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Changes in rainfall and temperature range patterns spanning several decades in Amherst, Mass. have been occurring. The data given below are May-October averages. This interval approximately spans the breeding seasons of *D. melanogaster* there (see Ives, 1970). The popu-

lation in that area has been investigated by Dr. P.T. Ives since the 1930's. Genetic changes toward a lower level of lethals and semilethals among second chromosomes in the population were observed in 1947 (Ives, 1954) and multiple genetic changes occurred in the 1960's (Band, 1964; Ives, 1970). Average daily temperature range and average total rainfall for the six months interval in the various periods have been:

average daily temperature range	<u>1930-1946</u> 23.0	<u>1947-1962</u> 24.0	<u>1963-1966</u> 26.4	<u>1967-1969</u> 24.5
total rainfall in inches	<u>1930-1946</u> 23.48	<u>1947-1961</u> 22.01	<u>1962-1965</u> 15.22	<u>1966-1969</u> 22.07

Furthermore, average daily temperature range for the summer months shows a regular 0.8° increase per decade, starting with an average of 22.5° between minimum and maximum daily temperatures in the 1930's and climbing to a 24.9° average for the 1960's. No other season shows such regular changes over the four decades though increases in temperature range in other seasons are also observed both in the 1940's and in the 1960's, particularly spring quarter of the latter decade.

The fairly simultaneous changes in both average daily temperature range and rainfall suggest the two are not independent climatic variables. Both had already been found to be significantly correlated with the level of lethals and semilethals found in the population. Summer rainfall is significantly positively correlated with *le + sle* frequency (Band and Ives, 1968) and temperature range prior to collection is highly significantly negatively correlated with *le + sle* frequency recovered in a sample (Band and Ives, 1961, 1968). The genetic changes detected in the population have been found to occur at each shift in one or both environmental variables.

The above climatic data strengthen the argument that genetic homeostasis accounts for the retention of *le + sle* frequency at 34% in 1964 (Band, 1969), the level around which these variants have fluctuated since 1947. No significant decline in lethals was detected until June 1966, when the frequency of these variants was only 17% (Ives, 1970). Yet after 1962 rainfall and after 1963 temperature range also maintained respectively their lowest and highest four-year averages during the forty-year period.

More climatic information than that available from averages per climatic period is needed to determine whether retention of *le + sle* frequency at 33.9% in 1962 is also an example of the resistance of *le + sle* frequency to decline. However, the increased developmental homeostasis present after 1961 (Band, 1964) does seem to have delayed the decline in *le + sle* frequency despite the persisting downward selection pressures exerted by both environmental variables from 1963 through 1965.

How widespread or how local these climatic shifts have been is not known at present. Tantawy et al. (1969) in Egypt and Hoenigsberg (1968) in Colombia both reported localities in which *D. simulans* supplanted *D. melanogaster* as the dominant species in the mid-1960's. Watanabe (1969) reported a significant decline in *le + sle* frequency in a Japanese *D. melanogaster* population in 1966. The possibility of climatic shifts was not considered; alternate hypotheses advanced in each case seemed more appropriate. Time-wise these may all be coincidental with climatic shifts and genetic changes in the S. Amherst *D. melanogaster* population.

References: Band, H.T., 1964 *Evolution* 18: 384-404; _____, 1969 *Japan. J. Genet.* 44 Suppl. 1: 200-207; _____ and P.T. Ives, 1961 *P.N.A.S.* 47: 180-185; _____ and _____, 1968 *Evolution* 22: 633-641; Hoenigsberg, H.F., 1968 *Amer. Nat.* 102: 389-390; Ives, P.T., 1954 *P.N.A.S.* 40: 87-92; _____, 1970 *Evolution*, in press; Tantawy, A.O., A.M. Mourad and A.M. Masri, 1969 *Amer. Nat.* 104: 105-109; Watanabe, T.O., 1969 *Japan. J. Genet.* 44: 171-187.