The Regulation and Properties of the Galactose Transport System in Escherichia coli K12*

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SUMMARY

This study of galactose-induced galactose transport in Escherichia coli K12 showed that in most strains two transport systems having the properties of "galactose permease" and "β-methyl galactoside permease" are responsible for the observed transport. Several strains contain only the galactose transport system but still grow normally on galactose. The regulation of this system was shown to be very similar to that of the galactose operon. That is, it is induced by galactose, shows only a slight repression by glucose, and is under the control of the galactose regulatory gene. The only differences between the regulation of the galactose transport system and that of the galactose enzymes are the level of induction (4-fold for the galactose transport system versus 15-fold for the galactose enzymes) and the fact that the galactose transport system is not under the control of the galactose operator locus. The β-methyl galactoside transport system is not regulated by either galactose regulatory gene in an agreement with the work of Lengeler et al. (Eur. J. Biochem. 19, 457-470). Galactose-binding protein is not essential for the activity of the galactose transport system as there is no correspondence between the level of the two. In fact, the galactose transport system is present at near normal levels under conditions where there is no measurable binding protein activity. Although the galactose and β-methyl galactoside transport systems have different genetic loci and are controlled by different regulatory systems, they do not function independently when they are both present in the same cell. Rather, they interact in a complex way that results in the rate of transport by the β-methyl galactoside system being reduced at high galactose concentrations and the $K_m$ of transport by the galactose system being reduced 3-fold.

Studies on the transport of galactose in Escherichia coli are made difficult by the presence of at least five different systems which can transport this substrate (2). Three of the systems, those for arabinose, lactose, and TMGII are not induced by growth on galactose and are transport systems for other sugars. The two remaining systems, those for β-methyl galactoside and galactose are both reported to function mainly in galactose transport (2, 3).

Horecker et al. (4) were the first to demonstrate that E. coli contained a system for the active transport of galactose. This was followed by the studies of Rotman and his co-workers who identified two galactose transport systems which they named "β-methyl galactoside and galactose permeases" (2, 5). These workers also attempted to study the regulation of these systems but they used galactokinase-negative strains in order to prevent metabolism of galactose; and in such strains all galactose-inducible systems are synthesized constitutively. Wu and Kalekar (6) have shown that this results from the fact that galactokinase mutants build up a high internal pool of galactose when grown in any media. Buttin reported that galactose transport was under the control of the galactose regulatory gene but did not show which transport system was involved (7). More recently, Boos and his coworkers have shown that the β-methyl galactoside transport system, though induced by galactose, is not under the control of the galactose regulatory gene (3). The present role of the galactose transport system in galactose transport is unclear as most workers have provided little evidence concerning this system, and one review article (8) stated that it might only be present in the strain originally studied by Rotman. Oxender in his review (9) states that galactose transport should be studied in isogenic strains which contain the different transport systems.

In this paper I have undertaken to identify the galactose transport systems in E. coli K12 which are induced by galactose and to study their regulation and possible interaction. For this purpose I have used several sets of isogenic strains. I find that galactose transport in most K12 strains results from the presence of two transport systems which correspond in their properties to "galactose permease" and "β-methyl galactoside permease." Several strains contain only the galactose transport system but they still grow normally on galactose. The galactose transport system is under the control of the galactose regulatory gene but not the galactose operator gene while the β-methyl galactoside transport system is not affected by either gene. Although galactose-regulated galactose transport in most K12 strains results from the combined activity of the galactose and β-methyl galactoside transport systems, the concentration dependence of galactose transport in such strains cannot be fitted by a model in which the measured transport is the sum of the transport by two independent systems. Rather there is a complex inter-
action that results in the measured rate of transport above $10^{-4} \text{m}$ galactose being lower than would be predicted if the two systems did not interact, which helps to explain the difficulties previous workers had in detecting the galactose transport system.

**EXPERIMENTAL PROCEDURE**

**Bacteria**

The strains used in this work are listed in Table I along with their genotype and source.

**Materials**

Most labeled substrates and Omnifuor were purchased from New England Nuclear. d-[3H]Fucose was bought from Amerbach Scientific and [1-14C]glycerol galactoside was synthesized from [1-14C]glycerol by the procedure of Boos et al. (16).

Unlabeled β-glycerol galactoside was the generous gift of Thomas Silhavy, a student of Dr. Boos. Other sugars and sugar analogues were purchased from Sigma Chemical Co. Rabbit antiserum against the galactose-binding protein was the gift of Dr. Heppel.

**Methods**

**Growth of Cells for Transport Measurements**—Cells were grown at 37° with shaking in Erlemeyer flasks in phosphate-buffered minimal medium (17). Carbon sources were always present at a concentration of 0.2%. Growth was stopped when the culture reached a density of $\sim 7 \times 10^8$ cells per ml (optical density at 600 nm is 0.80 in a Gilford spectrophotometer). The cells were centrifuged at room temperature ($7000 \times g$ for 7 min) and resuspended and recentrifuged either 2 or 3 times in 5 ml of 0.15 m NaCl. The final cell pellet from 10 ml of original culture was resuspended in about 1 ml of minimal medium lacking carbon source and stored at 0° up to 4 hours before making the transport measurements.

**Transport Measurements**—An amount of the above cell suspension which would take up from 1 to 25% of the substrate (usually 0.2 ml) was added to the volume of glycerol minimal medium needed to give 0.5 ml final volume in a tube (12 × 75 mm) and incubated 10 min at room temperature (21°). Labeled substrate was added, and 0.2 ml portions of the cell suspension were removed and filtered onto 25 mm of 0.45 μ nitrocellulose filters (presoaked in 0.15 m NaCl) 30 and 60 s after the substrate was added. The cells were washed with 4 ml of 0.15 m NaCl at room temperature. When a competing sugar was present it was added immediately before the addition of substrate. The filters were dried in a 150° oven and counted, with the use of a scintillation fluid containing 4 g of Omnifuor per liter of toluene, in a Packard Tri-Carb counter. All data are reported as nanomoles per min per mg of cell protein. Cell protein was determined according to Lowry et al. (18).

**RESULTS**

**Lack of Effect of Galactose Metabolism on Determination of Initial Rate of Transport** In order to show that metabolism of the transported galactose does not interfere with the measurement of the activity of the galactose or β-methyl galactoside transport systems, the activity of each of them was measured in an appropriate isogenic pair of strains one of which was a galactokinase mutant. Strain D115 was used for the β-methyl galactoside transport system and strain D116 for the galactose transport system. The results of these measurements are given in Fig. 1 and show that the initial rates of transport are nearly identical for both permeases regardless of whether metabolism is blocked or normal. The uptake by the β-methyl galactoside transport system appears to be linear for the first minute regardless of the strain or substrate used for measurement. Uptake by the galactose transport system is less linear especially in kinase-negative cells.

**Properties of Galactose Transport System**—The properties of the galactose transport system were first studied in strain D22 as it grows normally on 0.2% galactose (doubling time 67 min versus 56 min on glucose) and yet lacks the β-methyl galactoside transport system as shown by its inability to transport β-methyl galactoside and β-glycerol galactoside, and its $K_m$ of galactose transport. Measurements of galactose uptake as a function of galactose concentration are given in Fig. 2 and show a single $K_m$ for uptake with a value of $1.7 \times 10^{-4}$ M. The ability of various sugars to inhibit galactose uptake or to be transported in this strain is shown in Table II. Neither β-methyl galactoside nor β-glycerol galactoside is transported by these cells; however,

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**Table I**

**Bacterial strains**

| Strain | Genotype | Source |
|--------|----------|--------|
| 20SO K- | galK+ galp+ mglp- | Boris Rotman (7) |
| D115 | galK+ galp+ mglp+ | From 20SO K- by Ph transduction |
| D115 K- | galK+ galp+ mglp+ | From D115s |
| D115 | galK+ galp+ mglp+ | From 20SO K- by Ph transduction |
| D116 | galK+ galp+ mglp+ | From D116s |
| X208 | galK+ galp+ mglp+ lacY- proC- purC- trp- lys- metE- thi- | From Roy Curtiss III, University of Alabama, Birmingham |
| D22 | galK+ galp+ mglp+ lacY- proC- thi- purE- | From X208 by Ph transduction |
| D22 R+ | as D22 except galR+ | From D22+ |
| D22 OP+ | as D22 except galOP+ | From D22+ |
| W2244 | galK+ galp+ mglp+ lacY- | From Leon Heppel (5) |
| W2244R+ | galK+ galp+ mglp+ galR+ | From W2244+ |
| W2244OP+ | galK+ galp+ mglp+ galOP+ | From W2244+ |
| GSC4280 | F'129 His | From CGSC |
| GSC4249 | F'103 His | From CGSC |
| D22F'His | galK+ galp+ mglp+ | From Dr. Boos (14) |
| AWF500 | galK+ galp+ mglp- met | From Leon Heppel (15) |

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*Galactose-negative strains were isolated by penicillin selection (10) after nitrosoguanidine mutagenesis (11). Galactose-negative strains were identified on EMB galactose plates, grown up and assayed for the galactose enzymes as described before (12, 13).

* Strains constitutive for the synthesis of the galactose operon were isolated by two cycles of growth in minimal media containing $10^{-4}$ m galactose, $10^{-3}$ m thiomethyl galactoside after diethyl sulfate mutagenesis. Operator constitutive mutants were identified by their linkage to the galactose locus, as determined by Ph transduction.

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* A Hfr' derivative of D22 was isolated by penicillin selection after diethyl sulfate mutagenesis. This strain, grown on minimal media, was mixed with cells of strain GSC4280 or GSC4249 grown in L broth and the mixture plated on minimal glucose plates containing adenosine, thiamine, and proline. The colonies which grew were streaked for single colonies on the selection plates and then tested for their ability to transport β-methyl galactoside.
take so that the observed inhibition does not seem to result from the presence of contaminating glucose or galactose. The measured values of galactose transport were corrected for differences found in this way by multiplying the value of transport in D115 K- by 3.5, the ratio of β-methyl galactoside transport in the two strains.

d-fucose did seem to be transported to some extent. The only sugar which is a potent inhibitor of galactose uptake is glucose, while high concentrations of d-fucose also inhibit. Purifying the d-fucose by paper chromatography in 1-butanol-ethanol-water (6:4:3) did not affect its ability to inhibit galactose uptake so that the observed inhibition does not seem to result from the presence of contaminating glucose or galactose.

**Regulation of Galactose Transport System**—The regulation of this system was studied in strain D22 and in two isogenic strains which had fully constitutive synthesis of the enzymes of the galactose operon as a result of a mutation in either the galactose regulatory gene (D22 R°) or the galactose operator locus (D22 OP°). The cells were grown in medium containing either glucose, glycerol, or galactose as the carbon source, and the level of galactose transport was measured. The results are given in Table III and show that the regulation of this system is very similar to the regulation of the enzymes of the galactose operon. That is, it shows little repression by glucose, is induced by galactose, and is under the control of the galactose regulatory locus. Since the galactose operator mutant has the same constitutive level of the galactose enzymes as the galactose regulatory constitutive strain, the fact that the regulation of the galactose transport system is the same in D22 OP° as in strain D22 proves that the derepression seen in D22 R° is a direct result of the mutation in the galactose regulatory gene and not a secondary result of the constitutive synthesis of the galactose enzymes.

**Role of Galactose-binding Protein in Function of Galactose Transport System**—The above studies show that strain D22 contains a single transport system for galactose which differs from the β-methyl galactoside transport system in its substrate specificity, affinity for galactose, and in its regulation. Although strain D22 lacks the β-methyl galactoside transport sys-

| Competing sugar       | Concentration | Inhibition |
|-----------------------|---------------|-----------|
| α-Methylglucoside     | 1.67 × 10⁻⁴  | 13.1      |
| β-Galactoside         | 2.8 × 10⁻⁴   | 0.19      |
| D-Fucose              | 3.3 × 10⁻⁴   | 3.5       |
| D-Methylgalactoside   | 3.3 × 10⁻⁴   |           |
| D-Glycerol-galactoside| 3.3 × 10⁻⁴   |           |
| D-Glycerol-galactoside| 3.3 × 10⁻⁴   |           |
| α-Methylglucoside     | 1.67 × 10⁻⁴  | 13.1      |
| β-Galactoside         | 2.8 × 10⁻⁴   | 0.19      |
| D-Fucose              | 3.3 × 10⁻⁴   | 3.5       |
| D-Methylgalactoside   | 3.3 × 10⁻⁴   |           |
| D-Glycerol-galactoside| 3.3 × 10⁻⁴   |           |

TABLE II

Specificity of galactose transport in strain D22 (galp°, mglp°)

Transport assays were run as described under “Experimental Procedure.” The concentration of [¹⁴C]galactose used in the inhibition experiments was 3.3 × 10⁻⁴ M.

| Substrate                  | Concentration | Rate of uptake |
|----------------------------|---------------|----------------|
| Galactose                  |               |                |
| β-Methyl galactoside       | 1.67 × 10⁻⁴  | 13.1           |
| β-Glycerol galactoside     | 2.8 × 10⁻⁴   | 0.19           |
| d-Fucose                   | 3.3 × 10⁻⁴   | 3.5            |

TABLE III

Regulation of galactose transport in strain D22 (galp°, mglp°)

Cells grown on the indicated carbon sources were harvested, washed, and assayed for galactose transport as described under “Methods” with the use of [¹⁴C]galactose with a specific activity of 1550 cpm per nmole at concentrations ranging from 5 × 10⁻⁵ M to 5 × 10⁻⁴ M.

| Strain        | Vmax for the transport of galactose in cells grown on |
|---------------|------------------------------------------------------|
|               | Glucose | Glycerol | Galactose |
| D22           | 5.0     | 7.0      | 30        |
| D22 OP°       | 4.6     | 5.5      | 34        |
| D22 R°        | 16      | 33       | 35        |
tem, it contains the galactose-binding protein (19) which has been shown to be an essential component of this system (20). However, the regulation of the binding protein is different from the regulation of the galactose transport system (Table IV). In particular, when strain D22 R" is grown on glucose, galactose transport is present at 60% of its fully induced rate while there is no measurable galactose-binding protein. Furthermore, strain AW550 which is mutated in the galactose-binding protein (14) still has galactose transport activity (Table IV). Finally, osmotic shock (22) carried out on strain 7 (Table IV) releases strain AW550 which is mutated in the galactose-binding protein is no measurable galactose-binding protein. Furthermore, the regulation of the galactose transport system (Table IV). However, the regulation of the binding protein is different from particular, when strain D22 R" is grown on strain 7 (Table IV) releases more than 90% of the galactose-binding protein, causes a greater than 20-fold reduction in the activity of the β-methyl galactoside transport system, but only reduces the activity of the galactose system 3-fold. These results indicate that the galactose-binding protein is not an essential component of the galactose transport system.

Effect of Introducing β-Methyl Galactoside Transport System into Strain D22—The β-methyl galactoside transport system was introduced into strain D22 with the use of either of two episomes (P129 or F100) which overlap only in the region near histidine, from 38 to 41 min on the E. coli map, the region which contains the genes for the β-methyl galactoside transport systems (2). Strains containing either episome were able to transport β-methyl galactoside and contained an additional galactose transport system with a $K_m$ of $\sim 5 \times 10^{-6}$, the value reported for the β-methyl galactoside system. The rate of galactose transport as a function of its concentration for strain D22 and D22F'His are shown in Fig. 3. Although strain D22F'His shows an increased rate of galactose transport over strain D22 at low galactose concentrations due to the presence of the β-methyl galactoside transport system, there is only a small increase in transport at high concentrations. The galactose transport seen in strain D22F'His is identical with the transport seen in strains D115 and W2244 (Fig. 4) which are typical of wild-type K12 strains. The galactose transport data obtained in these strains cannot be fitted by a model in which galactose transport is due to two independent systems. This is shown in Fig. 5 where the data at higher galactose concentrations are used to determine the $K_m$ of galactose uptake; a linear plot is obtained only if the data are plotted directly. If they are corrected for the transport expected from the β-methyl galactoside transport system, the plot is nonlinear. These results could be explained if transport of galactose by the β-methyl galactoside system is inhibited at high galactose concentrations.

It is possible to distinguish between galactose transported by the β-methyl galactoside transport system and that transported by the galactose transport system by adding unlabeled β-glycerol galactoside which is a competitive inhibitor of galactose transport by the β-methyl galactoside system but has no effect on the galactose system (Table II). The results of such experiments are presented in Table V and indicate that galactose transport by the β-methyl galactoside transport system is inhibited about 33% at high galactose concentrations. An even more surprising result is that the $K_m$ of the galactose transport system in D22F'His is $5.5 \times 10^{-4}$ m compared with a value of $1.6 \times 10^{-4}$ m for strain D22. This result indicates that there is an interaction between the two systems which affects the properties of both.

**Table IV**

**Noncorrespondence between level of galactose-binding protein and galactose transport system**

The level of the galactose-binding protein was measured on lyophilized sonic extracts of cells, grown on the indicated carbon source, by the procedure of Rotman and Ellis (21) with the use of $4.5 \times 10^3$ m $^{14}$Cgalactose with a specific activity of $3.4 \times 10^4$ cpm per pmole. This method uses antibody against purified galactose-binding protein to improve the affinity of galactose binding. Galactose transport was measured on the same cultures by the procedure described in Table III with the use of $1.6 \times 10^{-4}$ m galactose.

| Strain | Growth medium | Galactose | Galactose-binding protein |
|--------|---------------|-----------|---------------------------|
|        |               | transport | protein in cell extract   |
| D22    | Glucose       | 3.4       | 0                         |
| D22    | Glycerol      | 4.0       | 93                        |
| D22    | Galactose     | 16.4      | 190                       |
| D22 R' | Glucose       | 12.9      | 0                         |
| D22 R' | Glycerol      | 22.6      | 45                        |
| D22 R' | Galactose     | 23.8      | 106                       |
| Strain 7| Glycerol + fucose | 9.9 | 58 |
| Strain 7| Osmotically shocked | 3.0 | 4 |
| AW550  | Galactose     | 10.5      | 4                         |

**Fig. 3.** Galactose uptake in strains D22 and D22F'His. Strain D22 (mglp\(^-\), galp\(^+\)) was grown in minimal galactose medium while D22F'His (mglp\(^+\), galp\(^+\)) was grown in minimal glycerol medium containing $10^{-4}$ m β-fucose. The cells were harvested and assayed for galactose transport as described under “Methods.”

**Fig. 4.** Galactose uptake in strains D115 and W2244. Strains D115 and W2244 (mglp\(^+\), galp\(^+\)) were grown in minimal glycerol media containing $10^{-4}$ m β-fucose as an inducer, and the rate of galactose uptake was measured at the indicated concentrations of galactose as described under “Methods.” •, D115; ×, W2244.
Fig. 5. Nonadditivity of galactose transport by galactose and \(\beta\)-methyl galactoside permeases in strain D115 (mglp\(^+\), galp\(^+)\).

The data at high galactose concentrations from Fig. 4 on strain D115 were plotted directly (●) and after subtracting the value of transport expected from \(\beta\)-methyl galactoside permease (x) with the \(V_{\text{max}}\) of 14.3 and \(K_m\) of \(5 \times 10^{-7}\) M calculated from the data at low galactose concentrations.

**Table V**

Galactose and \(\beta\)-methyl galactoside transport systems interact with each other

| Strain (Glucose 1 Glycerol 1 Galactose 1 N-fucose) | D22 | DD2F'His | D115 |
|---------------------------------------------------|-----|----------|------|
| Properties of \(\beta\)-methyl galactoside permease |     |          |      |
| \(K_m\) | \(4.9 \times 10^{-7}\) | \(5 \times 10^{-7}\) |          |
| \(V_{\text{max}}\) | 16.1 | 13.0 |          |
| Properties of galactose permease | 11.6 | 6.9 |          |
| \(V_{\text{max}}\) | 29.4 | 29.6 | 25.4 |
| \(K_m\) | \(1.6 \times 10^{-4}\) | \(9.3 \times 10^{-8}\) | \(8.9 \times 10^{-8}\) |

**Table VI**

Regulation of galactose and \(\beta\)-methyl galactoside permeases in strain W2244 (galp\(^+\), mglp\(^+\))

Cells grown on the indicated carbon source were harvested, washed, and assayed for galactose and \(\beta\)-methyl galactoside transport as described under "Methods" with the use of \(^{14}\)C-galactose with a specific activity of 1550 cpm per nmole and \(\beta\)-methyl \(^{14}\)C-galactoside with a specific activity of 6000 cpm per nmole, respectively. All values are in units of nanomoles per min per mg.

| Strain | Glucose | Glycerol | Galactose | Glycerol + \(10^{-5}\) M fucose |
|--------|---------|----------|------------|--------------------------------|
| W2244  | 6.3     | 8.6      | 33         | 37                             |
| W2244 Op\(^+\) | 4.5 | 8.4      | 16.5       | 24                             |
| W2244 R\(^+\) | 14.2 | 29.5     | 32.5       | 39                             |

\(\beta\)-methyl galactoside transport

| Strain | Op\(^+\) | R\(^+\) |
|--------|---------|--------|
| W2244  | 0.03    | 0      |
| W2244 Op\(^+\) | 3.5 | 2.2 |
| W2244 R\(^+\) | 17.4 | 5.8 |

\(\beta\)-methyl galactoside transport

**Discussion**

It is often stated that one cannot study the transport of a metabolizable compound because its metabolism would affect the initial rate of uptake. However, there are several cases where metabolism has been shown to have no effect; the initial rate of glutamine transport is the same whether or not its metabolism is prevented by an inhibitor of glutamine transaminase (23). Glucose-6-P transport is the same in a wild-type strain and in one which contains mutations in the genes for glucose-6-phosphate dehydrogenase and phosphoglucomutase (24). It has been shown that the initial rate of transport of galactose is the same for the \(\beta\)-methyl galactoside transport system in a wild-type strain and in a galactokinase mutant (4). Metabolism does not interfere with the determination of the initial rate of galactose uptake by either the galactos or \(\beta\)-methyl galactoside transport system (Fig. 1).

The galactose transport system studied in strain D22 has properties that are nearly identical with those reported for "galactose permease" by Rotman et al. (5). The \(K_m\) for galactose is \(1.7 \times 10^{-4}\) M compared with the reported value of \(1.4 \times 10^{-4}\) M, and it shows the same substrate specificity except that \(\alpha\)-fucose appears to be a substrate.

This paper shows that the transport of galactose in most K12 strains is due to the combined activities of the galactose and \(\beta\)-methyl galactoside transport systems. I have studied 12 separate K12 strains and find that each contains the galactose transport system. The proportion of galactose transport which is due to each system varies depending on the strain and the growth medium, presumably because the two systems are regulated independently. Thus I find that the galactose transport system is under the control of the galactose regulator gene confirming the report of Buttin (7) that galactose transport is regulated by the galactose regulator gene, while the \(\beta\)-methyl galactose transport...
port system, as reported by Lengeler et al. (3), is not under the control of the galactose regulator gene and also differs from the galactose system in that it is strongly repressed by growth on glucose. Recently Saier et al. reported that the galactose transport system in Salmonella is also regulated by the galactose regulatory gene (25). A further difference between the two systems is that the galactose transport system is unrelated to the level of the galactose-binding protein which is an essential component of the \( \beta \)-methyl galactoside transport system (20). This finding as well as the reported \( K_m \) of galactose transport indicates that the transport system studied in membrane vesicles by Kerwar et al. (26) is probably the galactose system. Although the properties of the two galactose transport systems are strikingly different, they do appear to interact when they are both induced in the same cell. This interaction may be similar to that reported for lactose and glucose permeases (27–30) where it was shown that the presence of a substrate of glucose permease would inhibit the transport of substrates of lactose permease if and only if the cells were induced for glucose permease. Such an interaction has also been found for substrates of the phosphotransferase system and the transport systems for melibiose, maltose, and glycerol (31) and for the glycerol phosphate and glucose 6-phosphate transport systems. In the case of the two galactose systems the situation is greatly complicated by the fact that all known substrates of the galactose system are also substrates of the \( \beta \)-methyl galactoside system. The mechanism of these interactions is not yet known for any case. The widespread occurrence of such interactions suggests that they probably are important. One possible function of such interactions would be to allow the orderly metabolism of carbon sources when several are present in the media. Thus it has been shown that when cells are grown in glucose and lactose the glucose is used up first and only then does lactose metabolism occur. If this suggested function is correct for the two galactose systems, one would find a similar diauxie between \( \beta \)-glycerol galactoside and galactose.

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