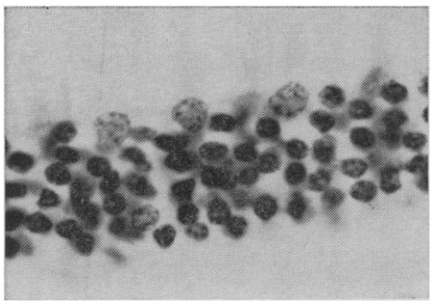

Fig. 1. Retinal layer of male eye, showing nuclei of rods and cones. The nuclei of cone cells are large and vesicular with multiple, minute masses of chromatin.

plantation (*8, 9*). In one other tissue, the bone marrow, somatic cells fail to show a condensed X (*10*); this fact appears to be in contradiction to the finding of two populations of red cells in individuals heterozygous for glucose-6-phosphate dehydrogenase deficiency (*11*).

A number of examples are known of "patches" of cells in which one or the other of the sex chromosomes is inactivated, confirming the X-inactivation hypothesis. The most notable are those resulting in variegated coat color in the mouse, in which coat color genes are present on an X-chromosome. In fact, examples of these have been used by Russell (*12*) and Lyon (*13*) as evidence for the X-inactivation hypothesis. Also, ocular albinism results in a finely mottled fundus in the heterozygote (*14*).

Krill and Beutler (*15*), using the dark-adapted fundus, found the red-light threshold of female protan heterozygotes to be no different from that of the male hemizygote in all areas. Using the light-adapted fundus (the technique bleaches the rods and therefore makes them less sensitive), they found (*16*) intermediate degrees of threshold sensitivity to red light in 1-degree

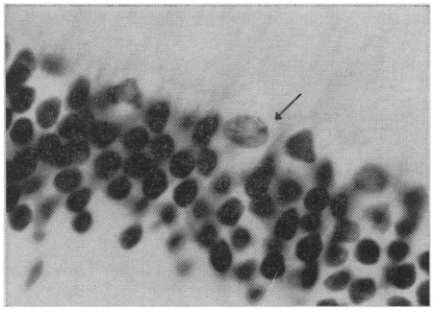

Fig. 2. Retinal layer of female eye. The arrow marks the only cone-cell nucleus that is in focus; the nucleus contains a distinct sex-chromatin body.

targets. Although some variation was detected, it did not exceed that found in normal individuals. This work suggested that, while inactivation of sex chromosomes may occur in the cone cell of the retina, patches, if they develop, must be smaller than 1 degree.

Recalling the lack of a condensed X-chromosome in bone-marrow cells, I suggested that an alternative view might be taken for the results obtained by Krill and Beutler. The possibilities were: (i) Patches of cone cells in the retina are so minute as to be undetectable. (ii) No patches occur because the function of the cone cells requires that both X-chromosomes be extended. If the second alternative were true, the cone cells of the retina, like the cells of bone marrow, would be deficient in sex chromatin in females. Therefore, I studied the status of sex chromatin in the cone cells of the human eye.

Sections of eye tissue which had been removed from ten adult males and six adult females because of various pathological conditions but which were nevertheless considered suitable for study were stained by routine hematoxylin and eosin procedures after fixation and embedding in paraffin. None of the cells in the males, but all those in the females, showed a distinct sex-chromatin mass at or near the nuclear membrane (Figs. 1 and 2). The masses showed the usual variations in shape, from a disk to a bent rod, and seemed quite prominent.

There appear to be some differences in "patch" development among various tissues with respect to X-chromosome inactivation. In the skin and pigmented layer of retina, patches regularly occur in heterozygous female mammals in which X-linked genes for pigmentation are present (*12–14*). However, retinal cone cells do not demonstrate patch formation in heterozygotes for protanopia, nor is patch formation detectable (by glucose-6-phosphate dehydrogenase determination) in uterine muscle of heterozygotes for G-6-PD deficiency. However, uterine myomas consist of cells of one type with respect to G-6-PD (*17*). This fact indicates that X-chromosome inactivation occurs in cells of uterine muscle and that the myoma originates from a clone established from a single cell.

Pigmented cells of skin and retina are derived from cells which migrate from the neural crest (*18*). These cells will have already undergone X-chromosome inactivation (*9*). Individual cells, therefore, may arrive at their final destination widely separated from their sister cells and then proceed to establish a clone. This would enable them to develop patches derived essentially from a single cell. Structures or tissues developed *in situ*—that is, without the migration characteristic of melanocytes and of other cells—would inevitably be a mixture of cells with respect to X-chromosome condensation. Mosaicism would be preserved, but patch formation would be on a cell-to-cell basis.

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Differential Enzymatic Activity in Ecological Races of Typha latifolia L.

Abstract. *Glycolic acid oxidase activity in chloroplast fragments from ecotypic populations of* T. latifolia *L. was much greater, particularly at lower temperatures, in populations adapted to a semimaritime climate than in populations adapted to a continental climate.*

The accumulated data on physiological patterns in ecological races of plants (*1*) provide circumstantial evidence for the existence of profound enzymatic differences between plants from conspicuously different habitats,

Table 1. Glycolic acid oxidase activity (oxygen consumption) in chloroplast fragments from ecotypic populations of *Typha latifolia* L. at three temperatures. The results represent oxygen consumption of two replications, each consisting of six flasks, expressed as milliliters of oxygen per flask per 5 minutes.

Temp. (°C)	Oxidase activity			
	Beaverton		Redmond	
	A	B	A	B
17	31.9	18.8	8.9	8.7
27	30.0	16.3	11.8	9.9
37	19.9	9.7	12.6	9.2

but no data have been provided documenting directly an enzymatic difference between ecotypic populations.

In an investigation designed to determine whether there are definite enzymatic differences between plants adapted to different habitats, four populations of broad-leaved cattail, *Typha latifolia* L., were selected. Two populations were from sites near Beaverton, Oregon, in a semimaritime climate characterized by a long growing season (264 days), cool midsummer high temperatures (about 25°C), and mild midsummer lows (15°C). The other two populations were from Redmond, Oregon, in a continental climate characterized by a short growing season (130 days), warm midsummer highs (35°C), and cool midsummer lows (10°C). Although the mean daily temperatures are similar, diurnal variation is much greater in the Redmond location.

Standard techniques for the isolation of chloroplasts were used (*2*) except that plastids from greenhouse-grown plants were sedimented under conditions between 1000*g* for 1 minute and 37,000*g* for 10 minutes. Chloroplasts were fragmented by suspending the pellet in 0.067*M* phosphate buffer, *p*H 7.8, to a concentration of 0.08 g of plastid fragments per milliliter; 2.5 ml of this suspension was added to each Warburg flask. Enough glycolic acid was in the sidearm to make the suspension 0.008*M*. Standard manometric techniques with KOH were used (*3*).

Data presented are from two experimental sets, each of which consisted of six flasks for each population at each temperature. The data indicate that glycolic acid oxidase activity differed markedly, depending upon the type of climate to which the plant population was adapted (Table 1). Student's *t*-test on paired samples showed that Beaverton A was much more active than all other populations at all temperatures ($P << 0.001$ in all comparisons). The Beaverton B population was more active than either Redmond population at 17° and 27°C ($P << 0.001$) but was less active than Redmond A at 37°C ($P < 0.01$). The two Redmond populations were not significantly different except at 37°C where population A was more active. These data provide direct evidence that enzymatic activity is conspicuously different and that temperature dependence is different in populations occupying distinct habitats.

Although considerable controversy surrounds the role of glycolic acid in the carbohydrate metabolism of higher plants (*4*), there can be little doubt that at normal concentrations of CO_2 a significant proportion of the CO_2 fixed is metabolized by glycolic acid oxidase (*5*). The important function of this enzyme in carbohydrate metabolism suggests that the conspicuous differences in glycolic acid oxidase activity between populations from different habitats probably reflect selective processes that are oriented by climatic adaptation of assimilatory metabolic pathways.

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Succinate: Protective Agent against Hyperbaric Oxygen Toxicity

Abstract. *When succinate is used to protect rats against the toxicity of oxygen at high pressure, 100 percent survive, with normal or above normal concentrations of adenosine triphosphate being present in the cerebral hemisphere, liver, and kidney. In contrast, 90 percent of the nonprotected animals died during exposure. In corresponding tissues of surviving nonprotected animals adenosine triphosphate concentrations are markedly reduced.*

The convulsions and depressed metabolic changes which accompany exposure to oxygen at high pressure were first described by Bert in 1878 (*1*). There has been at least one death attributed to the clinical use of hyperbaric oxygenation (*2*). The problem of toxicity to oxygen at high pressure is an obstacle to more widespread clinical application of hyperbaric oxygenation (*3*).

Studies on cellular metabolism have led us to believe that succinate should offer protection against the toxic action of oxygen at high pressure. A series of experiments was performed on four groups of fasted male Sprague-Dawley rats (160 to 225 g). Group 1 (controls) was given intraperitoneal injection of 7.5 ml of 0.4*M* succinate solution (*p*H 7.4) 2½ hours before they were killed; groups 2, 3, and 4 were given intraperitoneal injections of 7.5 ml isotonic saline, 0.4*M* dextrose solution (*p*H 7.4), and 0.4*M* succinate solution (*p*H 7.4), respectively, 1 hour before exposure (1½ hours) to 100 percent oxygen at 5 atm (absolute pressure).

Nine of the ten animals given isotonic saline (group 2) were dead before the end of the exposure period, and the tenth died before tissues could be removed for analyses. Five of the ten animals receiving dextrose (group 3) died during exposure, three exhibited symptoms of oxygen toxicity (convulsions, loss of consciousness, frothing at the mouth), and two animals appeared normal. All of the 22 animals receiving succinate injections (group 4) were normal in appearance, alert, and active. Six animals were observed for 6 days and showed no aftereffects.

The concentrations of energy stores