

control experiments, normal rats metabolized 50 percent of the administered C¹⁴ to respiratory C¹⁴O₂ during the same period.

Although several hydrazine derivatives will force flowering in pineapple, many will not (3). In our laboratory we have found that methylhydrazine is converted rapidly to methane and CO₂ after administration to intact rats (11). These results may suggest that many hydrazine derivatives are not sufficiently stable to remain intact for very long periods after administration to animals and possibly plants.

Of considerable importance is the effect the 1,1-dimethylhydrazide growth retardants—B-nine (B-995) *N*-dimethylamino succinamic acid and *N*-dimethylaminomaleamic acid (C-011)—may have upon plant diamine oxidase. In this regard the parent compound 1,1-dimethylhydrazine is a potent inhibitor of animal diamine oxidase (12). Work by Martin *et al.* (13) with *N*-dimethylamino succinamic acid-2,3-C¹⁴ (B-nine) shows that although this growth retardant is quite mobile and stable in apple seedlings it does undergo slow decomposition. Dahlgren and Simmerman (14) have reported that C-011 undergoes a slow hydrolysis in aqueous solutions to yield 1,1-dimethylhydrazine and 1,1-dimethylhydrazinium hydrogen maleate. Recent work by Reed, Moore, and Anderson (15)

shows that inhibition of shoot elongation in dwarf and tall peas by B-995 can be correlated with inhibition of tryptamine-2-C¹⁴ oxidation to indoleacetaldehyde-2-C¹⁴ in homogenates prepared from epicotyls of young B-995-treated plants.

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Autosomally Determined Polymorphism of Glucose-6-Phosphate Dehydrogenase in *Peromyscus*

Abstract. *Glucose-6-phosphate dehydrogenase commonly occurs in at least two forms in most tissues of the deer mouse, Peromyscus maniculatus, as demonstrated by starch-gel electrophoresis. An autosomally determined genetic polymorphism in one of the enzymes was discovered. A hybrid molecule occurs in the heterozygote; this suggests a dimeric structure of this enzyme.*

Genetic variants of glucose-6-phosphate dehydrogenase (G-6-PD), demonstrated by altered electrophoretic mobilities in starch gel, have been reported in man (1) and *Drosophila* (2). In both organisms, the gene controlling this enzyme is located on the X chromosome. It has been suggested (2, 3) that X-linkage of the G-6-PD gene may be a general phenomenon and that it may confer some biological advantage which is not now apparent. We report here a G-6-PD polymorphism in the deer mouse, *Peromyscus*

maniculatus, which is controlled by an autosomal gene.

The animals were obtained from a

laboratory population. Some, descended exclusively from mice trapped in Washtenaw County, Michigan, by Van T. Harris in 1946 and 1947, belong to the subspecies *P. m. bairdi*. Others are hybrids between members of this stock and other subspecies of *P. maniculatus*. Animals from four distantly related sibships were examined, together with their parents. All animals were at least 2 months old, and females were not pregnant or lactating.

The mice were killed by decapitation; the organs were removed immediately, washed in cold physiological saline solution, blotted, weighed, and homogenized in a Kontes glass tissue-grinder with a measured amount of distilled water. Cell walls were ruptured by high-frequency sound. Solid material was removed by centrifugation at 25,000g for 10 minutes at 4°C. The clear supernatant was pipetted into the starch-gel insert slots in 25- μ l amounts.

At least two different zones of G-6-PD activity were found in all tissues examined except erythrocytes. These were arbitrarily designated as A and B enzymes, the A form being the more anodally migrating (Fig. 1). Both occurred in liver, kidney, testis, muscle, lung, heart, and brain. In erythrocytes, only the A form was present.

In three of the four families studied, a polymorphism occurred which involved the B enzyme. Three phenotypes were observed: in two, B-a and B-b, there was a single band at one of two different positions; in the third, phenotype B-ab, bands appeared at both of these two positions with a third halfway between (Fig. 1). In this type, the middle band was relatively more intense than the other two.

Table 1 shows the results of the four matings. Although the hypothesis of partial sex-linkage of the gene controlling the B enzyme of G-6-PD cannot be excluded, complete sex-linkage involving a single locus is ruled out because in one sibship, from a mating between B-ab animals, males of all three phenotypes appeared. Additional

Table 1. Glucose-6-phosphate dehydrogenase patterns of parents and offspring in four matings.

G-6-PD phenotypes of parents		G-6-PD phenotypes of offspring							
		B-a		B-ab		B-b		Total	
♀	♂	♀	♂	♀	♂	♀	♂	♀	♂
B-b	B-b					4	2	4	2
B-a	B-ab	1	3	1	1			2	4
B-ab	B-b				4	2	1	2	5
B-ab	B-ab	1	2	2	5		2	3	9

evidence against this hypothesis is the fact that B-ab males when mated with either B-a or B-ab females sired B-a as well as B-ab daughters. This result is not expected under any simple hypothesis in which males are hemizygous for the gene under consideration. All of the observed results, on the other hand, are in agreement with expectation for single-locus, autosomal inheritance.

Genetic evidence supports the hypothesis that the gene producing enzyme B occurs as two alleles, the polypep-



Fig. 1. Starch gel showing liver glucose-6-phosphate dehydrogenase of three animals. Anodal direction is upward. A and B enzymes are indicated, with the three different B types shown: B-a and B-b are the two homozygotes; B-ab is the heterozygote. Vertical electrophoresis was carried out for 18 hours at a gradient of 8 volt/cm at 40°C. The gel buffer was a tris-EDTA-borate mixture, pH 8.0, total molarity 0.05. The same buffer at 0.5M was used in the bridge boxes. Incubation mixture for demonstration of the enzyme activity contained per 100 ml: triphosphopyridine nucleotide, 70 mg; nitro-blue tetrazolium, 50 mg; phenazine methosulfate, 2 mg; NaCN, 5 ml, 0.1M; tris buffer, pH 6.8, 10 ml; glucose-6-phosphate, 5 ml, 1.0M; gels were incubated for 2 to 4 hours at 35°C.

ptide products of which are here called the B^a and B^b subunits. The two single-band phenotypes, B-a and B-b, are considered to be homozygous forms and the three-band type, B-ab, the heterozygous form, with the intermediate band representing a "hybrid" molecule, B^{ab}.

The occurrence of a hybrid molecule in the heterozygote indicates that the B enzyme is probably a dimer composed of randomly associating subunits (4). In such a system, the hybrid dimer would theoretically be present in twice the amount of each of the homogeneous dimers in the heterozygote, and the increased intensity of the middle band in B-ab phenotypes fits this scheme. In the G-6-PD variants of human erythrocytes, heterozygotes do not have an intermediate, hybrid molecule; this is evidence that that enzyme and the B enzyme of *Peromyscus* are not homologous.

The fact that the B enzyme variation is not correlated with any alteration in the A enzyme indicates that the two molecules are controlled by different genetic sites. Whether the A enzyme is X-linked cannot now be determined. That *Peromyscus* erythrocytes contain only the A enzyme suggests that the A form might be homologous with the X-linked G-6-PD of human erythrocytes.

Both B-b and B-ab phenotypes were found in the stock of *P. m. bairdi*. The observed polymorphism, therefore, is present within this subspecies and is not entirely the result of bringing together genes from different subspecies by hybridization.

The enzyme 6-phosphogluconate dehydrogenase (6-PGDH), which catalyzes a subsequent metabolic step, sometimes produces a faint zone of activity in gels incubated with glucose-6-phosphate. To check the possibility that the B enzyme might be 6-PGDH, the specific substrate, 6-phosphogluconate, was used in place of glucose-6-phosphate. No activity occurred at the B position, but a strong zone developed at a position anodal to the A enzyme, an indication that 6-PGDH is distinct from both of the G-6-PD forms.

Tissue culture studies were done to investigate the applicability of these enzyme systems as genetic markers in cell culture. Diploid fibroblast lines were established from skeletal muscle of a B-a homozygote and from kidney of a B-b homozygote. Both A

and B enzymes were present in the cultured cells, and the B polymorphism was clearly demonstrated.

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Geoelectric Effect and Geotropic Curvature

Abstract. *A transverse electrical potential develops in many plant organs after they are reoriented from a vertical to a horizontal position. This phenomenon has been the basis for both a geoelectric hypothesis of geotropic curvature and its subsequent refutation. In both arguments a causal relation between the potential and curvature has been assumed. Curvature occurs in the absence of potential, as well as vice versa; it is concluded that both the hypothesis and its refutation are incorrect.*

A difference in electrical potential between the upper and lower sides of the shoot and root tips of many plant species develops after they are reoriented from a vertical to a horizontal position. This phenomenon is known as the geoelectric effect. In contemporary (1, 2) as well as earlier studies (3) experimenters have sought to relate this phenomenon causally to a lateral redistribution of the growth hormone, auxin, and thence to geotropic curvature. Recently, it was concluded that the geoelectric effect is a secondary consequence of the asymmetric distribution of auxin, and that the geoelectric hypothesis of geotropism is incorrect (4).