behavior accordingly. It appears therefore that a sensing function becomes attached to a function which has survival value, and the sensing function is then processed through a central system to affect behavior of the organism.

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Phosphoribosylpyrophosphate Synthetase Is Elevated in Fibroblasts from Patients with the Lesch-Nyhan Syndrome

Abstract. *In subconfluent cultures of fibroblasts from patients with complete or partial deficiencies of hypoxanthine-guanine phosphoribosyltransferase, phosphoribosylpyrophosphate synthetase activity is elevated. The abnormally high catalytic activity of the synthetase appears to account for the overproduction of purines by the cultured mutant cells and presumably for that by the patients.*

A deficiency of the catalytic activity of the purine salvage enzyme, hypoxanthine-guanine phosphoribosyltransferase (HGPRT), has been described in tissues and cultured cells from patients with the Lesch-Nyhan syndrome, a heritable syndrome of cerebral palsy, self-mutilation, and hyperuricosuria (*1, 2*).

The variable clinical and biochemical descriptions of HGPRT deficiencies in man (*3, 4*) include one consistent finding—the excessive urinary excretion of uric acid, a result of overactive de novo purine biosynthesis (*4–6*).

The biochemical basis of purine overproduction in human HGPRT deficiencies appears to be the increased intracellular concentrations of phosphoribosylpyrophosphate (PRPP) (*7, 8*), which serves as both a substrate and allosteric regulator (*9*) of de novo purine synthesis. The increased cellular concentration of PRPP has been ascribed to decreased utilization by HGPRT, the deficient purine salvage enzyme that has been considered to be a major intracellular consumer of PRPP. However, males with partial deficiencies of HGPRT enzyme activity overproduce purines and uric acid to extents (on the basis of body mass) equal to or even greater than many patients with the complete enzyme deficiency (*3, 5*). Thus, there is not a strong correlation between the degree of deficiency of HGPRT in the erythrocytes and the degree of purine overproduction.

We have observed increases of apparently normal PRPP synthetase enzyme in cultured rat hepatoma (HTC) cells with chemically induced HGPRT deficiency (*10*). We proposed that the enhanced rate of de novo purine synthesis in these mutant cells was due to increased PRPP synthetase activity rather than to the loss of activity of the HGPRT and any reduced PRPP utilization (*10*).

Our study was undertaken to determine whether mechanisms similar to those in mutant rat hepatoma cells might be responsible for the purine overproduction in cultured fibroblasts from patients completely or partially deficient in HGPRT activities. We demonstrate that the PRPP synthetase activity is greater in the mutant than in normal fibroblasts. This abnormality is present when the cultures are subconfluent but not when confluent. The rates of de novo purine synthesis measured during the growth phases of the cells change in parallel with the levels of PRPP synthetase activity. Furthermore, immunotitration data from two mutant cell cultures suggest that the increase of PRPP synthetase activity results from increased numbers of PRPP synthetase molecules.

The specific catalytic activities of PRPP synthetase were significantly elevated in gel-filtered extracts of cultured fibroblasts from patients with the Lesch-Nyhan syndrome when compared to those from normal young males (Fig. 1). In experiments in which cells were harvested 4 and 5 days after subculture, seven of the eight cultures from affected individuals had significantly greater PRPP synthetase than cultures from the matched controls (Fig. 1). The fibroblasts from patient S.S. were reexamined in a growth cycle experiment (see below) and were not exceptional. The levels of adenosine kinase, another soluble cytoplasmic enzyme, were the same in both the mutant and control cells.

However, further investigation indicated that the measurable levels of PRPP synthetase activity fluctuated in both mutant and normal fibroblasts and that the elevations in the mutant fibroblasts were not consistent. The previously described induction of PRPP synthetase during the activation of quiescent mouse spleen lymphocytes (*11*) suggested that the harvesting of fibroblasts at different stages of growth might be responsible for these fluctuations.

In three experiments we harvested and assayed mutant and control cultures simultaneously at three or four different stages of growth, where each stage was a fixed period after subculturing (by trypsinization and dilution) (Figs. 2 and 3). The consistent differences of activity in the examined mutant cells and that of their respective controls occurred between 2 and 5 days after dilution. However, the specific catalytic activities of the next enzyme in the sequence of the de novo purine pathway, PRPP glutamyl amidotransferase, varied by less than 25 percent both between mutant and normal cells and between cells harvested throughout the growth cycle. The rates of de novo purine synthesis were examined at various times throughout the growth cycle and compared to the rates in normal fibroblasts at similar times.

The rates of de novo purine synthesis also fluctuated throughout the growth cycle in a pattern parallel to that of PRPP synthetase (Fig. 2D). Thus, in the cultured fibroblasts from a series of unrelated patients with the Lesch-Nyhan syndrome the PRPP synthetase catalytic activity and the rates of purine synthesis were elevated during the rapidly growing or subconfluent stages after subculture and dilution. These observations seem analogous to those made in cultured rat hepatoma cells containing similar but chemically induced mutations (*10*). Furthermore, our results reported earlier (*12*) have been corroborated by Reem (*13*), who used cultured lymphoblasts deficient in HGPRT activity.

If, as has been proposed (*10, 12*), the HGPRT gene codes for both catalytic and regulatory functions, a mutation that results in partial deficiency of HGPRT catalytic activity might also alter the regulatory function of the gene, causing an increase of PRPP synthetase. This increase would not necessarily correlate with the degree of catalytic malfunction of the HGPRT enzyme. Fibroblasts from patient G.B., with gout and partial deficiency (20 to 25 percent residual) of HGPRT, and from patient C.M., with gout and a mutant HGPRT enzyme molecule with a normal V_{max} but elevated K_m for PRPP (*14*), were examined at subconfluency for PRPP synthetase activity. In comparison to control cells, the fibroblasts from these two patients also showed increased PRPP synthetase activity (650 and 790 nmole of product per hour per milligram of protein, respectively, compared with control values of 225 to 290 nmole of product per milligram of protein).

By analogy with HTC cells (*10*), it seemed possible that the increased PRPP synthetase activity in the mutant fibroblasts would result from increased numbers of normal enzyme molecules. If so, then the PRPP synthetase catalytic activity per enzyme molecule should be equal in control cells and in mutant cells. Specific antiserum against purified human PRPP synthetase (*15*) was used to determine the enzyme from mutant (G.B. and C.M.) and control (A-368) fibroblasts at subconfluency. The extracts were diluted to contain approximately equal catalytic activities per unit volume. The slopes of the curves in Fig. 4 are parallel, confirming the expectation that the increased enzyme activities in these mutant cells were accompanied by corresponding increases of PRPP synthetase antigen. Thus, the increased activity was apparently not the result of a mutation in the structural gene for PRPP synthetase conferring an enhanced V_{max} for the corresponding enzyme molecule. Such an alteration has been described in one family with overproduction hyperuricemia (*15*), in which the increased catalytic activity per enzyme molecule was demonstrable by the identical antiserum that we used (*15*).

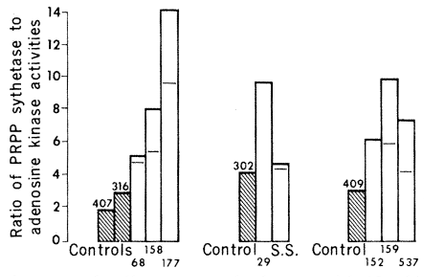

Fig. 1. The catalytic activities of PRPP synthetase relative to adenosine kinase in fibroblasts from Lesch-Nyhan patients and normal human males. Inocula of cultured fibroblasts (*21*) were grown at 37°C as monolayers in Falcon flasks (75 cm^2) in RPMI 1640 medium containing 15 percent fetal calf serum (Flow Laboratories) without antibiotics. The cultured cells were trypsinized and diluted tenfold; 3 to 6 days later, cells in duplicate flasks were chilled on ice and harvested by the abrasive action of about 30 glass beads (3 mm in diameter), the harvested cells were washed once with 10 ml of cold, buffered isotonic saline, centrifuged, quick frozen, and stored as pellets at −20°C for 4 to 36 hours. The pellets were thawed in the assay buffer [50 m*M* potassium phosphate, 6 m*M* $MgCl_2$, 0.1 m*M* dithiothreitol, 250 μM adenosine triphosphate (ATP), and 10 percent (by volume) glycerol, *p*H 7.4], frozen and thawed again twice, and then subjected to gel filtration at 4°C on 10-ml columns of Sephadex G25 (fine) equilibrated with the same buffer. The material eluting in the void volume of each column was then assayed for PRPP synthetase, adenosine kinase, HGPRT, and protein. The method of Kornberg (*22*) modified as described (*23*) was used to estimate the specific catalytic activity of PRPP synthetase. Adenosine kinase activity was determined by incubating 10 to 100 μg of protein of the crude extract in a volume of 300 μl containing 0.3 μmole of $MgCl_2$, 0.15 μmole of ATP, 15 μmole of potassium phosphate (*p*H 7.4), 0.03 μmole of dithiothreitol, and 0.3 μc of [^{14}C]adenosine (20 μc/μmole; New England Nuclear) at 37°C. At 0, 5, 10, and 20 minutes, 50-μl portions were removed and placed on DE-81 25-mm discs (Reeve-Angel). The discs were washed six times in 2 liters of deionized water and the adenosine monophosphate (AMP) retained was determined by direct counting in a liquid scintillation system. The assay was linear with time and quantity of protein added, and was dependent on the presence of ATP; the product was identified as AMP by thin-layer chromatography. The initial velocities were estimated from the slopes of the AMP accumulation. The means and standard errors (lower bars) of the PRPP synthetase activities are shown relative to adenosine kinase activities, which serve as controls for variable lysis and gel filtration recoveries. The adenosine kinase activities per milligram of protein varied less than 25 percent between samples. The results are grouped by donor age and cell passage number [see (*25*)].

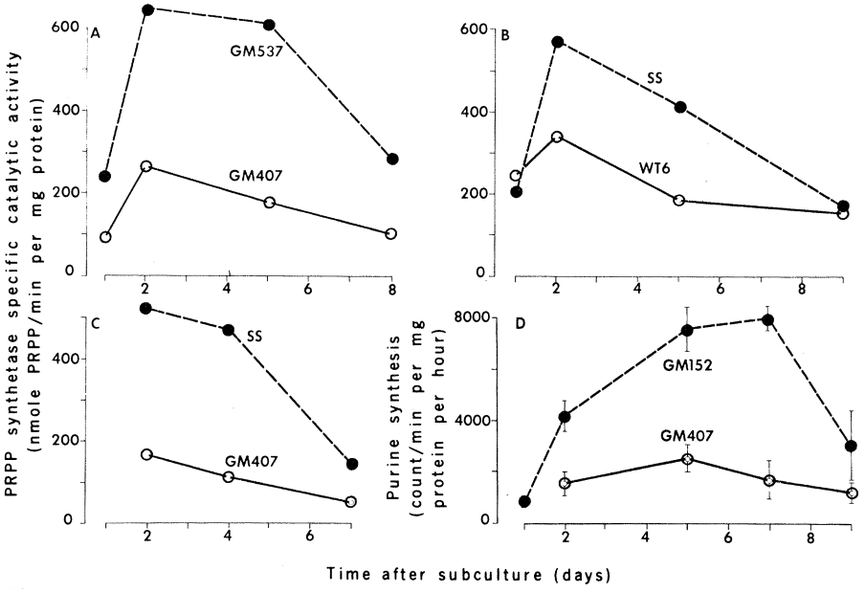

Fig. 2. The PRPP synthetase catalytic activities and rates of purine synthesis during fibroblast growth cycle. Monolayers of cultured fibroblasts from normal controls and Lesch-Nyhan patients (*25*) were subcultured (by trypsinization and tenfold dilution) and, by proper staging, were simultaneously harvested at various periods after the subculturing. Each culture had a complete change of medium within 48 hours of harvest. Extracts from the washed cell pellets were prepared and assayed for PRPP synthetase activities and protein concentrations (Fig. 1 legend). (A–C) The specific catalytic activities are plotted as a function of time (days) after subculture. (●--●) The values from the Lesch-Nyhan fibroblasts; (○——○) values from normal fibroblasts. WT6 cells are from a normal 3-year-old male donor, passages 9 through 10. (D) The rates (means and standard deviations) of purine synthesis from two independent experiments, each point involving duplicate monolayer cultures labeled with 10 μc of [^{14}C]glycine (uniformly labeled, 100 μc/μmole; New England Nuclear) per milliliter for 60 minutes. The accumulations of ^{14}C in purines were determined as described (*24*).

The discovery (*2*) of the deficiency of HGPRT activity in patients with the Lesch-Nyhan syndrome and the observations (*7*) of increased concentrations of intracellular PRPP in cells from affected patients were consistent with the conclusion that the enzyme deficiency directly caused the accumulation of one of its substrates, PRPP. In addition, the observations were taken as evidence that the salvage of hypoxanthine or guanine (or both) in humans is a major consumer of PRPP. The increased intracellular concentrations of PRPP can account for the observed enhanced rates of purine synthesis de novo in the mutant cells since PRPP serves as both a substrate and positive allosteric regulator (*9*) for purine synthesis. However, our observations of elevated concentrations of PRPP synthetase in HGPRT mutant fibroblasts readily account for the increase in PRPP, and can also explain why the PRPP concentration is as high in cells with 15 percent of HGPRT activity (*16*), as in those totally lacking HGPRT activity (*7*). The elevation of PRPP in erythrocytes of patients deficient in HGPRT is not easily explained by our observations. Therefore, the quantitative importance of purine salvage activity in the various human tissues must be reevaluated.

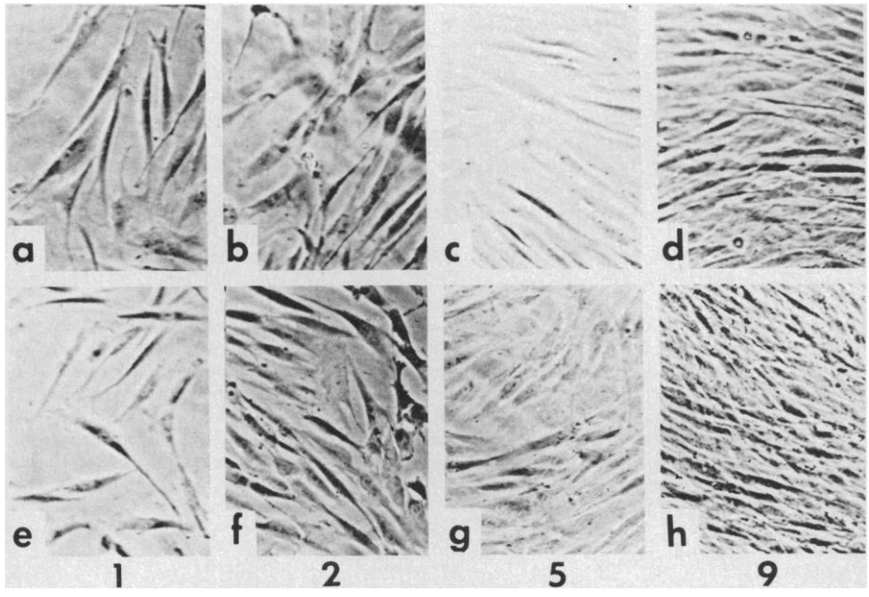

Fig. 3. Photomicrographs of representative fields of wild-type (WT6) and HGPRT-deficient (S.S.) fibroblasts. The monolayer cultures of fibroblasts used for the experiment in Fig. 2B were photographed a few minutes prior to harvest through an inverted phase contrast microscope (Nikon) with Tri-X Pan film (Kodak) (100 ×). (a to d) S.S., the mutant cultures; (e to h) WT6, the wild-type cultures. The numbers at the bottom indicate the time in days after subculture.

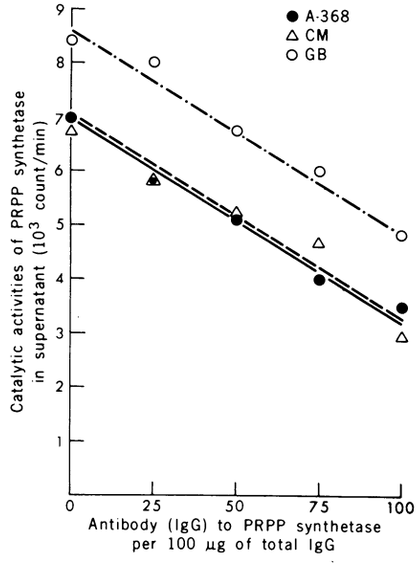

Fig. 4. The PRPP synthetases in fibroblasts from control and two patients with partial deficiencies of HGPRT activities. Subconfluent monolayer cultures of fibroblasts from a normal male fetus (A-368) and two male patients with partial functional deficiencies of HGPRT [C.M. has normal V_{max} but altered K_m values of HGPRT (*14*); and G.B. has HGPRT specific catalytic activity of 23 nmole of PRPP per hour per milligram of protein] were harvested, and crude extracts of these were prepared as described (Fig. 1 legend). The PRPP synthetase activities (in nanomoles of PRPP per hour per milligram of protein were A-368 cells, 255; C.M., 790; G.B., 650) were adjusted by dilution in assay buffer to be approximately equal per unit volume. To each of five tubes of each sample was added 100 μg of rabbit IgG with an increasing amount of specific antibody IgG to human PRPP synthetase (*15*). The tubes (final volume, 250 μl) were incubated at 37°C for 1 hour and chilled for 1 hour. To each was then added an appropriate quantity of goat antibody IgG to rabbit Fc fragment in order to precipitate the rabbit IgG. A further 15-minute incubation at 37°C was included and then 2 hours at 0°C. The precipitate was removed by centrifugation, and the PRPP synthetase activity remaining in each supernatant was determined. The remaining activities are plotted as a function of relative fraction of antibody IgG to PRPP synthetase added to incubations. The extracts were from A-368 (●), C.M. (△), and G.B. (○).

Our data support our previous hypothesis (*10*) that the gene coding for HGPRT has two functions. It codes for an enzyme that catalyzes the salvage of hypoxanthine and guanine to their respective ribonucleotides, and it regulates the intracellular concentration of the PRPP synthetase. The overproduction of purines could then be the result of an altered regulatory function of the HGPRT gene, a function which, according to our observations, might normally control the intracellular concentration of PRPP synthetase and thus purine synthesis de novo (*9*, *17*). The hypothesis predicts the existence of other HGPRT mutant phenotypes. If the HGPRT gene is bifunctional, one might expect to observe alterations in both functions, or in either separately, each with a distinctive phenotype.

The concentration of PRPP synthetase activity in cultured normal human fibroblasts fluctuates severalfold during the growth cycle. The level in fibroblasts possessing mutant HGPRT genes fluctuates to an even greater extent but the basal level of PRPP synthetase activity is quite similar to that in normal cells. This suggests that the altered regulatory system in the HGPRT mutants is normally responsible for controlling at least the maximum level of PRPP synthetase activity while other factors may be responsible for its regulation during cell growth. Our laboratory (unpublished observations) and others (*18*) have not observed any consistent fluctuation of HGPRT activity during the growth cycle of cultured normal fibroblasts. The fluctuation of PRPP synthetase activity during the cell growth cycle is not observed in the one established cell line (HTC) that we have examined closely, whether the HGPRT is mutant or not.

In the yeast *Saccharomyces cerevisiae* there is an excellent analogy to the proposed bifunctional nature of the HGPRT gene in humans and rats. The product of the *Ade 12* gene of *S. cerevisiae* is adenylosuccinate synthetase—an enzyme responsible for the first of two steps converting inosine monophosphate to adenosine monophosphate (*19*). The *Ade 12* gene product also regulates the de novo purine pathway, apparently by serving as an aporepressor of the same pathway in which it serves as a catalyst (*19*). The possible existence of a bifunctional catalytic and regulatory gene in humans has

interesting evolutionary implications, some of which have been discussed in the context of lower organisms (*20*).

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25. The descriptions of the fibroblast cultures are as follows:

Designation	Age (years)	Passages used	HGPRT (nmole/hr-mg)
		Normal donors	
GM 302	0.8	7 to 12	103
GM 316	7	4 to 10	88
GM 407	10	9 to 15	100
GM 409	12	9 to 14	107
		Lesch-Nyhan donors	
GM 29	0.7	9 to 14	< 2
GM 68	2	15 to 18	< 2
GM 152	9	11 to 16	< 2
GM 158	3	10 to 15	< 2
GM 159	10	12 to 17	< 2
GM 177	6	10 to 16	< 2
GM 537	12	9 to 15	< 2
S.S.	3	3 to 10	< 2

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Northern Fur Seal Diving Behavior: A New Approach to Its Study

Abstract. *A new type of depth-time recorder was used to monitor behavior of fur seals at sea. During 608 hours, 2957 dives were recorded for four animals. The deepest dive was 190 meters, and the longest submersion was 5.6 minutes.*

Recently, it became necessary to assess the behavior of the northern fur seal, *Callorhinus ursinus*, in the Bering Sea in order to predict the effects of potential oil spills near their breeding grounds. Remote acquisition of information about diving behavior has been done by only a few investigators. Evans (*1*) surveyed some small whales by attaching radio transmitters to their dorsal fins. The signal was monitored with a receiver on a nearby ship or aircraft. Similarly, Norris and Gentry (*2*) captured and placed harnesses and transmitters on gray whale calves, *Eschrichtius robustus*, in a breeding lagoon of Baja California, Mexico. The calves were then followed by ship as they left the lagoons. These methods require tracking by ship and aircraft, which is not only expensive, but also practical only in areas with good port and air facilities and where good weather is common. Such requirements are not met in the Bering Sea.

Depth recorders (*3*) and mechanical depth-time recorders (*4*) have been placed on the antarctic Weddell seal, *Leptonychotes weddelli*. When the seals, who were diving under ice, returned to the known ice holes in order to breathe, the instruments could be removed and the data recovered. The depth recorders provided only the maximum depth of all the dives made while the recorder was attached and therefore did not provide enough information for our purposes. The depth-time instrument's 4-hour recording duration was too short for the northern fur seal study.

We now present some preliminary results gathered with a new instrument. The instruments we designed and built for this project could continuously record every dive for 8 days (*5*). A depth transducer plots a record on pressure-sensitive paper, which is transferred from one spool to another by an electric motor. The mechanism is housed in an aluminum waterproof container weighing 650 g and measuring 5 cm in diameter by 17 cm long. The instruments were attached to a harness worn by the seals. The two measures (depth and time) provide a variety of information, such as the time feeding begins after the seals depart from the rookery, circadian activities, diving effort, and diving depth.

Lactating females seemed the best animals to equip with this recorder since (i) they form the largest segment of the population, (ii) at the time of our study (summer) they spend more time at sea than on shore, and (iii) their movements ashore are predictable in space and time (*6*). This third point was important for ensuring a low probability of instrument loss. In essence, the females transported our instruments and gathered data for us while we waited ashore for their return.

Our data are summarized as the number and depth of dives observed (Table

Table 1. Summary of diving depth and frequency from 608 hours of monitoring at sea.

Fur seal	Body weight estimate (kg)	Recording time (hours)	Number of dives to: 0 to 20 m	21 to 50 m	51 to 80 m	81 to 110 m	111 to 140 m	141 to 170 m	171 to 200 m	Total dives
1	35	167	301	314	37	9	13	2		676
2	35	92	1148	1	10	9	49	7	2	1224
3	35	200	54	478	89	1	4			626
4	50	149	201	200	14	7	6	3		431