al.*, 1991). In such situations, the yield of a mixed culture can be expected to be greater than that of a pure culture. It has been shown, however, that differential exploitation of an environment by competitors need not always result in overcompensation (Case *et al.*, 1979; Nunney, 1980). Conversely, there may be a higher yield in mixtures than in pure cultures even in the absence of differential resource utilization (Nunney, 1983). Overcompensation has been invoked as an important mechanism to account for the maintenance of enzyme polymorphisms in *Drosophila*. Examples are the *Mdh-2* locus in *Drosophila pseudoobscura* (Tosic & Ayala, 1980), and the *Sod* locus in *Drosophila*

melanogaster (Peng *et al.*, 1991), although nonrandom associations with alleles at other closely linked loci cannot be rejected. I show here, however, that there are serious objections to the view that this form of environmental heterogeneity can account for a significant amount of genetic variation. To put it in a paradoxical way, the more empirical work is published that supports overcompensation as a form of balancing selection at individual loci, either arising as a consequence of differential exploitation of the available resources or differences in resource preference (Nunney, 1983), the nearer we will be to the pure drift situation because these individual effects would cancel out each other across breeding sites (Fig. 1). Notice that in the simulations involving various loci, condition 1 of Cockerham *et al.* (1972) for a protected polymorphism is satisfied for any locus taken individually and, therefore, a stable equilibrium should in theory be attainable at $p = 0.5$ for both alleles. Genotypic diversity at one locus is not, however, correlated with that at another locus, and the level of genetic heterogeneity within breeding sites tends to be the same as the number of loci increases.

In summary, genotype diversity may have an effect on juvenile survival in *Drosophila*, but I doubt that it can count as a general mechanism to maintain genetic variation. Nevertheless, the mechanism may still be important in preserving linked gene complexes such as inversions. There is evidence that chromosomally polymorphic populations outyield monomorphic ones (Dobzhansky, 1970), and that different inversion types may show frequency-dependent interactions (Levene *et al.*, 1954; Kojima & Tobari, 1969; Anderson *et al.*, 1986). The inversions involve a sizable part of the genome, and recombination in a structurally homozygous individual would tend to destroy interacting polymorphic genes. This may cause a recombination load which is frequency dependent (Wasserman, 1968, 1972; Charlesworth & Charlesworth, 1975), and the population may remain chromosomally polymorphic. A higher productivity of chromosomally heterogeneous breeding sites may superimpose, and the point of equilibrium would be a function of the joint effect from the two mechanisms.

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