A-15d

Biology
Aug 57

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Property of _____

Class _____

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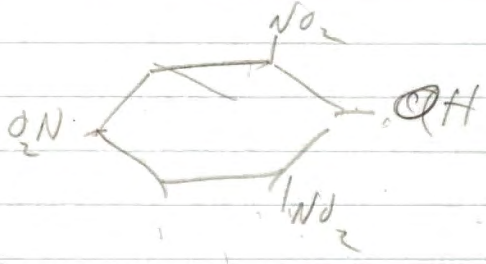
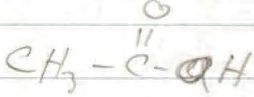
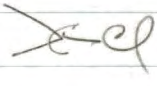
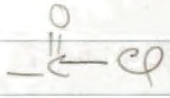
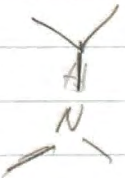
Test 8 Question Golden, Washington
in the understand this matter in the public media
Remarks that there has been a failure
to make sufficiently clear the distinction
between a citizens evident right to differ and
~~criticize~~ criticize a decision and policy of the
Govt and the equally evident obligation
as a citizen not to weaken or undermine
any such ~~policy~~ decision or policy. —











Stocker:
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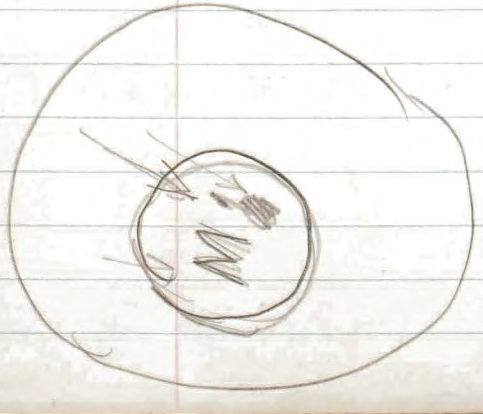


or if there is competition ~~between~~ ~~activator~~ an "inducer" with the repressor

$$1 - \theta = 1 - \frac{\frac{c}{K}}{1 + \frac{c}{K} + \frac{c_2}{K_2}}$$

$$= \frac{1 + \frac{c_2}{K_2}}{1 + \frac{c}{K} + \frac{c_2}{K_2}}$$

$$\bar{\iota} = \bar{\iota}(AA) \left(\frac{1 + \frac{c}{K} + \frac{c_2}{K_2}}{1 + \frac{c_2}{K_2}} \right) \quad (1)$$

$$\alpha = \frac{180}{\frac{c}{K}} = 180$$

$$K = 10^{-6} \qquad \frac{c}{K} = 180$$

$$c = 180 \times 10^{-6} \qquad 10^{-4} \text{ molar}$$

10^4 genes | X enzyme molecules

$$K_1 = \frac{y_1}{\pi_1} \qquad y_1 = K_1 \pi_1$$

1 Aug 57 H

Repressor : some trouble as

No 12.

Exp. ~~Grow~~ B/+ with arginine added between 0 and 1 unit (1 unit just enough for supply normal Arg content of B)

~~Vary~~ Vary τ !

$$s = \frac{1}{\tau(+A)}(1-q) = \frac{q/k}{\tau(Rep)}$$

This gives old result I but if we assume that repressor stays long (compared with $\tau(+A)$) then we get type II result || In case of adaptive ~~enzyme~~ ~~protein~~ mRNA is unstable ~~and~~ and this raises the question: how does enzyme level depend on τ if we have low inducer conc to ensure molecules per cell; $\tau(Ind) > \frac{3 \times 60}{180} sec$ must then have to be assumed for τ independence.

{ Exp. with ~~beta~~ β-galactosidase
Exp with arginine, using strong repression

$$\tau = \tau(+A)\frac{1}{1-q}$$

because $q = \frac{c/k}{1+c/k}$ | $1-q = 1-\frac{c/k}{1+c/k}$

$= \tau(+A)(1+c/k)$

4

$$\frac{1}{2d} + \frac{1}{3} = \frac{4}{\frac{2a}{4}} + \frac{4}{9}$$

10^{4} impure molecules

$S = 2 \times 10^{-7}$ mol/liter $\qquad \frac{S}{K} = 100 \qquad K = 2 \times 10^{-9}$

$$\frac{10^{4} \text{ impures} \times 10^{3} \times 10^{5}}{6 \times 20^{23}} = \frac{10^{12}}{6 \times 10^{23}} = \frac{1}{6 \times 10^{11}} = \frac{1}{6} 10^{-11} = 2 \times 10^{-12}$$

ζ_1

With phenomenon of Supermundo when τ is switched to temper τ_0, —

does not happen with Maxwell limitation (or Poincaré)

$\frac{\tau_1}{E}$

$$\frac{d\zeta}{dt} = -\frac{\tau_1}{E}\zeta + a - \frac{\zeta}{\tau_0}$$

$$\frac{d\zeta}{dt} = a - \frac{\zeta}{\tau_1} - \frac{\zeta}{\tau_0}$$

$$0 = a - \frac{\zeta_1}{\tau_1} - \frac{\zeta_1}{\tau_{0(1)}}$$

$$0 = a - \frac{\zeta_1}{\tau_d} - \frac{\zeta_1}{\tau_{0(1)}}$$

$$\begin{cases} v = \\ \\ 0 = \frac{a}{b} - \frac{E_1^2}{\tau_d} - \frac{E_1}{\tau_{0(1)}} \\ 0 = \frac{a}{b} - \frac{E_2}{\tau_d} - \frac{E_2}{\tau_{0(2)}} \end{cases}$$

$E_1 = \zeta_1$

$E_2 = \zeta_2$

$$\frac{E_1}{\tau_d} + \frac{E_1}{\tau_{0(1)}} = \frac{E_2}{\tau_d} + \frac{E_2}{\tau_{0(2)}}$$

$$\frac{E_2}{E_1}$$

$\frac{\zeta}{\tau_d}$

To old paper page 11*

what we really is ΔH and obtain this

$\frac{\rho}{K} = \sqrt{\frac{2m}{\ln m}} \simeq \sqrt{\frac{2000}{7}}$ two terms in (11) are equal) (16.5)

$$\tau_0 = \frac{2}{2AK\frac{\rho}{K}} \{ \} = \tau_{evap} \left\{ \frac{2m}{1+\frac{\rho}{4}} \frac{1}{\frac{\rho}{K}} + \ln m \right)$$

290

~~11~~ $\tau_0 \simeq \tau_{evap} \left\{ \frac{2m}{(\frac{\rho}{4})^2} + \ln m \right\}$

$\frac{2000}{290} = 7$ $\quad \ln \sim 16\frac{1}{2} + 17\frac{1}{2}$

$$\boxed{\tau_0 = \tau_{evap}\, 2 \ln m}$$

$\tau_0 = 14\, \tau_{evap}$

$\tau_0 = 1\,sec \quad \tau_{ev} = \frac{1}{14}\,sec = 13 \; e^{-\frac{\Delta H}{RT}}$

$\frac{\tau_0}{\tau_{ev}} = \alpha = 14 = \frac{10}{f} e^{\;} =$

$f \sim 1$ or 10

$1 < f < 55$

$1 = 7.14 \frac{10}{f} e^{11 - \frac{\Delta H}{RT}}$

$1 = e^2 \frac{10}{f} e^{11 - \frac{\Delta H}{RT}}$

$0 = 2 + 11 \cdot 2.3 - \frac{\Delta H}{RT} - (\ln f)$

$\frac{\Delta H}{RT} = 2 + 25 - \ln f$

$24 < \frac{\Delta H}{RT} < 27$

$140000 < \Delta H < 160000$

$2\tau_{evap} = \frac{10^{13}}{f} e^{-\frac{\Delta H}{RT}}$

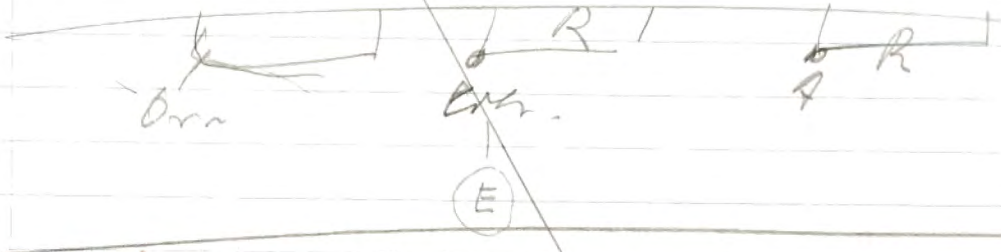
$2A\rho K \frac{1}{f} = 10^{13} e^{-\frac{\Delta H}{RT}}$

$2A\rho \frac{K}{\rho} \frac{1}{f} = 10^{13} e^{-\frac{\Delta H}{RT}}$

Aug 20 5

Antibodies:

In case of trigger theory:
Antigen, blocks enzyme and thus induces by
Anamnestic reaction! (blocking repressor)
damaged enzyme still combine
with trigger for good destroys template
therefore more number enzymes are produced and
these can combine with antibody. If much
damaged enzyme diffuses to gene or paragene
even those other resembling triggers
are destroyed. —



Antibodies:

1.) Antigen combines with enzyme
but does not say only 9/10 of enzyme.

2.) Antigen combines with enzyme
while enzyme still sticks to
paragene; breaks paragene-
paragene bonds, produces now
on combining antibody but
no enzyme. —
— From here on enzyme level
will be lower. (Anamnestic reaction

14/00

from p. 6)

8

contd. (to old paper p 11*). —

$$\tau_0 = 2 \ln m \times \tau_{ev}$$

$$\frac{\rho}{k} = \sqrt{\frac{2m'}{\ln m}} \quad \left(\text{for } m = 1000;\ \frac{\rho}{k} = 16.5 \right)$$

$$\frac{1}{\tau_{ev}} = \frac{10^{13}}{\rho} e^{-\frac{\Delta H}{RT}}$$

$$\Delta H = \frac{1}{2} \frac{1}{\tau_{ev}}$$

$$\tau_{ev} = \frac{1}{2\Delta H}$$

$$\Leftrightarrow \frac{\Delta k}{\rho} \rho = \frac{1}{\tau_{ev}}$$

$$2A\rho = \frac{1}{\tau_{ev}} \sqrt{\frac{2m'}{\ln m}}$$

$$\rightarrow 2A\rho = \frac{2 \ln m}{\tau_0} \times \sqrt{\frac{2m'}{\ln m}}$$

$$2A\rho = \frac{1}{\tau_0} \underbrace{\sqrt{2m \ln m}}_{120}$$

$$A = 6 \cdot 10^{23} \nu \sigma \rho$$

$$\rho = \frac{120}{\tau_0} \frac{1}{A}$$

ρ in mol/cc

for $\sigma\rho = \frac{1}{3} 10^{-17}$

$A = 10^{10}$ $\quad \rho \cong 10^{-8}$ mol/cc $= 10^{-5}$ mol/l

for $\sigma\rho = \frac{1}{3} 10^{-16}$

$A = 10^{11}$ $\quad \rho = 10^{-9}$ mol/cc $= 10^{-6}$ mol/l

contd p. 11

H

$$\tau_{emp} = \frac{\tau_0}{14} = \frac{1}{2AK}$$

$$\tau_0 = \frac{2 \ln m}{2AK} \qquad \boxed{2 \ln m = 14}$$

$$14AK = \frac{14 \ln m}{\tau_0}$$

Invest: τ dependence of weak const. if real inducer simply attached to enzyme and enzyme combines with trypsin:

$$\frac{d\beta}{dA} = \frac{\frac{C}{K(P_1)}}{1+\frac{C(I)}{K(P_2)}} \left(1 + \frac{2}{K(2)} \cdot \frac{1}{1+\frac{C(I)}{K(2)}}\right) = \frac{2}{\tau}$$

$$\frac{C(I)}{K(P_2)} \frac{1}{1+\frac{C(I)}{K(P_2)}} + \frac{2}{K(2)} \frac{1}{1+\frac{C(I)}{K(2)}} = 2$$

Exp. on Antibody formation

(The ovalbumin which is extracted in 6 hr chums may be immunes just as good)

1.) Inject antigen
2.) Inject antibody (unlabelled) to remove antigen
3.) Look physiol* and absence production of radioactive antibody! —

Repression

$$\frac{\frac{S_1}{K(S)}}{1+\frac{S_1}{K(S)}+\frac{S_2}{K(R)}}$$

If $\frac{S}{K(S)} \ll 1$ and if S and R proportional to each other say $R = 100S$

$$\frac{\frac{S}{K}}{1+\frac{S}{K}+100\frac{S}{K}}$$

for large $\frac{S}{K}$ goes to $\frac{1}{10}$

Induction

If S is increased and enzyme is induced we get induction even if S can be coupled to R

Rate of Bact. synthesis = H

uninduced $\quad 13 - \frac{44}{R\Delta}$

$$\frac{1}{\tau_{evap}} = \frac{10}{\ell} e = \frac{2 \ln m}{\tau_0}$$

$$2Ak = \frac{1}{\tau_{evap}}$$

$$2A \frac{k}{\rho} \rho = \frac{1}{\tau_{evap}}$$

$$2A\rho = \frac{1}{\tau_{evap}} \cdot \frac{\rho}{k} = \frac{1}{\tau_{evap}} \sqrt{\frac{2m}{\ln m}}$$

$$2A\rho = \frac{2 \ln m}{\tau_0} \sqrt{\frac{2m}{\ln m}}$$

$$\Delta\rho = \sqrt{2m \ln m} \frac{1}{\tau_0}$$

$\rho =$

for $\tau_0 = 1$ sec

for evap. Amp since less provision
in 10^{-5} M Arg. inside $\frac{\text{mgm}}{\text{liter}} = 10^{-5}$ M/L

Induction - Repression

active or inactive gyes:

Case of Induction

$\frac{g}{k} \ll 1$ $\frac{R}{k} \ll 1$

Fraction producing

$$\frac{\frac{g_i}{k(P_g)}}{1 + \frac{g_i}{k(P_g)} + \frac{R_a}{k P_R}}$$

If g raised and enzyme close to saturation R goes up more slowly than g. —

If both g and R raised induction

Repression

$\frac{g}{k} >> 1$ $\frac{R}{k} >> 1$ $R \gg g$ $R = \omega g$

If g and R go up no change

$\frac{g}{k} >> 1$ $\frac{R}{k} >> 1$

If R goes down, enzyme shuts up

Experiment: make with penicillin method from arginineless strain plate "arginine enzyptic" good for repressor experiments. —

Experiment. τ dependence of $E(\tau)$ in ~~irrepressible~~ an "arginine ~~suppressible~~" strain in chemostat, $a(arg.)$ in tank less than what is needed to ~~[illegible]~~ give full supply for protein of Cadenza. —

Prot. Synthesis!

How many Arg. molecules inside bacteria are at 10^{-5} M/l in

$6 \times 10^{+23} \quad 10^{-5} \quad 10^{-3}$ /cc

$6\,10^{23} \quad 10^{-5} \quad 10^{-3} \times 10^{-12}$ /B $= 6\,10^{3}$

Compute how many enzymes from Synthetase and Transp. rate of Arginine and Uptake rate of Citrulline.

---

outside 10/l

inside 100 X as much

~~10/l~~ 100×10/l $= 1$ mgm/l $\cong 10^{-5}$ M/l

could be five times higher —

How many E in Arg synthesis?

Transp. time $= \frac{1}{2AK} = \frac{10^{11}}{6\,10^{-8}} = 6000$/sec per

$A = 3\,10^{11}$ $\quad K = 10^{-8}$ mol/cc

number of E in cell = N

6000 N is made per sec

in 70 min $70 \times 60 \cong 4000$ sec

Arg is $\frac{1}{25}$ of net weights.

1 cc $\frac{1}{25}$ gm or $\frac{1}{2500}$ Mol or $\frac{6\,10^{23}}{2.5 \times 10^{3}}$ Molecules present

made per E molecule $24 \cdot 10^{6}$

$\frac{6 \times 10^{23}}{6\,10^{10}} = N$

$N = 10^{13}$/cc

25 total turnover at most could be built

~~Avogadro~~ Atomic wts are $= \frac{6\,10^{23}}{2.5\times10^{3}\times 4\text{ kms}} = \frac{6\,10^{23}}{10^{7}\times10^{11}}$

~~$\frac{6.10^{23}\cdot 6\,10^{23}}{10^{7}\times10^{4}\times10^{12}}$ Avogadro's~~

10^{-8} 10^{11} N/cc $= \frac{6\,10^{23}}{10^{7}}$ per cc

$N/cc = \frac{6\,10^{23}}{10^{7}\times10^{3}} = 6\,10^{13}\times10^{-12} = 60$

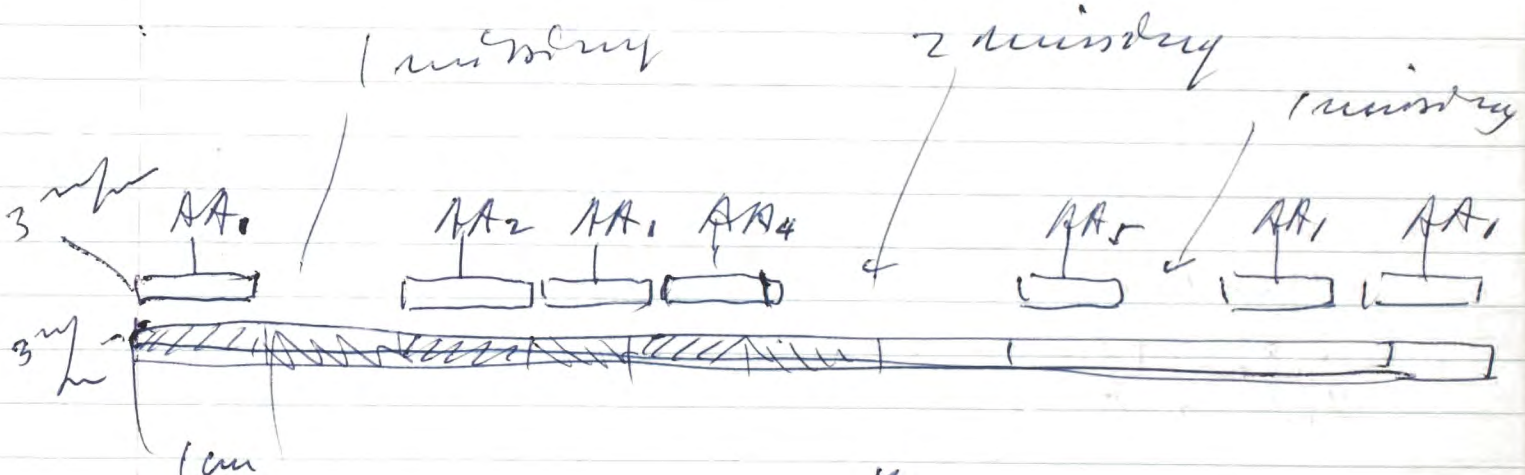
All this relates to mean factor 25 how long mos bits could it not be made longer ?-

$60.\times 25$

Repeat Morgan Prof. synthesis limitation μ K(E-Avg)

$\frac{S(E)}{K} < \frac{1}{10}$


AA
AA
AA
AA
AA
AA


1 miesiąc
2 miesiący
1 miesiąc
3
3
AA0
AA2
AA1
AA4
AA5
AA1
AA1
1 cm


y

Repressibility

$K_{rep} = \frac{K(\text{Mutant})}{K(\text{Parent})}$

pure repressor ~~plus~~ plus could one conjecture for kind of progeny ~~type~~? no.

In the B strain type wise case: —

assume $\begin{cases} rep & 10^{-6} \\ Any & 10^{-5} \end{cases}$

$$\frac{1 + \frac{n}{10^{-4}}}{1 + \frac{n}{10^{-4}} + \frac{rep}{10^{-6}\ 10^{-4}}} = \frac{1}{100}$$

$[rep] = 10^{-6}\,n$ $\quad rep = 10^{-6}\,n\,l$ $\quad 10^{-10} = 10^{-6} \times 10^{-4}$

$[rep] = \frac{1}{10}\,n$ $\quad$ (Mutant) (any)

if $n(Any) >> 1$ substantial repression

10^{-5} rep is important and in n

~~Which should indicate $E = \frac{n}{n}$~~

in B strain wild type suppressive does not suppress.

1 molecule in 10^{-12} cc

10^{12}/cc $\quad 10^{15}$/l $\quad$ ~~N~~ 6×10^{23}

$6\ 10^{-8}$

$\frac{1}{2}\ 10^{-7}$ mol/liter is limit.

lower

at limit at 10^{-7} mol/liter for cure

of rep. $\quad \frac{rep}{x} < \frac{1}{100} \quad x \geq 10^{-10}$

E_0 Problem:

$$[E_0] = \frac{N_0}{1 + \frac{r_{exp}}{K_0}}$$

$$r_{exp} = \beta \rho(A_{ext}) \frac{1}{1 + \frac{\rho(A)}{K(AE_0)}} [E_0]$$

$$(r_{exp})^2 \sim \beta \rho(A_{ext}) \frac{N_0}{1 + \frac{\rho(A)}{K(AE_0)}} \frac{K_0}{\tau}$$

Saturation is nonlinear because N_t ~~data~~ interacts to E_0

Repressor Theory

~~Inductor~~ $\rho' = \frac{1 + \frac{\gamma_A}{K(A)}}{1 + \frac{\gamma_A}{K(A)} + \frac{r_{exp}}{K_{exp}}}$

} for case of substrates

~~$1 + \frac{\gamma_A}{K(A)} \rho(E_0)$~~ ~~$\gamma_{exp}$~~

$r_{exp} = \frac{1}{1 + \frac{E_n}{\rho(n)}} r_{exp}(n+1)$

$\delta(r_{exp}^{n+1}) =$

from fitting (repressor) is ~~not~~ strongly termed 10^{-6} 10^{-7}

$K_{exp} = K K_0$

$\frac{1}{20}$ sec

τ_{exp} $10^{-4} 10^{-7} = 10^{-12}$ $\frac{1}{2} 10^{3}$ sec

10^{-7} $\frac{1}{20}$ 1%

further about arginine

control. —

$$\frac{1 + \frac{2}{K(0)} \frac{10^{-4}\ \text{m/l}}{10^{-5}} = 10}{1 + \frac{2}{K(0)} + \frac{\text{rep}(0)}{(K_{\text{rep}}(0)} = 1000} \simeq \frac{1}{100}$$

10^{-9} m/l

10 molecules = 10^{-6} m/l

Caution should protect ≡

Tentative solution

~~We~~ assume as altered that one of the later reps is able to bind it to promoter. —
that repressor must remain to "supress"

Applying

there is a basic Eo which is used for rabbits hang for man. —

Eq (o enzymes E ~~which~~ made types are ~~which~~ made by promoter I ~~which make~~.

promoter I is repressed by ~~an~~ enzyme E2 made by promoter II and vice versa

Promoter I also repressed by others so that E1 does not reproduce itself.

Theory

$$K_{12} > K_1 \times K_2$$

If he writes time he be touched

$$P_{12} < P$$

$$A_{12} < A$$

$$2AK = \frac{1}{\tau_{ew}} = A^{13} e^{-\frac{\Delta H}{RT}}$$

$$2A_{12}K_{12} = 10^{13} e^{-\frac{\Delta H_1 + \Delta H_2}{RT}}$$

$$\frac{1}{\tau_{1w}} = 10^{13} e^{-\frac{\Delta H_1}{RT}}$$

$$\frac{t}{\tau_{1w}} =$$

$$K_1 = \frac{10^{13}}{2A_1 f} e^{-\frac{\Delta H_1}{RT}}$$

$$K_2 = \frac{10^{13}}{2A_2 f} e^{-\frac{\Delta H_2}{RT}}$$

$$K_{12} = \frac{10^{13}}{2A_{12} f} e^{-\frac{\Delta H_1 + \Delta H_2}{RT}}$$

$$K_1 K_2 = \frac{10^{13}}{2f} \cdot \frac{10^{13}}{2f} \cdot e^{-\frac{\Delta H_1 + \Delta H_2}{RT}} \cdot \frac{1}{A_1 A_2}$$

10^{-9}

$$K_{12} = K_1 K_2 \frac{A_1 A_2}{A_{12}} \frac{2f}{10^{13}} 10$$

10^{-4}

$$K_{12} = K_1 K_2 \frac{A_1 2f}{10^{13}} \frac{A_2}{A_{12}} = 10$$

$$e^{+\frac{1400}{600}}$$

$A_{12} < A_2$

10^{-5}

10^{11}

Anyindre case:

How can we get strong induction in such a case:—

(Induction:) If K is true whenever E in B strain.—

~~The~~ Premise ~~of~~ rep to be formed must be most effective. This means competitive in terms of E_0 or protective to $M E_{n+1}$.—

But why should rep(n) not ~~express~~ represent E_{n+1} also?

rep n $\xrightarrow{E_{n+1}}$ rep(n+1)

This must have other reasons!

Post mortem:

$$\text{rep}(n+1) = A \frac{\frac{\text{rep}(n)}{K(\text{rep } n;\ E_{n+1})}}{1 + \frac{M(n)}{K(M(n);\ E_{n+1})}} + \frac{\text{rep } n}{K(\text{rep } n;\ E_{n+1})}$$

If M is large one loses information

This gives quadratic effect

A cell [illegible] ~~absorbed~~ dose
showing a period τ_0 the number of
enzymes n_0 becomes 0. assume n
enzymes on the average the probability
for this many given τ_0 is e^{-n}, for $n = 10$
$p = e^{-10} = 10^{-4.5}$ and if there are 6 such
systems $1/p = 10^{3.5} \simeq 3000$

Lifespan $\sim 3000 \tau_0 = 30,000$ days
or ~~not~~ $\tau_0 = 10$ days for man

If we X-ray and destroy 10% of enzyme
average man a probab of $0 \; e^{-9}$ or
e times more cells destroyed during period
or differently compounded. number
of cells with one enzyme $10 e^{-10}$
and of these 10% loses enzyme or X ray
kills e^{-10} or doubles aging.

So we get ~~non~~ linear ~~effect~~ ?
Yes $\frac{n^r}{r!} e^{-r}$.

100×10^{-10} time ; prob. of losing two
is 1% This gives same contribution
for quadratic and higher effects as big
or bigger than direct effect of
X ray doubles aging rate.

K

N ⟶ ● = $\frac{1}{2} 10^{13} e^{-\frac{\Delta H}{RT}}$

Arg + E $\rightleftharpoons$ E-Arg.

$K_{eqn} = \frac{[E\text{-}Arg]}{[Arg][E]}$

$K_{eq} = \frac{[E\text{-}Arg]}{K_{eqn}[E]}$

$K = 10^{13} e^{-\frac{\Delta H}{RT}}$

$K = 10^{2} e^{-\frac{\Delta H}{RT}}$

10^{11}

$\ln K = 4.6 - \frac{\Delta H}{RT}$ $\quad \frac{14000}{600}$

$RT \ln K = 4.6 \cdot RT - \Delta H$

agony

Anders

H

10 days

$\frac{n^r}{r!} e^{-n}$

-4.5

$1 \quad 10$

3000 probability that something happen in 10 days interval between gene pairs

$10 e^{-10}$

e^{-10}

$\frac{15}{5} 10^{13}$

10^{-9} cc

7×10^{12}

W

Anthylen F(-2) with F(1) to Ar

B

Ang - pntd

R

$K = 10^{-5}$ m/l

$10^{12} - \frac{\Delta H}{RT}$

$10 e$

$\frac{1}{100}$

$1 + \frac{2}{K} + \frac{2}{K} - 1000$

$\frac{2}{K}$ 10

$\frac{2}{K}$ 10

$\frac{1}{15}$

10^{-9}

$\frac{\rho}{K} = 100$

$\frac{1}{10}$

Optimum temp for growth could cause about this way:
With increasing T $\frac{L}{}$ could increase and Tetap decrease! —

Exp: Grow ~~hypoplaneless and~~ wild type strain with arginine control in thermostat. Let E(-1) grow as high as possible. —
What is temperature dependence. —
Exp: at high arginine with hypoplaneless B strain and hypoplaneless control. E(-1) goes to a certain level $E_0(-1)$ which can not be further depressed by increasing arginine. What is T dependence of $E_0(-1)$. If fully repressed promgene strength releases E(-1) at a certain rate than E(-1) should increase ~~xxxxxx~~ proportional with T. —

Inhibitor reaction

Augmenter reaction causes it a different rate

forms but is not retained if it does not pass

Concentration of $AA = \mu$

S - prod AA - pathway

b for repressor

a for Inducer*

$$\frac{1 + \sum_{(0,1)} \frac{s(S)}{K(S)} + \frac{[i^*]}{K(i^*)}}{1 + \sum_{(0,1)} \frac{[S]}{K(S)} + \frac{[i^*]}{K(i^*)} + \frac{[r^*]}{K(r^*)}}$$

Expo. Highly inhibited enzyme.

Temp coeff of enzyme level. If enzyme pres up it means that Temp coeff does lower than optimum.

Temp coeff of growth rate of compound X in mycoplasmas strain would be different by.

Benoit: {Compte Rendus 245. p 448
{Vol. 244, 1957 July 15-7
p. 2320

If $\rho(A) = 10^{-4}$ mol/liter a

$$f = \frac{1}{1 + \frac{\rho}{K}} \qquad \frac{\rho}{K} > 1$$

$$\therefore f = \frac{K}{\rho}$$

$$n = \frac{K}{\rho(A)} \rho(rep)$$

~~This method would~~

If we now note $\tau(AA)$ longer ~~than~~ ~~time to form a unrepressed enzyme~~ is independent of B hence as long if in order to reduce enzyme production by factor two

~~If $\tau(AA) = \tau(BB)$~~

$$\frac{1}{\tau} = \frac{1}{\tau(AA) + \Delta\tau_{ind}(E) + \rho\,\tau_{rep}}$$

reduce if rep missing plus

$$\frac{1}{\tau} = \rho \frac{(1-\rho)}{\tau_{ind}(E)}$$ Xprobab. that Enz. is getting there

$$\frac{1}{rec} - \frac{\rho\,\tau_{ind}(rep)}{\tau(AA)}$$

Prob that enzyme is not there

$$q = 1 - \rho\,\tau(AA)$$

Repression

Repressor-trp products. $K = 10^{-5}$ M/l

Half time (removed with trp) $t(\frac{1}{2}) = 10^{-9}$ l

Lac trp represses in $\frac{1}{1000}$ sec $\tau_{av} = 10$ sec. —

How often does rep. bind successfully. 70 min = 4000 sec $\frac{[rep]}{K} > 100$

if 10 sec. stay for rep. $[rep] > 10^{-7}$

each time ($\tau_{av} = 10$ sec)

of means 400 hits

needed, marking passed for these hits

if 10 enzymes are made in 4000 sec;

assume enzyme after being made

stays on 1 sec. is made 400 times

n hits by rep product of no hit

from scratch

enzyme there

all the time

arrived by rep most of the time

but each time rep goes away, enzyme

still stay on 1 sec

$n = K[rep]$

$400 e^{-n} = 10^4$

$400 \cdot 10^{-\frac{n}{2.3}} = 10^4$

$10^{-\frac{n}{2.3}} = \frac{1}{100}$

$10^{\frac{n}{2.3}} = 40$

$n = 4$

$A = 10^{10}$ / $\rho = 10^{-9}$ mol/cc

$[rep] = 10^{-6}$ mol/l

Instructions:

$$\frac{\text{new}}{1-\rho} = \frac{1 + \frac{y}{K(y)}}{1 + \frac{y}{K(y)} + \frac{\rho_{resp}}{K_{resp}}}$$

$$\frac{1}{1-\rho} = \frac{1 + \frac{y}{K(y)} + \frac{\rho_{resp}}{K_{resp}}}{1 + \frac{y}{K(y)}}$$

$$\tau_E = \tau_{ext}(E)\,\frac{1 + \frac{y}{K(y)} + \frac{\rho_{resp}}{K_{resp}}}{1 + \frac{y}{K(y)}} + \tau(AA)$$

$$\tau_E = \tau_{ext}(E)\left(1 + \frac{\frac{\rho_{resp}}{K_{resp}}}{1 + \frac{y}{K(y)}}\right) + \tau(AA)$$

If $\frac{y}{K(y)} \gg 1$

then if we show the growth, τ_E ~~and it not fully induced~~ does not change or that enzyme goes up. [ρ_{resp} goes up, y goes up]

but if we have outside influence y = Const. — then $\frac{\rho}{y}$ goes up

~~do this presupposes~~

$$s = \frac{(1-\rho)}{\tau_{en}(E)}\left(1 - s\,\tau(AA)\right)$$

$$s + \frac{1-\rho}{\tau_{en}(E)}\, s\,\tau(AA) = \frac{1-\rho}{\tau_{en}(E)}$$

$$s = \frac{\frac{1-\rho}{\tau_{en}(E)}}{1 + \frac{1-\rho}{\tau_{en}}\tau(AA)} =$$

$$\tau_E = \frac{\tau_{en} + \left(\frac{1-\rho}{\tau_{en}}\right)\tau(AA)}{1-\rho}$$

$$\boxed{\tau_E = \frac{\tau_{en}}{1-\rho} + \tau(AA)}$$

H

$$s \left(1 + \frac{\tau(AA)}{\tau_{en}}(1-\rho)\right) =$$

$$= \frac{1-\rho}{\tau_{en}}$$

$$s = \frac{\frac{1-\rho}{\tau_{en}}}{1 + \frac{\tau(AA)(1-\rho)}{\tau_{en}}}$$

$$\frac{1}{s} = \tau_{en} + \frac{\tau(AA)(1-\rho)}{(1-\rho)}$$

$$s = \frac{1-\rho}{\tau_{en} + (1-\rho)\tau(AA)}$$

s = growth rate (H)

σ_{gen} = entropy

does this violate (independent law and forces as such be bigger theory? —

assume repressor amt. increases with gen time,

$$1-\rho = \frac{1}{1 + \frac{\rho_{rep}}{k_{rep}}} \approx \frac{k_{rep}}{\rho_{rep}}$$

$$\tau_E = \frac{\tau_{en}(E)}{1/\gamma}\,\frac{\rho_{rep}}{k_{rep}} + \tau(AA)$$

If gen. time doubles ρ_{rep} doubles. τ_E doubles, but what does $\tau(AA)$ do? you may say that $\tau(AA)$ also doubles because more repressed enzymes must retain there load

Non classical

$$N = \frac{\tau_{gen}}{\tau_{rep}} \cdot \frac{\tau_{ev}}{A_{rep}\rho_{rep}} \cdot A_{rep}\rho_{rep}$$

$$N = \frac{\tau_{gen}}{\tau_{rep}} \cdot \frac{\tau_{ev}}{\tau_{rep}} \cdot \frac{\rho_{rep}}{k_{rep}}$$

$$N = \frac{\tau_{gen}}{\tau_{rep}} \cdot \frac{1}{\tau_{ev}} \cdot \frac{1}{A_{rep}\rho_{rep}} = \frac{\tau_{gen}}{\tau_{rep}} \cdot \frac{1}{\tau_{ev}} \cdot \frac{1}{A_{rep}k_{rep}} \frac{\rho_{rep}}{k_{rep}}$$

$$\frac{1}{\tau_{rep}}$$

$$N = \frac{\tau_{gen}}{\tau_{ev}} \frac{k_{rep}}{\rho_{rep}}$$

$$4 = 40{,}000 \cdot \frac{10^{-5}}{\rho} \quad (M/cc)$$

$$\rho = 10^{-5}\ M/l$$

Inner: if inner present:

$$N = \frac{\tau_{gen}}{\tau_{rep}} \cdot \frac{1}{\tau_{ev}} \cdot \frac{1}{A_{rep}\rho_{rep}} \cdot \frac{1}{1 + \frac{\rho(M)}{K(M)}} \approx \frac{k(M)}{\rho(M)}$$

$$N \approx \frac{\tau_{gen}}{\tau_{ev}} \frac{k_{rep}}{\rho_{rep}} \Big/ \frac{k(M)}{\rho(M)}$$

Classical

$$N = \tau_{gen}\, s = \frac{(1-p)\,\tau_{gen}}{\tau(EE) + (1-p)\,\tau(AA)}$$

for obtaining expected enzyme $N = 4$

$$N \approx \frac{k_{rep}\,\tau_{gen}}{\rho_{rep}} \cdot \frac{1}{\frac{1}{\tau_0} + (1-p)\,\tau(AA)} = 4$$

$$(1-p) \approx \frac{k_{rep}}{\rho_{rep}} \qquad p = \frac{\beta_{kk}}{1+\beta_{kk}}$$

$$\frac{k_{rep}}{\rho_{rep}} = \frac{40}{4000} = \frac{1}{100}$$

$$k_{rep} = 10^{-9}$$

$$\rho_{rep} = 10^{-7}$$

15
10 molecule/l 10^{-8}

$$\frac{10^{15}}{6\,10^{23}} = \frac{1}{6}\,10^{-7} = 2\,10^{-7}$$

Back advance:

$$s = \frac{1-p}{\tau_{gen}(E)} \times q$$

or $= 1 - s \times \tau(AA)$

$$s = \frac{1-p}{\tau_{gen}}\,(1 - s\,\tau(AA))$$

$$s\left(1 - (1-p)\frac{\tau(AA)}{\tau_{gen}}\right) = \frac{1-p}{\tau_{gen}}$$

$$s = \frac{1-p}{\tau_{gen} - (1-p)\,\tau(AA)}$$

$$N = \tau_{gen}\, s \approx (1-p)\frac{\tau_{gen}}{\tau(E)}$$

$$\sim \frac{k_{rep}}{\rho_{rep}} \times 4000 = 4$$

$$k_{rep} = \frac{s}{10^4}$$

$$\rho_{rep} = 10^{-5}$$

$$k_{rep} \simeq 10^{-9}$$

The minimum oxygen level is determined by [R] for even zero k_2

The growth of hyperplasmodes.

f/e outside ~ 100 f/e inside

~~10^{-6} mol/l~~ 10^{-7} mol/l =

$= 10^{-5}$ gm/l $\frac{1}{2}$ conc of hyperbolic

70 min = 4200 sec

I 2mm = 35 min if τ goes to 2×70 min

I mm becomes 3 fold

Arginine is 5% or gm $= \frac{1000}{20} = 50$

$10^4 \tau = 3 \times 35$ min $= \left| \frac{1}{AS} \frac{50}{1 + \frac{S}{K}} \right| 10^4 =$ 6000 sec

$\rho = 10^{-9}$ 10^{-6} 10^{-4} gm/l $\frac{1}{AS} \frac{50}{1 + \frac{S}{K}} = \frac{1}{2}$ sec

$\frac{1}{3 \times 10^4}$ $\frac{1}{AS} \frac{1}{1 + \frac{S}{K}} = \frac{1}{100}$ sec

Antibody formation: 4
Small injection of phenol into
should produce in mouse annual
anamnestic response on first
injection. —

~~Points for discussion~~

~~K for repressor~~ **

Assay: $\tau(R)$, and timing and

any $K = 10^{-12}$ mol/l

$\frac{\tau(E)}{\tau(HA)} \frac{R}{K}$ $\frac{R}{10 \cdot 10^{-12}} = 10^4$

$R = 10^{-6}$

$\frac{1}{10^{-4}}$

(in mem τ HA)

this is it

when $\tau(E)$ small R finite
suppression can be made large
but timing more
probability of escape!
each time when R loses.

$R = 10^{-6}$ mol/l

~~$P = \tau(E) \cdot R$~~

$e^{-\tau(E) \cdot R}$ $3 \cdot 10^{10}$ $10^{-9} = 3$ ~~$\tau(E)$~~

Mammalian cells have no mitochondria!

If Bill's ~~enzyme~~ protein is one enzyme and is made by 1 gene and if cell is 100 times as large as bacterium it should take 100 times as long to divide

20 min × 60 = 2,000

a day is 24 × 60 = 1500 min

In bacteria ~~1 protein~~

1 protein molecules of one enzyme

is = ~~1000~~ 10^{-13} gm Protein

$\frac{10^{4}\ 10^{5} = 10^{9}}{6.10^{23}\ 6.10^{23}} = \frac{1}{6}\ 10^{-14} = 60$

or $\frac{1}{60} \sim 2\%$

~~How Sept 1/57~~ 4

Can we really assume that R is not destroyed (in order to get law of growth rate independence) this would mean that if we start producing more repressor the repressor level would rise for a generation. - ~~stop~~ Experiment reinvestigate) How would one also explain the non linear (with TMG some) enzyme level (sigmoid type curves) in crypte. Could one say that enzyme destroys R and otherwise it is stable? TMG phenomenon, could it be explained by saying T.M.G slows production of precursor to inhibitor but as enzyme grows up ~~the~~ more inhibitor is produced, could this take a sigmoid type curve?

Answer see in other book

New Paper

N+K

KH 15 mm Cal $K = 10^{-6}$ m/s

$m = 1000$

$$= \frac{1}{AS}\frac{m}{1+\frac{S}{K}} + \frac{1}{AK}\ln m$$

I II

1.3×10^{-5} m/s

$Q_{CO} = 2.7$ 10%

100 1000 1000.0 1000

1000/1m

factor 6 gained by minimum

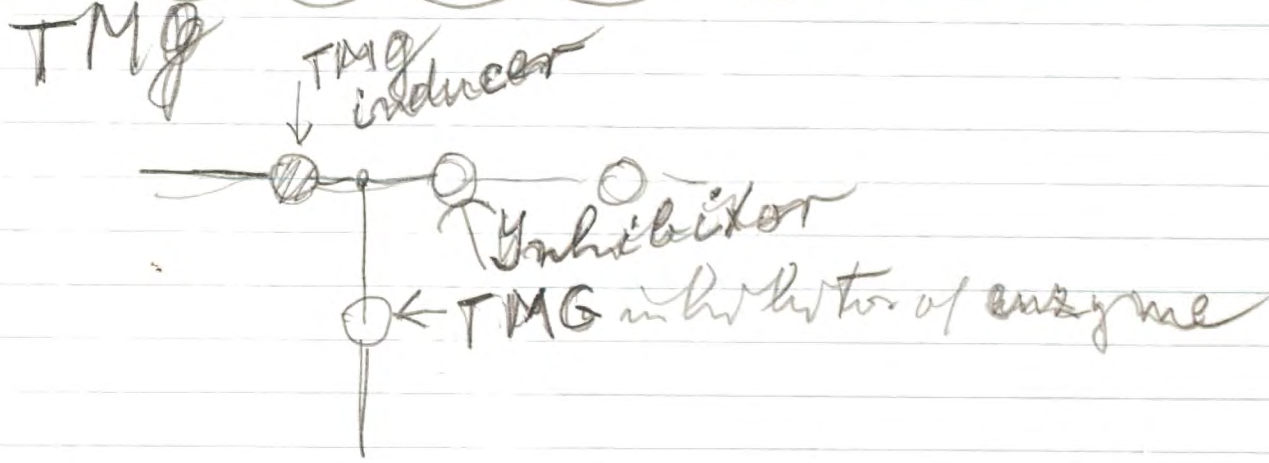
Lowest growth rate.
Is it due to denaturation
of the enzyme? - What
about lowest growth rate
in thermostat at 27°C?
Why is Q10 in my case
" " Q10 in denaturation
~~Do~~ Do enzymes ~~not~~ lose
which denature at a new
temperature have a
higher Q10 than those which
denature slowly at Room
temperature?

antibody formation
Is there a threshold. If
we feed radioactive glycine
will any mice in there
a threshold dose for
injected produce an

Enzymes!
What happens to enzyme
level / activity if cultured at T = 15 hrs

Notebook 4

~~Does~~ If one injects on to the antigen (just below the threshold) (Is there really a threshold? Use indicator method with early antigen removal!) that no primary response occurs is there then still an ~~anam~~ anamnestic response. There should be!



all other annular cases in abundance

At low pressure, the first term is limiting

At high pressure first power rate / when the second term is limiting and so

$$\frac{1}{\rho_{(temp-error)}} \times \frac{\dot{m}_{(temp)}}{A} + \sim = \tau_{(A,A_2)}$$

$$\begin{cases} \rho = \rho \\ \rho_1 A_1 + A_2 = \tau_{(A,A)} \\ \rho_2 A_1 + A_2 = \tau_{(A,A)} \end{cases}$$

$A_2 = \tau_{min\,(A,A)}$

from this we get A_2 and (K)

from A_2 we compute ~~temps~~ each of A_2

one can move from both and predict temps each.

Post synthesis.

Something wrong

(4)

In breath apparatus at 37
When all unnecessary high
pressure can only fall off
Overbreathing occurs.

Exp engine (Haldane)
Do you get CO2 if you
suppress enzyme with KCN.

Exp Effect of Post. Synthesis
Take fully induced enzyme
how much is produced in
unit time
in broth
in individual } at two different
temperatures.

Try high concentration of Urea
in Broth

This could test be done in
dialysis tube - say with arginine
control using no permeases
present and measuring
the enz. conc.

Antibodies

What about diphtheria toxin? Is neutralizing antibody present for active use? If we are could inject toxin, follow it with horse anti toxin. ~~and assume horse anti-toxin with~~ When testing for rabbit anti toxin the horse serum can be removed with rabbit anti horse in test tube:

Antibody: 3 kg Rabbit A. 1 mgm threshold

10 cc lymph tissue

at antigen 30 days in blood.
50 in extracellular fluid [ubiquitous throughout volume]
0.1 mgm tissue a much as 3 week
4 not detectable

1 mg/kg

1 or 2 % might go into cells. —

10% could be all in lymphoid tissue x —

(10 g) sites 10^{-5} gm in 10 cc
10^{-2} gm in 1 l
12 kg ~~in motor~~

0.1 % mgm gives 10% of maximum
secondary response
under conditions of Antibody excess. —

$h = \frac{\%}{100} \frac{1}{\text{sec}} A[R] = (0.3) \frac{1}{\frac{M}{K}} e$

10^{-5}

$\frac{ARK}{M}$

$10^{-7} < R < 10^{-5}$ mol/l

Protein synthesis and catalytic turnover. ——

$$\frac{1}{A\beta(A_{xy}^{*})}\,\frac{q\,m}{}+\frac{\rho(\quad\quad\quad)}{K\,(N+\alpha)}$$

$$\rho(A_{xy}^{*}) = \omega\,\beta(A_{xy})$$

$$N = \frac{C^{[R]}}{[R]}\,\frac{1}{\beta} + C$$

$$N = \frac{1}{A\alpha C(A_{xy})} + C$$

Rep.

$$\frac{2}{[A\,A_{xy}]} \qquad \frac{q\,m}{1 + K^{[A_{xy}]}}$$

C can be measured when [Axy] = ∞

$$K = \alpha$$

!!

$$A_{xy}^{*} = A_{xy}^{**}$$

represor = A_{xy}^{*}

A^{+} is reactor

A^{**} is answer

$[A_{xy}]$ can be measured in mutant lacking permease, this gives us A_{xy} as a test with α

we assay (in mutant where $[A_{xy}]$ can be measured) $[A_{xy}]$ and measure $\Delta\sigma$

We succeed so far

Shutdown Unit. —

$\frac{\text{Open}}{\tau(A) + \tau(AA)}$ = rate

14000

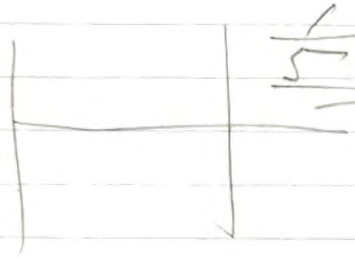
4000 sec

N_1

600 $\quad \tau(AA) = \frac{1}{As} \quad \frac{1}{1 + \frac{s}{4}}$

1 sec / 5 =

1000



600

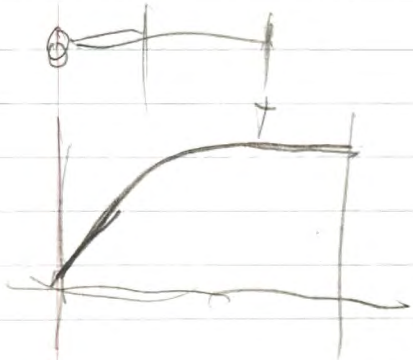
1 emp in 40 sec

1 emp in 4 sec

Works 5 employees

N_1 ∘ T

800 seconds



1000 seconds

Not enough

$\frac{N_1}{N_2}$

4000

10000 = 2000

5

$K(M) = 10^{-3}$ means time for half reaction

free $= \frac{1}{1000}$ M would come down in 30 sec

TMG / ~~glucose~~ shall elute in ~~two min~~, 100 sec

100 sec. sticks to 40000

$\frac{1}{35}$ sec

if TMG 10 $\frac{10^{-2}}{10^{-5}}$ = OK

time

~~1/10~~ ~~OK~~ $\frac{1}{10^{-3}}$

How much to R is less interact than TMG in 100 sec

R ~~amines~~ amines

$AR_3 = 10$

$\frac{K_{R3}}{K_R} = \frac{100}{35} = \frac{1}{3}$

$K(M) = 10^{-2}$ How often does M to R break away?

$\frac{1}{1000}$ of time at

for TMG to elute in 100 sec ⇒

$\frac{10^{-2}}{10^{-5}}$

$\frac{1}{10^{-3}}$

$K(R) = 10^{-9}$ mol/l

free at NH and 1 in 10.

$\frac{1}{1000}$ free

Eq: Instruction very short time elute after 11 sec etc.

E. Cohen

4

1 in 10^{-21} cc

1 cc 10^{-21} molecules

1 liter 10^{24} molecules

or conc 1 mol

in diameter is 10^{-2} cm

Assume $K = 10^{-6}$ binding of TMG

Time occupied by M* $= \frac{1}{35}$ sec

$\therefore$ fraction of time occupied by M

$$\frac{\frac{10^{-2}}{10^{-6}}}{1 + \frac{10^{-2}}{10^{-6}} + \frac{10}{10^{-7}}} = 10^{-4}$$

ten molar

assume 10^{-3} TMG does not elute

elution time 100 sec would

mean it is lowered at M and

$$100 \frac{1}{35} = \sim \frac{1}{3000} \text{ of time}$$

$N = 10^8$ / NV (1 cm) $\rightarrow$ * $\frac{1}{5000}$ sec

$V = 5 \times 10$ cm/sec.

$\lambda \sqrt{N_A} = 10^{-4}$ cm

$\lambda \nu = \text{diff} = 10^{-5}$

$\frac{\lambda}{3} \lambda = \frac{3 \times 10^{-5}}{5 \times 10^3} \sim 10^{-8}$

Electron cycle

Switch time = 40000 sec or 1 cycle

Lag in electron 100 sec

Electron must reduce time at M-N of being attached to enzyme by factor 400

1/1 fraction of time in Michaelis blocks.

$$\frac{[M^*]}{K(M^*)}$$

$$\frac{[R^*]}{K(M^*)}$$

$$[M^*] = \frac{1000}{Vol \times 6\,10^{23}}$$

$$Vol = 10^{-21} \qquad [R^*] = \frac{1000}{600} \sim 1$$

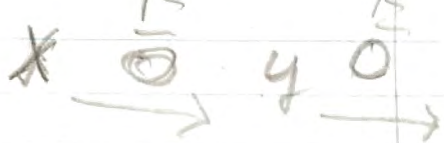
$$\frac{[M^*]}{[M_X]} \sim (N+M) \times \frac{M_X}{K_X} = 10^5$$

$$\frac{[M^*]}{K(M^*)} \Big/ \frac{[M_X]}{K(M_X)} \times \bar{c}_{ev} = 100$$

A[R] c(E)

How many ways can TMG act?

1. (1,1) ~~robust transcription~~ promoter M'
2. (2,1) + decrease of ~~Rep~~ on M
3. 3.) Inhibitor u of ~~rep~~ to a
4. 4.) acts as [illegible] in trans
5. 5 can compete for NTP with R.



$\frac{dy}{dt} = ax - by$ $ax = by$ by

I wish by is Restricted 4 fold action.

shortening elution time with a more indirect ? ! possible

With ~~most~~ robust power does TMG inhibit

Clustron

when inductor present

time freq at M and:

$$f = \frac{1}{1 + \frac{M_{carrier}}{K_x}}$$

$$\frac{\tau(\text{ew NM})}{f} = 10^5 \text{ sec} \simeq \tau(\text{ew N\&R}) \; / \; \frac{M_{carr}}{K_x}$$

complementary:

$$\frac{\tau_{ew}(M)}{} \quad \frac{1}{\frac{M_{carr}}{K_y}} + \frac{1}{\tau(\text{ew N\&R}) \frac{M_{carr}}{K_x K_y}} + \tau(\text{ew } M^*) + \frac{M_{carr}}{K_y}$$

$M_{carr}(NM) = M_{carr}(M)$

1.) $\frac{10^5}{10^2} = \frac{M^*}{K}$ sec.

$$\frac{M^*}{K^*} \frac{K_x}{M_{carr}} 10^2 = \tau(\text{ew N\&R})$$

Future tech 10^5

$$K_x = \frac{10^5}{\tau_{ew}(NM)} \times M_{carr}$$

$\tau_{ev} = 10^5 \frac{[K_x]}{[M_x]}$

$\bar{\sigma}_{ev} = 10^0 \frac{[M_x]}{K_x} \frac{K(M^*)}{[M^*]}$

$10^5 = \tau_{ev}$

$10^3 \left(\frac{K_x}{M_x}\right)^2 = \frac{K(M^*)}{[M^*]}$

$\frac{1}{1+\frac{M}{K}}$

$\frac{K_x}{M_x} = \left[\frac{1}{1000} \frac{K(M^*)}{M^*}\right]$

$\bar{\tau}_{ev} = 10^5 \frac{K_x}{M_x}$

$\bar{\tau}_{ev} = \left[\frac{1}{1000} \frac{K M^*}{[M^*]}\right]$

$\frac{K(M^*)}{[M^*]} = 10^{-5}$

$\frac{[M^*]}{K(M^*)} = 10^{-2}$

$M_x = \frac{\bar{\tau}_{ev}}{10^5 K_x}$

$\frac{\bar{\tau}_{ev}}{\text{Time base}} =$

$\frac{\text{Time base}}{\tau_{ev}} = 10^{-5}$

$K_x = \frac{\tau_{ev}}{M_x 10^5}$

$K_x = \frac{10^{-5} \tau_{ev}}{M}$

$\frac{K_x}{M_x} = \frac{10^5}{\tau_{ev}(M_x)}$

$\frac{K_x}{M_x} 10^5 = \bar{\sigma}_{ev}$ ok

$\frac{K_x}{M_x} \frac{M^*}{K(M^*)} \frac{1}{\tau_{ev}} =$

$\frac{M^*}{K(M^*)} = \frac{10^5}{10^2}$

$\frac{M^*}{K(M^*)} \frac{K_x}{M_x} 10^{+2} = \tau_{ev}$

(5) $\frac{1}{\tau(ew-M)} \frac{K_y}{M_{cupst}} < 10^{-5}$

(6) $\frac{1}{A_{NRd} K_y} = \tau(ew\text{-}NRd)$ $\quad K_y = \frac{1}{A(NRd)(\tau ew NRd)}$

from (5) from (4)

(5a) $\frac{K_y}{M_{cupst}} \; 3\,10^{10}(ew)\, K_x < 10^{-5}$

10

A_M

with (5a) from (6)

(5b) $\frac{A(M)}{A(NRd)} \frac{1}{\tau(ew\,NRd)} \frac{K_x}{} < 10^{-5} M_{cupst}$

(1) $\frac{K_x}{M_{cupst}} 10^5 < \tau(ew)(NRd$

$\frac{A(M)}{A(NRd)} \frac{K_x}{M_{cupst}} 10^5 < \tau ew (NRd)$

smaller → 10

M_{cupst} = [illegible] more

10 A gives $M=2$

Electron

De Novo:

transmittance free at M end = f

$$\frac{f}{\tau(ev\ N+d)} = 10^{-5}\ sec^{-1}$$

$$T = \frac{1}{1 + \frac{M\ capt}{K_x}} = \frac{K_x}{M\ capt}$$

(1) $$\frac{K_x}{M\ capt} 10^5 = \tau(ev\ N+d)$$

$$K_x = \tau(ev\ N+d)\ 10\ M_{capt}$$

transmittance M cap 10^{-2}

$$K_x = 10^{-2} \frac{M\ capt}{\tau(ev\ N+d)}$$

lag = 100 sec

$$T\ free = \frac{\frac{M^*}{K^*}}{1 + \frac{M\ capt}{K_x}}$$

$$\frac{T}{\tau(ev\ N+d)} = 10^{-2}\ sec^{-1}$$

(2) $$T \simeq \frac{M^*}{K^*} \frac{K_x}{M\ capt}$$

$$\frac{M^*}{K^*} \frac{K_x}{M_{cap}} \times 10^2 = \tau(ev\ N+d)$$

$$\tau ev \simeq \frac{10^5 K_x}{M\ capt}$$

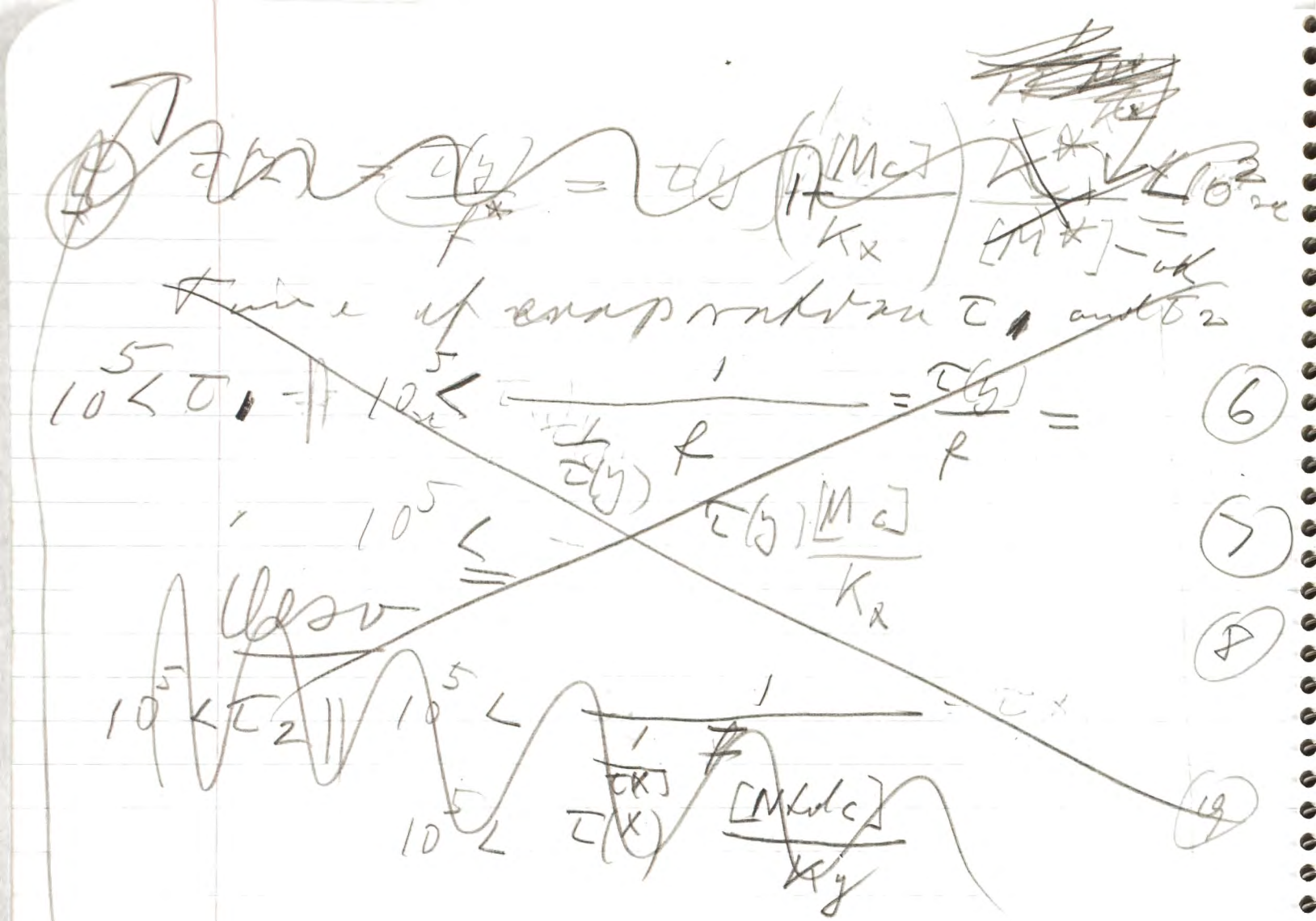
$$\frac{M^*}{K^*} \frac{K_x}{M_{cap}} 10^2 = \frac{K_x}{M\ capt} 10^5$$

(3) $$\frac{M^*}{K^*} = \frac{10^5}{10^2} = 10^3 = \log$$

1, V.K.

(4) Complementary

$$\tau(ev\ M^*) = \frac{1}{3 10^{10} K_x^*}$$



Time of exposure between τ_1 and τ_2

$$10^{-5} < \tau_1 = \frac{1}{\frac{\tau(y)}{R}} = \frac{\tau(y)}{R} = \quad (6)$$

$$10^{-5} < \frac{\tau(y)[M_c]}{K_x} \quad (7)$$

$$10^{-5} < \tau_2 = \frac{1}{\frac{1}{\tau(x)} + \frac{[M_x d_c]}{K_y}} \quad (8)$$

$$\frac{1}{\tau_0} = \frac{1}{\tau_1} + \frac{1}{\tau_2} \quad (9)$$

(5) $\tau(2) < 10^{-2}$ $\quad \tau(2) = \frac{\tau(y)}{R^*}$

(5a) $\tau(2) = \tau(y)\left(\frac{1 + \frac{[M_c]}{K_x} + \frac{M^*}{K^*}}{1 + \frac{M^*}{K^*}}\right) < 10^{-2}$

Dynamic Equilibrium — 4

part (3) $k^* \quad \frac{10}{10^2} \quad M^*$

we set

(10) $\tau^*(x)$ = evap of M moiety

$\tau(y)$ = evap of N+d moiety

f probability of time M moiety is base

F " " " " N+d moiety is base

we set $\frac{1}{\tau(x)} F \quad < 10^{-5}$

$\frac{1}{\tau(y)} f \quad < 10^{-5}$

(1) $F = \frac{1}{1 + \frac{[N+d]_c}{k_y}} \simeq \frac{k_y}{[N+d_c]}$

(2) $f = \frac{1}{1 + \frac{[M]_c}{k_x}} \simeq \frac{k_x}{[M_c]}$

Time for electron $\tau(z)$

(3) $f^* \frac{1}{\tau(y)} = \frac{1}{\tau(z)}$

(4) $f^* = \frac{1 + \frac{M^*}{k^*}}{1 + \frac{[M_c]}{k_x} + \frac{M^*}{k^*}} \quad \frac{[M^*]}{k^*} \frac{k_x}{[M_c]}$

With equal signs

from (7a) and (5c)

(10) $$10^2 \frac{M^*/K^*}{\frac{M_c}{K_x} + M^*/K^*} = 10^5 \frac{K_x}{M_c}$$

(11) $$10^2 \frac{M^*/K^*}{1 + \frac{M^*}{K^*}\frac{K_x}{M_c}} = 10^5$$

or if $\frac{M^*}{K^*}\frac{K_x}{M_c} \ll 1$ { $\frac{M^*}{K^*} < 1000$, $\frac{K_x}{M_c} < 10^{-4}$

(12) $$10^2 M^*/K^* = 10^5$$

$$M^*/K^* \approx \frac{10^5}{10^2}$$ e.g. { $M^* = 10^{-2}$, $K^* = 10^{-5}$, $M_c \sim 1$, $K_x < 10^{-4}$

From 9 we get 9*

9(a) $$10^5 < \tau(x) \frac{[N+M_c]}{K_y}$$

(with $\frac{[N+M_c]}{K_y} \gg 1$)

~~and complementary~~ ~~also~~ 4

~~(6)~~ Further we have

$$10^5 < \tau_1 \;\Big|\; 10^5 < \frac{1}{\frac{1}{\tau(y)} f} \quad (6)$$

$$10^{+5} \leq \tau(y)\left(1+\frac{[M_c]}{K_x}\right) \quad (7)$$

and complementary

$$10^5 < \tau_2 \;\Big|\; 10^5 < \frac{1}{\frac{1}{\tau(x)} \neq} \quad (8)$$

$$10^5 < \tau(x)\left(1+\frac{[N+d_c]}{K_y}\right) \quad (9)$$

from (5a) and (7) we obtain (with $1+\frac{[M_c]}{K_x} \approx \frac{M_c}{K_x}$)

(7a) $\boxed{10^5 \frac{K_x}{M_c} \leq \tau(y)}$

~~and with $1+\frac{M^*}{K^*} \approx \frac{M^*}{K^*}$~~

5b $$10^2 \times \frac{1+\frac{M^*}{K^*}}{1+\frac{M_c}{K_x}+\frac{M^*}{K^*}} \geq \tau(y)$$

and with

$$\frac{1+\frac{M^*}{K^*}}{1+\frac{M_c}{K_x}+\frac{M^*}{K^*}} \approx \frac{\frac{M^*}{K^*}}{\frac{M_c}{K_x}+\frac{M^*}{K^*}}$$

5c $$10^2 \frac{M^*/K^*}{M_c/K_x + M^*/K^*} \geq \tau(y)$$

Try to satisfy more accurate formula with larger k^* !!!

(11) shows it can not be done;

(11a) $$10^2 \frac{M^*}{K^*} = \left(1 + \frac{M^*}{K^*} \frac{K^*}{Mc}\right) 10^5$$

$$\frac{M^*}{K^{**}} \geq \frac{10^5}{10^2}$$

For the correlation for enough repressor (2) in absence of M^*. It we must only impose conclusions to be produced

$D[E] R[R] \sim 1$ $\quad (3\,10^{-7})$

$$\begin{cases} D[E] = \frac{1}{10} \text{ sec} & \Delta R = 10 \\ D[E] = \frac{1}{100} \text{ sec} & \Delta R = 100 \end{cases} \quad \begin{cases} R = \frac{1}{3\,10^6} \\ \left(3\,10^{-6}\right) \\ R = \frac{1}{3\,10^5} \end{cases}$$

$A = 3\,10^7$ mol/liter

Number of spots per $[M^*]$
Growth are factor known not
1) Continuously supply of R
2) Competing with M_c for NEBS
3) Preemptive effect

---

and from 5 & 6 we get 4 & 6

9(4) $\mathcal{O}(x) \geq 10^5 \frac{K_y}{[M+M]_c}$

(7a) $\mathcal{O}(y) \geq 10^5 \frac{K_x}{[M_c]}$

From (7a)

setting $\mathcal{O}(y) = 100 \text{ sec} > \mathcal{O}(y) \geq 10^5 \frac{K_x}{[M_c]}$

is fulfilled if

$K_x = 10^{-5}$

and $[M_c] \geq \frac{1}{100}$

OK.

We could have $M_c = \frac{1}{10} = 10^{-1}$

we must have to have solved formula (12)

$M^* = 10^{-2}$

$K^* = 10^{-3}$

$K_x = 10^{-6}$

$\frac{M^*}{K^*} \frac{K_x}{M_c} \ll 1$ we escape from $K^* = 10^{-5}$

(11) Theorem can not be done

Hex p replaces
Bunsen-Kirchhoff method
by Hydrazine-azide
method / mechanism
not due! — paper Seymour S. Cohen
& Horst D. Bauer
Journ. of Biol. 7, 500, 56

Insert page 5 (second draft)

~~As we shall presently see either~~
~~of these mechanisms that are~~
~~and is brought to bear~~
As we shall presently ~~see~~ if one wishes
to describe a mechanism that may
account for the phenomenon of
enzyme ~~activation~~ one ~~immediately~~
runs into the following difficulty.
~~Either of the two~~ mechanisms that ~~are~~ may be
~~the~~ proposed because at first sight
they appear most reasonable, would
flagrantly violate the principle of
growth rate independence. ~~Both~~ Either
of these ~~the~~ mechanisms ~~would~~ would lead
to the following prediction:
~~If~~ Let us assume that ~~at~~ at first growth ~~rate for~~
~~when the minimal conditions is when~~
~~supplemented with~~ the ~~required~~
~~growth factor~~ concentration of the controlling growth
factor is ~~kept at a high level~~ made a limit high
an enzyme Ea ~~is present in a~~
~~growth~~ two is ~~maintained~~ formed in the
steady state in
a growing culture at a rate ~~by~~ 20 ~~to~~ 100
times higher ~~than~~ than another
enzyme Eb. When by ~~keeping~~
~~of the time~~ lowering of the ~~[illegible]~~
curve of the controlling growth
factor the rate of the formation
of Eb is reduced in the steady
state ~~of~~ by a factor 2 then
~~or those mechanisms predict~~
the enzyme Ea ~~is formed~~ will be
formed only twice as fast as
enzyme Eb.—

Insert page 2 (second draft)

In E. coli for instance ~~there~~ the cell maintains in a growing culture about 1000 molecules of the enzyme β-galactosidase when this enzyme is fully induced and ~~any~~ ~~other~~ the same number of molecules might be maintained of any one other enzyme provided only one knew how to induce it fully. ~~Most enzymes in the average~~ Assuming 10^4 gms/mol and an average molecular weight of 1000 a bacterium like E. coli which contains about 10^{-13} gm of protein per cell, maintains on the average 60 molecules ~~of each~~ of each of the different enzymes. How does the bacterium regulate the level at which each enzyme is maintained? If ~~we can~~ we ~~answer this~~ have the answer to this question we probably also have the answer to the problem of enzyme induction. —

Insert page 3 (Second draft)

This principle ~~of gradual change in proportions~~ here — which I am postulating — (— as a general rule — demands that the ratio of the quantities of the different enzymes contained in the bacterial cell shall be independent of the quantities of the bacteria.

Insert 4 page 5 (second draft)

The experimental ~~my~~ evidence which I cited above in support of the principle of growthrate independence ~~is not to be~~ might be regarded as not compelling because the distribution of the enzyme β-galactosidase is in many respects ~~not~~ typical of ~~the~~ ~~the~~ of enzyme distribution in general. There are however I believe ~~my~~ compelling considerations of a ~~rather~~ general nature which ought to convince us that ~~models~~ ~~tests~~ the above mentioned flagrant violations of the principle of growthrate independence ~~are not~~ is not likely to exist in nature

Insert III. page 5 second draft

In the circumstances I shall only briefly discuss these theories ~~and~~ and I shall do so mainly because they are shopping shows for a theory that ~~conforms~~ to the principle of growthrate independence ~~that at least is~~

at fast growth we have
~~If~~ We shall ~~now~~ consider the case where ~~N₁ >>> N₂~~ ; ~~we have~~ and $\Delta(2) >>> \Delta(1)$

$\frac{N_1 \text{ fast}}{N_2 \text{ (fast)}} >>> 1$ and $\Delta(2) >>> \frac{1}{\tau(E-M^*)} + \tau(AA \text{ fast})$

and ask what happens to the ~~when we~~ we reduce the rate of production of E_2 ~~to~~ by factor 2. ~~we have by [illegible]~~ (which ratio $\frac{N_1}{N_2}$)

by growing the bacteria sufficiently slowly. ~~[illegible]~~ In order to accomplish this ~~[illegible]~~ we must making $\tau(AA, \text{slow})$ sufficiently large ~~[illegible]~~ so that we have:

~~[illegible]~~

$$\Delta(2) = \tau(E-M^*) + \tau(AA; \text{slow})$$

~~and we then have~~ If we do this we have

$$\frac{N_2(\text{slow})}{t(\text{gen, slow})} = \frac{1}{2\Delta(2)}$$

and

$$\frac{N_1(\text{slow})}{t(\text{gen slow})} \cong \frac{1}{\Delta(2)}$$

Therefore at this slow growth we have

$$\frac{N_1}{N_2} = 2$$

$\Delta(M)$ does not depend ~~[illegible]~~ ~~[illegible]~~ ~~[illegible]~~ on t gen but M which may be ... with M*

This is probable: that our ... is ... with M^*

Paper

New paper ~~[illegible]~~

$$N = \frac{t \text{ gen}}{\Delta + \tau(AA)}\text{; where } \Delta = \frac{1}{\Phi(M)}\tau(E^*M)$$

Δ (independent of $\tau(AA)$ or t gen) $\Phi(M)$ is one for high M and ... for low M

Appendix to Paper 9 (H)

second draft

For Paper Collected for Printing. — Appendix

Made Not

$$N = \frac{\tau_{gen}}{\tau(E-M^*) + \tau(AA)} \qquad \Delta$$

with M^* employed

(perhaps is E with up to complement with M^*)

$$N_0 = \frac{\tau_{gen}}{\tau(E-M^*) + \tau(AA)}$$

$N(H) >> N(f)$; perhaps $N(H) >> N(s)$

$\Delta(2) >> \Delta(1)$; $\Delta(2) >> \tau(E-M^*)$

f stands for fast

s for slow

If we

and for rate of production if only we have

$$\frac{N}{\tau_{gen}} = \frac{1}{\Delta + \tau(E-M^*) + \tau(AA)}$$

γ is clear

We may now see how the ratio N_1/N_2 changes if we change $\tau(AA)$ by staining the growth rate of bacteria

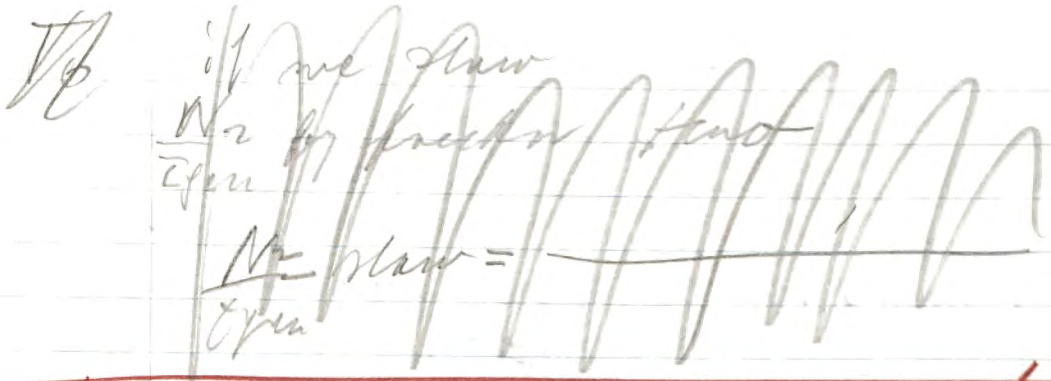
We have when the bacteria grow fast:

$$\frac{N_1(fast)}{\tau_{gen}(fast)} = \frac{1}{\Delta(1) + \tau(E-M^*) + \tau(AA; fast)}$$

$$\frac{N_2(fast)}{\tau_{gen}(fast)} = \frac{1}{\Delta(2) + \tau(E-M^*) + \tau(AA; fast)}$$

10

molecule R may be combined
with ~~the~~ an ~~enzyme~~
Acetyl coenzyme A molecule
~~that has been formed as a~~ working
on the enzyme at the
same time as the NAD moiety
~~of the material~~ is combined
~~with a receptor part of the~~
~~receptor that part of the~~
~~of the enzyme~~ to which it
has chemical affinity. ~~This~~
~~part of~~
to a portion of the enzyme
to which it has specific
chemical affinity. - This
portion of the enzyme might
be composed of ~~perhaps three~~ several
nucleotides and the NAD
moiety might also be composed
of several nucleotides and
~~be at opposite ends of the~~
~~to a receptor~~ the NAD moiety
might attach to the enzyme
through hydrogen bonds
- that may form between the
purines and pyrimidines of
the NAD moiety and ~~the~~ the
complementary purines and
pyrimidines on the enzyme,



Ms I page 12 (second draft)

In order to account for the ~~transmission of the~~ repression of the formation of the enzyme beta-galactosidase by arginine I shall ~~hypothesize~~ postulate the following: the cell is capable of ~~making~~ forming from Arginine a ~~two headed~~ molecule R which may be symbolically written as

R = Arginine — NH-C ~~and~~ ~~a molecule~~ such molecules is present in the cell at a sufficient concentration may repress the formation of the enzyme really quickly provided that the ~~other~~ metabolic ~~conditions~~ conditions are such the Arginine moiety of the

and we assume that it enzymatically at the same rate if it is not free but combined with a metabolite M (such as for instance arginine) or its precursor M' or a dependent analog M* at site M*.—

Accordingly we may rewrite for the number of enzyme molecules per cell growing exponentially

Rules mentioned in the steady state at a

go back to pages 12, 13, 14 and 15

Insert bottom of page 12

where p is the fraction of the time $\tau(E)$ during which an enzyme molecule that acts on the metabolite is free from M*

Hiramoto: (4)

$$p = \frac{1 + \frac{\tau(E)}{\tau(M)}}{1 + \left(\frac{M}{K(M)} + 1\right)\frac{\tau(E)}{\tau(M)}} = \frac{1 + \frac{\tau(M)}{\tau(E)}}{1 + \frac{\tau(M)}{\tau(E)} + \frac{M^*}{K_{M^*}}}$$

(1) $N = \frac{\sigma \mu}{\Delta + \tau(E) + \sigma(\Delta R)}$

(2) $\Delta = (1 - e^{-h\tau(E)})\sigma R$ at least one

(3) Hit rate $h = \Delta R \tau(E) p$

$\Delta = 1 + e^{-h\tau(E)} \sigma R$

is probable at least in that

~~The cross sections here presented~~
are not ~~Beta~~ The designation
Which was chosen with this possibility
in mind, but the general
cross sections to be presented
are not dependent on this ~~assumption~~
~~assumption~~.

~~It will be important to keep~~
~~in mind at all times the molecules~~
~~concentration for the enzyme~~
~~of the molecule R which is~~
~~produced~~ Eo defining at Lake Page 13 Town

If we now say that the molecule
R represents the formation of an
enzyme we mean that ~~when~~
~~the precursor enzyme~~ if a molecule
R is combined with the enzyme
molecule that sits on the pre-
pere and the presence the enzyme
molecule cannot cause the pre-
pere. ~~When~~ When the enzyme
molecule is not so combined ~~or~~
~~but is either free or is~~
~~combined with a molecule~~
~~A or its chemical analog~~
A* (or Alan) it evaporates ~~at the~~
~~cake~~ - so we shall assume -
at ~~rate~~ some rate, ~~with~~ which
but we know it evaporates
at some rate, which we
shall also make ~~with the~~ $\frac{1}{\tau_E}$.

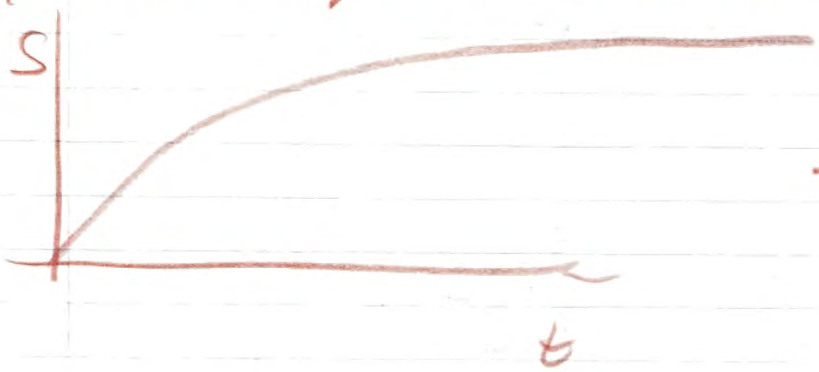
10 days to dissociate off. Let us then examine what we obtain if we assume that a repressor molekule R once it is combined with an enzyme molekule sitting on the promgene remains combined until the cell nucleus divides and is then somehow "shaken off." ~~and further let us assume that the time when the nucleus divides is determined~~ ~~In the absence of repressor for~~ Whenever an enzyme molekule is formed on the promgene is it likely to $A R \Sigma(E)$ ~~times~~ ~~by a repressor molekule~~ by repressor molekules and $A R \Sigma(E)$ times before it evaporates and ~~it~~ each time ~~when~~ the probability $d(p(M))$ of being placed by the repressor molekule to the promgene for the rest of the generation time. ~~the~~ ~~working hypothesis $A R \Sigma(E) p(M) = h$~~

~~We plot~~ ~~No promgene can become~~ ~~In order to obtain the number of~~ we write $A_0 R \Sigma(E) p(M) = h$

the average number of enzyme molekules No which we obtain from a promgene ~~which~~ ~~enzyme~~ molekule formed during its generation time but ($N_0 = \frac{\sigma + \mu}{\sigma(E) + \sigma(A)}$) We then obtain for the average number of molekules of the specific enzyme that the promgene will produce

Exp^o determine lag for Galactose at saturating concentrations.

Rate of production. — Initial slope should be proportional to — not to conc. of M*



~~What is Δ in Model 2?~~

Assuming. —

Each ~~unique~~ ~~sharp~~ in free state from repressor ~~state~~ ~~binds to~~ ~~Repressor~~ ~~molecule~~ Δ

τ(E) Second, during this time is is caught by Repressor

$AR\,\tau(E)\,p$ (Hirando) times (when p molecule comes not connected with R) each time it stays connected τ(R) sec long, therefore

(2) $\Delta = AR\,\tau(E)\,p \times \tau(R)$ sec

(3) $\Delta = AK_R \frac{R}{K_R}\,\tau(E)\,p \cdot \tau(R) = \frac{R}{K_r}\,\tau(E)\,p$

(10) $N = \frac{\tau_{gen}}{\alpha(M)\frac{R}{K}\tau(E) + \tau(E) + \tau(AA)}$

Turn to page 14

Here N_0 designates the number of enzyme molecules which an ~~organism~~ a paragene ~~is~~ in the culture of the bacterium ~~which~~ N_0 assigned for the sake of argument that N_0 has the same value for every paragene

It may be seen from this equation that if $N < \frac{N_0}{2.3}$ the number of enzyme molecules for each cell maintained in the steady state of a growing culture is independent of the growth rate. If r is increased beyond $\frac{N_0}{2.3}$ the number of enzyme molecules per

If for a particular organism r is increased beyond $\frac{N_0}{2.3}$ the N is moving asymptotically towards N_0. — If we now further assume that the point in at which the nucleus divides is determined by the concentra-

By applying the Poisson formula

$$N = \sum_{m=0}^{N_0-1} m e^{-hm}(1-e^{-h}) + N_0 e^{-hN}$$

where N_0 designates the number of enzyme molecules produced when by chance none of the enzyme molecules formed in the enzyme were caught by a repressor molecule. The ~~value of N_0 is taken by set~~ ($N_0 = \frac{\text{enzyme}}{\tau(E) + \tau(M^*)}$ ≈ 10,000)

$$\bar{N} = e^{-h}\frac{1-e^{-N_0 h}}{1-e^{-h}}$$

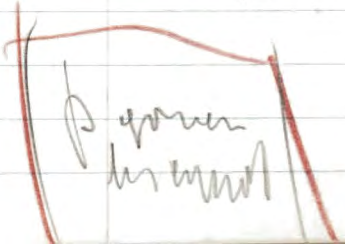
if we write $h = \frac{1}{n}$

~~then we may write~~ ~~with an error of less than 10%~~ ~~is a number~~

for $r > 5$ and up to $r \approx \frac{N_0}{2}$ within a margin of error of 10% $\bar{N} \sim r$ within these limits

Therefore we may write

$$\bar{N} \approx \frac{1}{h} = \frac{1}{k_R[R]\,p(M^*)\,\tau(E)}$$



$$p = \frac{1+\frac{\tau(E)}{\tau(M)}}{1+\left(\frac{M^*}{k_{M^*}}+1\right)\frac{\tau E}{\tau M}}$$

What we assume here is

~~Perhaps we should also say~~

~~that~~ when we speak these

other

~~the~~ name of ~~these~~ "enzymes" are produced by these organisms ~~at the~~ ~~enable them~~ ~~to grow~~ ~~at the just rate~~ throughout and thus ~~to~~ ~~maintain~~ ~~at the~~ ~~just~~ rate

~~and~~ ~~just~~ catalysts of any

these enzymes be

~~such~~ chemical reaction

but ~~structure~~ of structural

problems ~~clearly~~ the term enzyme

as it is ~~as~~ used here ~~in the broad~~ including

such ~~also~~ ~~as~~ "inactive" proteins.

However it might devise

If the division of the

nucleus is controlled in either

~~this~~ of these two ways than No is

independant of the generation

time and so is N as

given by equation ( ) .—

Thus we have ~~here~~ a

~~most~~ developed here

a mechanism that

conforms to the principle

of growth rate independence

for an of one of the enzymes E_d for which $K_{Ed} < K_{No}$ ~~for which~~ ~~is purely~~ ~~enzyme so that~~ ~~$N_d < K_{No}$~~ then ~~we may say that~~ the quantity ~~production~~ of this ~~enzyme~~ ~~drops rather~~ reaches its full value rather soon after the formation of the nucleus and therefore its concentration will increase as some of the other "enzymes" increase in quantity until a certain critical min. value $[E_d]_{min}$ is reached at which point the ~~egg~~ nucleus — we ~~will divide~~ shall assume — ~~Perhaps it~~ ~~would be better to say~~ might be that what is "critical" is for instance the number of the nucleus not the absolute cause of the enzyme E_d but the ratio of the cause of this enzyme and some other enzyme or enzymes which keep open to the ~~one produced by this process~~ this is a point we may come at the full (unopposed) rate.

Planar case

R biased M grows up

N grows up

Impedance case

M^a prop to R

$[M] = C_0[R]$ $\quad [R] = C_0 M$

$$\frac{1}{h} = r = \frac{1}{A_R \sigma(E)} \left[\frac{1}{[R]} + \frac{1}{C_0 \left(\frac{1}{\sigma(M)} + \frac{1}{\sigma(E)} \right)} \right]$$

$\frac{1}{C_0 M}$

Non proper

~~Can we understand~~ Do we obtain "pantbrake independence" if undercut TMG dukes and we are say at 1/10th of cycling? Yes, we make thom β-Galactosidase at 1/10 rate of a fully induced enzyme!

For Diane

$$C^{+\alpha \tau_d} = 2$$

$$\alpha \tau_d = \ln 2$$

$$\tau_d = \ln 2 \times \tau_{gen}$$

$$\tau_d = \frac{\tau_{gen}}{1.44}$$

Doubling number

τ_g

$$N_g = \frac{N_d}{\ln 2}$$

In how many ways does an H
increase act? — TM, M.

Lag. and TMG
$k \neq K$
the precursor as inducer —
the metabolite M as repressor. —

the precursor as inducer

from ( ) and ( ) we obtain

$$\bar{N} \sim \frac{1}{A_R \tau(E)} \frac{1 + \left(\frac{M^*}{K^*} + 1\right) \frac{\tau(E)}{\tau(M)}}{[R]\left(1 + \frac{\tau(E)}{\tau(M)}\right)}$$

$$\frac{1}{A_R \tau_E} \frac{1 + \frac{\tau(M)}{\tau(E)} + \frac{M^*}{K^*}}{[R]\left(1 + \frac{\tau(M)}{\tau(E)}\right)}$$

$$\bar{N} = \frac{1}{A_R \tau(E)} \frac{1}{[R]} \left(1 + \frac{M^*/K^*}{1 + \tau(M)/\tau(E)}\right) = \frac{1}{\rho}$$

for K^* we must take $A_M K^* = \frac{1}{\tau(M)}$ — when enzyme is saturated with M

$$K^* = \frac{1}{A_M \tau(M)}$$

$$\frac{1}{\rho} = \left(1 + \frac{A_M [M^*] \tau(M)}{1 + \tau(M)/\tau(E)}\right) \to 0$$

when enzyme is not saturated with M

$$\text{or} \to \frac{1}{\rho} = \left(1 + \frac{A_M [M^*]}{\frac{1}{\tau(M)} + \frac{1}{\tau(E)}}\right)$$

when enzyme is saturated with M

$$\tau = \frac{1}{A_R \tau(E)[R]} \frac{1}{\rho}$$

when enzyme is not saturated with M

Final model

$$N = \frac{g}{(\text{Ar}R\,\tau(E)\,\tau_2(R_{\text{Ar}})) + \tau(E) + \tau(R_A)}$$

Better go to M model and ~~assume~~ that for β-galactosidase when not at ~~ceiling~~ R prop. to τ; What is changed by the time $\tau(R_A)$ means short within gen time R is up, even so there ought to be an initial increase in enzyme level; may be there is.

Try some M models

All R propto τ gen controlling enzymes, one of them ~~does~~ has τ

4

10^{-7} cm

5×10^{-7} cm = 1

10^{10} 10^{-11}

Rhubarb and Grunthrake
in dependence.

Rhubarb in THIS case is key
with Rhubarb if β(THIS) falls for
help but by then R is up and [E] is very
low. What about Grunthrake
independence

If we halve the grunthrake:
the same number of Rhubarb
are produced in twice the time.
How many molecules of β grunthrakase
are produced by org at 1/3 saturation
of just grunthrake and 1/2 maximum
grunthrake | 1/3 Sat means fill up
1 time each time for 1 min in Lotor
3 threshing for at 70 min grunthrake
half up for 120 min at
3 caught in Dino
for 40 min
80 min
caught

240 min
caught
actually stunned
the 20%
160 min grunthrake

Paper (Antibodies)

As long as the membrane protecting the pathogen remains ~~impermeable~~ impermeable to the antigen ~~it~~ and through the process level falls no antibody will appear because the antigen continues with the "unique" molecules ~~which~~ on the pathogen and blocks further ~~synthesis~~ synthesis by the pathogen. Thus it would become understandable that ~~autoimmunity~~ in the mechanism (Rabbit ~~the~~ the possibility of producing tolerance almost ~~disappears~~ at the same time when ability to form proteins antibodies appears.

~~When~~ Since the suppressor cells could appreciably only if the cells of the antigen carrying

If all R-s prop. to τ_{gen} H...

and controlling enzyme has $\frac{1}{t_R} = \frac{1}{10}$

and if second controlling enzyme unrepressed and inducted that it is irrelevant that all R proportional to τ_{gen}. If second controlling enzyme both repressed and slated than when we change τ_g what happens? Perhaps it is not unreasonable to assume that ~~—~~ strongly repressed enzymes ($V(R) \ll$) have long $\tau(R)$,

~~β-galactosidase~~ ~~τ_{gen} ≠~~ $N_2 = \frac{\tau_{gen}}{A_1 R_1} L_1(R) + \tau(E) + \tau(AA)$

$N_2 = \frac{\tau_{gen}}{\frac{\tau(E)}{A_2 R_2} + \tau(E) + \tau(AA)}$

$N_3 = \frac{N_2 \left[\frac{\tau(E)}{A_1 R_1} + \tau(E) + \tau(AA) \right]}{\frac{\tau(E)}{A_1 R_1} \tau_1(R) + \tau(E) + \tau(AA)}$

$N = \frac{\tau_g - N A R \tau(E) \tau(R)}{\tau(E) + \tau(AA)}$ } etc.

~~$N(\tau(E) + \tau(AA)) = \tau_{gen}$~~

× if one multiplies τ_g with 2 and R with 2 and $\tau(E) + \tau(AA)$ with 2 N remains unchanged but $\tau(R)$ should it become longer? In this case $\frac{1}{2} N_0$, — O.K. in steady state

TMG problem:

Central (abundant) enzyme } $\tau(R) < \tau_{gen}$
R for non-abundant central enzyme indep. of fluctuations and

$N_c \sim 2000$
(R proportional)

Assume ρ-polycondensate

$$\Delta = \frac{A R \tau(E) \tau(R)}{N}$$

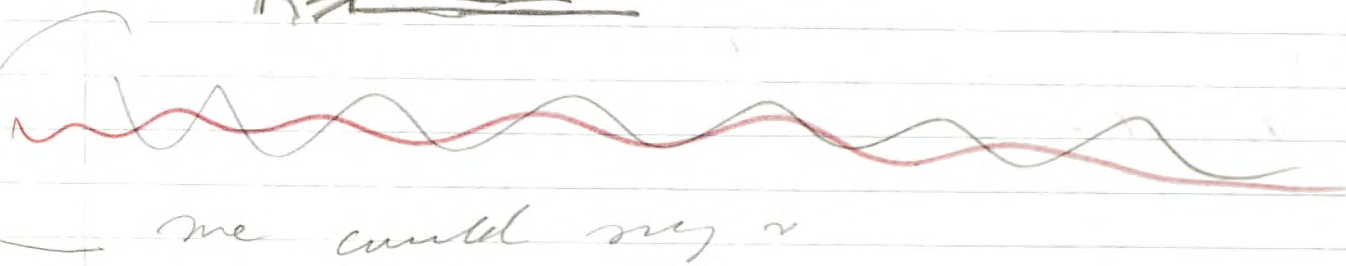
Let us assume that TMG inhibition apparent ceiling comes of saturation of elongation process but their remains $\tau(R)/N$)

This might be ~ 10 times higher than M_0

the chemical analog M^* becomes high enough to take up an appreciable fraction of the enzyme E.

free enzyme = $\frac{1}{1 + \frac{M}{K} + \frac{M^*}{K^*}}$ ~~time~~

~~might be, thresholded~~ the repressor concentration as a function of ~~the~~ $[M^*]$ is ~~might~~ to be an S shaped curve R



we could say ~

$[R] \sim$ prop to $\frac{\frac{M}{K}}{1 + \frac{M}{K} + \frac{M^*}{K^*}}$

and threshold where M^* begins to turn appreciably level if R is $\frac{M^*}{K^*} = 1 + \frac{M}{K}$

M increases with M^*

$$A\frac{1}{R} = \frac{1}{A}\,\frac{1 + \frac{M}{K} + \frac{M^*}{K^*}}{\frac{M}{K}} = \frac{1}{1000} \text{ for } M = 0$$

when we raise $\frac{M^*}{K^*}$ we also raise M to $\frac{M_{sat}}{K+1}$ when we set $\frac{M^*}{K^*} = \frac{M_{sat}}{K+1}$ so only

low effect is the same as if a number of Jews were cut off!!!

Enzymes: paper

~~How~~ We assume that non abundant enzymes are repressed by $\bar{c}(R) > \bar{c}_{gen}$ and 80% of enzymes not repressed at all. IMG induced β-galactosidase (cytosolic) maintained say at $\bar{c} = 3$ hrs at 1/3 level of ecoli. If we shift to 6 hrs steady state would go up relative to total proteins except if repressor conc. prop to $\bar{c}$. In that case in the transition to $\bar{c} = 6$ hrs the enzyme would first rise and then fall back to ~~zero~~ intrinsic value. This is so, because absence

Antibody formation

Exp. Does Dr Rose through whole Rabbit? If after a few weeks after the first injection a small dose of antigen is given in the footpad of the Rabbit and also compares the antibody production at the ~~nearest~~ ~~the~~ lymph glands ~~with~~ which drain this footpad with the production of other lymph glands do they all produce equivalently well? Cannot be!

Aging!?

e^{-n} may give pumpers ~~amount~~ ~~on~~ ! / n goes like a number e^{-kt}

X-ray effect: of thousands of chromosomes are destroyed ~~in that Brain~~ ~~In~~ in that cell the rate of aging is increased. The age is not increased if it because of pumpers

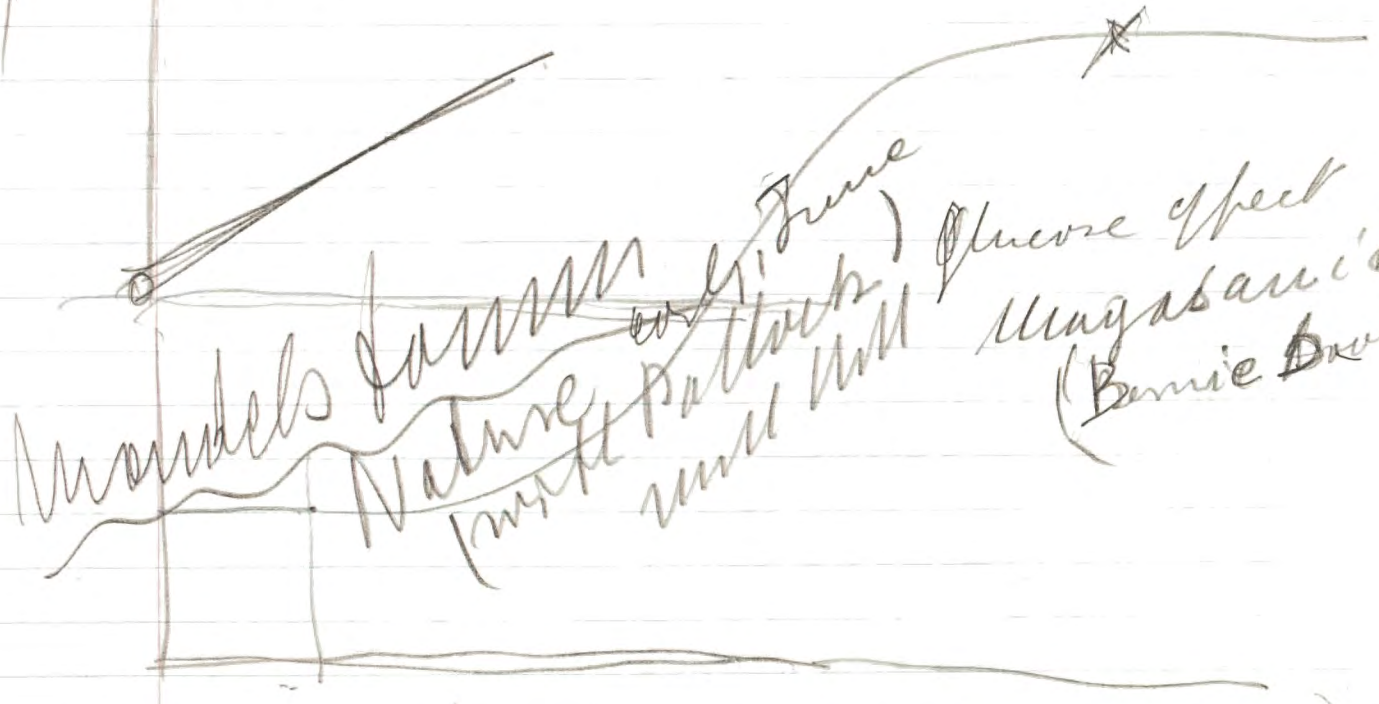
Nowak

Duke Kosinski
(Skeet)

Nowak

explanation
fast growing hardwood
loose broken discussion

Energy



Nowak

Bumerang Fries (Jim Watson)

(MIT Wall)

Leonard Hamilton Sloane Kettering (Francis Watson)

(Ted Mendelsohn
Point production

of key forces as to assume T(R)LL open
In general when T(R)LL open
for a given enzyme and
~~dogs~~ R proportional to T open
we get this kind of behaviour
of the enzyme when it is
more repressed than the
"total proteins" and when it
is less repressed the opposite
happens i.e. ~~the enzyme~~
upon shifting to lower
concentrations time the
enzyme first must fall
and then rise back to
the initial level (R prop.
to T open)

~~Ans to page 11~~ New
Preface: next book (II term 57)

10,000

60 10,000 10^{-13} gm

number of antibody forming cells.