

activity and composition are influenced by temperature, relative humidity and rainfall. There are many primary and secondary alcohols present in fruit: long chain primaries such as hexanol and octanol are present in apples, bananas and oranges, which also contain many secondary alcohols (Hulme 1970; McKechnie and Morgan 1982). These include isopropanol (in apples) 2-Butanol (in apples and oranges), 2-pentanol, 2-hexanol and 2-heptanol (in apples and bananas) (Hulme 1970). The *in vitro* activity of ADH has been found to be greater with secondary alcohols than with primary alcohols as substrates (Sofer and Hatkoff 1972; Vigue and Johnson 1973; Day *et al.* 1974; Morgan 1975; Oakeshott 1976; Chambers 1981; Daggard 1981).

Thus, *in vivo*, ADH allozymes are presumably good catalysts for the oxidation of secondary alcohols. This appears to be an evolutionary quirk. Although primary alcohols can be used by *Drosophila* as food, secondary alcohols cannot (van Herrewege *et al.* 1980; Sánchez-Cañete *et al.* 1986). However secondary alcohols are more toxic than the corresponding primary ones (David and Bocquet 1976). (Other evidence however, showed that the presence of ADH in non-mutant flies increased the short term (1-3 days) physiological tolerance to isopropanol (David *et al.* 1981).)

As with primary alcohols, the *in vitro* activity of the *Adh^F* homozygote was found to be considerably greater than that of the *Adh^S* homozygote when the substrate was isopropanol and this difference in activity between the two *Adh* genotypes was found to be greater than the difference in their respective activities

with ethanol (Day *et al.* 1974; Lewis and Gibson 1978; Winberg *et al.* 1985). Further, *Adh^F* homozygotes showed significantly greater *in vivo* maximum oxidation rates with isopropanol as a substrate than did *Adh^S* homozygotes (Heinstra *et al.* 1987). It therefore seems likely that, in the presence of secondary alcohols the fast allele would be selected against due to the high activity of the corresponding allozyme and the resultant build up of toxic products.

An explanation for the toxicity of secondary alcohols is that, whereas primary alcohols are oxidized to aldehydes and can be further oxidized to acids, secondary alcohols are oxidized to ketones which cannot easily take an extra atom of oxygen and thus the process of oxidation is terminated. This results in a build up of toxic ketones. Experimental *in vivo* evidence of this was obtained when exposure of both *Adh* positive and *Adh* negative (null homozygous) flies to the vapours of the secondary alcohols, 1-pentene-3-ol (Sofer and Hatkoff 1972) and pentyne-3-ol and 1-hexene-3-ol (O'Donnell *et al.* 1975) resulted in the death of the *Adh* positive flies leaving negative mutants unaffected.

As regards the different alleles, Morgan (1975) found that exposure of flies to pentene-3-ol (which is reported to be oxidized by ADH to give a lethal ketone product, ethyl-vinyl-ketone (Sofer and Hatkoff 1972)), selectively favoured the *Adh^S* allele. Morgan (1975) also showed that, for primary alcohols the ratio of ADH^F to ADH^S activities *in vitro* was related to the degree to which

the *Adh^F* homozygote was selected for in the presence of that alcohol. This was not the case for isopropanol (a secondary alcohol which is broken down into acetone by ADH). Despite an extremely high (ADH^F:ADH^S) activity ratio, a surprisingly large proportion of *Adh^S* homozygous flies survived.

It is possible, however, that different secondary alcohols are metabolically handled in different ways. Garrido *et al.* (1988) suggested that pentene-3-ol (an unsaturated secondary alcohol) and isopropanol (a saturated secondary alcohol) may be detoxified by slightly different (ADH-activity-dependent) processes. They also suggested that ethyl-vinyl-ketone is further transformed (by means of some ADH-independent mechanism) into some other compound, and consequently does not accumulate in the fly.

Finally, a difference in the number of carbon atoms in longer chain alcohols also changes the toxicity of the alcohol. David and Bocquet (1976) found that the higher the number of carbon atoms, the more toxic the alcohol. They suggested that this toxicity could be due to either the slow metabolism of these alcohols or to their metabolism into even more toxic products.

7.1.1.3 Latitudinal clines and temperatures

Many of the *Adh* investigations have revealed a latitudinal clinal pattern with the frequency of *Adh^S* increasing with decreasing distance from the equator. This was found in the Eastern United States (Vigue and Johnson 1973; Smith *et al.* 1978), in France (Charles-Palabost *et al.* 1985), in Japan (Watada *et al.* 1986), in

the Peoples' Republic of China (Jiang *et al.* 1989) and in Australasia (Oakeshott *et al.* 1982a). Further, clines in North America and Europe/Asia (Oakeshott *et al.* 1982a), in North America and France and West Africa (Singh *et al.* 1982) and between Europe and equatorial Africa (David *et al.* 1986) have been shown to exist (see also Section 6.3.6.1 for a description of the worldwide geographic distribution of the *Adh* alleles). The initial response to these clines was that they were caused by temperature changes and this has at times proved to be correct. For example Pipkin *et al.* (1973) found the regression of *Adh^S* (*Adh* II) frequency on extreme minimum temperature to be statistically significant and Oakeshott *et al.* (1982a) found a positive correlation between *Adh^S* frequency and maximum temperature in North America - but not in Asia or Australasia. Further, an altitudinal cline in frequencies of *D. melanogaster* populations of Southeastern Mexico was believed to be temperature induced (Pipkin *et al.* 1976).

Laboratory evidence suggests that the *ADH^S* allozyme is more thermostable than the *ADH^F* allozyme *in vitro* (Gibson 1970; Day *et al.* 1974; van Delden and Kamping 1980). Several exceptions have been reported, however; Oakeshott (1976) found that extracts of the *Adh^F* homozygote and the *Adh^F/Adh^S* heterozygote were more stable at 40°C than those of the *Adh^S* homozygote. Further, Oakeshott (1979) found that the frequency of the *Adh^S* allele decreased in laboratory populations exposed to high temperatures (29°C). Van Delden and Kamping (1980) found that adult mortality at high temperatures (35°C) was not associated with the differing heat stability of the allozymes. In natural populations Singh

et al. (1982) found a surprisingly high frequency of Adh^F in populations in Taiwan and Vietnam, which are at 11° N and 25° N respectively. Places this close to the equator are expected to have a high frequency of Adh^S in their populations. For example, the same authors found Adh^S frequencies of 0.861 and 0.971 in Texas (25.8° N) and West Africa (6.3° N) respectively. Thus it is possible that temperature is not the chief causative agent of the Adh latitudinal clines.

7.1.1.4 Rainfall

Another environmental variable which has been investigated as a possible causal agent for variation in Adh allele frequency is rainfall. Anderson (1981) found that the latitudinal cline in the frequency of Adh^F in Australia could be explained largely by variation in rainfall. The author found a positive correlation between Adh^S frequency and the amount of annual rainfall. A negative correlation between Adh^F frequency and rainfall for populations from Europe and Equatorial and Southern Africa was found by David *et al.* (1986). Oakeshott *et al.* (1982a) found the same to be true for populations from Australasia, North America and Asia. On the other hand, a more recent study by Anderson *et al.* (1987) found no such correlation to exist. However, it does appear that moisture in the atmosphere has some selective effect since Adh^S homozygotes survived better at high humidity in laboratory populations (Balsma-Meeles and van Delden 1974; van Delden *et al.* 1978).

between the *Adh* locus and chaetae-controlling loci. Furthermore, they suggested that individuals with intermediate sternopleural chaetae numbers had an advantage in mating. Theoretically, heterozygous intermediacy can lead to a balanced polymorphism in highly variable environments where the selective advantage of one allele over the other changes frequently (Gillespie and Langley 1974).

7.2. Materials and Methods

The electrophoretic data obtained as described in Chapter 6 were used to test for correlations with two geographic variables and three environmental variables as follows:

The Spearman rank correlation statistic was used on the common allele of the five most polymorphic loci (*Adh*, *Dip-A*, *Hes-1*, *Hes-2* and *Idh-NADP*) to test for associations with (1) latitude, (2) longitude, (3) extreme minimum temperatures, (4) extreme maximum temperatures and (5) precipitation. The temperature and rainfall data were averaged over the last four months of the year in which the collections were made. All samples were collected in October, November or December. Only 14 of the 17 locations could be used for correlation with temperature and rainfall as there was no weather data for the three remaining locations, Agatha, Maclear and Swakopmund. Climatic data were obtained from the weather bureaux at Pretoria (South Africa), Windhoek (Namibia) and Harare (Zimbabwe).

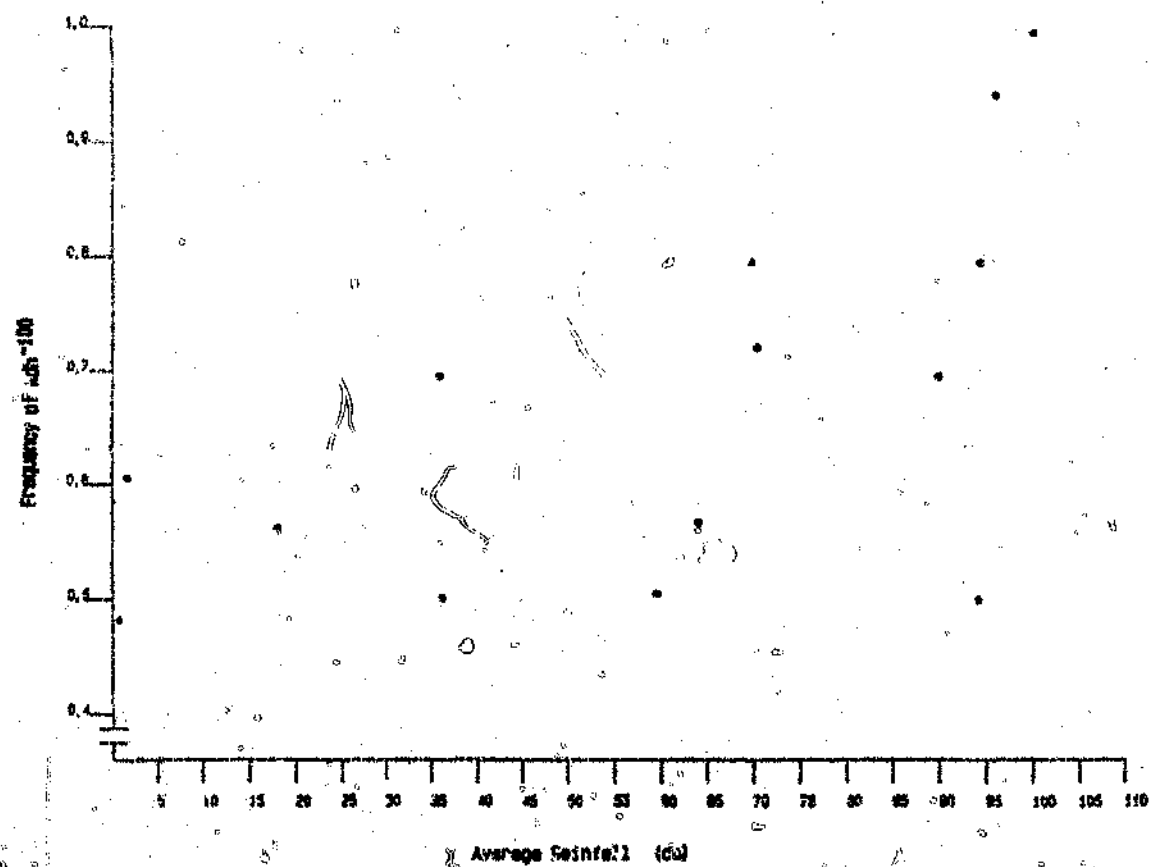


Fig. 7.3

Variation in the frequency of
Adh-100 with average monthly
 rainfall.

7.4.1 Dipeptidase-A

The frequency of *Dip-A*¹⁰⁰ decreased with increasing longitude. Because of the lack of any environmental gradient running from west to east it is difficult to postulate any adaptive significance for this cline. The strength of this correlation was weak ($0.01 < P < 0.05$) indicating that this cline may be an artificial by-product of stochastic effects.

7.4.2 Isocitrate Dehydrogenase-NADP

The frequency of *Idh-NADP*¹⁰⁰ varied significantly ($P < 0.01$) with extreme minimum temperature. As the temperature increased, so the frequency of *Idh-NADP*¹⁰⁰ decreased. Oakeshott (1979) noted a correlation of *Idh-NADP*^{SS} with temperature, its frequency was significantly lower at 29°C than at 25°C. To my knowledge no other variations in *Idh-NADP* allele frequency (in *D. melanogaster*) with temperature have been reported. There are numerous reasons as to why an enzyme could vary with temperature in a selective sense. A biochemical property which may be relevant is enzymatic activity. Isocitrate dehydrogenase is involved in the third step of the citric acid cycle. This enzyme catalyzes the oxidative decarboxylation of isocitrate to α -ketoglutarate and CO₂ (Lehninger 1982). Consequently, various alleles may be favoured at various times depending on the level in the food supply of the major organic fuel molecules of the cell i.e. carbohydrates, fatty acids and amino acids. These in turn will be partly determined by the environmental temperature.

has been observed in *in vitro* experiments, it has often been under conditions of extremely high temperature such as 35°C (e.g. van Delden and Kamping 1980), temperatures which were not attained in the Southern African sites investigated.

The correlation between the frequency of the "slow" allele and rainfall which has been observed in this and several other studies (see Section 7.1.1.4) may not be universal. If rainfall does have a selective effect, then the following model may explain why this is so. Firstly, the *Adh^S* allele has been shown to be advantageous in laboratory populations where humidity is high (Bijlsma-Meeles and van Delden 1974; van Delden *et al.* 1978). Thus, the concomitant increase in humidity with rainfall apparently confers some (unknown) benefit on *Adh^S* homozygotes. Secondly, changes in moisture availability result in changes in the microenvironment of *Drosophila* eggs and larvae. I do not know what the direct effect of increased rainfall on the variety and concentration of alcohols produced by ripening and fermenting fruits would be. It seems likely however that increased moisture results in lower evaporation rates of alcohols and also possibly in an increase in the activity, abundance and diversity of fermentative microorganisms. Different strains of yeasts are responsible for the production of different alcohols depending on available substrates (Cavazzo *et al.* 1989). Thus an increase in, for example, the production of secondary alcohols and their subsequent efficient oxidation by the more active ADHF allozyme could lead to the accumulation of toxic ketones

7.1.1.5 Non-environmental factors

Apart from environmental factors, several other genetic factors may be responsible for the *Adh* polymorphism. These include linkage disequilibrium and heterosis.

The *Adh^S* allele has frequently been observed to be in linkage disequilibrium with the chromosomal inversion, *In (2L)t* (Kojima *et al.* 1970; Langley *et al.* 1974; Voelker *et al.* 1977; van Delden and Kamping 1989). This suggests that the geographic patterns in allele frequency may reflect linkage with a polymorphic chromosomal inversion or may reflect selection at a closely linked, but unidentified locus.

The worldwide polymorphic nature of the *Adh* locus suggests that some kind of balancing selection may be operating. Evidence of heterozygote superiority has been obtained in normal laboratory populations (Bijlsma-Meeles and van Delden 1974; van Delden *et al.* 1978), in laboratory populations kept on ethanol-supplemented media (Oakeshott *et al.* 1984) and in wild populations (Franklin 1981; McKenzie and McKechnie 1981). An indirect reason for the maintenance of the *Adh* polymorphism has been proffered by Sherief and Skibinski (1988) who found a significant association between heterozygosity at the *Adh* loci and sternopleural chaetae numbers. Individuals heterozygous for the *Adh^F* and *Adh^S* alleles had chaetal numbers that were close to the population mean or mode. These authors suggested that the association was due to the pleiotropic effect of *Adh* alleles on chaetae number or was due to strong linkage disequilibrium

7.3 Results

An examination of the correlation between the most common allele of the 5 polymorphic loci (*Adh*, *Dip-A*, *Hex-1*, *Hex-2* and *Idh-NADP*) and the parameters latitude, longitude, extreme minimum and maximum temperature and precipitation using Spearman's rank correlation test revealed the following:

1. The frequency of *Dip-A*¹⁰⁰ varied inversely with longitude i.e. as the longitude increased and the environment changed from more temperate clines in the west to more tropical clines in the east the frequency of *Dip-A*¹⁰⁰ decreased ($0,01 < P < 0,05$; $r_s = -0,494$) Fig. 7.1.
2. *Idh-NADP*¹⁰⁰ varied inversely in frequency with extreme minimum temperature. As the temperature increased, so the frequency of this allele decreased. ($0,005 < P < 0,01$; $r_s = -0,714$). So this allele was present in greater proportions when extremely low temperatures were attained, Fig. 7.2.
3. *Adh*¹⁰⁰ showed no latitudinal or longitudinal cline. Further extremes of temperature did not appear to affect the frequency of this allele although there was a weak correlation with extreme minimum temperature ($0,1 < P < 0,2$; $r_s = 0,425$), as the minimum temperature increased so the frequency of *Adh*¹⁰⁰ increased. However, there was a highly significant correlation between the frequency of *Adh*¹⁰⁰ and recent rainfall ($0,005 < P < 0,01$; $r_s = 0,680$) Fig. 7.3.

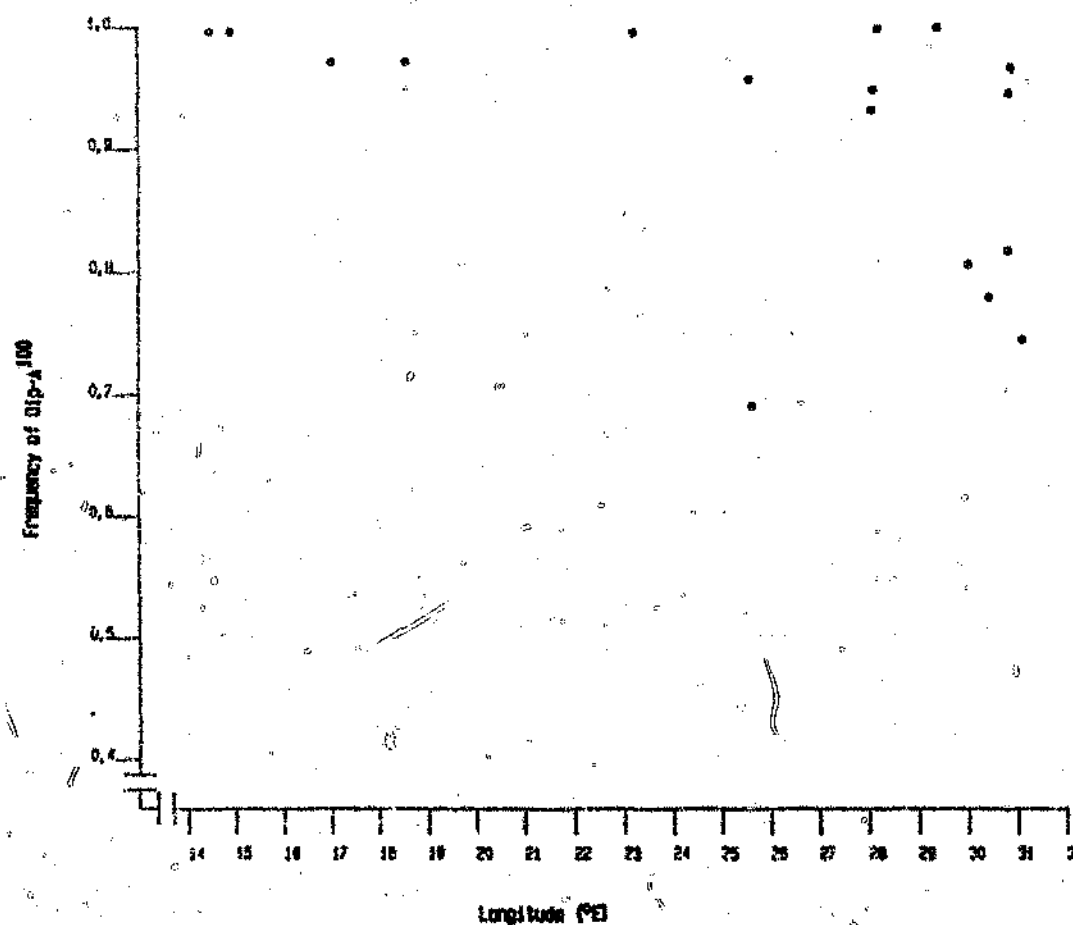


Fig. 7.1 Variation in the frequency of Dip-A100 with Longitude.

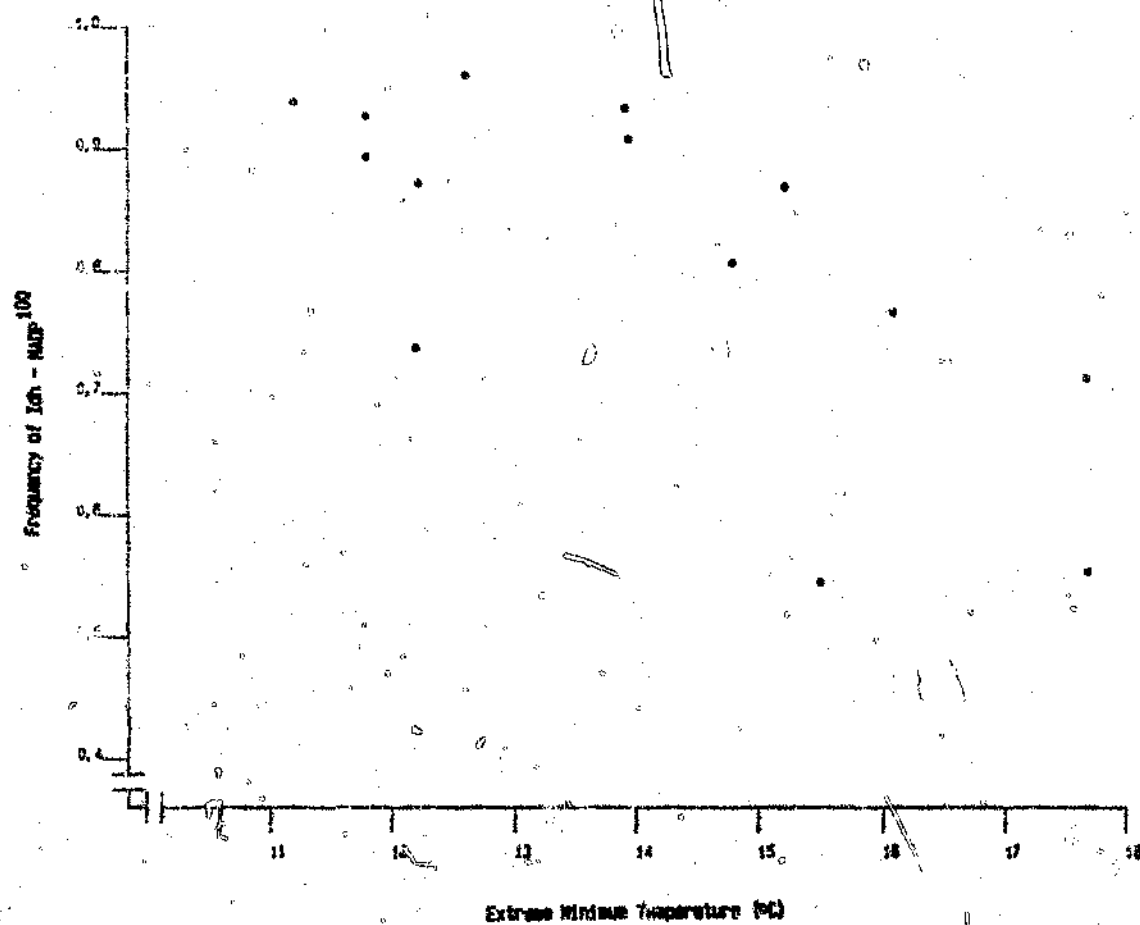


Fig: 7.2

Variation in the frequency of
Idh-NADP100 with extreme minimum
 temperature.

7.4 Discussion

The assumption that a latitudinal clinal pattern paralleled on different continents, is strong evidence of adaptive responses to climatic changes is not always realistic. The facts are persuasive when the continents under scrutiny have continuously varying north to south environmental gradients which are in turn, mirrored between them. However, this may often not be the case. In the Southern African subcontinent, where the 17 collections of this study were made, there is no simple latitudinal climatic gradient. This subcontinental region consists of three separate and relatively homogeneous areas: a western arid area, a central dry plateau and tropical and subtropical eastern lowlands. On the north west coast lies the Namib desert, unique in the world's deserts in that it is located next to the Benguela current and its associated cold water upwelling system. The latter is influenced by the anticyclonic winds and is directly responsible for the maintenance of cool water near the coast, the fog, the high humidity and the steep climatic gradient from the coast to the eastern escarpment (Seely 1987). The province of Natal on the eastern coast, washed by the warm waters of the Indian ocean, has a subtropical climate. Between the west and east coast, collections were made in the temperate climes of the Cape and the Transvaal and in the more tropical Zimbabwe. Further, there is a great deal of variation in the altitude of which collections were made - ranging from 0 to 2000 meters above sea level. In view of this geographic heterogeneity it was not surprising that no latitudinal clines were found. The three significant correlations obtained in this study are discussed below.

An interesting aspect is that Fox (1971) noted that, in *Drosophila*, *Idh*-NADP has the same developmental profile as many other *Drosophila* enzymes in that, enzyme activity was found to increase throughout larval development, to decrease during the pupal period and then to increase again in anticipation of adult emergence. Amongst these similar enzymes are *Adh*, *Gpdh* and *G6pd* - all of which have exhibited some correlation with temperature and/or latitude (e.g. Anderson, 1981; Singh *et al.* 1982). Environmental conditions which are better suited to different stages (such as egg, larval, pupal or adult stage) in the development of the fly may be instrumental in producing a selective pressure on enzymes which vary in their kinetic properties during developmental time.

7.4.3 Alcohol Dehydrogenase

Unlike many of the previous studies no latitudinal clines were observed in the allele frequency variation of *Adh*. In a study which involved populations from Europe, and tropical Africa as well as four Southern African populations (one from Swaziland and three from South Africa), a latitudinal cline in the *Adh* allele frequency between Europe and equatorial Africa was found, and the authors (David *et al.* 1986) suggested that a symmetrical cline in the southern hemisphere from the equator to South Africa may exist. Although the frequencies in this study were similar to those of David *et al.* (1986) for collections from the same area (frequencies of *Adh*^F in Johannesburg and Cape Town were respectively 0,337 and 0,519 for David *et al.* (1986) and 0,280 and 0,440 in this study), nonetheless, no clear latitudinal cline emerged. (I am assuming that the *Adh*¹⁰⁰ and *Adh*⁰ alleles

in this study correspond to the common *Adh^S* and *Adh^F* alleles that occur worldwide - see section 6.3.1.1). If environmental selection of some sort is responsible for the *Adh* cline then, the lack of such a pattern in Southern Africa is not surprising since the geographic heterogeneity of the areas from which collections were taken in this study precludes any kind of latitudinal climatic gradient as previously mentioned.

There was a significant correlation of the "slow" allele (*Adh⁻¹⁰⁰*) with recent rainfall and also a weak correlation with extreme minimum temperature. The temperature correlation indicated a decrease in the frequency of the fast allele with increasing minimum temperature. Now, as pointed out by Pipkin *et al.* (1973), enzyme activity is generally higher in cold acclimated (*viz*) than in warm adapted poikilotherms such as *D. melanogaster*. Since, the fast allele is the more active of the two common alleles (see Section 7.1.1.1) it therefore seems logical that this allele would be selected for in cooler areas.

The correlation observed was not very strong, but this can also be accounted for by the lack of extremely cold temperatures in Southern Africa. (The average minimum temperature in the summer months was not lower than 11°C.) This lack of extreme variation in temperature could also explain the absence of correlation with maximum temperature since the average maximum temperature for the summer months in the collection sites analysed did not exceed 31°C. When increased thermostability of ADHS

for which there are no known metabolic outlets. Consequently flies homozygous for *Adh^F* would be selected against and a subsequent increase in the frequency of the *Adh^S* allele would result.

In conclusion, the highly polymorphic nature of the *Adh* locus is most probably due to a complex interaction of both environmental selective forces such as temperature and alcohol as well as some form of superimposed balancing selection which has been shown to be present in some laboratory and wild populations (see Section 7.1.1.5).

CHAPTER EIGHT

8. Summary and General Conclusions

The genetic differences within and between certain species have been extensively investigated. These investigations have led to the formulation of many theories and to the understanding of many phenomena regarding the mechanisms of change and its concomitant evolutionary consequences. Genetic variation can be studied on many levels. Up to about 25 years ago the study of phenotypic diversity was the only available method of gaining insight into population differences. However, with the advent of molecular techniques geneticists are now able to look more deeply into the genome.

The ultimate source of genetic variation is at the level of DNA sequences. Although ambitious projects such as sequencing the human genome are being undertaken, for the most part DNA sequencing is time consuming and labour intensive and a great deal of knowledge can be gained by working at a less refined level.

Two techniques that have been utilized very successfully to investigate hidden variation at the gene and at the gene product levels are restriction enzyme analysis of DNA and protein electrophoresis. The former has been used to detect restriction fragment length polymorphisms (RFLPs), i.e. polymorphisms in DNA (genomic, mitochondrial or chloroplastic) which occur due to the absence or presence of restriction sites. The latter has been used to detect differences in the electrophoretic mobilities of allozymes. A third area of study which overlaps those previously

mentioned is that which deals with sections of DNA which are able to move about from one location to another within the genome. These segments of DNA are capable of bringing about tremendous changes within the organism and may prove to be the ultimate mutational event. In this study I have examined the variation in Southern African populations of *Drosophila melanogaster* in two different respects, mobile P elements and proteins.

8.1 P elements

8.1.1 GD Sterility and Geographic Distribution

Two hundred and six (206) isofemale lines collected from Namibia, South Africa and Zimbabwe were tested for their ability both to induce and withstand P factor activity. A system of classification was introduced using joint confidence regions which, as well as retaining the system of classifying flies as P, Q or M (Kidwell 1983), also introduced intermediate (P-Q, Q-P, Q-M and M-Q) and indefinite (P?, Q? and M?) categories. When this system was used, only two out of 206 strains were completely unclassifiable.

All collection sites where more than five isofemale lines were established showed a central tendency towards either P, Q, M, P/Q or Q/M type strains. Collections from the western side of the continent in Namibia were of type Q/M. Collections from Southern Namibia and the Southern Cape were mainly of type M. On the eastern side of the continent in Natal, Q type populations predominated, while further north in the Transvaal, P type

populations started to appear and the northernmost collection site (Harare) in Zimbabwe consisted of 89% P type lines. Statistical tests (Spearman's rank correlation) show that P factor activity and regulatory ability decreased steadily and highly significantly (respectively) in a southerly direction. This clinal pattern, together with other clinal patterns in P element distribution which have been described elsewhere in the world, can be explained by invoking the stepping stone model of Anxolabéhère *et al.* (1986) and the invasion theory of Kidwell (1975, 1983).

8.1.2 *an^W* Mutability

Seventy eight (78) Southern African isofemale lines were assayed for P factor activity using the *an^W* mutability test. The statistical correlation between *an^W* mutability and cross A GD sterility was examined using Kendall's τ . A highly significant correlation was obtained between *an^W* mutability rates and frequencies of induced GD sterility, thus confirming the common origin of these two effects.

8.1.3 Molecular Analysis

The 2.4 kb *Aac* I fragment of the plasmid p π 25.1 was used to probe for P elements in 31 wild South African strains of *D. melanogaster*. Two control strains were routinely employed, Canton-S as an example of M type strain and π_2 as an example of a P type strain. The π_2 strain gave a very strong signal on each blot. The Canton-S strain consistently emitted a very

weak signal. This latter signal may have been due to non-specific hybridization or it may have been due to the presence of P elements in this strain. I interpreted it as being the latter in view of the observation of Woodruff *et al.* (1987a) that P elements were present in the Canton-S strain used by these researchers. All the wild strains tested gave a positive signal when probed with the *Acc* I fragment.

Overall, the signal intensity was greater for P strains, less for M strains and intermediate for Q strains. Only three P lines were tested, but all of those tested, gave either a strong or a medium hybridization signal.

8.1.4 Somatic Activity

The strain *mmwh* (constructed by N van Schaik) is homozygous for the *mwh* gene and is a moderately strong P strain. This strain was used to test for somatic P element activity in dysgenic hybrids by crossing males of this strain with females of the M strain *f1r³/Ser*. Wings of the trans-heterozygous *mwh/f1r³* progeny from this cross were scored for mutant clones. All crosses were performed at both 25°C and 29°C and the *mmwh* strain was used as both male and female parent in order to produce dysgenic and nondysgenic progeny. As a further control, the standard *mwh* line (without P elements) was crossed to the *f1r³/Ser* line and wings of the progeny were analysed.

No significant heterogeneity was obtained between the experiments performed at 25°C thus revealing that P activity was probably not taking place somatically at that temperature. At 29°C, no significant heterogeneity was perceived in the male progeny of the various tests.

However, the frequency of mutant clones was significantly higher in females derived from crosses between M strain females and P strain males than in females derived from the reciprocal crosses or from crosses where P elements were entirely absent at 29°C. It therefore seems plausible to suggest that P element activity may be taking place in wing imaginal disc tissue in female dysgenic flies reared at 29°C.

B.2 Allozymes

B.2.1 Allozyme Heterogeneity

Populations collected from different areas were geographically subdivided as naturally as possible into six different regions. A hierarchical population structure was then obtained by further subdividing regions into areas, the areas into districts and the districts into subpopulations. Using this hierarchical population model, allele frequency differences among samples were compared using a nested contingency-table analysis. The G-statistic (Sokal and Rohlf 1981) for six of the eight loci was computed and used to test for significant differences between the various units of subdivision.

There was a significant amount of heterogeneity at the lower levels which precluded pooling in order to make comparisons at higher levels. Consequently an approximate F ratio was obtained by summing the G-statistics over loci, dividing the sum by its associated degrees of freedom and comparing this

standardized measure with the standardized measure at the next lower level (Smouse and Kojima 1972). These tests showed that, although the amount of heterogeneity between districts was not significantly greater than that between collection sites within a city, the amount of heterogeneity among areas was significantly greater than that among districts. There was not, however, a greater degree of heterogeneity in the regions to areas comparison.

The same model of hierarchical population structure was used to estimate gene diversities. The gene diversity analysis showed that the average sample heterozygosity H_S (for all eight loci combined) was 0,18 and this represented 87,75% of the total variation. The remaining 12,25% of the variation was therefore due to population subdivision because of geographic separation. The degree of diversity increased with increasing distance between samples.

In order to estimate gene flow, the conditional average frequencies of alleles (Slatkin 1981) in the 17 samples examined in this study were plotted as a function of the proportion of the populations in which they were found. A comparison of the shape of this curve with those for other species (Slatkin 1981) pointed to a comparatively high level of gene flow for Southern African populations of *D. melanogaster*. Further, two other rough measures of gene flow estimated the migration of *D. melanogaster* across the Southern African subcontinent to range from 1,8 to 6,9 individuals per generation. This high rate of gene flow is theoretic-

cally sufficient to prevent differentiation among populations due to genetic drift (Spieth 1974). The significant degree of heterogeneity observed in the present study is apparently at odds with this high rate of gene flow. However, it is possible that heterogeneity may have been overestimated due to the dynamics of *Drosophila* populations and the sampling techniques employed. Also, it is possible that estimates of gene flow are based on models which do not appropriately reflect the natural history of *Drosophila*.

Finally, I was able to use this study in conjunction with the works of other researchers in this field to obtain information on the origin of Southern African populations of *D. melanogaster*. A comparison of the allele frequencies and the number of different alleles found in this study with that found in other parts of Africa and the world indicated that Southern African populations form a single genetic entity which shows a close affinity to other sub-Saharan African populations.

8.2.2 Clines and Correlations

The Spearman rank correlation statistic was used on the most common allele of each of the five polymorphic loci (*Adh*, *Dip-A*, *Hex-1*, *Hex-2* and *Idh-NADP*) to test for associations with latitude and longitude as well as with extreme minimum and maximum temperature and precipitation. Three results were significant:

The frequency of *Dip-A*¹⁰⁰ varied inversely with longitude;

The frequency of *Idh-NADP*¹⁰⁰ varied inversely with extreme minimum temperature;

The frequency of Adh^{100} increased with increasing rainfall.

The longitudinal variation in $Dip-A^{100}$ is difficult to explain in view of the lack of any west to east environmental gradient. The variation in the frequency of $Idh-NADP^{100}$ with temperature could be accounted for in terms of enzymatic activity. However, the most interesting of the three results is the variation in frequency of Adh^{100} with rainfall. This could be explained by the following (selective) hypothesis: Changes in moisture availability affect the microenvironment of the *Drosophila* eggs and larvae. It is not clear what the direct effect of increased rainfall is on the concentration of alcohols present in ripening and fermenting fruits and vegetables. However it is possible that an increase in, say, the production of secondary alcohols, may result in the more active ADHF allozyme being selected against, due to the accumulation of toxic ketones produced in the fly by the efficient oxidation of these alcohols. Consequently flies homozygous for Adh^S may be selected for under these circumstances. (I am assuming that the Adh^{100} and Adh^0 alleles in this study correspond to the common Adh^S and Adh^F alleles that occur worldwide).

8.3. P elements and Allozymes

In this study I examined genetic variation in Southern African populations of *D. melanogaster* in both a Mendelian and a non-Mendelian system. There are various ways in which the two systems could be interrelated. Firstly, if any of the enzymes examined were target sites for the P elements then one might expect to

perceive an altered gene product. This could manifest itself in the appearance of a rare allele at places which had higher frequencies of P elements and not elsewhere. Consequently I looked for the exclusive appearance or the higher frequency of rare alleles in places like Harare, Thohoyandou, Nelspruit and Johannesburg where the incidence of P elements was relatively high, but found no evidence for this. In this study, only eight enzymes were investigated and it is likely that none of them were "hot-spots" for P element insertions. The *Adh* locus apparently does not attract P elements. Engels (1983) mentions that in the *Adh* locus "no P insertions were detected in a screen of more than ten million gametes from dysgenic parents". Although in this connection it has been suggested by Mukai *et al.* (1984) that transposon-like elements — possibly P elements — are related to the induction of changes in the specific activities in alcohol dehydrogenase. These mutational events which control this activity are thought to occur in regions different from the alcohol dehydrogenase locus itself — mainly in the non-coding DNA region. Further defective P elements have been shown to be present in the *G6pd* locus (Eanes *et al.* 1988; Itoh *et al.* 1988).

A second area where one might look for connections between the two systems is in geographic distributions. For example, a latitudinal pattern in an allele frequency could be paralleled by a clinal distribution of P elements. Although P element distributions in this study correlated well with both latitude and longitude, the only such parallel in the enzymes was for

*Dip-A*¹⁰⁰ which exhibited a longitudinal variation. However, an examination of the allele distribution of *Dip-A* reveals no obvious reason why this should be so and the two patterns are probably coincidental. Many other researchers have detected clines (mainly latitudinal) in allele frequencies (see Chapter 7). In Australia, Boussy (1987) noted that the P-M cline found there was the third such latitudinal cline, the other two were a latitudinal cline in the frequencies of the common cosmopolitan inversions (Knibb *et al.* 1981) and many latitudinal clines in allozyme frequencies for several enzymes particularly *Adh*^S (see Chapter 7 for details). Despite the commonness of these clines, Boussy (1987) concluded that the P-M hybrid dysgenesis, cosmopolitan inversion frequencies and *Adh*^S frequencies seemed to be independent systems.

In this study the latitudinal and longitudinal clinal distribution of P elements can be accounted for by the high gene flow observed in Southern African populations of *D. melanogaster* (Chapter 6) in conjunction with the rapidity with which P elements are able to spread through populations (see Kidwell *et al.* (1981, 1988); Kiyasu and Kidwell (1984); Daniels *et al.* (1987a); and Good *et al.* (1989) for examples of the rapidity of this spread in both laboratory populations and transformed lines. Theoretical simulation studies also indicate that, provided the efficiency of transposition is high, transposable element contamination in a population of randomly mating sexual diploid organisms will take place rapidly (Hickey 1982).

Thirdly, the presence of P elements at a collection site might influence the genetic variability in that site in the form of polymorphic loci, heterozygosity, or both. A statistical analysis using the Spearman rank correlation test on cross A and cross A* GD sterility and the percentage of polymorphic loci on both the 95% and 99% criteria, at each collection site revealed no correlation between the two.

Using the proportion of polymorphic loci to measure genetic variability is not precise (highly polymorphic loci and those with low levels of polymorphism are grouped into the same class), and is influenced by sample size (a small sample may not detect the presence of a polymorphism) as well as sampling methods. A better measure is that of average heterozygosity. In this study eight loci were examined and this number of loci is statistically too small to be able to detect differences in average heterozygosities between samples unless these differences are very large (Archie 1985).

In a similar investigation to this one about 120 strains from the Languedoc-Roussillon area of France were tested for P-M characteristics as well as for enzymatic polymorphism (Anxolabéhère et al. 1988a). In that study an attempt was made to compare levels of heterozygosity within a three zone system which was found to partition statistically the P activity of the French strains. The authors found no significant difference between the three areas for either individual loci or combined

loci heterozygosity. They further found no statistical correspondence between the gradient observed in the P-M system and the allele frequencies of the enzymes examined. It therefore appears from this study and those of Anxolabéhère *et al.* (1988a) and Boussy (1987) that the two systems (P elements and enzymes) are separate entities, having very little interaction between them.

The spread of P elements can be likened to a ripple-like process which radiates outward rapidly from a central infectious source. The size and structure of the infecting P elements are determined by the transpositional activities which took place previously within the carrier genomes. On the other hand, the geographic patterns of allelic variants among different populations are influenced by such factors as environmental selection, gene flow, mutation and stochastic effects.

APPENDIX A

Estimated GD sterilities (P_A and P_{A^*}) obtained in crosses A and A^* together with 95% confidence regions ($P_A \pm S_A$, $P_{A^*} \pm S_{A^*}$).

$$S_A = 2,235 (P_A (1-P_A)/n_A)^{1/2}; S_{A^*} = 2,235 (P_{A^*}(1-P_{A^*})/n_{A^*})^{1/2}$$

n_A and n_{A^*} are the numbers of ovaries examined in crosses A and A^* respectively.

Cross A : $sn^W(M) \text{ } \varnothing \times U \text{ } \sigma$

Cross A^* : $U \text{ } \varnothing \times \pi_2 \text{ } \sigma$

U is the unknown strain; $sn^W(M)$ and π_2 are described in section 2.2.3.

The P-Q-M classification system is described in Table 2.2

Appendix A

Location	Strain	Cross A				Cross A*				P-Q-M Classi- fication
		PA-SA	PA	PA+SA	NA	PA*-SA*	PA*	PA*+SA*	NA*	
1. Swakopmund	Swak 1	0	0	0	150	0	0	0	150	Q
	Swak 3	0	0	0	150	0	0	0	150	Q
	Swak 4	0	0	0	150	0,662	0,731	0,800	208	M
	Swak 5	0	0	0	150	0	0	0	104	Q
	Swak 7	0	0	0	150	0	0	0	96	Q
	Swak 8	0	0	0	150	0,019	0,062	0,105	160	Q-M
	Swak 9	0	0	0	150	0	0	0	106	Q
	Swak 10	0	0	0	150	0,305	0,390	0,475	164	M
	Swak 12	0	0	0	150	0	0	0	64	Q
	Swak 14	0	0	0	150	0	0	0	124	Q
2. Gobabeb	Gob 1	0	0,012	0,031	178	0,004	0,037	0,070	160	Q
	Gob 2	0	0,003	0,019	178	0	0,027	0,057	148	Q
	Gob 4	0	0	0	150	0,014	0,061	0,108	132	Q-M
	Gob 5	0	0	0	150	0	0,015	0,038	136	Q
	Gob 7	0	0	0	150	0	0,011	0,029	176	Q
	Gob 10	0	0	0	150	0,231	0,306	0,384	182	M
	Gob 11	0	0	0	150	0,088	0,141	0,200	198	M-Q
	Gob 13	0	0,027	0,057	150	0,170	0,234	0,297	222	M
	Gob 14	0,030	0,079	0,178	152	0,004	0,037	0,070	160	Q-P
	Gob 15	0,004	0,037	0,070	162	0	0,033	0,069	122	Q
	Gob 16	0	0,012	0,031	162	0	0	0	150	Q
	Gob 17	0	0,014	0,036	140	0	0,026	0,055	154	Q
	Gob 19	0	0	0	150	0,493	0,574	0,655	188	M
	Gob 20	0	0	0	150	0,004	0,040	0,076	150	Q
	Gob 21	0	0	0	150	0,351	0,444	0,536	144	M
	Gob 22	0	0	0	150	0,093	0,155	0,217	168	M-Q
	Gob 24	0	0	0	159	0,012	0,053	0,094	152	Q
	Gob 25	0	0	0	150	0,107	0,173	0,239	162	M
3. At Ais	Ais 1	0	0,013	0,033	160	0,503	0,583	0,662	192	M
	Ais 3	0	0	0	200	0,141	0,220	0,299	136	M
	Ais 4	0	0	0	164	1,000	1,000	1,000	164	M
	Ais 5	0	0	0	156	0,132	0,198	0,254	182	M
	Ais 6	0	0	0	160	0	0	0	166	Q
	Ais 7	0	0,015	0,038	136	0,213	0,295	0,377	156	M
	Ais 8	0	0	0	150	0,090	0,151	0,212	172	M-Q
	Ais 9	0	0	0	150	0,490	0,581	0,672	148	M
	Ais 12	0	0	0	150	0,912	0,948	0,984	192	M
	Ais 14	0,047	0,101	0,155	158	0,858	0,915	0,972	118	?
	Ais 16	0	0	0	106	0,180	0,265	0,350	136	M
	Ais 17	0	0,064	0,133	62	0,234	0,348	0,446	100	M?
	Ais 19	0	0,013	0,034	150	0,347	0,425	0,503	200	M
	Ais 20	0	0,014	0,036	146	0,497	0,581	0,665	172	M
	Ais 21	0	0	0	150	0	0	0	24	Q
4. Cape Town	CT 1	0	0	0	126	0,127	0,197	0,267	162	M
	CT 2	0	0	0	84	0,403	0,488	0,573	172	M
	CT 3	0	0	0	84	0,635	0,723	0,811	130	M
	CT 4	0	0	0	56	0,289	0,373	0,457	166	M
	CT 5	0	0	0	86	0,370	0,454	0,538	176	M
	CT 6	0	0	0	90	0	0	0	150	Q
	CT 7	0	0	0	144	0,265	0,351	0,437	154	M
	CT 9	0	0	0	150	0,264	0,345	0,425	174	M
	CT 10	0	0	0	150	0,357	0,447	0,537	152	M
	CT 11	0	0	0	150	0,425	0,512	0,599	164	M
	CT 12	0	0	0	150	0	0,025	0,053	160	Q
	CT 13	0	0	0	150	0,004	0,038	0,072	158	Q
	CT 15	0	0	0	142	0,461	0,541	0,621	196	M
	CT 17	0	0	0	150	0,039	0,091	0,143	154	Q-M

Location	Strain	Cross A				Cross A*				P-Q-M Classi- fication
		PA-SA	PA	PA+SA	NA	PA*-SA*	PA*	PA+SA*	NA*	
5. Gordons Bay	GB 1	0	0,011	0,028	182	0,187	0,255	0,323	204	M
	GB 2	0	0	0	180	0,136	0,260	0,323	238	M
	GB 3	0	0	0	180	0,575	0,650	0,725	200	M
	GB 4	0	0	0	180	0,129	0,200	0,271	160	M
6. Plettenberg Bay	Plet 6	0	0,033	0,069	120	0,406	0,485	0,564	200	M
	Plet 8	0	0	0	180	0	0	0	180	Q
	Plet 10	0,205	0,283	0,361	166	0	0,010	0,026	200	P
	Plet 12	0,035	0,085	0,134	164	0,253	0,337	0,424	160	M?
7. Port Elizabeth (Summerstrand)	SS 1	0	0	0	86	0	0	0	126	Q
	SS 3	0,005	0,049	0,093	122	0,082	0,179	0,276	78	M-Q
	SS 4	0	0,029	0,074	68	0,620	0,720	0,820	100	M
	SS 5	0	0	0	86	0,621	0,727	0,833	88	M
	SS 6	0	0	0	92	0,859	0,925	0,991	80	M
	SS 10	0	0	0	114	0,743	0,842	0,936	76	M
	SS 12	0	0	0	64	0,776	0,845	0,914	136	M
	SS 13	0	0	0	66	0,364	0,454	0,544	152	M
	SS 14	0	0	0	62	0,212	0,319	0,426	94	M
	SS 15	0	0,021	0,054	96	0,498	0,614	0,730	88	M
	SS 16	0	0	0	74	0,724	0,807	0,890	124	M
	SS 17	0	0	0	110	0,504	0,625	0,746	80	M
	SS 18	0	0	0	120	0	0,018	0,047	108	Q
	SS 19	0	0	0	80	0,693	0,795	0,897	78	M
	SS 20	0	0	0	76	0,253	0,360	0,467	100	M
	SS 21	0	0	0	60	0,525	0,610	0,695	164	M
	SS 23	0	0	0	112	0,258	0,367	0,476	98	M
	SS 24	0	0	0	72	0,701	0,776	0,851	156	M
	SS 25	0	0	0	70	0,669	0,759	0,849	112	M
8. Port Elizabeth (Walmer)	Wal 1	0	0	0	150	0,943	0,973	1,000	150	M
	Wal 2	0	0,025	0,053	158	0,315	0,405	0,495	148	M
	Wal 3	0	0	0	150	1,000	1,000	1,000	150	M
	Wal 4	0	0	0	150	0,219	0,300	0,381	160	M
	Wal 6	0	0	0	150	0,853	0,904	0,955	166	M
	Wal 7	0	0	0	96	0,047	0,103	0,159	146	M-Q
9. Maclear	Mac 1	0	0,010	0,026	200	0,077	0,130	0,183	200	M-Q
	Mac 3	0	0	0	200	0,119	0,180	0,241	200	M
	Mac 4	0	0	0	204	0	0	0	200	Q
	Mac 5	0	0	0	216	0	0	0	200	Q
10. Durban (Berea)	Ber 4	0,002	0,067	0,132	74	0	0,017	0,044	118	Q-P
	Ber 5	0,111	0,233	0,355	60	0	0	0	150	P
	Ber 6	0	0	0	118	0,278	0,388	0,498	98	M
	Ber 8	0,109	0,177	0,245	158	0	0,026	0,067	76	P
	Ber 9	0,006	0,054	0,102	110	0	0	0	132	Q-P
	Ber 10	0,009	0,075	0,141	80	0	0	0	110	Q-P
	Ber 12	0	0	0	54	0	0	0	150	Q
	Ber 14	0,034	0,088	0,142	136	0,009	0,073	0,137	82	Q?
	Ber 15	0,045	0,104	0,163	134	0	0	0	8	P-Q
	Ber 18	0	0	0	128	0	0	0	104	Q
	Ber 19	0,056	0,119	0,181	134	0	0	0	108	P-Q
	Ber 20	0,073	0,131	0,189	168	0	0	0	104	P-Q
	Ber 22	0,047	0,104	0,161	144	0	0	0	74	P-Q
	Ber 23	0,007	0,059	0,111	102	0	0	0	60	Q-P
	Ber 24	0,047	0,118	0,189	102	0	0	0	134	P-Q
	Ber 25	0	0	0	150	0	0,023	0,059	88	Q

Appendix A (continued p.3)

Location	Strain	Cross A				Cross A*				P-Q-M Classification
		PA-SA	PA	PA+SA	na	PA*-SA*	PA*	PA*+SA*	na*	
11. Durban (Addington Beach)	AB 2	0	0	0	150	0	0	0	140	Q
	AB 3	0	0,013	0,034	143	0	0	0	150	Q
	AB 5	0	0	0	150	0	0	0	120	Q
	AB 6	0	0	0	106	0	0	0	140	Q
	AB 7	0	0	0	150	0	0	0	134	Q
	AB 9	0	0	0	150	0	0	0	70	Q
	AB 10	0	0	0	144	0	0	0	120	Q
	AB 11	0	0	0	150	0	0	0	130	Q
	AB 12	0	0	0	150	0	0	0	150	Q
	AB 13	0	0	0	150	0	0	0	114	Q
	AB 15	0	0	0	124	0	0	0	100	Q
	AB 16	0,036	0,078	0,120	204	0	0	0	64	Q-P
	AB 17	0	0	0	150	0	0	0	150	Q
	AB 18	0	0	0	150	0	0	0	150	Q
	AB 20	0	0	0	150	0	0	0	150	Q
	AB 23	0	0,026	0,056	154	0	0	0	86	Q
12. Johannesburg (Northern suburbs) (1988)	JHB 1	0	0	0	150	0,018	0,125	0,232	48	M-Q
	JHB 4	0	0	0	150	0	0	0	150	Q
	JHB 5	0,111	0,169	0,234	166	0	0,024	0,051	164	P
	JHB 7	0,016	0,059	0,102	152	0	0	0	150	Q-P
	JHB 12	0	0	0	150	0	0	0	150	Q
	JHB 15	0	0	0	150	0,005	0,043	0,081	140	Q
	JHB 18	0	0	0	150	0	0	0	150	Q
	JHB 20	0	0	0	176	0	0	0	150	Q
13. Johannesburg (Northern suburbs) (1984/1987)	Rut 1	0,237	0,310	0,343	200	0,015	0,066	0,117	120	P?
	Rut 2	0,059	0,108	0,157	204	0,073	0,137	0,201	146	?
	Rut 3	0,111	0,170	0,229	200	0,030	0,070	0,110	200	P?
	Rut 4	0,289	0,365	0,441	200	0,056	0,083	0,139	120	P?
	Br	0,001	0,038	0,075	130	0,063	0,140	0,217	100	M-Q
14. Johannesburg (Southern suburbs)	Den 1	0	0,033	0,069	120	0,005	0,050	0,094	120	Q
	Den 2	0	0,008	0,026	120	0	0	0	120	Q
	Den 3	0	0	0	120	0,039	0,100	0,161	120	Q-M
	Den 4	0	0,008	0,026	120	0,039	0,100	0,161	120	Q-M
15. Pretoria	Pta 1	0	0	0	120	0	0,016	0,042	120	Q
	Pta 2	0	0	0	120	0	0,016	0,042	120	Q
	Pta 3	0	0	0	120	0	0,008	0,026	120	Q
	Pta 6	0	0,016	0,042	120	0,084	0,158	0,232	120	M-Q
16. Hammanskraal	H'K 1	0	0	0	140	0	0	0	180	Q
	H'K 2	0,027	0,083	0,139	120	0	0	0	220	Q-P
	H'K 3	0,001	0,050	0,094	120	0	0	0	140	Q
	H'K 4	0	0,016	0,042	120	0	0	0	180	Q
17. Nelspruit	Nel 1	0,018	0,052	0,086	212	0,015	0,065	0,115	122	Q-M
	Nel 2	0,051	0,103	0,154	174	0	0	0	56	P-Q
	Nel 4	0,089	0,145	0,191	228	0	0	0	128	P-Q
	Nel 5	0	0,018	0,038	220	0	0,009	0,023	214	Q
	Nel 8	0,003	0,026	0,049	234	0	0	0	80	Q
	Nel 9	0	0	0	214	0	0	0	54	Q
	Nel 10	0,121	0,176	0,231	238	0	0	0	206	P
	Nel 11	0	0,010	0,026	202	0	0,004	0,013	232	Q
	Nel 12	0,284	0,345	0,406	304	0	0	0	146	P
	Nel 13	0,162	0,159	0,215	208	0	0	0	136	P
	Nel 15	0,006	0,029	0,052	272	0	0	0	83	Q
	Nel 16	0	0,078	0,020	262	0,005	0,029	0,053	242	Q
	Nel 17	0,019	0,045	0,071	310	0	0	0	230	Q
	Nel 18	0	0	0	268	0,237	0,315	0,393	178	M
	Nel 19	0,063	0,103	0,143	290	0	0	0	108	P-Q
	Nel 20	0,466	0,528	0,590	322	0	0	0	298	P

Appendix A (continued p.4)

Location	Strain	Cross A				Cross A*				P-Q-M Classi- fication
		PA-SA	PA	PA+SA	NA	PA*-SA*	PA*	PA**SA*	NA*	
18. Pietersburg	P'B 1	0.364	0.432	0.500	264	0	0	0	200	P
	P'B 3	0.030	0.063	0.096	270	0	0	0	200	P
	P'B 4	0.150	0.203	0.256	286	0	0	0	180	P
	P'B 5	0.046	0.086	0.126	244	0	0	0	200	Q-P
19. Agatha	Ag 1	0.022	0.058	0.094	208	0	0.019	0.047	206	Q
	Ag 2	0	0.009	0.023	212	0	0	0	200	Q
	Ag 3	0	0.018	0.030	224	0	0	0	200	Q
	Ag 5	0	0.009	0.023	216	0	0	0	200	Q
20. Thohoyandou	Tho 1	0.298	0.370	0.442	224	0	0	0	156	P
	Tho 2	0.942	0.097	0.152	144	0	0	0	150	Q-P
	Tho 3	0.051	0.103	0.154	174	0	0	0	64	P-Q
	Tho 5	0.031	0.076	0.121	170	0	0	0	124	Q-P
	Tho 6	0.146	0.213	0.290	188	0	0	0	150	P
	Tho 9	0	0	0	150	0	0	0	150	Q
	Tho 10	0.101	0.161	0.221	186	0	0	0	68	P
	Tho 11	0.012	0.054	0.095	148	0	0	0	63	Q
	Tho 17	0	0.006	0.020	152	0	0	0	54	Q
	Tho 19	0	0.027	0.057	150	0	0	0	68	Q
	Tho 20	0.032	0.076	0.120	184	0	0	0	104	Q-P
	Tho 21	0	0.011	0.028	178	0	0	0	100	Q
21. Harare	Har 2	0.181	0.253	0.325	182	0.019	0.061	0.103	164	P?
	Har 3	0.099	0.161	0.223	174	0	0	0	138	P-Q
	Har 5	0.197	0.276	0.362	134	0	0	0	150	P
	Har 6	0.27	0.362	0.453	138	0	0	0	150	P
	Har 8	0.087	0.150	0.213	160	0	0	0	114	P-Q
	Har 9	0.119	0.207	0.295	106	0	0	0	150	P
	Har 10	0.176	0.253	0.330	158	0	0	0	102	P
	Har 11	0.228	0.302	0.376	190	0	0	0	74	P
	Har 12	0.097	0.165	0.213	160	0	0	0	150	P-Q
	Har 13	0.169	0.244	0.319	164	0	0	0	126	P
	Har 14	0.221	0.295	0.369	190	0	0	0	150	P
	Har 15	0.209	0.283	0.357	184	0	0	0	150	P
	Har 16	0.056	0.111	0.166	162	0	0	0	150	P-Q
	Har 18	0.143	0.214	0.285	168	0	0	0	150	P
	Har 19	0	0.014	0.036	138	0	0	0	144	Q
	Har 20	0.146	0.210	0.274	200	0	0	0	150	P
	Har 22	0.011	0.050	0.088	160	0	0	0	150	Q
	Har 23	0.216	0.294	0.372	170	0	0	0	144	P
	Har 25	0.063	0.119	0.175	168	0	0	0	150	P-Q
Controls										
enW(M)		0	0	0	70	1.000	1.000	1.000	100	M
T ₂		1.000	1.000	1.000	100	0	0	0	140	P

APPENDIX B

Percentage GD sterilities obtained in crosses B and B* and in intra/train crosses.

Cross B : $U \times \pi^W(M)$

Cross B* : $\pi_2 \times U$

U is unknown strain; $\pi^W(M)$ and π_2 are described in Section 2.2.3.

Appendix B

Part 1 - GD sterility from crosses B and B*

Location	Strain	Cross B		Cross B*		P-Q-M classi- fication
		n ¹	%GD sterility	n ¹	%GD sterility	
Gordons Bay	GB 1	140	0	140	0	M
	GB 2	174	13,8	180	0	M
	GB 3	200	6,0	160	0	M
	GB 4	160	0	160	0	M
Plettenberg Bay	Plet 6	128	3,1	144	3,5	M
	Plet 8	160	0	120	0	Q
	Plet 10	180	0	180	0	P
	Plet 12	180	0	180	0	M?
Maclear	Mac 1	180	3,3	180	0	M-Q
	Mac 3	200	2,5	180	0	M
	Mac 4	200	0	200	0	Q
	Mac 5	200	0	200	0	Q
Johannesburg (Northern suburbs) (1987/1988)	Rut 1	180	1,1	120	3,3	P?
	Rut 2	180	0	190	7,9	?
	Rut 3	180	0	180	0,5	P?
	Rut 4	180	0	180	0	P?
	Br	100	0	100	0	M-Q
Johannesburg (Southern suburbs)	Den 1	122	9,0	120	2,5	Q
	Den 2	120	0	120	0	Q
	Den 3	120	1,6	120	0	Q-M
	Den 4	120	0	120	0	Q-M
Pretoria	Pta 1	120	2,5	120	0	Q
	Pta 2	120	0	120	1,6	Q
	Pta 3	120	0	140	0	Q
	Pta 6	158	1,9	120	0,8	M-Q
Hamanskraal	H'K 1	120	1,6	120	0	Q
	H'K 2	120	0	120	0	Q-P
	H'K 3	120	0	120	0	Q
	H'K 4	120	0	120	0	Q
Pietersburg	P'B 1	200	0	206	0	P
	P'B 3	200	0	160	0	Q
	P'B 4	180	0	200	0	P
	P'B 5	200	0	180	0	Q-P
Agatha	Ag 1	200	0	200	0	Q
	Ag 2	200	0	200	0	Q
	Ag 3	200	0	200	0	Q
	Ag 5	200	0	200	0	Q

¹n is the number of ovaries examined.

Appendix B Part 2 Intrastrain GD sterility

Location	Strain	Intrastrain		P-Q-M Classi- fication
		n ¹	% GD sterility	
Swakopmund	Swak 1	70	0	Q
	Swak 3	70	0	Q
	Swak 4	70	0	M
	Swak 5	64	0	Q
	Swak 7	70	0	Q
	Swak 8	70	0	Q-M
	Swak 9	70	0	Q
	Swak 10	70	0	M
	Swak 12	58	5,1	Q
	Swak 14	70	0	Q
Gobabeb	Gob 1	66	3,0	Q
	Gob 2	50	0	Q
	Gob 4	50	0	Q-M
	Gob 5	50	0	Q
	Gob 7	68	5,9	Q
	Gob 10	94	8,5	M
	Gob 11	80	0	M-Q
	Gob 13	60	3,3	M
	Gob 14	60	0	Q-P
	Gob 15	70	0	Q
	Gob 16	70	0	Q
	Gob 17	50	0	Q
	Gob 19	60	0	M
	Gob 20	70	0	Q
	Gob 21	60	3,3	M
	Gob 22	50	0	M-Q
	Gob 24	86	0	Q
	Gob 25	70	0	M
Ai Ais	Ais 6	100	0	Q
	Ais 7	84	11,9	M
	Ais 8	124	6,4	M-Q
	Ais 9	60	0	M
	Ais 12	50	0	M
	Ais 14	82	14,6	?
	Ais 16	50	8,0	M
	Ais 27	54	0	M?
Cape Town	CT 1	40	0	M
	CT 9	50	0	M
	CT 10	50	0	M
	CT 11	70	0	M
	CT 12	50	0	Q
	CT 13	44	0	Q
	CT 15	70	0	M
	CT 17	60	0	Q-M

¹n is the number of ovaries examined

Appendix B Part 2 (continued p.2)

Location	Strain	Intrastrain		P-Q-M Classi- fication
		n ¹	% GD sterility	
Port Elizabeth (Summerstrand)	SS 1	70	0	Q
	SS 3	66	0	M-Q
	SS 4	78	5,1	M
	SS 6	84	0	M
	SS 10	74	2,7	M
	SS 12	86	0	M
	SS 13	74	0	M
	SS 14	52	0	M
	SS 15	62	0	M
	SS 16	90	0	M
	SS 17	82	0	M
	SS 18	100	0	Q
	SS 19	64	0	M
	SS 20	64	0	M
	SS 21	100	0	M
	SS 23	70	0	M
	SS 24	60	0	M
	SS 25	64	0	M
Durban (Berea)	Ber 4	108	6,5	Q-P
	Ber 5	100	0	P
	Ber 6	128	21,9	M
	Ber 8	96	4,2	P
	Ber 9	90	0	Q-P
	Ber 10	100	0	Q-P
	Ber 12	100	0	Q
	Ber 14	80	0	Q?
	Ber 15	96	4,2	P-Q
	Ber 18	100	2,0	Q
	Ber 19	100	2,0	P-Q
	Ber 20	100	0	P-Q
	Ber 22	80	0	P-Q
	Ber 23	100	0	Q-P
	Ber 24	100	0	P-Q
	Ber 25	100	0	Q
Durban (Addington Beach)	AB 2	70	0	Q
	AB 3	40	0	Q
	AB 5	50	0	Q
	AB 7	100	0	Q
	AB 9	50	0	Q
	AB 10	50	0	Q
	AB 11	70	0	Q
	AB 12	50	0	Q
	AB 13	50	0	Q
	AB 20	40	0	Q
	AB 23	50	0	Q

¹n is the number of ovaries examined

Appendix B

Part 2 (continued p. 3)

Location	Strain	Intrastrain		P-Q-M Classi- fication
		n ¹	% GD sterility	
Johannesburg (Northern suburbs) (1988)	JHB 12	50	0	Q
	JHB 15	50	0	Q
	JHB 18	50	0	Q
	JHB 20	50	0	Q
Nelspruit	Nel 1	100	0	Q-M
	Nel 2	100	0	P-Q
	Nel 4	100	1.0	P-Q
	Nel 5	100	1.0	Q
	Nel 8	100	0	Q
	Nel 9	100	0	Q
	Nel 10	100	0	P
	Nel 11	100	0	Q
	Nel 12	100	0	P
	Nel 13	100	0	P
	Nel 15	100	0	Q
	Nel 16	100	0	Q
	Nel 17	100	0	Q
	Nel 18	100	2.0	M
	Nel 19	100	0	P-Q
	Nel 20	100	0	P
Thohoyandou	Tho 1	20	0	P
	Tho 2	40	0	Q-P
	Tho 3	44	0	P-Q
	Tho 6	50	0	P
	Tho 9	50	0	Q
	Tho 20	50	0	Q-P
Harare	Har 2	50	0	P?
	Har 3	50	0	P-Q
	Har 5	50	0	P
	Har 6	80	0	P
	Har 8	90	0	P-Q
	Har 9	80	0	P
	Har 10	50	0	P
	Har 11	30	0	P
	Har 12	80	0	P-Q
	Har 13	50	0	P
	Har 14	42	0	P
	Har 15	70	0	P
	Har 16	60	0	P-Q
	Har 18	50	0	P
	Har 19	42	0	Q
	Har 20	50	0	P
	Har 22	60	0	Q
	Har 23	50	0	P
	Har 25	34	0	P-Q

¹n is the number of ovaries examined

APPENDIX C

The sn^W mutation rate of 78 wild Southern African strains.
Crosses were performed as described in Section 3.2.

Line	Number of males				sn^W mutation rate	GD sterility Cross A
	sn^W	sn^+	sn^e	Total		
Gob 5	295	1	1	297	0,007	0
Gob 10	244	0	3	247	0,012	0
Gob 15	192	3	2	197	0,025	0,037
Gob 20	306	2	2	310	0,013	0
Gob 25	204	0	0	204	0	0
Ais 5	453	0	2	455	0,004	0
CT 1	187	0	0	187	0	0
CT 2	265	0	0	265	0	0
CT 3	241	0	0	241	0	0
GB 1	631	0	0	631	0	0,011
GB 2	782	0	0	782	0	0
GB 3	762	0	0	762	0	0
GB 4	806	0	0	806	0	0
Plet 6	666	2	2	670	0,006	0,033
Plet 8	483	9	7	499	0,032	0
Plet 10	443	37	33	513	0,136	0,283
Plet 12	526	1	0	527	0,002	0,085
SS 5	476	0	0	476	0	0
SS 10	311	0	0	311	0	0
SS 15	378	0	0	378	0	0,021
SS 20	365	0	0	365	0	0
SS 25	419	0	1	420	0,002	0
Wal 1	473	0	0	473	0	0
Wal 3	298	0	1	299	0,003	0
Wal 4	271	1	2	274	0,011	0
Mac 1	930	0	0	930	0	0,010
Mac 3	466	2	2	470	0,008	0
Mac 4	477	16	12	505	0,055	0
Mac 5	555	9	5	569	0,025	0
Ber 5	223	14	10	247	0,097	0,233
Ber 10	534	14	6	554	0,036	0,075
Ber 15	365	16	7	378	0,061	0,104
Ber 20	145	0	1	146	0,007	0,133
Ber 25	273	3	1	277	0,014	0
AB 5	453	6	5	464	0,024	0
AB 10	387	6	5	399	0,030	0
AB 15	529	2	3	534	0,009	0
AB 20	339	5	4	348	0,026	0
JHB 5	220	9	4	233	0,056	0,169
JHB 15	189	0	1	190	0,005	0
JHB 20	333	15	14	362	0,080	0
Rut 1	495	28	24	547	0,095	0,310
Rut 2	533	7	9	549	0,029	0,108
Rut 3	532	14	16	562	0,053	0,170
Rut 4	450	27	17	494	0,089	0,365
Br	624	26	21	671	0,070	0,038

Appendix C (continued p.2)

Line	Number of males				sn ^W mutation rate	GD sterility Cross A
	sn ^W	sn ⁺	sn ^e	Total		
Den 1	489	10	10	509	0,039	0,033
Den 2	491	17	10	518	0,052	0,008
Den 3	407	16	14	437	0,069	0
Den 4	656	25	17	698	0,060	0,008
Pta 1	523	24	24	571	0,084	0
Pta 2	612	13	17	642	0,047	0
Pta 3	567	33	28	628	0,097	0
Pta 6	647	19	30	696	0,070	0,016
H'K 1	569	17	20	606	0,061	0
H'K 2	632	25	13	670	0,057	0,083
H'K 3	332	5	4	342	0,029	0,050
Nel 5	412	16	8	436	0,055	0,018
Nel 10	532	34	15	581	0,084	0,176
Nel 15	589	16	15	620	0,050	0,029
Nel 20	510	19	18	547	0,068	0,528
P'B 1	347	12	12	371	0,065	0,432
P'B 3	602	10	6	618	0,026	0,063
P'B 4	381	12	9	402	0,052	0,203
P'B 5	421	11	13	445	0,054	0,086
Ag 1	390	7	7	404	0,035	0,058
Ag 2	390	6	7	403	0,032	0,009
Ag 3	600	17	19	636	0,057	0,018
Ag 5	543	22	19	584	0,070	0,009
Tho 5	488	13	12	513	0,049	0,076
Tho 10	467	17	15	499	0,064	0,161
Tho 11	494	10	13	517	0,044	0,054
Tho 20	185	8	6	199	0,070	0,076
Har 5	20	4	2	26	0,231	0,276
Har 10	120	10	7	137	0,124	0,253
Har 15	59	11	11	81	0,272	0,283
Har 20	94	4	2	96	0,062	0,210
Har 25	94	7	6	107	0,121	0,119
Controls						
sn ^W (M)	471	0	0	471	0	0
n ₂	742	137	125	1004	0,261	1,000

APPENDIX D

This Appendix re-evaluates the heterogeneity statistics and gene diversity analysis with *Hex-2* scored as a sex-linked locus.

Table D.1 shows the allele frequencies of *Hex-2* when each sample is taken to represent three genomes.

Table D.2 compares the likelihood ratios that would be obtained by the two different methods and

Table D.3 compares the heterozygosity and gene differentiation coefficients.

It is obvious from Tables D.2 and D.3 that the two methods of scoring give very similar results.

Table D.1 Allele frequencies of a sex-linked *Hex-2* for 16 Southern African populations of *D. melanogaster*

Location	N	Alleles		
		100	90	110
1. Swakopmund	45	1,000	-	-
2. Gobabeb	70	1,000	-	-
3. Ai Ais	72	0,972	0,014	0,014
4. Cape Town	63	0,968	0,016	0,016
5. Plettenberg Bay	12	0,667	0,167	0,167
6. Summerstrand	33	1,000	-	-
7. Walmer	24	0,958	0,042	-
8. Maclear	12	0,667	0,250	0,083
9. Berea	81	0,988	-	0,012
10. Addington Beach	66	1,000	-	-
11. Johannesburg	99	0,949	0,051	-
12. Pretoria/ Hamanskraal	24	0,875	0,083	0,042
13. Nelspruit	72	0,773	0,200	0,027
14. Pietersburg	12	0,750	0,250	-
15. Asitha	9	0,778	0,222	-
17. Harare	75	0,973	-	0,027

Table D.2 Comparison of log likelihood ratio statistics (*G* statistics) of *Hex-2* scored as autosomal and as sex-linked

Source of Variation	Case I <i>Hex-2</i> sex-linked	df	Case II <i>Hex-2</i> autosomal	df	Total			
					Case I	df	Case II	df
Among regions	29,15***	5	38,47***	5	369,64 ^c	30	378,95 ^c	30
Namibia								
Among areas	0,26	1	0,26	1	46,44 ^c	6	46,44 ^c	6
Between districts	0,13	1	0,13	1	45,25 ^c	6	45,25 ^c	6
Southeastern Cape								
Between districts	8,17*	1	11,35**	1	21,47 ^b	6	24,65 ^c	6
Between suburbs	0,05	1	0,07	1	26,84 ^c	6	26,86 ^c	6
Eastern Cape/Natal								
Between districts	14,61***	1	19,32***	1	23,50 ^c	6	28,22 ^c	6
Between suburbs	0,02	1	0,02	1	38,53 ^c	6	38,53 ^c	6
Transvaal								
Among areas	12,46**	2	19,43***	2	179,79 ^c	17	186,76 ^c	17
Between districts								
S. Transvaal	1,51	1	1,48	1	15,76 ^a	6	15,73 ^a	6
N. Transvaal	0,02	1	0,00	1	5,51	6	5,49	6

* $P < 0,05/6$
 * $P < 0,01/6$
 ** $P < 0,001/6$
 a $P < 0,05$
 b $P < 0,01$
 c $P < 0,001$

Appendix E

Allele frequencies for 17 Southern African populations of *D. melanogaster*

Location	Adh				Dip-A				Gpdh			Hex-1				
	N	-100	0	-120	N	100	92	108	N	100	75	N	100	115	130	90
1. Swakopmund	60	0.933	0.067	-	60	1.000	-	-	60	0.983	0.017	60	1.000	-	-	-
2. Gobabeb	100	0.450	0.520	-	100	1.000	-	-	100	1.000	-	100	1.000	-	-	-
3. At Ais	96	0.604	0.385	0.010	96	0.969	0.031	-	68	0.721	0.279	96	0.927	0.073	-	-
4. Cape Town	84	0.560	0.440	-	84	0.976	0.024	-	72	0.819	0.181	84	0.952	-	0.012	0.036
5. Flettenberg Bay Port Elizabeth	16	0.563	0.438	-	16	1.000	-	-	16	1.000	-	16	0.563	0.125	0.063	0.250
6. Summerstrand	108	0.500	0.500	-	108	0.963	-	0.037	108	0.722	0.278	108	0.917	0.009	-	0.074
7. Walmer	32	0.688	0.313	-	32	0.588	-	0.313	32	0.844	0.156	32	0.781	0.188	0.031	-
8. Maclear Durban	16	0.688	0.313	-	16	1.000	-	-	16	1.000	-	16	0.813	-	0.063	0.125
9. Berea	108	0.500	0.463	0.037	108	0.972	0.028	-	104	0.952	0.048	108	0.935	0.065	-	-
10. Addington Beach	88	0.795	0.205	-	88	0.955	-	0.045	64	0.813	0.188	88	0.818	0.159	0.023	-
11. Johannesburg	132	0.720	0.280	-	132	0.947	0.045	0.008	132	0.909	0.091	132	0.947	0.030	-	0.023
12. Pretoria + Hamanskraal	32	0.688	0.313	-	32	0.938	0.063	-	32	1.000	-	32	0.750	0.063	0.094	0.094
13. Neispruit	100	0.940	0.060	-	100	0.820	0.150	0.030	100	0.900	0.100	100	0.730	0.130	0.070	0.070
14. Pietersburg	16	0.688	0.313	-	16	1.000	-	-	16	1.000	-	16	0.563	0.188	0.125	0.125
15. Agatha	16	0.938	0.063	-	16	0.813	0.188	-	16	1.000	-	16	0.625	0.063	0.188	0.125
16. Thohoyandou	100	1.000	-	-	100	0.780	0.050	0.170	100	1.000	-	100	0.820	-	0.180	-
17. Harare	100	0.510	0.460	0.030	100	0.740	0.260	-	100	1.000	-	100	0.990	-	0.010	-

Appendix E (continued p.2)

Location	Hex-2				Idh-WADP						Pg4				Pg5			
	N	100	90	110	N	100	85	107	110	120	N	100	92	110	N	100	120	70
1. Swakopmund	50	1.000	-	-	60	0.800	0.183	0.017	-	-	45	1.000	-	-	50	1.000	-	-
2. Gobabeb	100	1.000	-	-	96	0.927	0.073	-	-	-	75	1.000	-	-	100	1.000	-	-
3. Al Ais	96	0.979	0.010	0.010	96	0.896	0.094	0.010	-	-	72	0.986	0.014	-	96	0.990	-	0.010
4. Cape Town	84	0.976	0.012	0.012	80	0.738	0.100	0.162	-	-	53	1.000	-	-	84	0.893	-	0.107
5. Plettenberg Bay Port Elizabeth	16	0.688	0.188	0.125	16	0.875	0.063	0.063	-	-	12	1.000	-	-	16	1.000	-	-
6. Summerstrand	44	1.000	-	-	108	0.926	0.019	0.056	-	-	81	0.951	0.049	-	108	0.926	0.028	0.046
7. Walmer	32	0.969	0.031	-	32	0.906	-	0.094	-	-	24	1.000	-	-	32	1.000	-	-
8. Maclear Durban	16	0.688	0.188	0.125	16	1.000	-	-	-	-	12	1.000	-	-	16	1.000	-	-
9. Berea	108	0.991	-	0.009	108	0.704	0.296	-	-	-	81	1.000	-	-	108	0.870	0.130	-
10. Addington Beach	88	1.000	-	-	88	0.557	0.432	0.011	-	-	66	1.000	-	-	88	1.000	-	-
11. Johannesburg	132	0.962	0.038	-	128	0.961	0.039	-	-	-	99	1.000	-	-	132	0.962	0.038	-
12. Pretoria Hammanskraal	32	0.906	0.063	0.031	32	0.875	0.031	-	0.094	-	24	0.958	0.042	-	32	0.844	0.125	0.031
13. Nelspruit	96	0.781	0.198	0.021	100	0.760	0.230	-	-	0.010	75	0.986	-	0.014	84	0.892	0.048	0.060
14. Pietersburg	16	0.750	0.250	-	16	0.813	0.188	-	-	-	12	1.000	-	-	16	1.000	-	-
15. Agatha	12	0.750	0.250	-	12	0.917	0.083	-	-	-	12	1.000	-	-	16	1.000	-	-
16. Thohoyandou	-	-	-	-	100	0.540	0.310	0.150	-	-	75	1.000	-	-	100	0.990	0.010	-
17. Harare	100	0.980	-	0.020	100	0.940	0.060	-	-	-	75	1.000	-	-	100	0.830	0.170	-

CHEMICALS⁺

Acc 1	Boehringer
Agar	Sigma
Agarose Seakem	EMC Bioproducts
Agarose Sea Plaque	EMC Bioproducts
Antibiotics	Boehringer
Boric Acid	Saarchem
BSA	Boehringer
Citric Acid	Sigma
CO ₂	Afrox
CsCl	Boehringer
Deoxycytidine 5'-[α - ³² P]	Amersham
DEP	Sigma
O-Dianisidine	Sigma
EDTA	Boehringer
Et Br	Boehringer
Ethanol	Boehringer
Ficoll (mw 400 000)	Sigma
Glucose ^a	Sigma
Glucose-6-P-dehydrogenase	Sigma
Glucose-1-phosphate	Sigma
Glycerol-3-phosphate	Sigma
HCl ^a	Saarchem
H ₂ PO ₄	Saarchem
DL-Isocitrate	Sigma
Isopropanol	Saarchem
Lambda molecular weight marker	Boehringer
Leucyl Tyrosine	Sigma
Liquid Nitrogen	Afrox
Lysozyme	Boehringer
MTT	Sigma
NaCl	Saarchem
NAD	Sigma
NADP	Sigma
Na ₂ HPO ₄	Saarchem
NaOH	Saarchem
Peroxidase	Sigma
6-Phosphogluconate gluconic acid	Sigma
PMS	Sigma
Potato starch	Sigma

Salmon Sperm DNA
SDS
Snake venom
Sodium citrate
Spermidine
Spermine
Sucrose

Trichloroacetic acid
Tris
Triton-X-100

Sigma
Saarchem
Sigma
Saarchem
Sigma
Sigma
Saarchem

Saarchem
Sigma
Sigma

*The source of all major chemicals used is listed. Chemicals which have not been listed are available from a number of sources and are standard in form. Their source will not in any way affect the outcome of the experiment in which they are used. (This also applies to several of the chemicals listed above).

PHOTOGRAPHY

1 Scanning Electron Microscopy Ovaries and Bristles (Fig. 2.1A; Fig. 3.1A)

Specimens were fixed for 24 hours at 4°C in 2.5% glutaraldehyde in a phosphate buffer (28,0 ml of 0,2 M monobasic sodium phosphate, 72,0 ml of 0,2 M dibasic sodium phosphate) (pH 7,2); rinsed briefly three times in the buffer and dehydrated in a graded ethanol series, 10 min per solution.

Specimens were critically point dried in CO₂ in a Hitachi HCP2 critical point dryer; then mounted onto stubs and sputter coated with gold/palladium in a Polaron Sputter Coater.

Specimens were observed in a JEOL JSM 840 scanning electron microscope using an accelerating voltage of 20 kV.

Wing Hairs (Fig. 5.3)

Scanning electron micrographs of wing hairs were obtained from Dr. H. Juon and Prof. F.E. Würzler from the Institute of Toxicology, Swiss Federal Institute of Technology and University of Zurich, Schwerzenbach.

2 Dissecting Microscope Photographs (Figs 2.1B,C; Fig. 3.1B)

Photographs of ovaries and bristles were taken with a Nikon Fx35 camera using a Nikon microscope. The film used was Fujicolor super HR100. Ovaries and whole flies were mounted on slides in Faure's solution (as described in Section 5.2.3.2).

3 Gel Photography and Autoradiograph Photography

Photographs of agarose gels and autoradiographs (Fig. 4.1; Fig. 4.2; Fig. 4.4; Figs 4.6 A-F) were taken with a polaroid CU-5 land camera. Type 665 black and white polaroid film was used.

Photographs of starch gels (Fig. 6.4B) were taken with a Nikon FM2 camera and Kodak plus x ASA 120 film was used. These photographs were taken by one of my supervisors (S. Grant).

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