possessed a medium-sized knob (K^M) was marked with the dominant *Sh* (plump kernel) factor, and the homolog possessing a small-sized knob (K^S) was genetically marked with the recessive *sh* (shrunken kernel) allele. Plants with the above cytogenetic constitution were employed as female plants in backcrosses to plants homozygous for both the *r* and *sh* alleles. It should be stated that preferential segregation is not confined to chromosome 10. Preferential segregation of other chromosomes occurs in K10/k10 heterozygotes when the two members of a pair differ by one possessing a knob while the other is knobless or if the two homologs have knobs of dissimilar size. If the K_T10 chromosome were as efficient as the K10 form in inducing preferential segregation, one would expect to find approximately 70 percent of the backcross kernels to be colorless (*3, 4, 7*) and about 60 percent to be "plump" in phenotype (*7*). The data presented in Table 1 clearly demonstrate the inability of the K_T10 chromosome to effect preferential segregation. Thus one would not expect to observe "meiotic drive" (*11*) in a population in which only the normal and the K_T10 chromosomes were segregating. Rather, these two forms of chromosome 10 should exist in stable proportions. On the other hand, a population containing equal numbers of the K10 and K_T10 forms should in time convert into a population in which the K10 chromosome is more prevalent.

Whether the absence of preferential segregation is related to the low level of neocentric activity is not known. Emmerling has noted that two modified forms of the K10 chromosome elicit a low level of neocentric activity and has suggested that this reduced neocentric activity could be responsible for the random segregation ratios she has observed in her experiments involving these modified chromosomes (*12*). A similar situation may very well exist here also (*13*).

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Metabolic Block in Utilization of Galactose by Strain L Tissue Culture Cells

Abstract. Evidence is presented which suggests that the utilization of galactose by mouse strain L-cells is blocked by the absence of epimerase activity in this strain.

Abnormalities in the metabolism of galactose occur in a variety of organisms. The precise nature of these abnormalities has been studied in humans and, more recently, in bacteria. Thus Schwarz *et al.* (*1*) showed that when erythrocytes of carriers of the heritable defect galactosemia were incubated with galactose, galactose-1-phosphate (Gal-1-P) accumulated in the erythrocytes. Kalckar and his collaborators (*2*) demonstrated that this accumulation of Gal-1-P was caused by the lack of Gal-1-P uridyl transferase (transferase) activity in galactosemics. Different mutants of the bacterium *Escherichia coli* unable to utilize galactose as a source of carbon have been shown to be deficient in galactokinase (*3*), transferase (*3*), and uridine diphosphogalactose-4-epimerase (epimerase) (*4*) activity, respectively. Triply defective mutants have also been found (*4*).

Eagle *et al.* (*5*) have reported that galactose does not support the growth of L-cells, a strain of mouse fibroblasts (*6*). We confirmed this observation and also found that L-cells do not respire galactose. In this report it is shown that the inability of L-cells to utilize galactose as a source of carbon may be explained by the absence of epimerase activity in these cells.

L-cells were grown as described earlier (*7*); lactalbumin hydrolyzate medium (*8*) was substituted for tissue culture medium 199. The determination of the presence of the enzymes required for the metabolism of galactose was based in part on the identification of intracellular galactose and in part on enzyme assays. L-cells which had been incubated for 180 minutes at 37°C in lactalbumin hydrolyzate medium containing 100 μmole 1-C^{14} labeled galactose per milliliter were harvested by sedimentation, washed, and boiled in a small amount of water; and the cellular debris was removed by centrifugation. The supernatant was then freed of protein by ultrafiltration through a dialysis membrane and chromatographed with the use of Leloir's ethanol-acetate solvent (*9*). A portion of this protein-free extract was retained for the enzymic determination of Gal-1-P which had been formed (see below). The ethanol-acetate solvent provided excellent separation of the three compounds, galactose, Gal-1-P, and uridine diphosphogalactose (UDPGal). Thus, the R_F value of galactose is about 0.65, Gal-1-P has a R_F value of about 0.32, and uridine diphosphoglucose (UDPG) of 0.18. Since UDPG and UDPGal have the same R_F value in this solvent (*9*), UDPG was used as the reference standard for UDPGal.

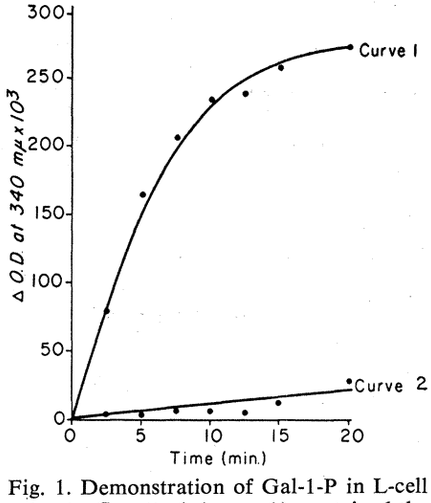

Fig. 1. Demonstration of Gal-1-P in L-cell extracts. Cuvette 1 (curve 1) contained the following assay mixture: 0.5 ml of buffer (0.006*M* histidine-HCl, *p*H 7.5); 1 μmole of $MgSO_4$; 0.6 μmole of TPN; 0.5 μmole of UDPG; yeast enzyme preparation, 0.05 ml; 0.2 ml of ultrafiltrate (see text) prepared from L-cells incubated in the presence of 0.005*M* galactose. Cuvette 2 (curve 2) contained 0.2 ml of boiled extract prepared from L-cells incubated in the absence of galactose. Cuvette 3 (control for endogenous reduction of TPN by yeast extract) contained 0.4 μmole of added Gal-1-P and no L-cell extract, and cuvette 4, which contained the yeast enzyme preparation (0.05 ml) and TPN, served as blank. The total volume in all cuvettes was 1 ml. The course of the reaction was followed by measuring TPN reduction at 340 mμ.

The radioactive material tentatively identified as UDPGal (R_F, 0.18) was eluted from the paper in 1*N* HCl, boiled 10 minutes, and rechromatographed in an isopropanol-water (4:1) solvent (*10*) which separates galactose from glucose ($R_{glucose}$ of galactose, 0.80). Over 70 percent of the radioactivity was then associated with the galactose area.

In the L-cell extract prepared according to the above procedures, 88 percent of the radioactivity was found to be associated with galactose, 8 percent with Gal-1-P, and the remaining 4 percent migrated with a R_F characteristic of UDPGal. As much as 6 μmole Gal-1-P and 3 μmole UDPGal accumulated per milliliter of packed cells.

When L-cells were incubated in the presence of lower concentrations of galactose in the medium (5 μmole/ml), approximately 10 percent of the total intracellular radioactivity was associated with UDPGal (0.59 μmole/ml of packed cells). Appropriate experiments indicated that less than 0.05 μmole of UDPG was present per milliliter of packed cells. In the presence of still lower concentrations of galactose in the medium (0.05 μmole/ml), 54 percent of the total intracellular radioactivity was associated with UDPGal (0.42 μmole/ml of cells).

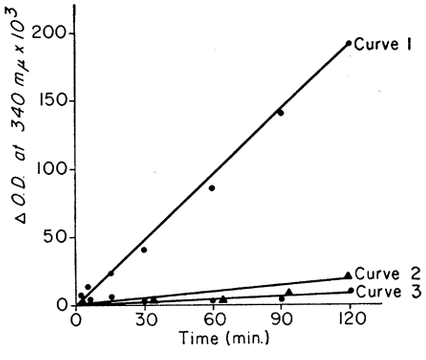

Fig. 2. Demonstration of transferase activity in L-cell extracts. The composition of the assay mixtures was similar to that used in the assay for Gal-1-P (Fig. 1). However, 0.01 μmole of diphosphopyridine nucleotide (*14*) was added to all reaction vessels in addition to the TPN; the yeast enzyme preparation was omitted. L-cell extracts were prepared by freeze-thawing and contained 8 mg of protein per milliliter: 0.05 ml of this extract was added to the cuvettes, and the total volume in all cuvettes brought to 1 ml with H_2O. Curve 1, L-cell extract incubated in the presence of 0.1 μmole of Gal-1-P. Curve 2, L-cell extract incubated in the presence of 0.4 μmole of Gal-1-P only. Curve 3, extract incubated in the presence of 0.01 μmole of UDPG and 0.4 μmole of Gal-1-P. No TPN reduction occurred when extracts were incubated with 0.1 μmole of UDPG only.

The presence of 28 μmole of galactose per milliliter of medium did not significantly inhibit the growth of L-cells when glucose served as a source of carbon. A slight increase in the lag period prior to the onset of exponential growth was observed.

The presence of Gal-1-P in extracts from L-cells incubated with galactose was also demonstrated by the reduction of triphosphopyridine nucleotide (TPN) (*2*) in the presence of an extract from yeasts adapted to galactose (Fig. 1). Attempts to measure galactokinase activity in crude L-cell extracts by the method of Horecker *et al.* (*11*) were unsuccessful. Transferase activity was determined by the method of Kurahashi and Anderson (*12*). Instead of the glycine buffer employed by these authors a histidine-HCl (0.006*M*) buffer at *p*H 7.5 was used. L-cell extracts prepared by freeze-thawing were found to be rich in phosphoglucomutase and glucose-6-phosphate dehydrogenase activity, and hence it was not necessary to add these enzymes to the assay preparation. Since UDPGal was not available to us, we could not measure epimerase activity directly by the method of Kalckar *et al.* (*4*). However, by limiting the amount of UDPG in the assay mixture to catalytic, rather than substrate, concentrations, the relative activity of epimerase could be measured: in the absence of epimerase, TPN reduction is dependent upon the concentration of UDPG, whereas in the presence of epimerase, TPN reduction requires only catalytic amounts of UDPG.

Comparison of curves 1 and 3 (Fig. 2) shows that the L-cell extracts had transferase activity (0.10 μmole UDPGal formed in 2 hours) but no detectable epimerase activity. The effectiveness of the assay procedure was demonstrated by the finding that, under identical assay conditions, freshly excised mouse liver displayed epimerase as well as transferase activity.

The results reported here suggest strongly that the inability of L-cells to use galactose as a source of carbon reflects the absence of epimerase activity in this cell strain (*13*).

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Relationship of Blood Type to Histocompatibility in Chickens

Abstract. Evidence shows that the *B* blood group locus in chickens, which controls red cell antigens, is associated with tolerance of skin homografts. Three other blood group loci studied did not show this effect.

The various factors involved in transplantation biology have been studied by workers in several scientific areas. Investigations have been most fruitful in those species where inbred lines are available.

In the case of chickens, tolerance to skin homografts is associated with degree of kinship between donor and host (*1*) as well as with age at grafting (*2*). Early workers, using heteroimmune hemagglutinins, reported that homograft tolerance and blood type were independent (*3*). Experiments involving induced homograft tolerance by embryonic transfusion of whole blood or its separate components have led to contradictory conclusions. Red blood cells were held to be ineffective in inducing tolerance in one case (*4*) but were found to be the most effective cell fraction in another (*5*). High incidence of homograft acceptance has been observed in one inbred line of chickens known to be segregating at the *A* blood group locus (*6*).