Rotman permease 8/26/60

1. galactose - P: Specific for ~~galactoses~~ ; contra methylgalactosides
Not inhibited by thio compds.

2. galactoside - P. (MG-P). β megal. gal? probably yes (galactose inhibits MG).
iPr; Pr < Et < Me Et-gal much less than Me., α-gal not at all.

2. galactoside - P MONOP. { β-thiogal, β-gal, α-gal. } . gal probably not
lactose.

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3 - assay diff for lactose, maybe irrelevant.

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2 vs. 3 : lac_2^- { β-galactosidase must not be present.

Not induced: { galactose consumed, MG accumd., TMG not accumd.
~~glucose~~ glycerol cells:

Inducers: (any gal, galactoside; TMG also induces TMG accumulation).

Mutants:

all (galactose) so far are permease-positive. Gal-permease always present.

Glucose always accumul. Even lac_3: glu is accumul. (but Cohn says glucose may inhibit synth of glucose permease).

	CONST.	MG-IND. (presumably in lac_3)
W3092. (wild⁻)	Gal P^+, MG P^-, TMG⁻	P^{++} TMG P induced. P^+ TMG⁺. ← inhibited by alkyl galactosides.

Gal_1 – Gal_9. Gal_2 Gal_8: MG permease-negative also 2 others.? are these wild⁻

Cells: D(glu) – gal_{C14}... Inc. 15 minutes. Filter Millipore & washing. Washing removes many counts. Control other kinds of cells, e.g. oxide. These backgrounds are rather high.

600	80	unw.
200	6	w.
exp.	oxide	

Exit reaction. ONP.Gal*; B-galactosidase (presumed) excess; $Gal^+ \rightarrow$ exit, at high ONPG, permease hydrolysis rate, and gal* will accumulate until $v_{en} = v_{ex}$. Gal* itself outside very low. Separate permeases for unity.

Thus

$ONPG^*_{ex} \rightarrow ONPG_{in}$ lac permease

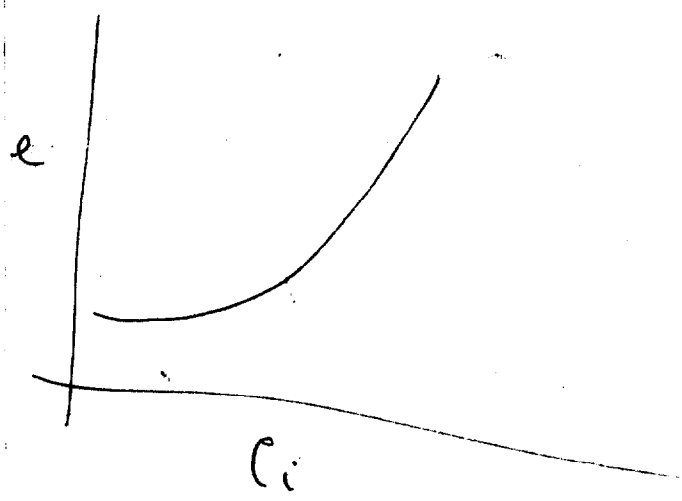
$ONPG_{in} \rightarrow NP + Gal^*_{in}$

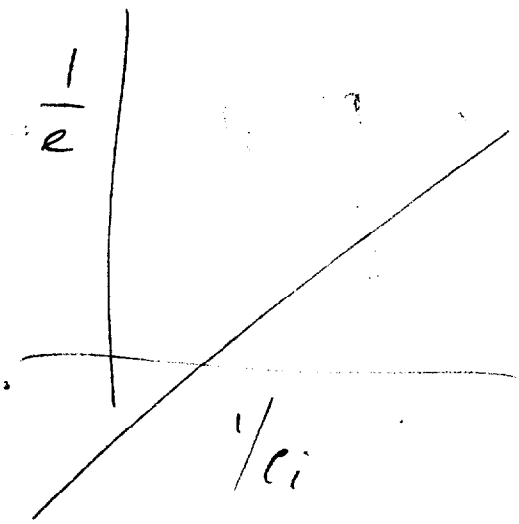
$Gal^*_{in} \rightarrow Gal_{ex}^*$

at steady state

$$\dot{C}_i = k_1 y - k_2 C_i = 0$$

$$C_i = k_3 y$$





$$e \neq K C_i$$

but $e = \dfrac{K_1 C_i}{K_2 - C_i}$.

Study exit reaction.

Azide: no effect on rate of NPG hydrolysis but stops accumulation. ∴ under effect exit reaction.

" Permease is a shaped hole., facilitated diffusion azide relaxes the structure, and faster exit. "

..... Induce cells with