Table 1. Activity of fractions from *Solanum dulcamara* against Sarcoma 180 in mice.

Dose (mg/kg)	Survivors	Change in animal weight (g)*	Tumor weight in mg (test/control)
	Fraction A		
266	0/4		
177	1/4	−4.1	275/935
118	4/4	−1.9	346/935
	Fraction B		
300	3/4	−4.5	0/1015
150	4/4	−4.4	356/938
75	4/4	0.6	629/938
37.5	3/4	−1.4	826/938
	Fraction C		
200	3/4	−1.7	348/804
100	4/4	−0.4	543/804
50	4/4	−1.2	791/804
	Fraction D		
300	4/4	1.9	1571/867
150	4/4	0.1	1203/867
75	4/4	−1.0	1349/867
	Fraction E		
300	0/4		
150	0/4		
75	3/4	−3.2	180/804
	Fraction F		
60	0/4		
30	4/4	−1.8	274/1285
15	4/4	−1.6	806/1285

* The difference, in grams, between weights of test and control animals.

The major component of one of the fractions was separated from the remainder of the material by countercurrent distribution (upper phase, 50 percent butanol in ethyl acetate, and lower phase, *p*H 4 citrate-phosphate buffer) and further alumina chromatography. The compound (*F*) separated from methanol-acetone as a microcrystalline solid which melted at 267° to 270°C (decomp.) and showed a specific rotation ($[\alpha]_D^{28}$) of −81 deg in pyridine solution. There was no depression of the melting point on admixture with authentic β-solamarine (m.p. 256° to 259°C) [literature m.p. 275° to 277°C (softens, 270°C), $[\alpha]_D^{20}$ = −85.6 deg (pyridine) (*15*)] and the infrared spectra of the respective samples (KBr pellets) could be superimposed. The respective samples showed identical R_F values upon thin-layer chromatography on silica gel with butanol, acetic acid, and water (4 : 1 : 5, upper phase); ethanol, chloroform, and 1 percent ammonium hydroxide solution (2 : 2 : 1, lower phase); and ethyl acetate, pyridine, and water (3 : 1 : 3, upper phase). Hydrolysis of our sample with methanolic 1*N* hydrochloric acid and recrystallization of the resulting aglycone from acetone gave a product of m.p. 236° to 238°C and $[\alpha]_D^{29}$ −34 deg (methanol). The aglycone was characterized as Δ^5-tomatidenol [literature m.p. 238° to 240°C and $[\alpha]_D$ −37.9 deg (methanol) (*15*)] by mixed melting point and mixed thin-layer chromatographic comparison with an authentic sample (m.p. 237° to 239°C).

β-Solamarine has previously been isolated from *Solanum dulcamara* L. (*15*) and has been assigned the steroid alkaloid glycoside structure shown in Fig. 2 by P. M. Boll (*16*). The tumor-inhibitory activity of steroid alkaloid glycosides does not appear to have been reported previously.

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13. Evaluation of assay results by CCNSC on a statistical basis in sequential testing is such that a material is considered active if it causes reduction of tumor weight to 42 percent or less; for further details see *Cancer Chemotherap. Rep.* **25**, 1 (1962).
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Sex Chromatin of Cone Cells of Human Retina

Abstract. *Each retinal cone cell of the female contains a sex-chromatin body not present in comparable material from the male. Therefore the lack of detectable "patch" formation in female heterozygotes for red color-blindness (as expected from the X-inactivation hypothesis) cannot be attributed to failure of Barr-body formation.*

Most cells of the mammalian female are functionally mosaic for the X-linked genes. This is the result of heteropyknosis of one of the two X-chromosomes in each somatic cell of the female. The evidence for this was originally derived from the finding of a deeply stained body (sex-chromatin mass) in the nuclei of cells of female cats; this body is not present in males (*1*). Moore and Barr (*2*) later suggested that the heteropyknosis of the X-chromosome known to occur in certain insects (*3*) might also occur in mammalian cells, causing the stainable nuclear mass. This suggestion was shown to be essentially correct when it was demonstrated that only one of the two X-chromosomes was condensed at prophase and thus was probably responsible for the sex-chromatin mass (*4*). The cytologic data were substantiated when experiments with tritiated thymidine tracer revealed that one of the X-chromosomes in mammals is labeled asynchronously with respect to the other (*5*).

Under certain circumstances condensation of an X-chromosome in cells of the female does not occur. For example, neither oogonia nor oocytes show a heteropyknotic X-chromosome (*6*). In somatic cells, the condensed X-chromosome is extended during some phase of the mitotic cycle, presumably when replication of DNA takes place (*7*). In the fertilized ovum, the female zygote shows no condensed X until some time after blastulation and im-

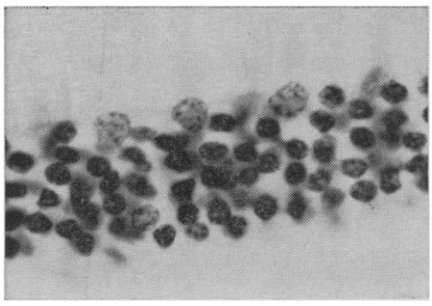

Fig. 1. Retinal layer of male eye, showing nuclei of rods and cones. The nuclei of cone cells are large and vesicular with multiple, minute masses of chromatin.

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

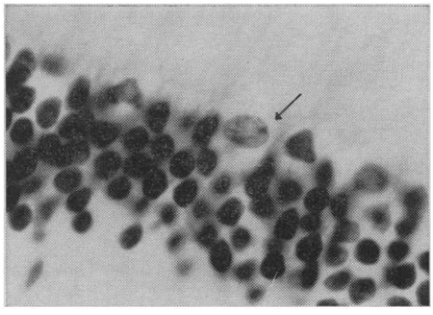

Fig. 2. Retinal layer of female eye. The arrow marks the only cone-cell nucleus that is in focus; the nucleus contains a distinct sex-chromatin body.

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,