Leenders, H.J. University of Nijmegen, The Netherlands. Temperature induced puffs in Drosophila: their possible physiological origin.

A sudden rise in environmental temperature (25-37°C) induces characteristic changes in the chromosomal puffing pattern of Drosophila[1,2,3]. Various suggestions have been made with respect to the intracellular mechanism responsible for the activation of the responding loci. Since in vitro treatments of larval salivary glands with 10^{-3}M dinitrophenol (DNP) affected the puffing pattern in essentially the same manner as a temperature treatment[1], it was supposed that either a deficiency in ATP or an accumulation of ADP might be the trigger for the activating mechanism of the responding loci.

In order to test this possibility, the effect of in vitro administration of ATP, ADP, malate and succinate to mid-third instar glands of D. hydei was studied. All incubations were performed under Arachis oil and the response of the loci 2-48C, 2-36A, 2-32A and 4-81B (temperature puffs) was taken as an indication for the activation of the induction mechanism. Neither addition of 10^{-2}(or 10^{-3})M ATP, nor of 10^{-2}(or 10^{-3})M ADP to the incubation medium resulted in the activation of these loci. Addition of 10^{-3}M DNP resulted, as expected, in their activation. However, a slightly increased concentration 3.10^{-3}M DNP appeared to be ineffective. If 10^{-3}M ATP is present in the medium 10^{-3}M DNP has no effect upon the puffing pattern, whereas addition of 3.10^{-3}M DNP to the same medium results in the induction of the puffs. A further increase in DNP concentration ($>5.10^{-3}$M) appears to be ineffective again.

Incubation media containing ATP were also used to investigate the effect of a temperature treatment, CO_2 and N_2 treatments. These treatments all resulted in the appearance of temperature puffs[4]. Thus it seems that, though a deficiency of ATP (or accumulation of ADP) cannot be excluded as possible intracellular trigger for puff induction, it may well be that the change in the level of these metabolites is a consequence of an enhanced metabolism.

Support for this assumption came from experiments in which malate (10^{-2}M), which upon metabolization may supply hydrogen to the coenzyme NAD^+, or succinate (10^{-2}M) which may support the hydrogenation of quinones, was supplied to the incubation medium prior to the addition of 10^{-3}M DNP. These substances did inhibit puff formation of the loci 4-81B, 2-32A and 2-36A but not of 2-48C. It was further found that oxymethylene blue (10^{-2}M) and menadione (10^{-3}M) (vit. K3), compounds which may serve as intermediates in the electron transfer system can induce temperature puffs in vitro. These data may be interpreted as support for the assumption that temperature puffs are induced by an increase in oxidation rate, leading to a temporary shortage of hydrogenated metabolites of the respiratory chain (or a temporary accumulation of oxidized compounds). The effect of DNP may be explained in terms of a common property of uncouplers, the stimulation of oxidation.

References: 1. Ritossa, F.M., 1964 Exptl Cell Res. 35: 601; 2. Berendes, H.D., F.M.A. van Breugel and T.K.H. Holt, 1965 Chromosoma 16: 35; 3. Ashburner, M., 1970 Chromosoma 31: 356; 4. van Breugel, F.M.A., 1966 Genetica 37: 17.

Schalet, A.* University of Connecticut, Storrs, Connecticut. Suppressor of forked: insertion into a sc^8 chromosome; high frequency of X-ray-induced deficiencies.

Since the proximal heterochromatic region of the X chromosome in Drosophila melanogaster, especially when it is distal or (partly) absent, has been a favorite playground for Drosophila geneticists of many persuasions, the insertion of the heterochromatically located mutant su(f) into a sc^8 inversion chromosome should prove to be a useful tool in different types of experiments. When ♀♀, y^+ sc^8 Df(1)mal^{10} B/1J1 y^{J1} sc^{J1} v f mal^1 su(f) are crossed to ♂♂, Y/y v sw mal^2, the only non-crossover class to survive are ♀♀ carrying the y^{J1} marker. This is because mal^{10} which includes sw is lethal in the combination mal^{10}/sw, and ♂♂ are inviable because of mal^{10} or 1J1. Regular offspring marked with y^+ come from a crossover chromosome that must have had one exchange between y^+ and mal^{10}. Of 59 ♂♂ observed, 1 proved to be y^+ sc^8 su(f)mal^1 f v. Of 75 y^+ ♀♀, 51 gave sufficient offspring in crosses to B^SY/y ac In49 v f mal^1 su(f) ♂♂ to show that only 1 carried su(f) in the sc^8 chromosome. The 2 crossovers of independent origin were found among a crudely estimated total of 57,000 chromosomes.

A number of lines of genetic evidence, (Lindsley and Sandler, 1958; Zimmering, 1959; Herskowitz, Schalet, and Reuter, 1962; Schalet, 1963; Schalet and Finnerty, 1968), have pointed to the probable penultimate position of su(f), i.e., close to the left of bb, in the proximal X. The mitotic X cytology of Cooper (in Lindsley and Grell, 1968) and the polytene