2-oxoglutarate, the proportion of these two compounds depending on the initial redox state of the intramitochondrial NADP.

This system is currently being investigated.

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COMMUNICATIONS

Proton Movements Coupled to the Transport of β -Galactosides into *Escherichia coli*

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It is now accepted that β -galactosides are transported into the intracellular water space of *Escherichia coli* by a genetically determined transporter (the γ -gene product), where they can accumulate as the unmodified β -galactosides. In view of the 100-fold concentration gradients that can be established, it is clear that the inflow of β -galactoside must be coupled to another process capable of making it spontaneous, but it has not hitherto been obvious what that other process was. Phosphorylation of the β -galactosides by a phosphoenolpyruvate-dependent phosphotransferase is not observed (cf. glucosides), nor does ATP hydrolysis appear to be involved (for review see Kaback, 1970). However, evidence is given in the present communication that suggests that a flow of protons into the cell is coupled to the inflow of β -galactoside, in support of the suggestion (Mitchell, 1963) that the accumulation of β -galactosides by *E. coli* is due to the operation of a proton- β -galactoside symporter, analogous to the Na^+ -glucose and Na^+ -amino acid symporters thought to occur in animal cells.

(1) When β -galactoside is added to a suspension of non-metabolizing cells (anaerobic, and depleted of fermentable substrates), there is an alkalification of the extracellular medium, corresponding to an inflow of protons (or outflow of OH^- ions) observable with a glass electrode.

(2) Similarly, with non-metabolizing cells made permeable to K^+ ions by treatment with tris, EDTA and valinomycin, the addition of β -galactoside causes the outflow of K^+ ions, observable with a K^+ ion-sensitive glass electrode, stoichiometric with the inflow of H^+ ions, indicating that β -galactoside induces an electrogenic inflow of H^+ ions.

(3) When a transmembrane pH gradient is established in non-metabolizing cells by the injection of a small amount of O_2 (dissolved in KCl solution), the subsequent slow rise of extracellular pH towards its original value is accelerated by the presence of β -galactoside, as expected if the inflow of protons is coupled to the transport of β -galactoside.

(4) A transient diffusion potential across the cell membrane (inside negative) can be established by the addition of KSCN to non-metabolizing cell suspensions because of their greater permeability to SCN^- than to K^+ ions. The inflow of protons caused by the electrical force is increased by the presence of β -galactoside, indicating that β -galactoside participates in the electrogenic movement of H^+ ions across the cell membrane.

All these effects of β -galactosides are completely removed by inhibiting the β -galactoside porter with *N*-ethylmaleimide.

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The Ion-Specific Increases in Membrane Permeability with a Group of Membrane-Active Antibacterial Agents

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In order that the various agents known to cause uncoupling of oxidative phosphorylation in mitochondrial systems may be used as experimental tools in the study of energy-transducing mechanisms in bacteria, the specificity of the increases in ion permeability that they produce must be verified for the systems under study.