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SECOND CHROMOSOME VIABILITY GENETIC LOAD AND LETHAL ALLELIC FREQUENCY IN DROSOPHILA MELANOGASTER IN EWEKORO, OGUN STATE, NIGERIA.

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ABSTRACT

The frequencies of recessive lethal, semi-lethal, sub-vital and quasinormal chromosomes-2 in Drosophila melanogaster from the cement factory at Ewekoro, Ogun State were determined by making one chromosome 2 from each trapped male, homozygous. The frequency of allelism among the lethal chromosome isolated was also determined. The frequency of lethal, semi-lethal, subvital, quasinormal and normal chromosomes in the population were 44.9%, 22.5%, 12.2%, 2.0% and 16.3% respectively. Although the frequency of lethals was lower than expected for a lowland population located close to the equator, the proportion of lethals among drastics (lethal + semi-lethals) was not different for values for a number of populations in higher latitudes. The low frequencies indicated a large population and a free flow of gene was suggestive. Comparison with previous data on chromosomes – 2 and 3 suggest that generalizations on genetic load in population must be based on data on all major chromosomes in the species.

INTRODUCTION

Muller (1950) described genes that carry deleterious mutations, which reduce viability or fertility of organisms carrying them as constituting genetic load of the population. Many of such mutations are retained and transmitted in populations, thus lowering the fitness of the organisms carrying them (Temin, 1966).

The mutations lead to gene frequency changes (Barker and East, 1980) and are raw materials of evolution (Strickberger, 1985). They may cause death when dominant (Hadorn, 1961) and when recessive may be carried in heterozygous condition for infinite length of time without exerting lethal effects (Wallace, 1966).

Rieger et al (1965) defined lethal sensu strictu as those, which cause death in all carriers of the correct dose, but also defined to include death in at least 90% of those carrying the active dose, semi-lethals cause death in 50-90%, sub-vitals in 10-50% and quasi-normals in less than 10%.Studies on recessive lethal chromosome-2 of Drosophila melanogaster have shown that the frequency may vary not only in different populations in same country (Ives, 1954; Crumpacker, 1967; Allen, 1969) for American populations and (Williams and Akpabio, 1993a; Williams and Bamboye, 1996; Adekoya and Williams, 1998) for Nigeria populations. The frequencies also vary in different parts of the world (Paik, 1966; Hoenigsberg, et al, 1968, Watanabe, 1969, Adekoya, 1991,Adekoya and Williams, 1998 and Dawood, 1961).

There have only been a few reported studies on genetic load on chromosome – 2 in Africa: Dawood (1961) in Egypt, Williams and Akpabio (1993) and Adekoya and Williams (1998) in Nigeria. The first and second studies investigated only lethal chromosome, while the third report studied other various categories (semi - lethals, subvitals and quasi-normals) of Chromosome – 2 reducing viability in Nigeria population of Drosophila melanogaster.

This study sampled flies in and near the Ewekoro Cement factory in Ogun State South Western Nigeria as well as the fields around Faculty of Science in Lagos State University. The study determine the frequencies of different types of chromosomes affecting viability for information on gene flow as well as determine the mutagenic effects of the cement dust on the genetic load of D. melanogaster in the wild. It is also intended to compare the data recorded for Ewekoro with those from LASU.

MATERIALS AND METHODS

Flies were trapped using decomposing banana baits in bottles about 150ml in volume and mouth about 3cm in diameter. The bottles were placed near garbage collection centers. After about 6-10hrs, the bottles were covered with cotton plugs and taken to the laboratory where D. melanogaster males were removed and used for crosses. The flies were raised on a banana and cassava meal medium (Williams and Akpabio, 1993a) at 28±3°c in a room air conditioned only during the day.

The collections were made from three locations in Lagos State University (Faculty of Science Buttery) and three locations in the environments of Ewekoro cement factory food stalls. The collection was made between the months of May and June 1998. The straight-line map between LASU and Ewekoro is 65km.

A total of 104 male flies from the wild (48 from Ewekoro and 56 from LASU) were tested for presence of detrimental factors on chromosome – 2. By using one marked chromosome – 2 isolated from each wild male and made homozygous in a series of three crosses in order to determine it's recessive viability effect(Adekoya and williams,1998).

The progeny of cross – 3 (F3) were scored for 7 days only in order to avoid inclusion of F4 progeny. A wild chromosome – II was classified lethal if the observed ratio of curly (II/SM5) to wild type wing (II/II) was equal to or significantly greater than 20:1 at the 1% level of significance in a $?^2$ – test.A chromosome was however classified as semi – lethal if the ratio was not only significantly less than 20:1 but also significantly greater than 4:1. Chromosomes were either sub vitals or quasinormals if the ratios were significantly lower than 4:1 but higher than 2.8:1 or between 2.8:1 and 2:1 respectively. The analysis was processed using Fortran application programme developed for this study (Appendix I)

The lethal chromosomes detected were maintained as balanced lethal stocks, SM5/L where L is the lethal chromosome. This lethal chromosome was tested for allelism by carrying out a partial diallelic cross(Adekoya,1991).The progeny of the crosses for allelism were scored also for not more than 7 days after the first eclosion. The lethal chromosomes were classified as allelic if the ratio was similar to that for lethals in the homozygosis cross. The frequency of allelism was determined as the percentage of allelic crosses in the total number of crosses made (Wallace, 1966.)

RESULTS

One – chromosome – 2 was tested for lethality from each of the fifty-six male flies trapped from Lagos and forty-eight from Ewekoro in Ogun State of Nigeria. The numbers detected for each of the five categories of chromosomes in each site are shown in Table 1. The lethals occurred in the highest frequencies in each of the sites. Semi – lethal were next in magnitude in both sites. The frequencies of lethals were almost double of the value for semi-lethals in both sites. The drastic (Lethals + Semi –lethals) and detrimental (all categories of chromosome except normal) were 66% and 80% of the total tested flies respectively.

Heterogeneity $?^2$ – test of the two sites and the five categories of chromosomes indicated no heterogeneity among the sites ($?^2$=1.196; 4df;P >0.1). Another heterogeneity tests of the two sites and drastic as well as sites and detrimentals showed no significant difference: ($?^2$ = 0.23, 1d.f. P>0.1) and 0.687;1 d.f.P>0.1) respectively.

Diallelic crosses among the isolated lethals made within and between sites were partial. This is because eleven of the lethals isolated from Ewekoro and twelve from Lagos died before the test and some of the crosses were infertile. In the intrasite crosses for Ewekoro lethals only one – cross 1.82% (1/55) was allelic for lethality and none was allelic for Lagos lethals (0/66). There was only one allelic lethal recovered in 132 intersite allelic crosses (0.76%) made between Lagos and Ewekoro lethals. The overall intersite frequency of allelism was not significantly different from the overall intersite frequency ($?^2$ = 0.0035, 1d.f;P>0.1)

APPENDIX I

FORTRAN APPLICATION PROGRAM FOR CLASSIFICATION OF CHROMOSOMES.

```
DIMENSION A (150, 2), B (150,2), C (150), D (150), E (150)
INTEGER A, B
CHARACTER *15 D,E
OPEN (1, FILE='AKOKA.DAT')
OPEN (3, FILE=' AKOKA.OUT')
READ (1, *)N
WRITE (*,*)N
4    FORMAT (13)
```

```
      DO 5 J=1,N
      READ (1,6)A (J,1),A (J,2), B (J,1), B (J,2)
6     FORMAT (6X,4 (I 3,1X)
      IF (B (J,1). NE.0.OR.B(J,2).NE.0)THEN
      C (J) = FLOAT ( (A (J,1) + A (J,2) ) )/FLOAT ( ( B (J,1) + B (J,2) ) )
      ELSE
      IF ( A (J,1).EQ.O. AND. A (J,2). EQ.0) THEN
      C (J) = 0.0
      ELSE
      C (J) = 22.0
      ENDIF
      ENDIF
      WRITE (*,*) C (J )
5     CONTINUE
      DO 7  I=1,N
      IF ( C (I). GE. 20.0) D (1) =' LETHAL'
      IF (C (I).GT.4.0. AND. C (I). LT. 20.0) THEN
      D (I)=' SEMI-LETHAL '
      IF (C (I). GT.12.0)THEN
      CHI=ABS (C(I)-20.0)-0.5
      CHI=CHI**2/20.0
      IF (CHI.GT.6.64) E (I)='  '
      IF (CHI.LT.6.64) E (I)=' LETHAL'
      ELSE
      CHI=ABS (C(I)-4.0) – 0.5
      IF (CHI.GT.6.64) E (I)='  '
      IF (CHI.LT 6.64) E (I)=' SUB-VITAL'
      END IF
      ELSE
      END IF
      IF (C(I).GT.2.8.AND. C (I).LT.4.0)THEN
      D (I)=' SUB-VITAL'
      IF (C (I). GT.3.4)THEN
      CHI=(ABS (C (I) –4.0)-.0.5
      CHI=CHI**2/4.0
      IF (CHI.GT.6.64)E (I)='  '
      IF (CHI.LT.6.64) E (I)='SEMI-LETHAL'
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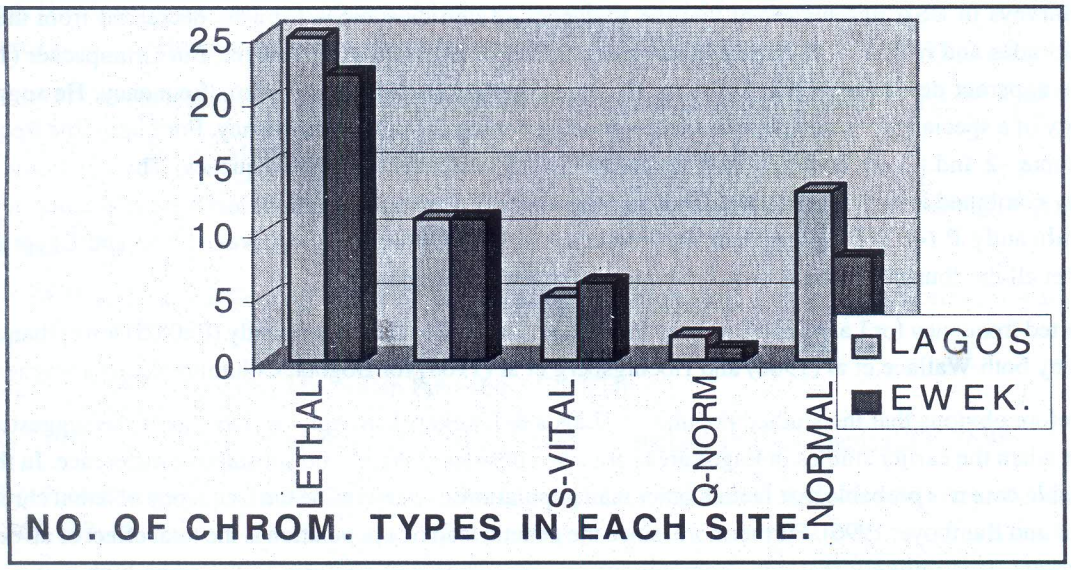
```
      ELSE
      CHI=ABS (C (I)-.2.8)-0.5
      CHI=CHI **2/2.8
      IF (CHI.GT.6.64) E (I)=' '
      IF (CHI.LT.6.64) E (I) =' QUASI-NORMAL'
      END IF
      ELSE
      END IF
      IF (C (I).GT.2.1 AND. C (I).LT.2.8) THEN
      D (I)=' QUASI-NORMAL)
      IF (C (I).GT.2.45)THEN
      CHI=ABS (C (I) –2.8)-0.5
      CHI =CHI**2/2.8
      IF (CHI.GT.6.64)E ( I )='  '
      IF (CHI. LT.6.64)E ( I )=' SUB-VITAL'
      ELSE
      CHI=ABS (C (I) –2.1)-0.5
      CHI=CHI**2/2.1
      IF (CHI.GT.6.64) E (I)=' '
      IF (CHI.LT.6.64) E (I) =' NORMAL'
      END IF
      ELSE
      END IF
      IF (C (I). LT. 2.1) THEN
      D (I)=' NORMAL
      IF (C(I).GT.1.5) THEN
      CHI=ABS (C(I)-2.1) –0.5
      CHI=CHI**2/2.1
      IF (CHI.GT.6.64) E (I)=' '
      IF (CHI. LT.6.64) E (I)=' QUASI-NORMAL'
      ELSE
      E (I)='UNCLASSIFIED'
      END IF
      ELSE
      END IF
      IF (C (I). EQ.0.0) D(I)=' UNCLASSIFIED'
7     CONTINUE
```

```
      DO 16 I=1,N
      IF (D ( I ).EQ. 'LETHAL' )THEN
      WRITE (3,23) A (I,1),A(I,2), B (I,1),B (1,2),C( I), D (I)
      ELSE
      WRITE (3,17) A (I,1),A (I,2), B (I,1), B (I,2),C (I), D (I), E (I)
      END IF
23    FORMAT (2X,4 (2X,13),2X,F5.1,2X,A15)
17    FORMAT (2X,4 (2X, 13), 2 X F5.1, 2X, 2 (A15,2X)
16    CONTINUE
      STOP
      END
```

SITE	NUMBER OF CHROMOSOME TESTE	LETHAL (%)	SEMI LETHAL (%)	SUB VITAL (%)	QUASINOR MAL (%)	NORMAL (%)
LAGOS	56	25(44.64)	11(19.64)	5(8.92)	2(3.57)	13(23.21)
EWEKORO	48	22(45.83)	11(22.45)	6(12.24)	1(2.04)	8(16.33)
TOTAL	104	47(45.19)	22(21.15)	11(10.58)	3(2.88)	21(20.19)



DISCUSSION

The no significant differences recorded between the two study sites strongly suggest an unimpeded gene flow among the sites (Lagos and Ewekoro), consequently making the sites one large homogeneous population.

The high frequencies of drastic (lethals + semi lethals) chromosomes in this study (66%) and detrimental (80%) implies that ~ 73% of the chromosomes – 2 have a negative effect on viability, a very large genetic load is therefore suggestive. Moreover, the low frequency of subvitals and quasinormals, which only slightly depress viability, does not have a direct effect on the genetic load.

The conclusion of high genetic load and free flow of genes in a large homogeneous population is strongly supported by the results of test of allelism. The frequency of allelism recorded in this study is not significantly ($P>0.5$) different from the frequency of 0.76% reported by Adekoya and Williams (1998) and 0.95% reported by Williams and Akpabio (1993a). It is also not significantly different ($P>0.5$) when compared to the values reported for chromosome – 3 (Williams and Bamboye, 1996). The low frequency of allelism is therefore consistent with the expectation of large D. melanogaster population in the area.

Although the frequency of lethals in this study is significantly (P<0.005) higher than the 8% reported for 2 sites in Lagos by Williams and Akpabio (1993a) but not significantly different (P>0.1) different from those recorded for 6 sites by Adekoya and Williams (1998). The differences in frequencies observed could be as a result of the fact that Williams and Akpabio considered only balanced lethals stocks as lethals but this study conforms with the sensu strictu consideration of lethals by Rieger et al (1965). Another possible reason for the deviation could be as a result that Williams and Akpabio did collection during dry season.

The frequency of lethals in this study is not significantly different (P>0.1) from those reported for comparable parts of the world such as the American Samoan (Allen, 1969) and Turbo, Columbia (Hoenigsberg et al, 1968). The frequency is however significantly higher than reported for Egypt (Dawood, 1961) and far less than 55% reported for Bogota, Columbia close to the equator but on a high cold Plateau (Wallace et al 1966)

Examinations of the frequency of lethal and semi-lethal chromosome – 2 compiled by Watanabe (1969) and those by Adekoya (1991) show that frequency of lethals among drastic could be calculated in 14 studies. The frequency was between 13% and 78% except in 2 cases 8% in a study in Lagos and 82% in Israel. In spite of the fact that the frequency of drastics in Egypt, Bogota and earlier report in Lagos were different, a 2 x 2 contingency $?^2$ tests showed that the frequency of lethals among drastics in these populations were not significantly different from the frequency in the present study.

Williams and Bamboye (1996) had reported that from their study and others of other investigators, lethal chromosome – 3 constituted 63-79% of drastic in most studies. It seems that for each chromosome, there is some equilibratory relationship between the frequency of lethal and semi-lethal chromosomes, in spite of the overall frequency of drastic in the population.

Earlier Surveys in USA has suggested that the frequency of chromosome – 2 drastic decreased from the lower to higher latitudes and results from Korea, Japan and USSR seemed to be in agreement. But Crumpacker (1967) also noted the apparent deviation of the American Samoan populations, which had a low frequency. He suggested the peculiarity of a specialized island population as a possible reason for the low frequency. For Lagos, the frequency for chromosome –2 and –3 are contradictory; chromosome – 3 has expected high frequency. The Lagos and Ewekoro frequency combined is not significantly different from those of Korea (Paik; 1966) and Japan (Watanabe, 1969) but it is significantly P (<0.005) lower than the frequency in both USA (Band and Ives, 1968) and Egypt (Dawood, 1961). Yet all the four populations occur at higher latitudes than Lagos.

The reported frequency for Lagos and Ewekoro for chromosome – 2 is also significantly (P<0.05) lower than frequency reported by both Wallace et al (1966) and Hoenigsberg et al (1968) for Bogota, Columbia that is at higher latitude.

It is therefore obvious that the studied population (Lagos & Ewekoro) strongly deviates from the suggested pattern. However when the earlier studies in Lagos areas are also considered there is no substantive difference. In the light of the available data it is probable that factors other than population size determine the frequency of lethal chromosomes (Williams and Bamboye, 1996). Furthermore, since the genetic load of a population is the total effect of all detrimental genes present, it is unlikely that non- homologous chromosomes will conform to the same high pattern, rather it seem s that there is a corroborative mechanism. For example 70.6% for chromosome –3 detrimentals (Williams and Bamboye, 1996) may be ameliorated by 38:2% for chromosome –2 (Adekoya & Williams, 1998).

The no significant difference between the frequency of chromosome types in Lagos and that at Ewekoro suggests that the expectedly high genetic load of Ewekoro due to the mutagenic effects of the cement dust of the factory on the flora & fauna situated in the environs, were not accounted for in the study. According to data on frequency of inversion in chromosomes-2 and 3 (Williams and Akpabio, 1993b; Williams and Bamboye, 1996) one expects a higher frequency of detrimentals or drastics in Ewekoro than Lagos.

The differences in the frequency of inversion report from different chromosomes and the differences in data of frequency of lethals and detrimental reported for different times and the difference from population to population suggests therefore that different types of chromosomes do not individually give a true picture of the genetic load in a population, hence any generalizations must be based on all the major types of chromosomes in the species as well as the different types of detrimentals in the population.

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