Scientific societies should foster discussion of the means of scientific communication, develop clear codes of scientific ethics, and publicize information on the actual status of claims or discoveries, particularly when widespread publicity is being given to unproved or false claims.

Science should be interpreted to society generally through the use of "progress reports" on science which are aimed at demonstrating the methodical processes of research, the pitfalls and disappointments, and the philosophy of objective reasoning. Science news consists as much in the processes as in the final results. The effort required for such interpretation is not the obligation of scientists alone, but must be augmented by all the means described in the preceding paragraphs and by better interpretations in the mass media. It is an encouraging sign that science newswriters generally recognize these obligations and problems. Closer cooperation between scientists and science writers would result in a better general public understanding of science and scientific evidence.

Educators should explain the demands of scientific objectivity to their students. The mere teaching of subject content alone will not give assurance that the academically trained scientist is aware of the pitfalls of premature claims or the proper relations between science and society. Each future scientist should be taught the responsibilities of his position as a representative of science to society generally.

Every practicing scientist should reflect seriously on his own opportunities to assist in representing to the public the way in which science advances, the need for tests of the validity of conclusions, the logical processes of science, the demands for objectivity, the need for adequate and valid data, and the difference between claims and proved results. His own research reports should be models of objectivity and clarity. Performing this duty might not result in personal rewards, but scientists have a unique responsibility to see that false opinions do not eclipse the accurate information needed for progress.

Public confusion about the meaning of scientific work must eventually produce a negative reaction. Exaggeration and overselling in order to gain financial support for science will ultimately stand revealed. Unless such fringe practices have been publicly and specifically disowned by legitimate scientists, the reaction may affect all of science; then public confidence, understanding, and support may vanish. In that event, the scientific community may deeply regret having neglected to clarify the nature of science in the public mind. Scientific societies and individual scientists can lead to a solution by attracting more attention to the progress of science and by communicating with one another and with the rest of society in completely candid terms.

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Biochemical Mutations in Man and Microorganisms

Herman M. Kalckar

It is a well-known fact that normal development of mammals is possible without an external supply of galactose. Galactoside synthesis, especially of the complex galactosides that are constituents of cellular structures, is an essential feature of normal growth and development. Mammals, like most organisms, are able to convert glucose to galactosides. The galactolipids, for example, which constitute a large bulk of the brain, are examples of structural galactosides that are deposited exclusively after birth (*1*). As will be discussed in subsequent paragraphs, these compounds can be synthesized from dietary glucose as well as from dietary galactose.

The dispensability of galactose raises the question of why lactose is ubiquitous in mammalian species. It is possible that early in their evolution mammals were subjected to influences that made it advantageous for them to produce milk containing lactose for their progeny. The possible advantages of lactose in the diet of the progeny have not been explored. The influence of lactose on the bacterial flora of the gastrointestinal tract should certainly be considered, for the microorganisms in the intestine play a role in making certain vitamins available or unavailable to the host. It is known that replacement of lactose by sucrose in the diet greatly increases the need of the host organism for vitamins such as pyridoxine and riboflavin (*2*).

If galactose is administered externally, it is largely used as a fuel through conversion to glucose-6-phosphate. Most microorganisms use galactose for this purpose provided that they are able to adapt and that they cannot get access to glucose. Part of the administered galactose is used, as mentioned, for the synthesis of cellular galactosides.

Complexity of Galactose Metabolism

The activation of galactose, unlike that of glucose, is initiated through a direct phosphorylation of the reducing group, giving rise to α-galactose-1-phosphate (G-1-P) (Kosterlitz, *3*). The metabolic mobilization of galactose, also unlike that

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This article is one of a group of four articles on galactose metabolism that appear in this issue. The other three articles appear at the beginning of the "Reports" section.

of glucose, requires an additional step beyond the phosphorylation step. The galactose-1-phosphate formed must be incorporated into a specific nucleotide of the type first discovered by Leloir and his coworkers (*4*). This is a peculiar "mongrel" of a dinucleotide in which 5-uridylic acid is linked to the phosphate of either α-glucose-1-phosphate [Cori ester (G-1-P)] or α-galactose-1-phosphate (Gal-1-P). This mongrel nucleotide is called uridine diphosphoglucose (UDPG) or uridine diphosphogalactose (UDPGal) (*5*).

The enzymatic interconversion of uridine diphosphoglucose and uridine diphosphogalactose was first described in crude extracts of galactose-adapted yeast (*6*). The enzyme was called "galacto-waldenase" in accordance with the term *galacto-walden inversion,* which had been used for a long time in reference to the interconversion of galactose to glucose.

At this point it is necessary to spend a few lines on terminology. The term *galacto-walden inversion* was never satisfactory because it implied that an "umbrella" type of inversion (first described by Walden) was operating on the rearrangement of the 4-hydroxyl group of galactose (or glucose), but this has never been proved to be the case. On the contrary, recent studies in our laboratory on highly fractionated galacto-waldenase from calf liver have shown an absolute requirement for a hydrogen-transferring system, namely diphosphopyridine nucleotide (*7*).

This observation points strongly in favor of an oxidation-reduction mechanism as the basis of the rearrangement (compare also *8*). We therefore propose the more correct and descriptive name, uridine diphosphogalactose 4-epimerase, for the enzyme that catalyzes the inversion, to replace the old name, galacto-waldenase (*9, 10*).

Congenital Galactosemia in Man

It is known that infants afflicted with the heritable human disease, congenital galactosemia, which deprives them of the ability to metabolize galactose, improve dramatically when they are put on a strict galactose-free diet (*11*). A lack of "galacto-waldenase" has often been cited as the probable biochemical lesion in galactosemia; especially after the observation was made that galactose-1-phosphate accumulates in the blood cells of galactosemic subjects (*12*). Such a biochemical lesion would, however, deprive the infants of the ability to make brain galactosides from glucose or other nutrients (*13*) and limit them to using administered galactose, which is known to be able to serve as a source of galactolipids (*14*). Whether an organism with such a deficiency and without any external supply of galactose would undergo normal growth and development is doubtful. An alternative would be deposition of glucolipids instead of galactolipids in brain. Would this be compatible with normal brain function?

It seems worth while to try to explore these problems in properly designed animal experiments. However, this aspect has become irrelevant in a discussion of the disease congenital galactosemia, for it has recently been shown that the enzyme block does not affect the uridine diphosphogalactose 4-epimerase proper (*15*) but affects another totally different type of enzyme. This is the enzyme that catalyzes the step prior to the inversion—that is, the incorporation of galactose-1-phosphate into uridine nucleotides according to the equation (*16, 17*)

$$\text{Gal-1-P} + \text{UDPG} \rightleftharpoons \text{G-1-P} + \text{UDPGal}$$

This enzyme, which "trades" the glucose-1-phosphate moiety of uridine diphosphoglucose for galactose-1-phosphate, can best be classified as a uridyl transferase (*18, 19*) in which galactose-1-phosphate or glucose-1-phosphate take their turn as nucleophilic agents for the uridyl fragment.

Another type of uridyl transferase affecting uridine diphosphoglucose, which was first described in yeast that was not adapted to galactose, catalyzes the following reaction (*18, 19*):

$$\text{UTP} + \text{G-1-P} \rightleftharpoons \text{UDPG} + \text{PP}$$

Here inorganic pyrophosphate (PP) takes the place of galactose-1-phosphate as the alternating nucleophilic reagent. The enzyme catalyzing this reaction belongs to the class that we have called pyrophosphate uridyl transferases because inorganic pyrophosphate is involved. Since there are other pyrophosphate uridyl transferases (*20*), it would be best to name the specific catalyst of the reaction "uridine diphosphoglucose pyrophosphorylase," in line with Kornberg's diphosphopyridine nucleotide pyrophosphorylase (*21*).

New Terminology

Let us summarize the pertinent points to recapitulate the new terminology.

1) Galactokinase catalyzes the phosphorylation of the reducing group of D-galactose (Gal.)

$$\text{Gal} + \text{ATP} \rightarrow \text{Gal-1-P} + \text{ADP} \quad (1)$$

2) Galactose-1-phosphate uridyl transferase catalyzes the incorporation of galactose-1-phosphate into a nucleotide.

$$\text{Gal-1-P} + \text{UDPG} \rightleftharpoons \text{G-1-P} + \text{UDPGal} \quad (2)$$

This is the catalyst that is defective in congenital human galactosemia.

3) Uridine diphosphogalactose 4-epimerase, having diphosphopyridine nucleotide as coenzyme, catalyzes the epimerization of the 4-hydroxyl group of the hexose moiety, presumably through an oxidation-reduction process in which a 4-keto sugar nucleotide (and reduced diphosphopyridine nucleotide) is a transitory product.

$$\text{UDPGal} \rightleftharpoons \text{UDPG} \quad (3)$$

4) Uridine diphosphoglucose pyrophosphorylase catalyzes the release of glucose-1-phosphate from uridine diphosphoglucose.

$$\text{UDPG} + \text{PP} \rightleftharpoons \text{UTP} + \text{G-1-P} \quad (4)$$

However, this can also be accomplished in reaction 2. More important, presumably, is the reaction from right to left in which uridine diphosphoglucose is synthesized from uridine triphosphate (UTP) and glucose-1-phosphate (*18*) or from uridine diphosphate, adenosine triphosphate (ATP), and glucose-1-phosphate (*22*). This is probably how the key substance, uridine diphosphoglucose, is initially formed in cellular metabolism.

Genes and Enzyme Synthesis

As mentioned here and in our earlier publications, the enzymes that catalyze steps 1, 3, and 4 are present in blood cells of galactosemic subjects as well as in normal cases. Only the enzyme catalyzing step 2 is defective. It should be emphasized that, although we have been able to demonstrate this major defect of galactose-1-phosphate uridyl transferase in more than 15 cases of this disease, it would be premature to classify even these cases as necessarily belonging to a biochemically homogenous disease. This is well illustrated in Kurahashi's article in this series (*23*). Kurahashi has found that among a number of galactose-negative mutants of *Escherichia coli,* K_{12} strain (*24*), four different mutants are lacking galactose-1-phosphate uridyl transferase. Although they all show the same biochemical defect, Morse, Lederberg, and Lederberg were able to show by means of their genetic techniques (*25*) that they are due to alterations in different, though closely located genes.

Yanowsky (*26*), working on *Neurospora,* was able to resolve the manifestations of such mutants by immunochemical techniques. In this work, instances are described in which the enzymatic activity was lost but in which the antigenic activity—that is, the ability to bind antienzymes—was preserved. Such a study is planned for the bacterial mutants in order to show the possible existence of

incomplete enzymes. This study may provide additional information about gene action and protein synthesis. It is sufficient at present to state that at least four genes are needed in *Escherichia coli* in order to insure a normal functioning galactose-1-phosphate uridyl transferase. Offhand, there is certainly no reason to believe that man should require fewer genes for the same purpose. Furthermore, there is no reason either to believe that the same gene is affected in different, unrelated families. Hence, there is obviously margin for biochemical variations if methods with finer resolutions are found.

The immunochemical approach seems particularly promising, but there are other possibilities that can most readily be tried on the bacterial mutants. If, for instance, one of the mutants lacks the ability to synthesize a cofactor but can synthesize the protein, the biochemical defect would look like a lack of galactose-1-phosphate uridyl transferase if the standard technique was used. However, if extracts of this mutant were combined with protein-free filtrate of a normal strain which would supply this cofactor, activity might be restored.

These considerations have direct bearings on the approach in a further study of human galactosemia. We had earlier tried such a type of extract-recombination experiment in two galactosemic families, but we found no reactivation (*17*). However, for the reasons just stated, such reactivation attempts should be applied for each galactosemic family studied, for each family would count as a different case despite the seemingly identical enzymatic defect.

Congenital Galactosemia, a General Tissue Defect

The importance of assaying more than one type of cell or tissue is evident from the article by Anderson *et al.* in this series (*27*). In one case, a 24-year old boy with diagnosed congenital galactosemia showed a small but definite ability to incorporate galactose-1-phosphate into nucleotide. This was revealed by assaying a microsample (biopsy) of his liver tissue using carbon-14-labeled galactose.

The rate of incorporation amounted roughly to about 2 to 5 percent of the normal activity in human liver samples (*27*). Likewise, administration of a small sample of carbon-14-labeled galactose *in vivo*, together with a glucuronide trapping agent (menthol), revealed that the same individual was able to incorporate a significant amount of galactose into menthol glucuronide (*28*). However, this incorporation was also low, amounting to 1 to 3 percent of normal. Red blood cells from the same individual did not show detectable amounts of activity of galactose-1-phosphate uridyl transferase. However, the method employed here is not as sensitive as that using carbon-14 labeled galactose.

It is not inconceivable that the liver might have had a total defect of the galactose-1-phosphate uridyl transferase and that it had gradually developed an alternate adaptive and closely related pathway. An adaptive mechanism might be expected to develop more readily in liver cells, which are, metabolically speaking, more universally active than, for instance, the more specialized erythrocytes. Perhaps age also plays a role. A liver biopsy sample from an infant with congenital galactosemia showed no trace of galactose-1-phosphate uridyl transferase (*27*).

It is unlikely that inhibitors are at work in this disease. Mixing hemolyzed erythrocytes from a nongalactosemic subject and a galactosemic patient did not significantly suppress the activity of the "normal" enzyme. Likewise, a case in which a galactosemic child was transfused with normal blood showed activity corresponding roughly to the amount of donor blood administered. In this case, the disappearance of the normal donor cells could be followed very well by the decrease in titer of galactose-1-phosphate uridyl transferase. It took 45 days to reach 50 percent of the original titer. This is somewhat faster than the time required to destroy 50 percent of a population of erythrocytes *in vivo* (*29*). A detailed study of more of such transfusion cases might throw additional light on the problem of the life span of erythrocytes *in vivo*. The possibility of antienzyme formation in such transfusion cases is being studied.

Galactosemia, a Hereditary Disease

That the defect in galactose-1-phosphate uridyl transferase is full-fledged at birth is apparent from the fact that blood from the umbilical cord of a newborn infant (one that had galactosemic siblings) had no detectable amounts of the transferase, whereas normal cord blood has abundant enzyme (*27*).

It might well be possible to pursue a study of the genetics of the disease using the erythrocyte enzyme assay. Preliminary experiments showed that neither of the parents of galactosemic children showed any striking decrease in enzyme activity. However, a moderate lowering of activity might be picked up. A genetic study was initiated by the important observations by Holzel and Komrower (*30*). The most pertinent fact in this study was that in the majority of the cases only one of the parents of galactosemic children showed a lowering of the galactose tolerance test.

Richard Post of the Institute for Human Variation, Columbia University, has suggested to me that the inheritance of the disease might be based on at least two multiple alleles. In other words, if A represents the normal gene and A' the state that affects galactose-1-phosphate metabolism, A'' is a third state giving another type of manifestation and aggravating the defect in galactose-1-phosphate metabolism if it is combined with A' such as $A'A''$. In that event, we should not only look for defects of galactose-1-phosphate uridyl transferase in galactosemic families, but also for another type of metabolic pattern that hitherto has escaped our attention as a trait in this disease.

Uridine Diphosphogalactose Pathway Dominating in Man

It should not be forgotten that there may be pathways of galactose metabolism totally different from the aforementioned uridine diphosphogalactose pathway operating in the animal body. A direct oxidation of free galactose has been described to occur in certain microorganisms (*31*). The latter mechanism seems, however, to be irreversible. Lactose synthesis seems also to follow different pathways (*32*) even in the same species, although it is considered most likely at present that both of these pathways operate through uridine diphosphogalactose. The biochemical symptoms in congenital galactosemia strongly indicate that in the growing infant the uridine diphosphogalactose pathway is the major one in operation.

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News of Science

NSF Summer Institutes for High-School and College Teachers

Approximately 4500 high-school and 250 college teachers of science and mathematics will participate during the summer of 1957 in teacher-training programs sponsored by the National Science Foundation at summer institutes in 95 colleges and universities throughout the United States and its territories. Total support of the program amounts to $4.8 million.

Eighty-six of the institutes will be open only to high-school teachers of science or mathematics; four will be open to both high-school and college teachers; and five will be for college teachers only. Six institutes offering a course in radiation biology just for high-school teachers are being jointly sponsored by the foundation and the Atomic Energy Commission.

Through its widely expanded summer-institute program for 1957, the foundation hopes to provide opportunities for teachers of science to cooperate in improving the quality of their teaching and to learn at firsthand of recent research progress in their respective fields. By this means more students with aptitude for science may be motivated toward careers in science, mathematics, and engineering through improvement of the quality of instruction they receive in high school. The foundation's program of summer institutes was initiated experimentally in the summer of 1953 with two institutes and the number of institutes was increased gradually to a point where the foundation supported 25 last summer.

Congress this year specified that $9.5 million of its appropriation to the foundation be used for the supplementary training of high-school teachers of science and mathematics. The summer institutes are in addition to the 16 academic-year institutes for which the foundation recently announced support in the amount of $4,065,000 and an expected enrollment of 750 high-school science and mathematics teachers. In both programs, teachers will pursue a course of study planned especially for them and conducted by leaders noted not only for competence in their fields but for skill in presentation.

The foundation grants to each summer institute will cover costs of tuition and other fees for a specified number of teachers—from 10 to 200, the average number to be approximately 50. Most institutes will pay stipends directly to participating teachers at the rate of $75 per week. Additional allowances and travel grants for dependents, to a maximum of four, are provided.

Inquiries and applications should be addressed to the director of summer institutes at one of the colleges listed here. Applications for the summer institute program must be submitted *by 1 Apr. 1957*.

For high-school teachers. Allegheny College, American University, Arizona State College, Atlanta University, Baylor University, Bucknell University, Clarkson College of Technology, Colorado College, Duke University, Iowa State College, Kansas State Teachers College, Louisiana State University, Marshall College (Huntington, W.Va.), Michigan State University, Morgan State College (Baltimore, Md.), Murray State College (Murray, Ky.), North Carolina College, Oak Ridge Institute of Nuclear Physics, Ohio University (Athens), Ohio Wesleyan University, Oklahoma Agricultural and Mechanical College, Rensselaer Polytechnic Institute, San Jose State College, South Dakota State College, Southern Methodist University, Stephen F. Austin State College (Nacogdoches, Tex.), Syracuse University, University of Alabama, University of Alaska, University of Arizona, University of Arkansas, University of California (Berkeley and Los Angeles), University of Hawaii, University of Idaho, University of Maryland, University of Minnesota (Duluth and Minneapolis), University of Mississippi, University of Missouri, University of North Carolina, University of North Dakota, University of Oklahoma, University of Pennsylvania, University of South Dakota, University of Texas, University of Wyoming, Virginia Polytechnic Institute, Wesleyan University, and Western Michigan College.

Biology only. Howard University, Indiana University, Iowa State Teachers College, Purdue University, and Rutgers University.

Radiation biology. (sponsored jointly with Atomic Energy Commission). Duke University, Harvard University, University of California (Los Angeles), University of New Mexico, University of Tennessee, and Wayne University.

Chemistry only. New Mexico Highlands University (Las Vegas), St. Louis University, Tufts University, Tuskegee Institute, University of California (Berkeley), University of New Hampshire, University of Rochester, University of Wisconsin, and Utah State Agricultural College.