The total amount of DNA in the haploid set of chromosomes of man corresponds to 100,000 genes if we assume that, in general, genes produce proteins of the molecular weight of 100,000. Thus, there is a DNA in a mammalian cells for 90,000 genes which were left out of consideration; it is conceivable that these might be genes which are not important or perhaps incompetent and that, while they reproduce themselves, they don't produce anything else. One might perhaps argue that the important genes of which we have assumed 10,000 are 10 times as large as estimated but this is somewhat difficult to reconcile with the fact that the total amount of DNA in the haploid set in bacteria is 100 times less, and that the size of the gene which we estimated would then just give about 1,000 genes for bacteria, a number which appears just about to be right. Let us then for the sake of argument, accept the notion that 90,000 out of 100,000 genes don't have any relevance for the considerations with which we are here concerned. We might then say that exposure to x-rays causes with a certain probability a lesion in a gene and that if the lesion happens to be an important gene, it causes a fault or a handicap, and On this basis, we would then have to say that an exposure to an x-ray dose of 365 rep must cause lesions in the diploid set of genes of a human cell or which means that rep causes on the average one lesion.

On the basis of observations made on proliferating tissue cultures T. T. Puck has reported that it takes an exposure to x-ray dose of at least 30 rep to produce one chromosome break. Accordingly, it would seem that x-rays produce at least four times as many lesions -- which in the case of important genes, renders a gene incompetent -- than as the produced chromosome breaks. —

Our total load of mutations can be now computed if we adopt the kind of reasoning presented by H. J. Muller in an article "Our Load of Mutations". Assuming that in the past mutant forms of the

Demerec, M.
Frequency of cell lethals
P. N. A. S. 22: 350/54
1936

Bruce Wallace
May 1957
Princ. Wat. Acad.

33 25 26

33
25
26
84

n = 1.7

C. H. Waddington
The Strategy of the Genes
George Allen & Unwin
1957

Frederick Monnot

Michael Lerner
Berkeley.

James E. Birren

X-RAY CURB URGED BY GYNECOLOGIST

Check of Patients' Radiation History Asked in Report to College of Surgeons

BY MORRIS KAPLAN

Special to The New York Times.

CHICAGO, Oct. 10 — Specialists in gynecology and genetics recommended today that physicians check carefully into the radiation history of patients and use minimal dosages of X-ray in all cases, especially with expectant mothers. They voted that ten roentgens is the lifetime limit for a person.

A panel on the current knowledge of congenital defects, deplored particularly what was described as a wide-scale use of X-ray as a diagnostic and therapeutic aid at some university medical centers. While the specialists did not specifically link radioactivity with birth defects, they urged more discriminate use of X-ray equipment.

Dr. Robert E. L. Nesbitt Jr., of Albany, reported that more than 1 per cent of 4,000,000 live births annually in this country had congenital defects. Defects from whatever the cause ranged as much as one in sixty-five births, he said. The death rate from such defects was between 10 and 15 per cent in the first twenty-eight days of life.

He estimated that the total number of infants with congenital defects that accrued annually reached 250,000. He noted also that there were about 400,000 spontaneous abortions. It was believed that at present 50 per cent of these were related in some way to defective germ plasm.

Dr. Nesbitt, chairman of obstetrics at the Albany Medical College of Union University, moderated a closing session of the forty-fourth annual clinical congress of the American College of Surgeons in the Congress Hotel.

On the panel with him were Drs. H. Ingalls, professor of preventive medicine and epidemiology, University of Pennsylvania School of Medicine; Samuel B. Kirkwood, clinical professor of maternal health, Harvard University; Peter Gruenwald, director of laboratories, Margaret Hague Maternity Hospital, Jersey City, and William J. Schull, associate professor of genetics, University of Michigan Medical School.

Dr. Nesbitt observed that high energy radiation produced mutation and that genes thus became permanently altered. He criticized X-ray examination in pregnancy, saying they were doubly effective, influencing both the expectant mother and her child. Dr. Nesbitt said that such examinations should be stopped.

Words of Caution Offered

Dr. Schull suggested that no definite rule was possible, however; that it was each physician's responsibility to question whether X-ray procedure was "critically" needed.

He cautioned against stirring up increased fear among the public, noting in this connection that there were ten times as much so-called harmless radioactivity in water drawn from deep artesian wells in regions of Illinois, Wisconsin and Iowa than in other areas.

The college inducted tonight at the Conrad Hilton Hotel 1,100 surgeons as new fellows in a ceremony ending the five-day program. The organization, founded in 1913, has a total membership of more than 22,000.

The congress cited as honorary fellows Dr. William Arthur Mackey of Bearsden, Dumbartonshire, Scotland, and Hiroshi Miyake of Fukuoka, Japan.

Dr. Newell W. Philpott of Montreal was inducted as president of the college.

W. L. Russel, L B Russel Dec 19
& M Kelly / Science Vol 120 / 38

3 mpm mall

(γ)	861 rep	6.12×10^5	per lac/
X-ray	600 rep	13.29×10^5	"
	1400 rep	10.33×10^5	"

Ulrich Luft.
Dept. of Physiology
Lovelace Foundation
Albuquerque
Shows a relationship between a work function and ageing.

Coli	5×10^8 r/min	Demerec & Fetzer
Drosophila	3.6×10^7 rep/min	Alexander (M.)
Mouse	1.4×10^6	Russel (L.B.) & Major

Pneumo DNA mach, initial	5×10^5	various in Fuils including L & T
" " " final	3×10^6	Gold

W. J. Dunning
Cancer Research Lab
Univ of Miami
Coral Gables 46, Fla.

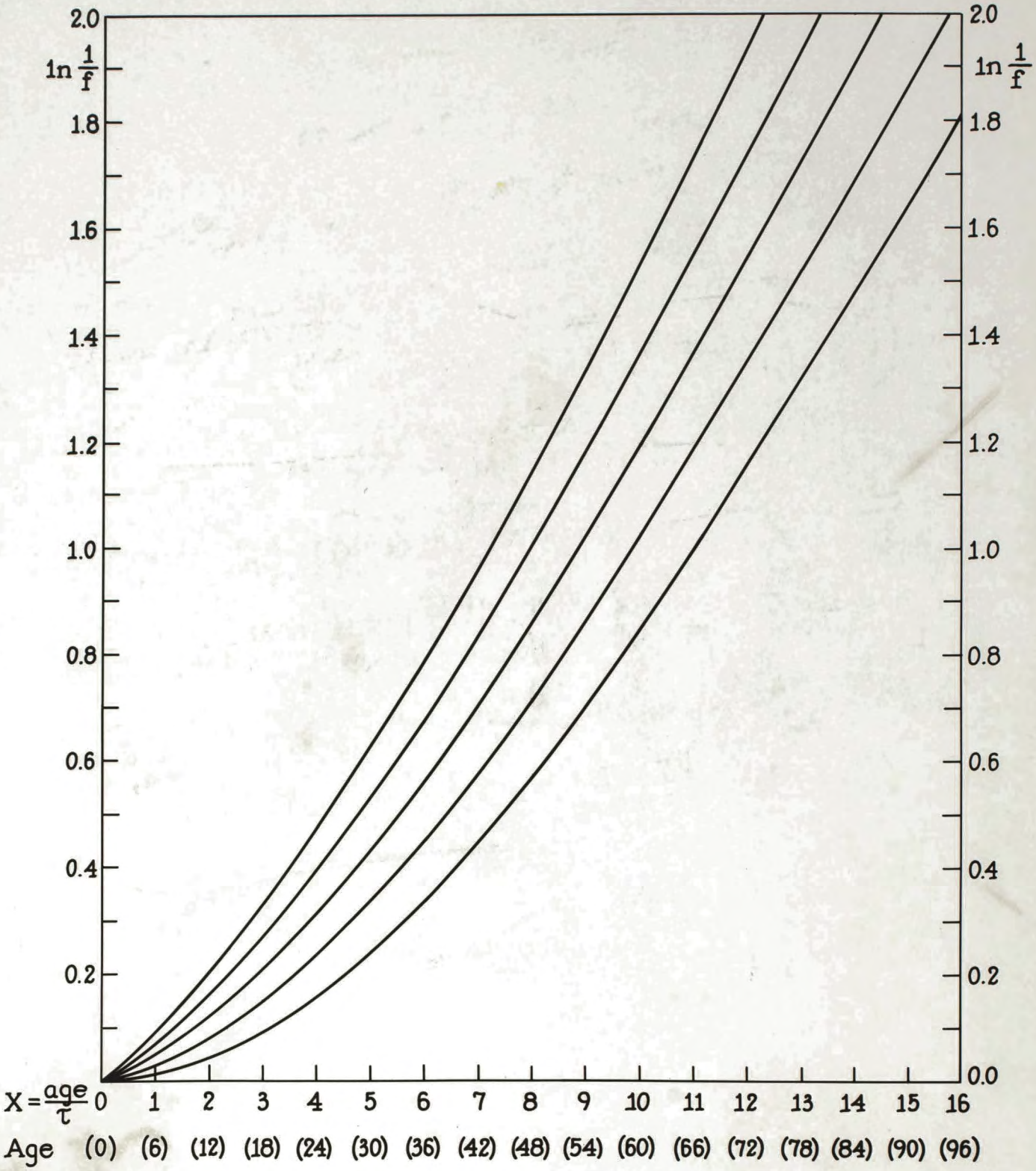
Rats

Douglas Grahn
Div. of Biology + Medicine
U.S. A.E.C.
Washington 25, D.C.

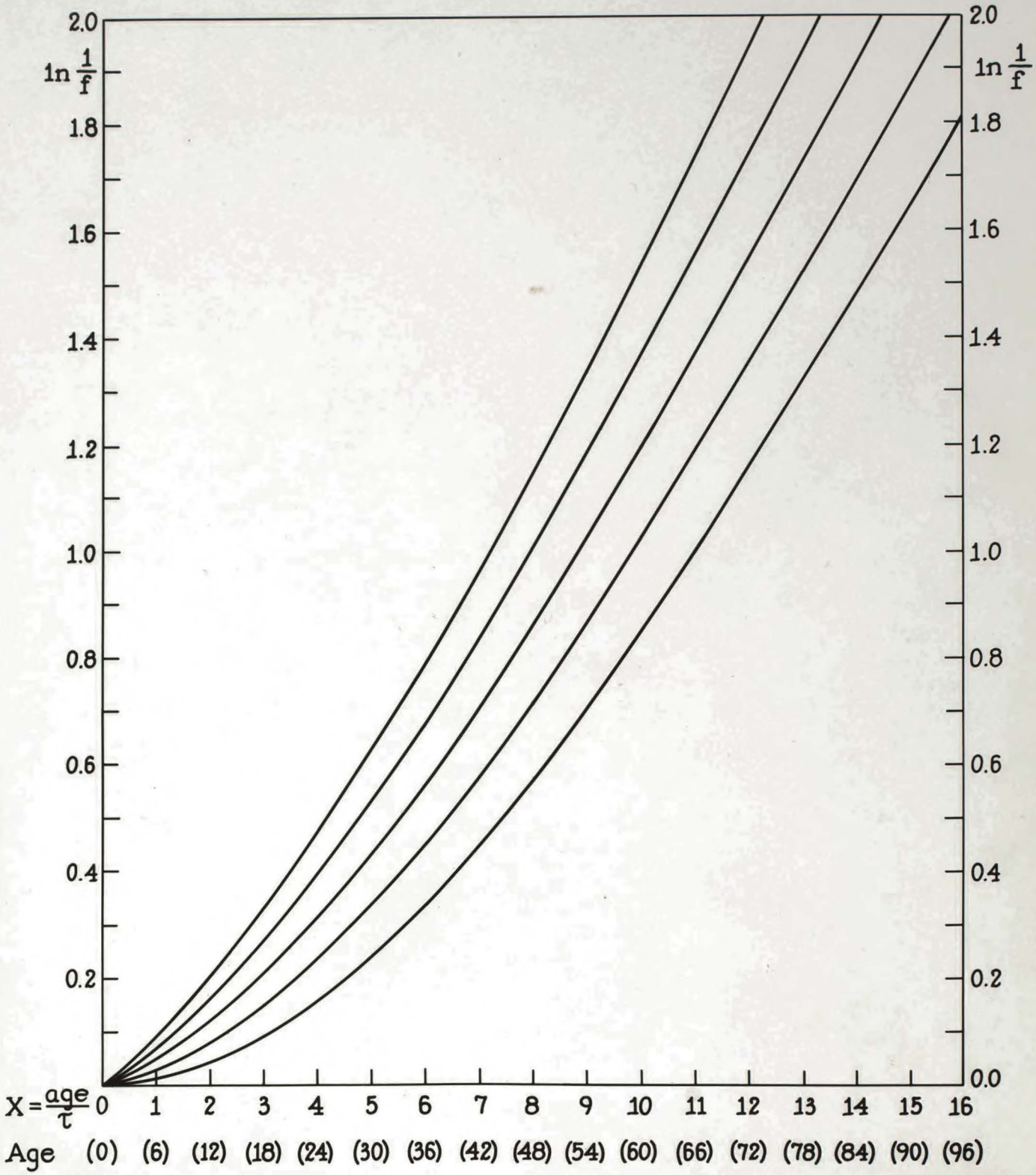
33/1

50 mp / 180 day / 50

3.5 days


2.0
ln 1/f
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
X = age/τ
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
Age (0) (6) (12) (18) (24) (30) (36) (42) (48) (54) (60) (66) (72) (78) (84) (90) (96)

PHOTOGRAPHY
by
Illustration Service
Rockefeller Institute
Neg. # 8834-7
Date 12/11/58
Credit


2.0
ln 1/f
1.8
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X = age/τ
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
Age (0) (6) (12) (18) (24) (30) (36) (42) (48) (54) (60) (66) (72) (78) (84) (90) (96)

PHOTOGRAPHY
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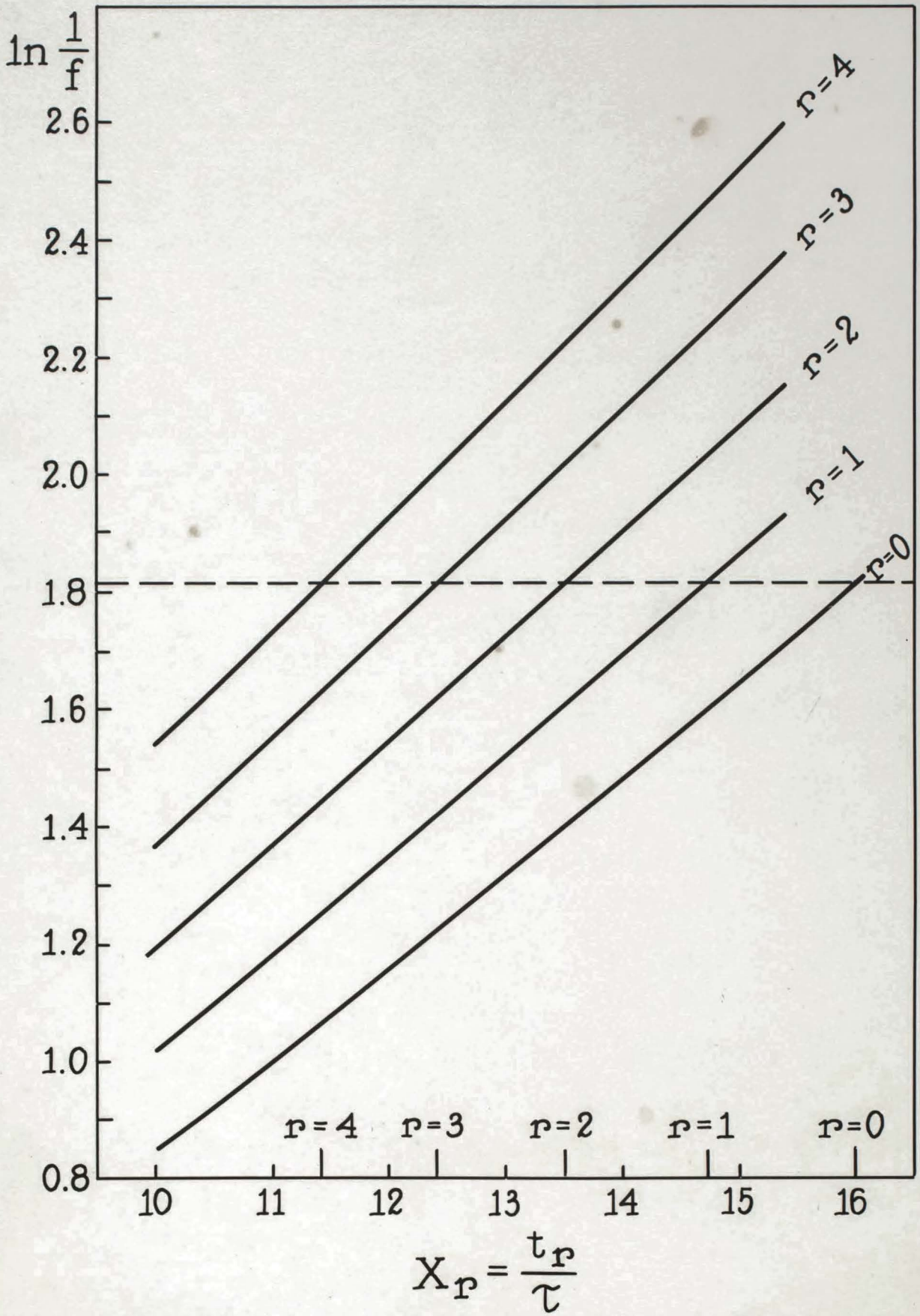
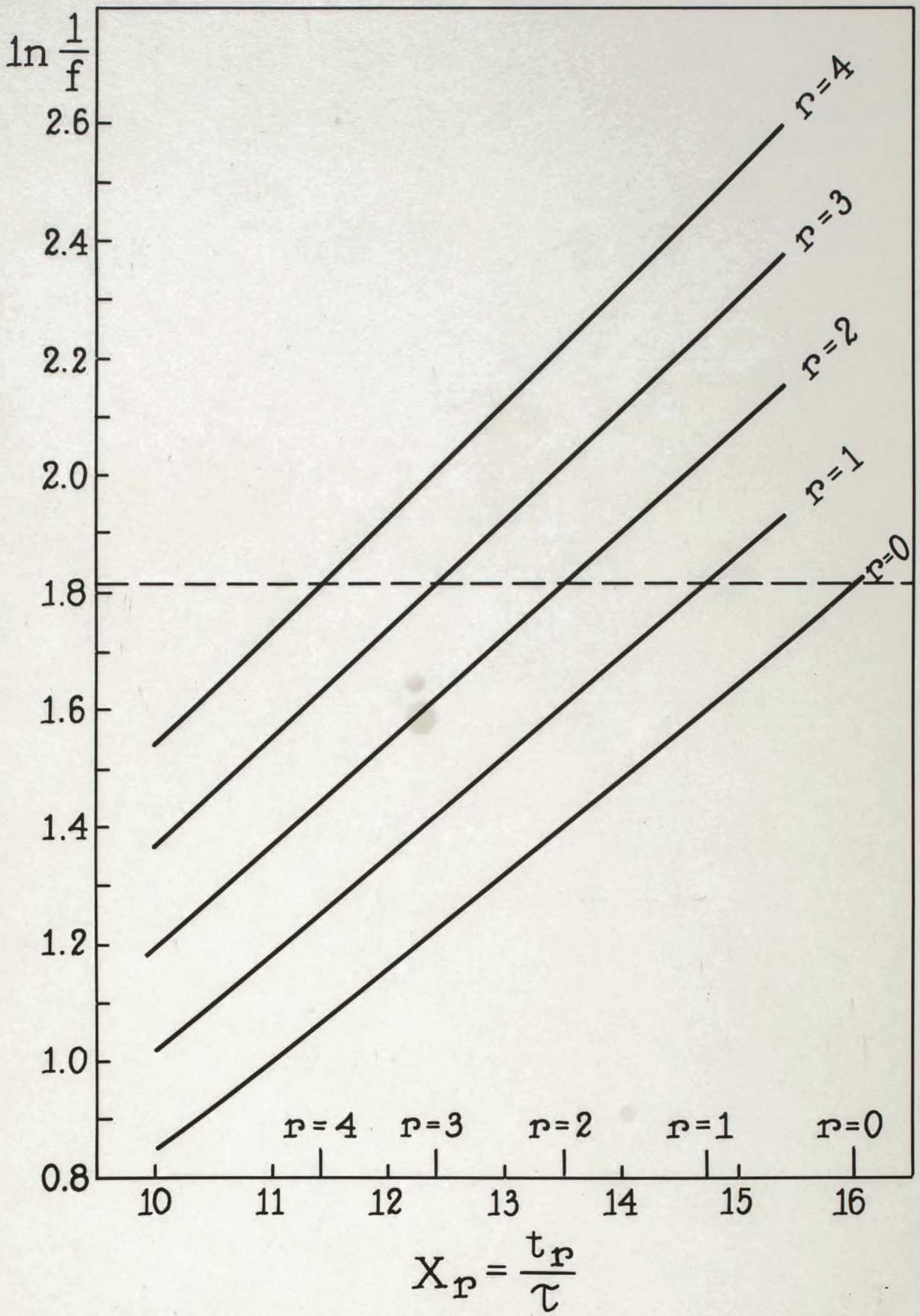
Date 12/11/58
Credit


ln 1/f
2.6
2.4
2.2
2.0
1.8
1.6
1.4
1.2
1.0
0.8
r=4
r=3
r=2
r=1
r=0
r=4
r=3
r=2
r=1
r=0
10
11
12
13
14
15
16
$X_r = \frac{t_r}{\tau}$

PHOTOGRAPHY
by
Illustration Service
Rockefeller Institute
Neg. # 8814-7
Date 12/1/58
Credit


ln 1/f
2.6
2.4
2.2
2.0
1.8
1.6
1.4
1.2
1.0
0.8
r=4
r=3
r=2
r=1
r=0
r=4
r=3
r=2
r=1
r=0
10
11
12
13
14
15
16
$X_r = \frac{t_r}{\tau}$

(20) $N = \dfrac{\tau \gamma_{es}}{4 + \tau(E) + \tau(HA)}$

(21) $A = \cancel{A_R K_R} \; A R \dfrac{\tau(E)}{\cancel{\tau(A)}} \; \tau(R) \, p(M^*)$ or

$A = \dfrac{R}{K_R} \tau(E) \, p(M^*)$

22 $\dfrac{1}{p} = 1 + \dfrac{[M^*]/K^*}{1 + \tau(M)/\tau(E)}$ shows what happens to $\frac{1}{p}$ as K^*, if $\tau(M) \to 0$

or

23 $\dfrac{1}{p} = 1 + \dfrac{A_M [M^*]}{\frac{1}{\tau(M)} + \frac{1}{\tau(E)}}$ shows what happens to $\frac{1}{p}$ if $\tau(M) \to \infty$

W. F. Ogburn

Pearle

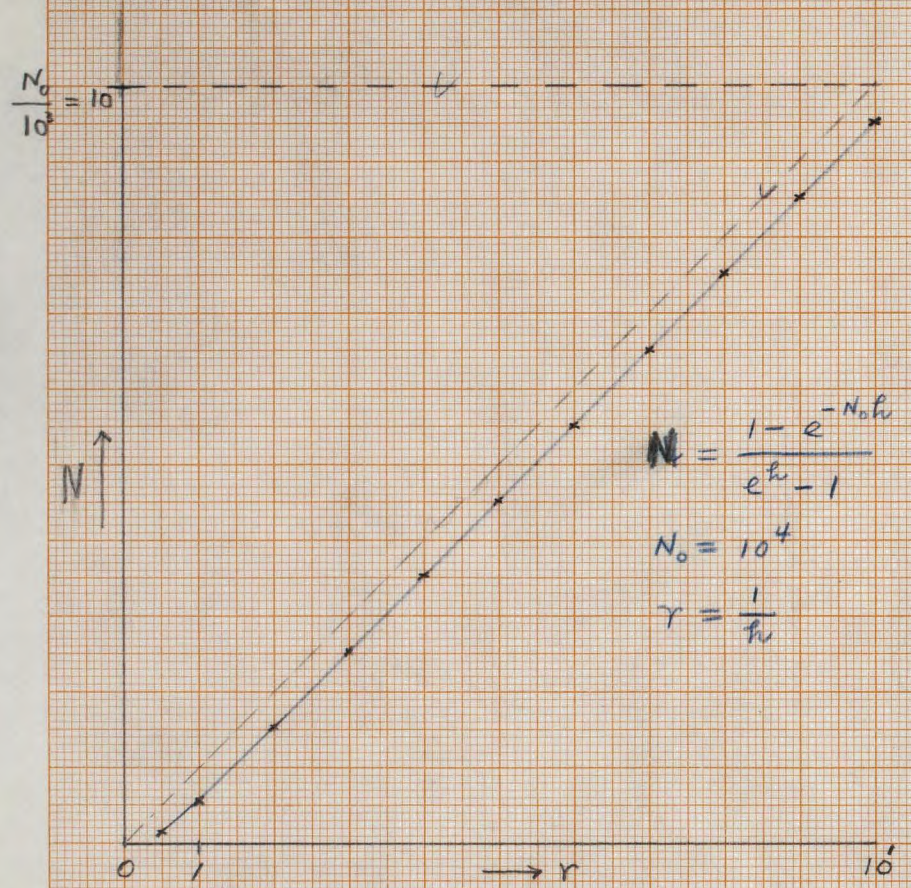
1661 Crescent Place N.W.,
Washington, (9) D.C.

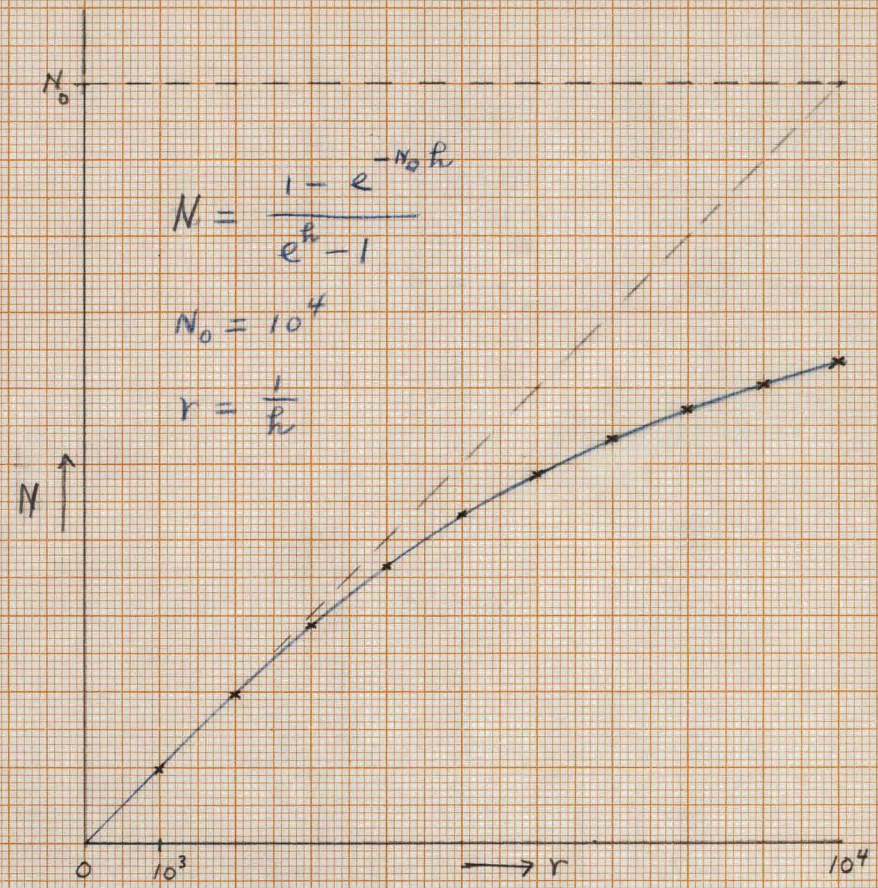
Telephone Unde Sixteen sixtie

Seymour S. Cohen & Hazel Bonner
Journ of Biochem. 69 p 59 1957 (Enzyme adenylate)

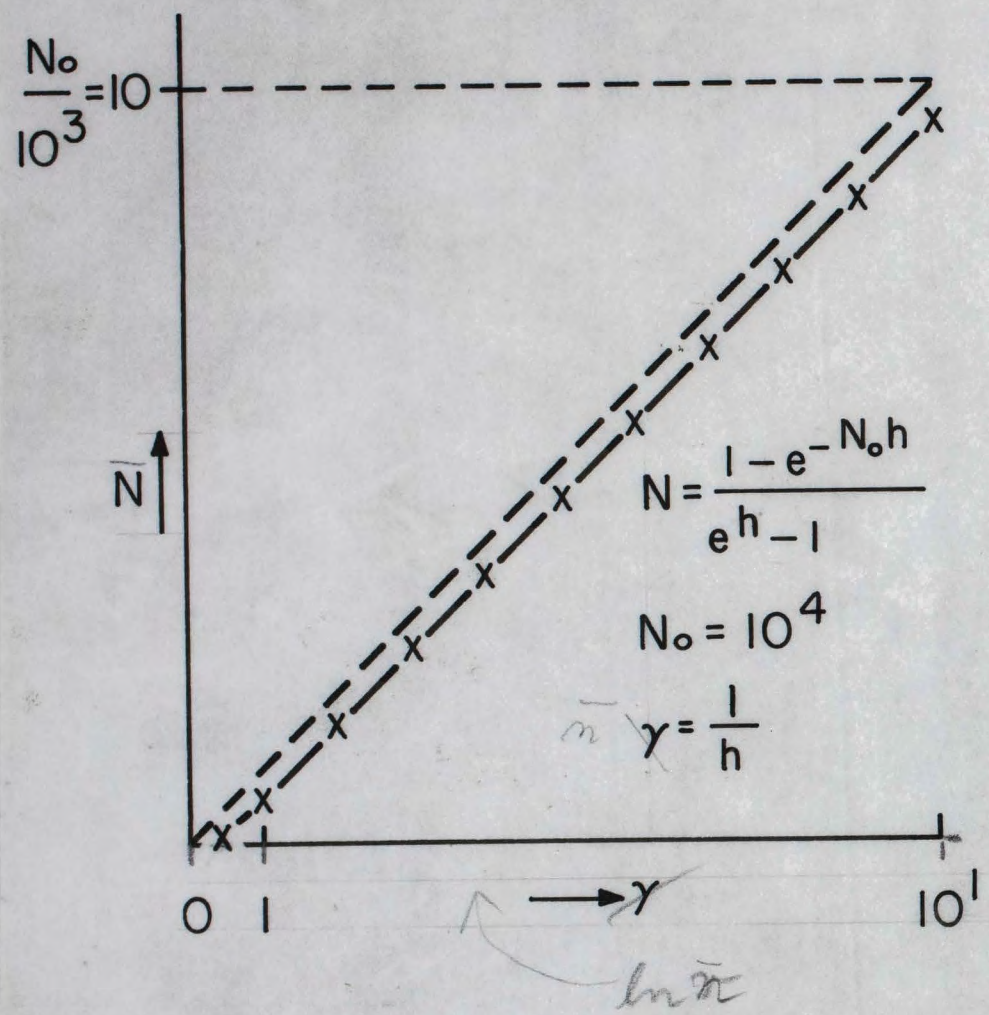
Dysadenylation: Bonner and Cohen
Journal of Biochem Vol 72 p. 1956

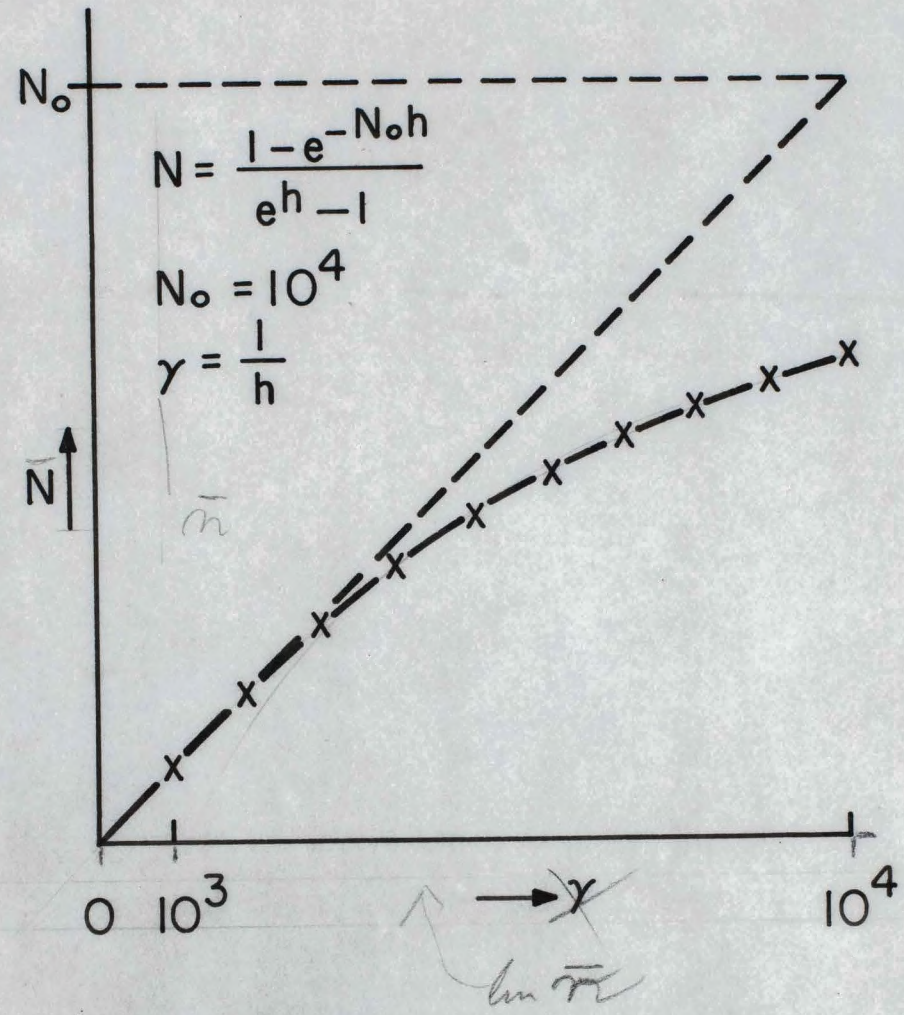
Thymidine:
Bonne V. G.; Lark K. G. and Maclae O.
Nature 176 563-64
Lark K. G. & Maclae O.
Biochem & Biophys Acta
15 345-346 1954



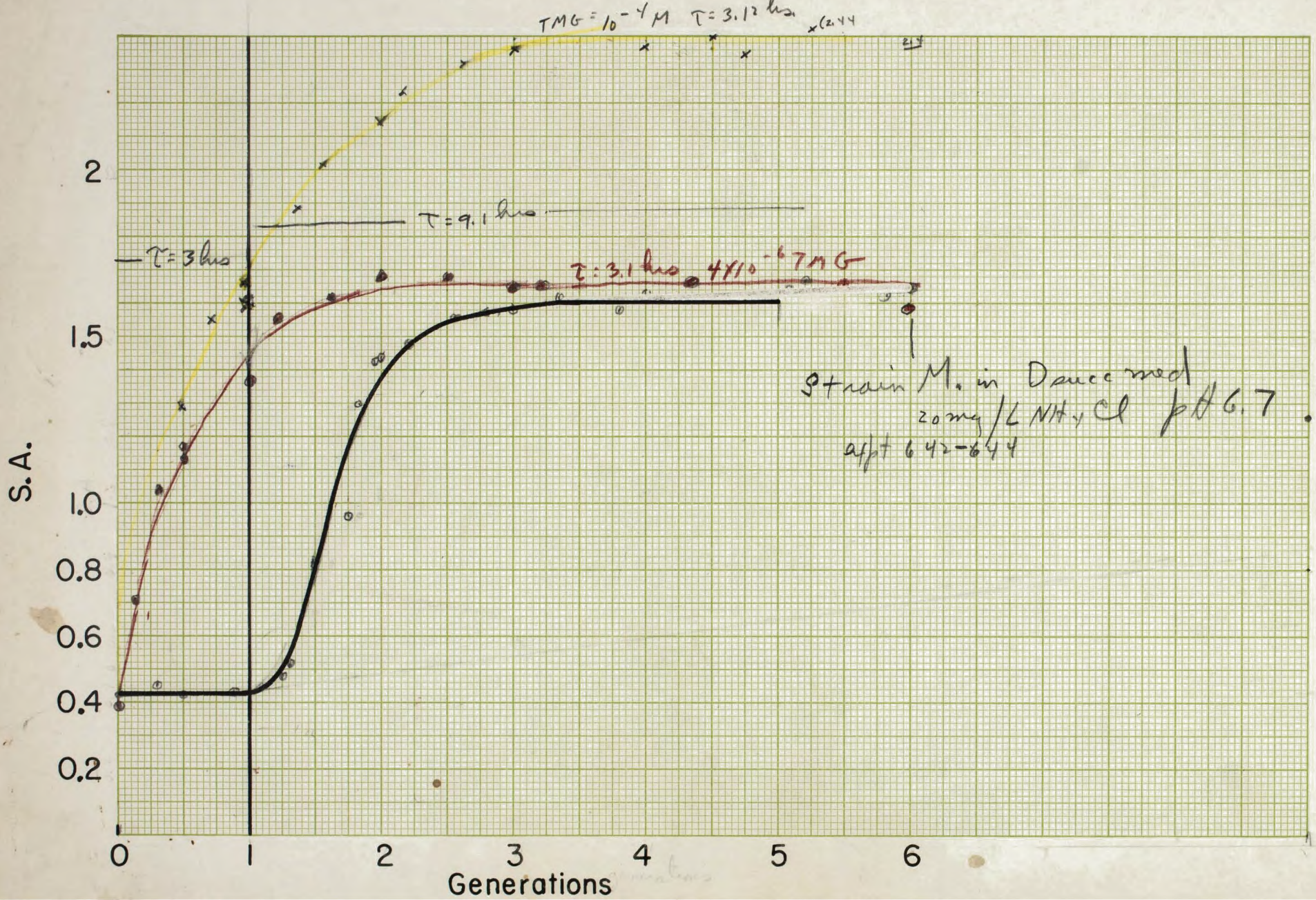


If $5 < r < \frac{N_0}{2.3}$ then $0.9 \leq \frac{N}{r} \leq 1$

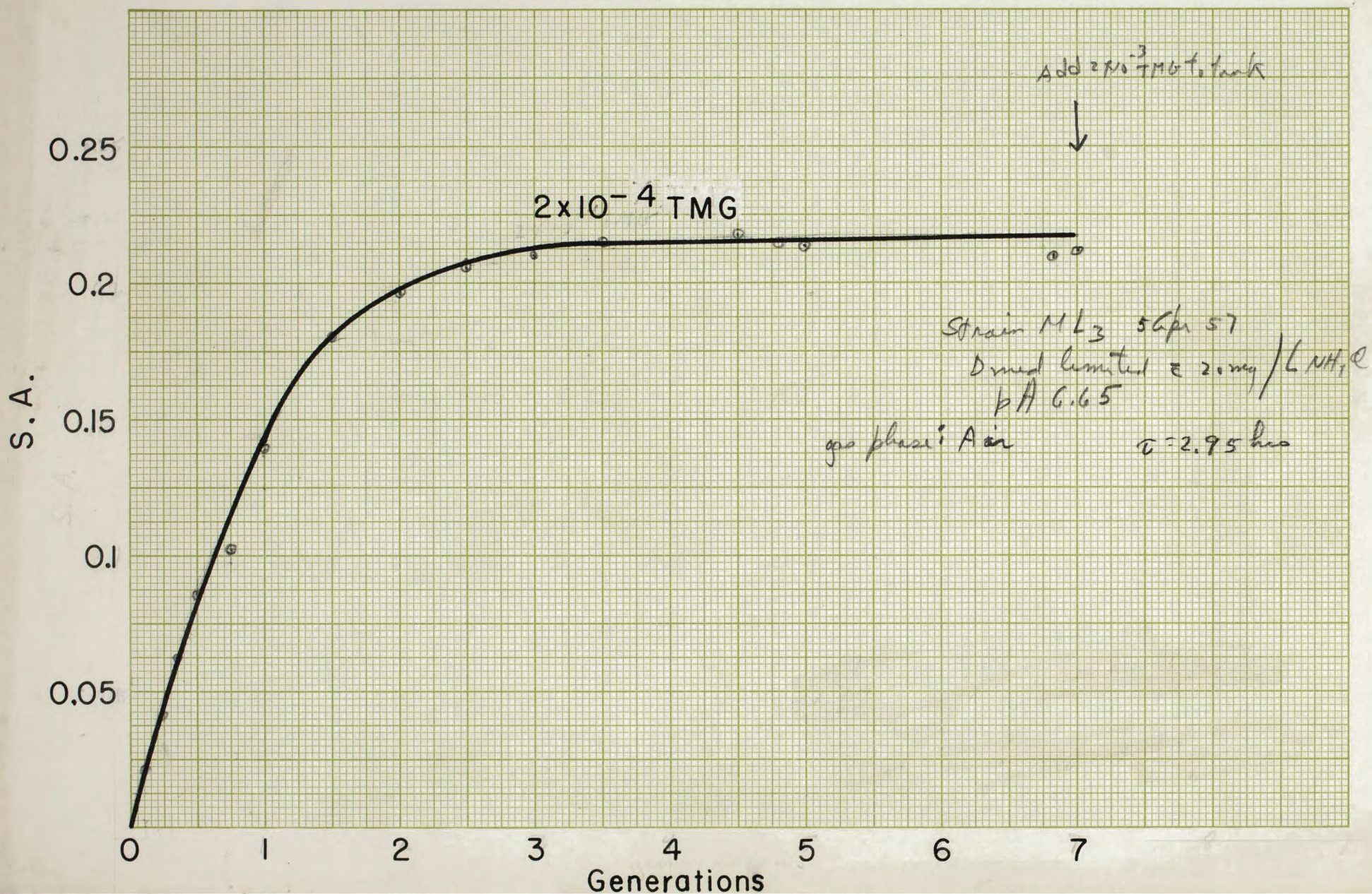

$\frac{N_o}{10^3} = 10$
$\bar{N}$
$N = \frac{1 - e^{-N_o h}}{e^h - 1}$
$N_o = 10^4$
$\gamma = \frac{1}{h}$
0
1
10^1
γ
$\ln \bar{n}$


N_o
$N = \frac{1 - e^{-N_o h}}{e^h - 1}$
$N_o = 10^4$
$\gamma = \frac{1}{h}$
$\bar{N}$
0
10^3
10^4
γ
$\ln \bar{n}$

A1359-1

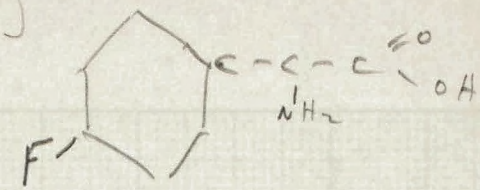


(.)1.25



~~XIII~~ β_2 thienylalanine

fluorophenylalanine



For with
Weak const.
Cryptic
is F.P.G. inhibition lower with conc.?

There is one weak const. which is induced by
not cryptic T.P.G.

Urocil limited makes $\frac{1}{3}$ of 2 in weak const
than when it is Arg. limited.—
for $\tau \times 3$ hrs.
For longer τ is % goes up with τ for Arg. limitation
tion / no change for Urocil limi—
tation

JAG (10^{-4} M) 20 gm for

Thiodi[illegible] galactoside

crystals to take over

$D = 3.5$ km

N. Wanitakon

Lactose full prism in THG

Hexan crystals

1.5

9.

MCP

Second version May 7/57

1.) $M + R \xrightarrow{E_0} \{M-R\}$

2.) $\{M-R\} = \text{rep}\,M$

3.)
$$\begin{matrix} E \\ | \\ T \end{matrix} + \begin{matrix} M \\ | \\ R \end{matrix} \rightleftharpoons \begin{matrix} E \sim M \\ | \quad | \\ T \sim R \end{matrix}$$

or $E-T + \text{rep} \rightleftharpoons E-T-\{M-R\}$

4.)

wild type	Mutant
$E \sim M$	$E \sim M$
$\vert \quad \vert$	$\vert \quad \vert$
$T \sim R$	$T \not\sim T$

weak bond

$K_{rep}(\text{wild type}) \ll K_{rep}(\text{mutant})$

5.)
$$\begin{matrix} E \\ | \\ T \end{matrix} + M^* \rightleftharpoons \begin{matrix} E \approx M^* \\ | \\ T \end{matrix} ; \quad \begin{matrix} E \\ | \\ T \end{matrix} + (M-R) \rightleftharpoons \begin{matrix} E \sim M \\ | \quad | \\ T \sim R \end{matrix}$$

or

$E-T + M^* \rightleftharpoons E-T-M^*$; $E-T+(M-R) \rightleftharpoons E-T-(M-R)$

6

$$\begin{matrix} E \approx Gal \\ | \\ T \end{matrix} \qquad \begin{matrix} E \sim Gal \\ | \quad | \\ T \sim UDP \end{matrix}$$

$0 \cdot 10^{-4}$

1) $M(-3) \xrightarrow{E(-2)} M(-2) \xrightarrow{E(-1)} M-1 \xrightarrow{E} M$

2.) $\begin{array}{c} E \\ | \\ T \end{array} \qquad \begin{array}{c} E \sim M \\ | \qquad | \\ T \sim R \end{array}$

~~3)~~

3.) $R+M \xrightarrow{E_{\alpha}} \{R-M\}$

4) $\begin{array}{c} E \\ | \\ T \end{array} \quad \begin{array}{c} E-M \\ | \\ T \end{array} \quad \begin{array}{c} E\ M(m) \\ | \\ T \end{array} \quad \begin{array}{c} E+M^{*} \\ | \\ T \end{array}$ | $\begin{array}{c} E \sim M \\ | \qquad | \\ T \sim R \end{array} \quad \begin{array}{c} E \sim M(m) \\ | \qquad | \\ T \sim R \end{array}$

5.) $\{M-R\} = \text{rep} \quad \{M(\alpha)+R\} = \text{rep}(1)$

6

7

8

9 $\begin{array}{c} E \sim M \\ | \qquad | \\ T \sim R \end{array} \longrightarrow \begin{array}{c} E \approx M \\ | \qquad | \\ T \approx R \end{array}$

KM $\qquad$ K_M ~~(natural)~~

10 take from Yamasty

11 $(H_n - R) \xrightarrow{E_n} (H_{n+1} R)$ $\qquad$ ATO

13 $\frac{dr}{dt}$

Summary

II verstehen

Ins I. Introduction

① $M(-3) \to M(-2) \to M(-1) \xrightarrow{E} M \xrightarrow{E_1} M(1) \xrightarrow{E_2} M(2) \xrightarrow{E_3} M(3) \ldots M(n)$

chemical analog.

Vorpel

M + 1° degradations

① a $M + R \xrightarrow{E_0} \{M-R\}$; $\{M-R\}$ = rep

1 corrder

① b $\begin{matrix} E \\ | \\ T \end{matrix} + \begin{matrix} M \\ | \\ R \end{matrix} \rightleftharpoons \begin{matrix} E \sim M \\ | \quad | \\ T \sim R \end{matrix}$; K_{rep}

$E-T + rep \rightleftharpoons E-T \sim rep$

assumes highly supposed

Basic assumption: everything reported is much around K

$\{M-R\}$ concentrations.

1.c) $\begin{matrix} E \sim M \\ | \quad | \\ T \sim R \end{matrix} \longrightarrow \begin{matrix} E \sim M \\ | \quad | \\ T \not\sim R \end{matrix}$ mutant ; K_{rep} (mut) >> K_{rep}

(wild type)

1.d.) $\{M-M\} \to M(1) \to M(2) \to M(3) \ldots M_{n-1} \; E_n \; M_n$

$rep \xrightarrow{E} rep(1) \to$ $rep(n)$

What is an "real" inductor [Werner Mus]

free | covered

$\begin{matrix} E \\ | \\ T \end{matrix}$ $\begin{matrix} E-M \\ | \\ T \end{matrix}$ $\begin{matrix} E-M^+ \\ | \\ T \end{matrix}$ || $\begin{matrix} E \sim M \\ | \quad | \\ T \sim R \end{matrix}$ $\begin{matrix} E \sim M(n) \\ | \quad | \\ T \sim R \end{matrix}$

$K(M)$ $K(M^+)$ K_{rep} $K_{rep}(n)$

Precursor as inductor ~~[illegible]~~ ~~[illegible]~~ ~~[illegible]~~ ~~[illegible]~~

Galactose

Stander [illegible] Summary II session

Chemical analog can act in two ways

complete with a repressor for the
template enzyme complex
complete with the repressor or a precursor
of the repressor for an enzyme that makes the repressor or its precursor

$E_n^* M^*(n) \rightleftharpoons (E_n \sim M^*)$

$T\text{-}E(n) + M^* \rightleftharpoons T\text{-}E \sim M^*$

$E(n-p) + M^* \rightleftharpoons E(n-p) \sim M^*$

β galactosidase system TMG
inducer of enzyme formation — Inhibitor
of enzyme formation TDG

as contradistinction from a repressor
inhibitor an enzyme that transforms a
repressor or a precursor of ~~the~~ a stronger
repressor into a ~~non~~ weaker repressor or a
~~weaker~~ non repressor. —

β galactosidase system

Mult Winner
observed by permease

UDP Gal $\xrightarrow{E}$ UDP Gal deg

UDP Gal der $\xrightarrow{E_K}$

Yanofsky

Hum did they originate

Stander — Antibody

TPG must introduce ~~an~~ an enzyme that converts Gal-R into something else

TMG ~~could this~~ ~~reduce the production~~ increase the level ~~by inhibi~~ ~~by interrupting~~ of Gal deriv. ~~that~~ by inducing an enzyme that transforms it or its precursor into something else

This it can do in two ways reduce the amount of Gal deriv. by interfering an enzyme that produces by influencing the formation of ~~an~~ an enzyme that converts it into something else

---

Template

$E_{n-2} \; E_{n-1} \; E_n \; E_{n+1} \; E_{n+2} \approx H$

between 99% – 100%

$T - E_{n-2} \approx H - Prot$

$T - E_{n-1} \approx H - Prot$

$T - E_n \approx (H - Prot)$

$T - E_{n+1} \approx (H - Prot)$

$T - E_{n+2} \approx (H - Prot)$

p. 287 Thieman

p. 182 Dann. of. Biochem

Anthranilic + diphosphoglyceric $\xrightarrow{E}$ A

$$A \xrightarrow{E} B \longrightarrow \text{Indolglycerolphosphate}$$

$$\downarrow E$$

Indol + Serin

$$\downarrow \text{Synthetase}$$

Tryptophane

Preparatory stage

$$\begin{cases} \dfrac{dE}{dt} = a T_0 \dfrac{1}{1+\frac{r}{k_1}} - \dfrac{E}{\tau_E} \\ \dfrac{dr}{dt} = bE - \dfrac{r}{\tau_r} \end{cases}$$

$a = T_0 = b = 1$

$\tau_E = 20$ days

$\tau_r = 1$ day

II Attempt

a) $\dfrac{r_0}{k_2} = 10$

Let E & r come to the steady state E_0, r_0, and choose k_1 so that $\frac{r_0}{k_1} = 10^4$

Then $E_0 = 2\times10^{-3}$, $r_0 = 2\times10^{-3}$, $k_1 = 2\times10^{-7}$.

After 1st injection of antigen, templates producing enzyme get affected in the following way.

$$\begin{cases} T_E = 0 & \text{for } 0 \leq t \leq 16. \\ T_E = \left(1 - e^{-\frac{t-16}{\tau_T}}\right)(1-p)(1-q) & \text{for } t \geq 16. \end{cases} \qquad \tau_T = 5 \text{ days}.$$

After 16th day, we set $p = 0.2$, $q = 0.1$. Then we get $T_E = 0.72\left(1 - e^{-\frac{t-16}{5}}\right)$.

~~For the~~

For $0 \leq t \leq 16$, we take

$$\left.\begin{aligned} E &= 0 \\ \frac{dr}{dt} &= bE - \frac{r}{\tau_r}. \end{aligned}\right\}$$

Then $r = 2\times10^{-3} \cdot e^{-t}$.

~~At~~ At $t = 16$ we have $E = 0$, $r = 2.25\times10^{-10}$.

For $t \geq 16$ we take

$$\left.\begin{aligned} \frac{dE}{dt} &= a T_E(t) \frac{1}{1+\frac{r}{k_1}} - \frac{E}{\tau_E} \\ \frac{dr}{dt} &= bE - \frac{r}{\tau_r} \end{aligned}\right\}$$

$\frac{d(r-1)}{dt} = b(r-1)E$

Then we get steady state value : $E_\infty = r_\infty = 1.697\times10^{-3}$.

Now let $\bar{A}$ denote the amount of antibody synthesized during the first 16 days.

Let A denote the amount of fresh antibody which were synthesized after 16th day and are still remaining.

We take : For $0 \leq t \leq 16$, $\dfrac{d\bar{A}}{dt} = \dfrac{a T_0 p(t)(1-q(t))}{1+\frac{r}{k_2}}$ $\qquad \bar{A}_0 = 0$

$k_2 = 2\times10^{-4}$ i.e. $\frac{r_0}{k_2} = 10$

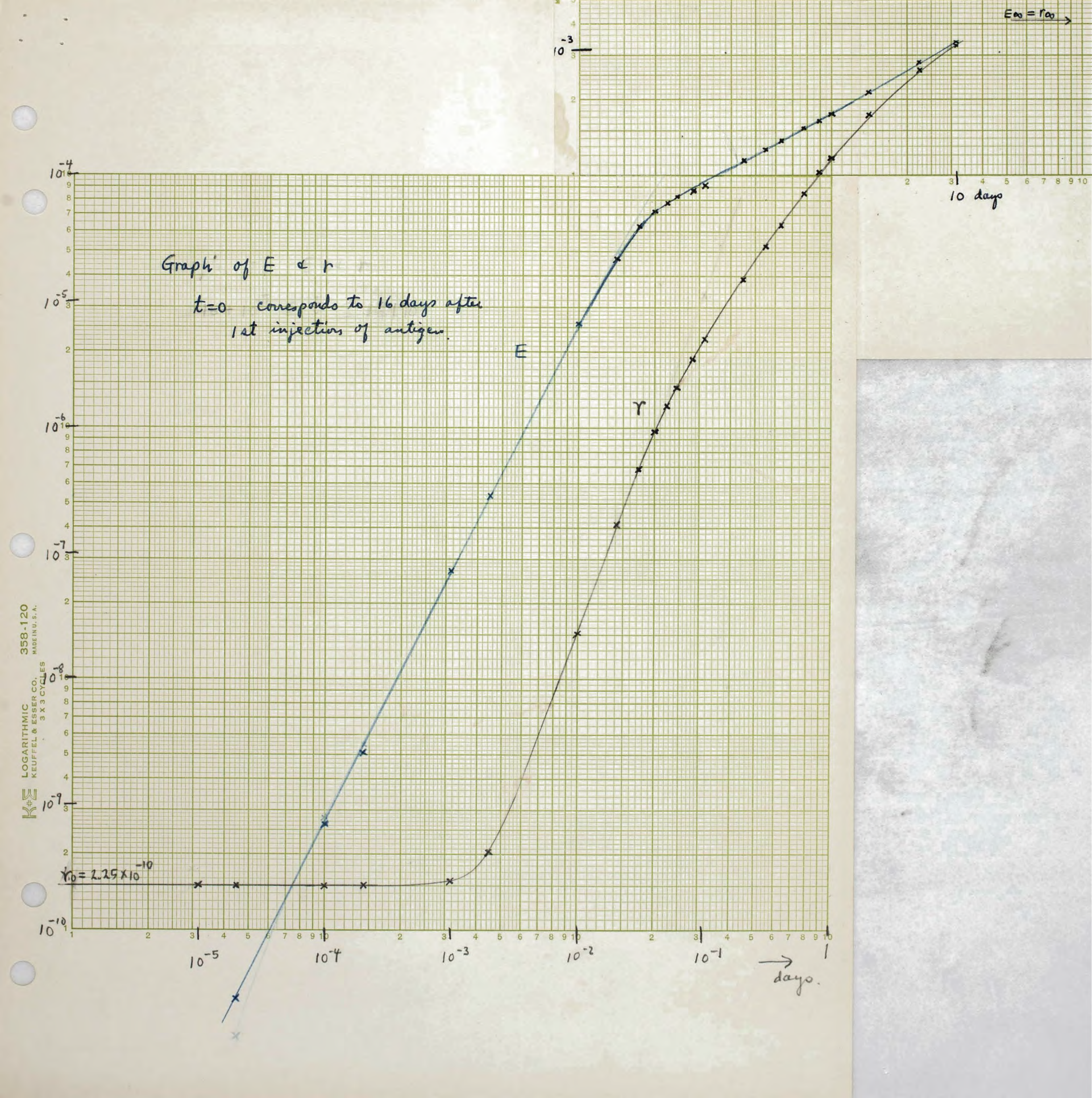
For $t \geq 16$, $\dfrac{dA}{dt} = \dfrac{a T_0 p(1-q)}{1+\frac{r}{k_2}} - \dfrac{A}{\tau_A}$ $\qquad \tau_A = 10$ days, $A_{t=16} = 0$.

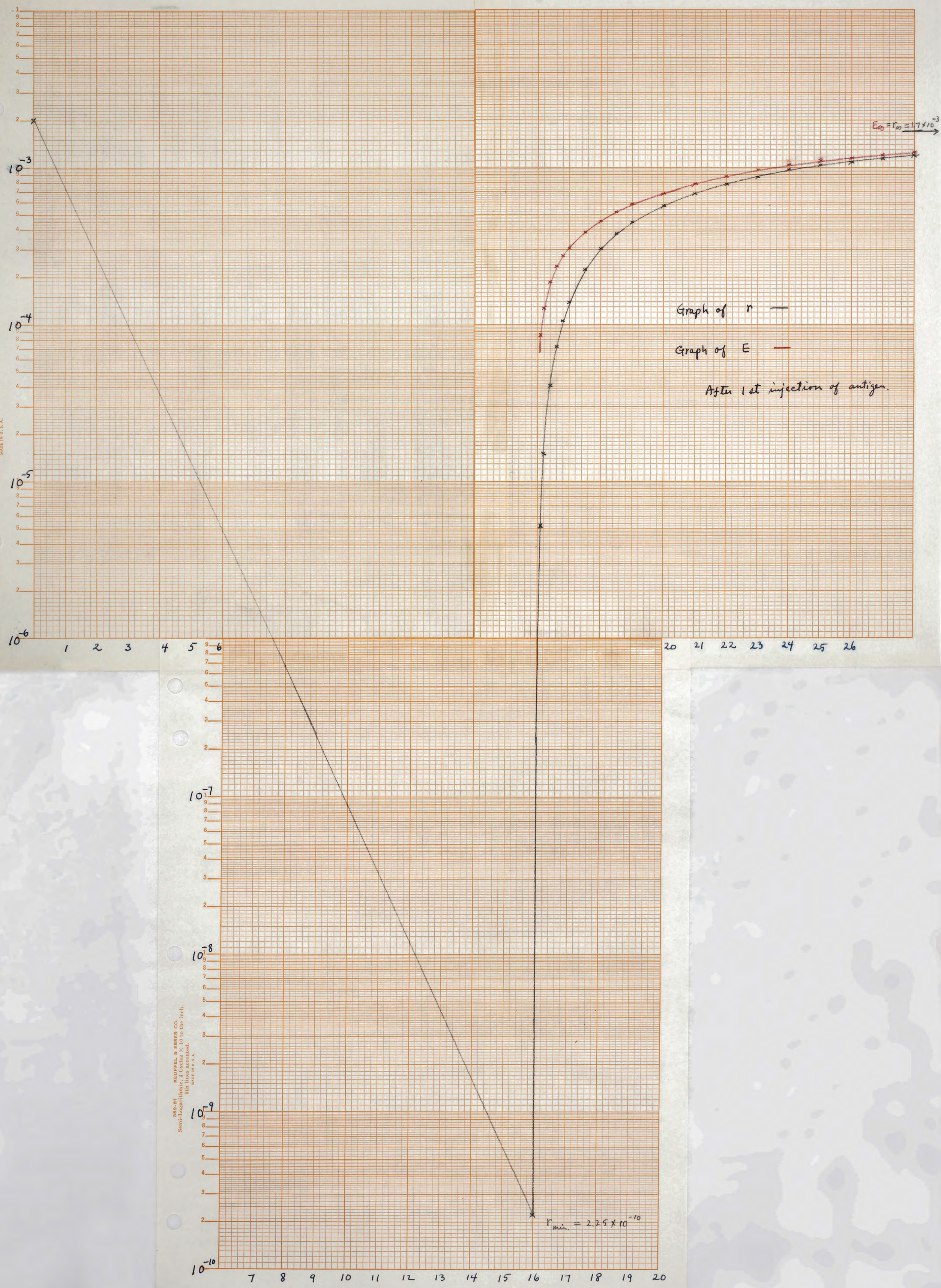
Here for $0 \leq t \leq 16$, we define

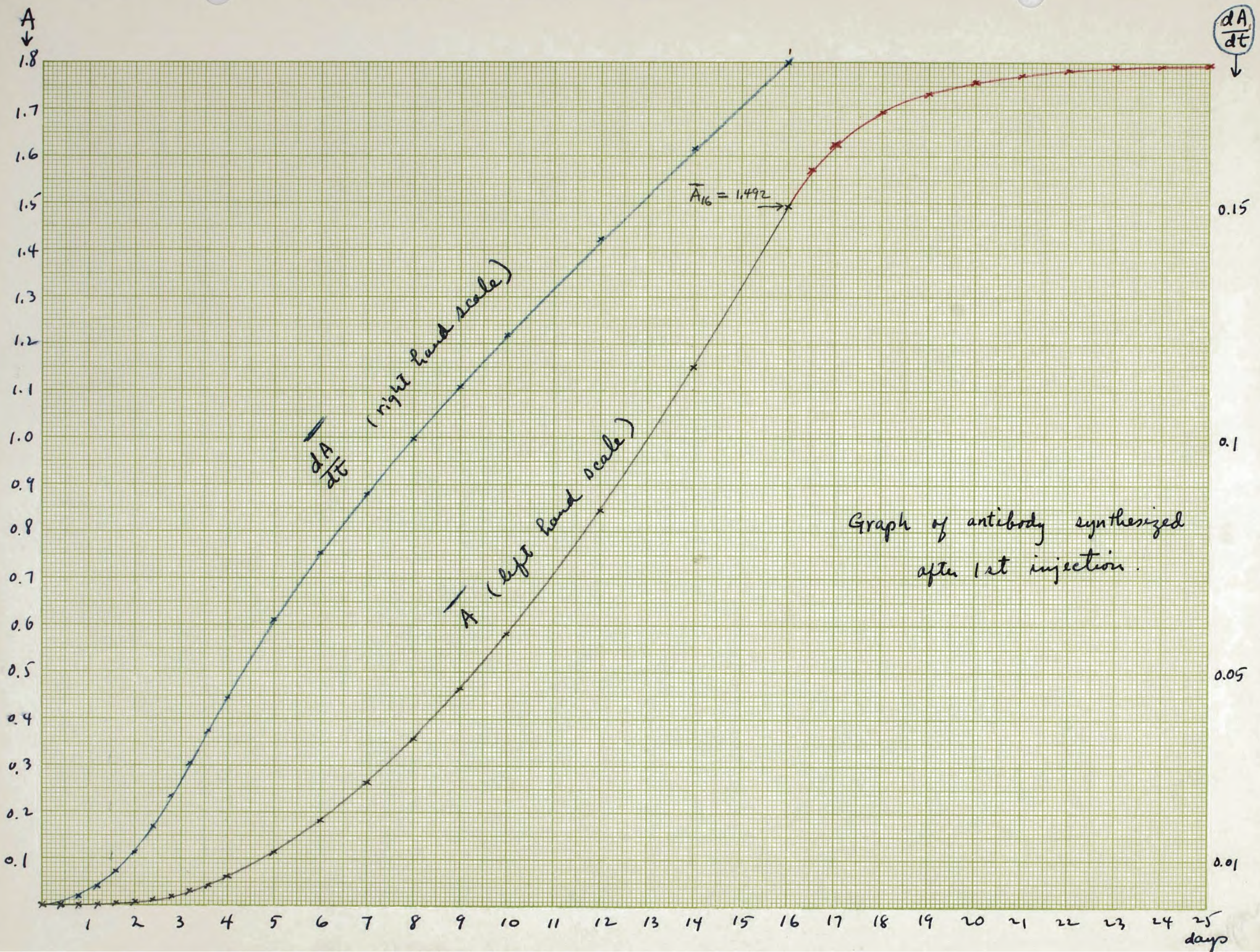
$$\left.\begin{aligned} p(t) &= 1 - e^{-0.01395t} \\ q(t) &= 1 - e^{-0.006585t} \end{aligned}\right\}$$

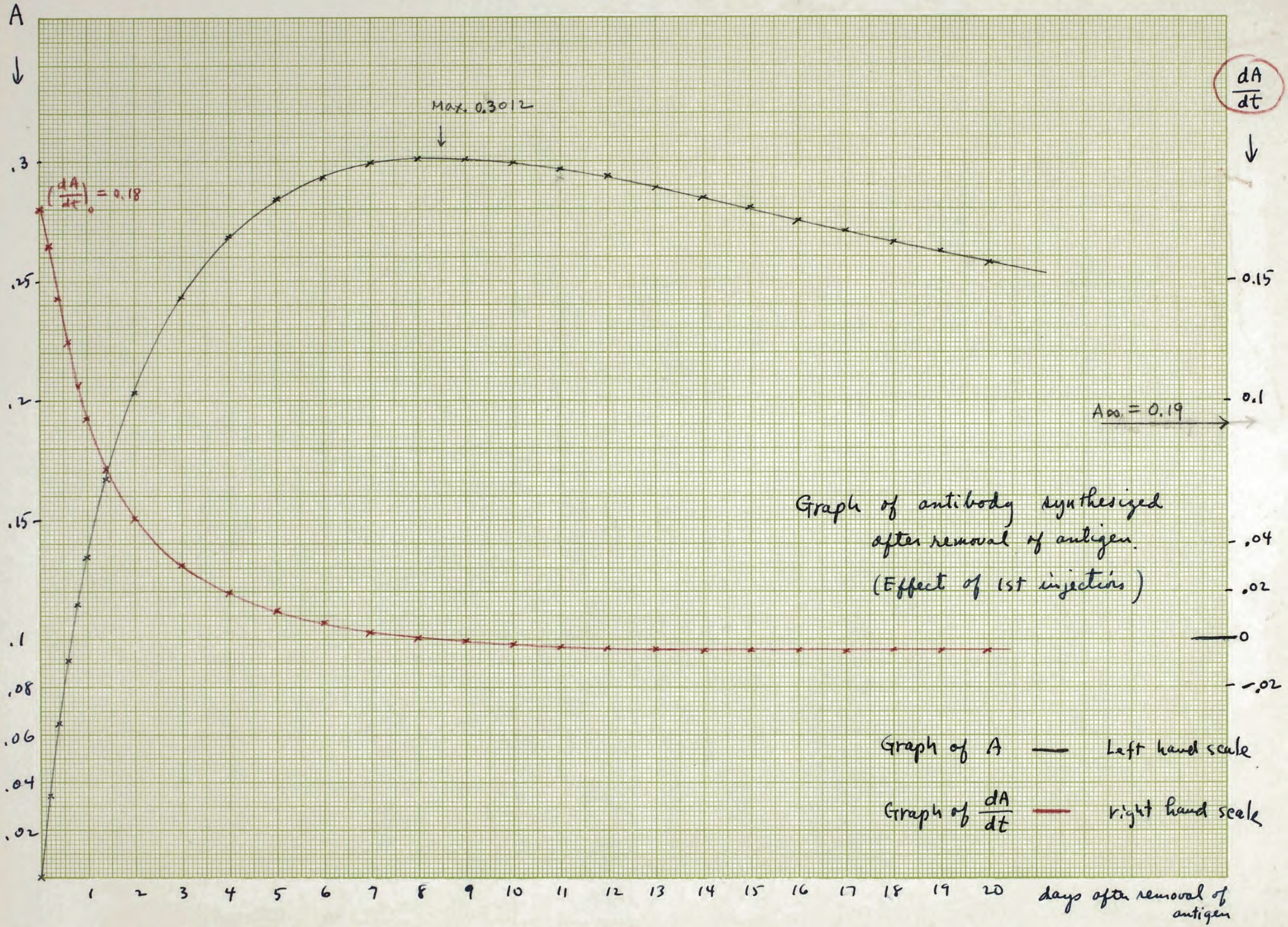
for $t \geq 16$, $p = 0.2$, $q = 0.1$.

Then we get (after putting $\frac{r_0}{k_2} = 10$) $A_\infty = 0.1898$.


Graph of E & r
t=0 corresponds to 16 days after 1st injection of antigen.
E
r
$r_0 = 2.25 \times 10^{-10}$
$E_\infty = r_\infty$
10 days
days.
LOGARITHMIC
KEUFFEL & ESSER CO.
3 X 3 CYCLES
358-120
MADE IN U.S.A.


$E_\infty = r_\infty = 1.7 \times 10^{-3}$
Graph of r —
Graph of E —
After 1st injection of antigen.
$r_{min.} = 2.25 \times 10^{-10}$
10^{-3}
10^{-4}
10^{-5}
10^{-6}
10^{-7}
10^{-8}
10^{-9}
10^{-10}
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26
KEUFFEL & ESSER CO., N. Y. NO. 359-81
Semi-Logarithmic, 4 Cycles × 10 to the inch.
MADE IN U.S.A.
359-81 KEUFFEL & ESSER CO.
Semi-Logarithmic, 4 Cycles × 10 to the inch.
5th lines accented.
MADE IN U.S.A.





Effect of 2nd injection of antigen.

a.) II Attempt $\frac{20}{k_2} = 10$

Suppose at time $t=0$, 2nd injection was given and also suppose at the time $t=t_1$ antigen was removed by antibody.

For $0 \leq t \leq t_1$, we assume $T_E = 0$, $E = 0$.

$r_{(t=0)}$ = steady state value after 1st injection $= 1.697 \times 10^{-3}$.

From $\frac{dr}{dt} = 0 - \frac{r}{1}$, we get $r = r_0 e^{-t} = 1.697 \times 10^{-3} e^{-t}$.

Now $\frac{d\bar{A}}{dt} = \frac{a T_0 p(t)(1-q(t))}{1 + \frac{r}{k_2}}$ with $\bar{A}_0 = 0$ $\left(\begin{array}{l} a,\ T_0,\ b = 1 \\ k_1 = 2\times10^{-7},\ k_2 = 2\times10^{-4} \end{array} \right)$

$p(t)$ and $q(t)$ are given by: $p(t) = 0.2 + 0.8(1 - e^{-0.01395t})$

$q(t) = 0.1 + 0.9\ (1 - e^{-0.006585t})$.

Integration gives $\bar{A} = 1.492$ when $t = 8.92$. Hence put $t_1 = 8.92$.

At the end of 8.92 days, antigen was removed and then:

$E_{(t=8.92)} = 0$, $r_{(t=8.92)} = 2.268 \times 10^{-7}$, $p(t=8.92) = 0.2937$, $q(t=8.92) = 0.1513$.

For $t \geq 8.92$, we have p and q both at constant.

And template recovery is given by: $T_E = (1 - e^{-\frac{t-8.92}{\tau_T}})(1-p)(1-q)$ with $\tau_T = 5$ days.

$$\begin{cases} \frac{dE}{dt} = a\, T_E(t) \frac{1}{1 + \frac{r}{k_1}} - \frac{E}{\tau_E} \\ \frac{dr}{dt} = b E - \frac{r}{\tau_r} \end{cases}$$

with $\tau_r = 1$ day, $\tau_E = 20$ days

Antibody synthesis is then given by:

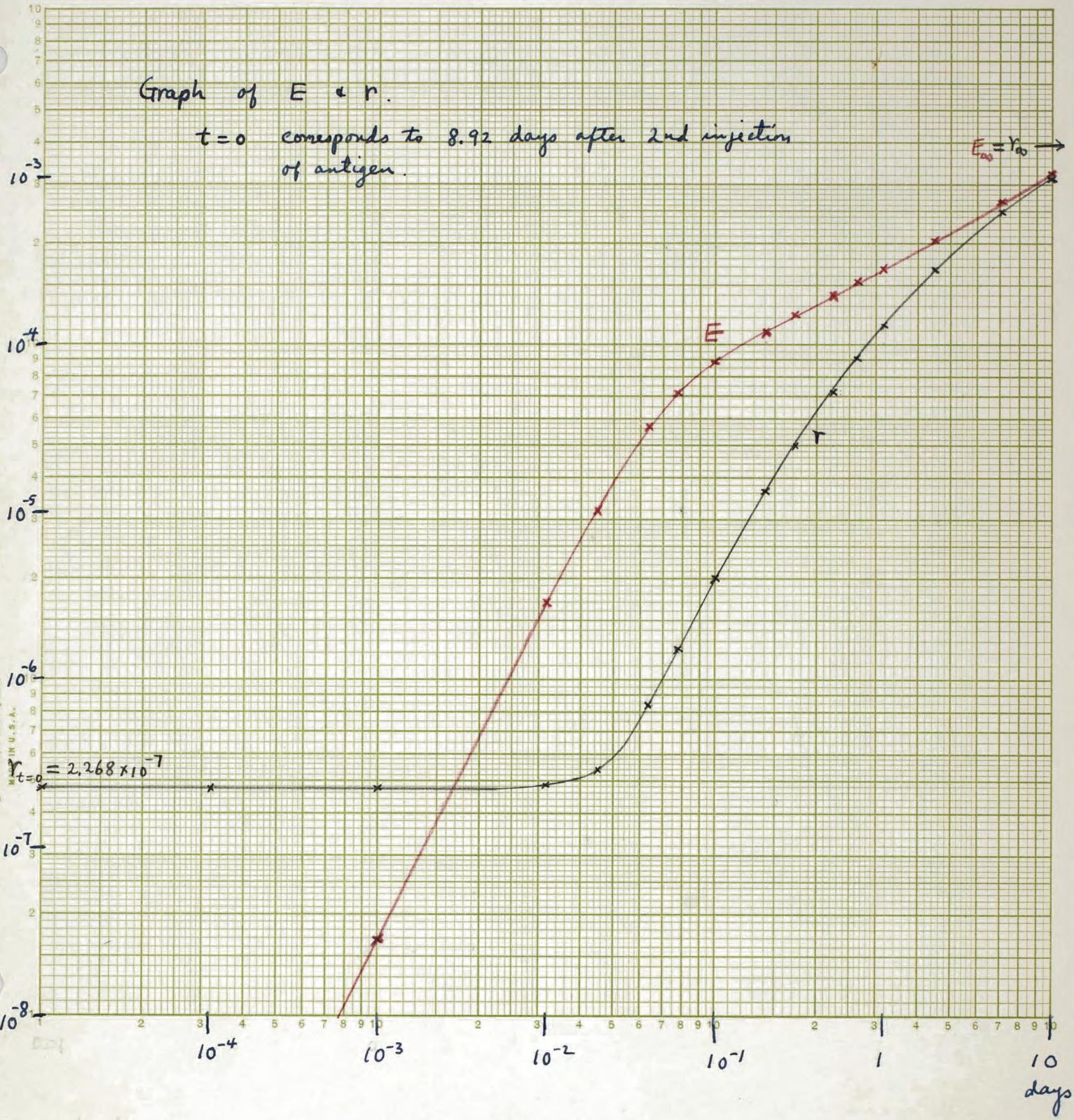
$$\frac{dA}{dt} = \frac{a T_0 p(1-q)}{1 + \frac{r}{k_2}} - \frac{A}{\tau_A}$$

with $A_{(t=8.92)} = 0$, $\tau_A = 10$ days.

For steady state values, we get:

$$r_\infty = E_\infty = 1.55 \times 10^{-3}$$

$$A_\infty = 0.285$$


Graph of E & r.
t = 0 corresponds to 8.92 days after 2nd injection of antigen.
$E_\infty = r_\infty$
E
r
$r_{t=0} = 2.268 \times 10^{-7}$
10^{-3}
10^{-4}
10^{-5}
10^{-6}
10^{-7}
10^{-8}
10^{-4}
10^{-3}
10^{-2}
10^{-1}
1
10
days
KEUFFEL & ESSER CO. N. Y. NO. 358-120
Logarithmic, 5 X 3 Cycles.
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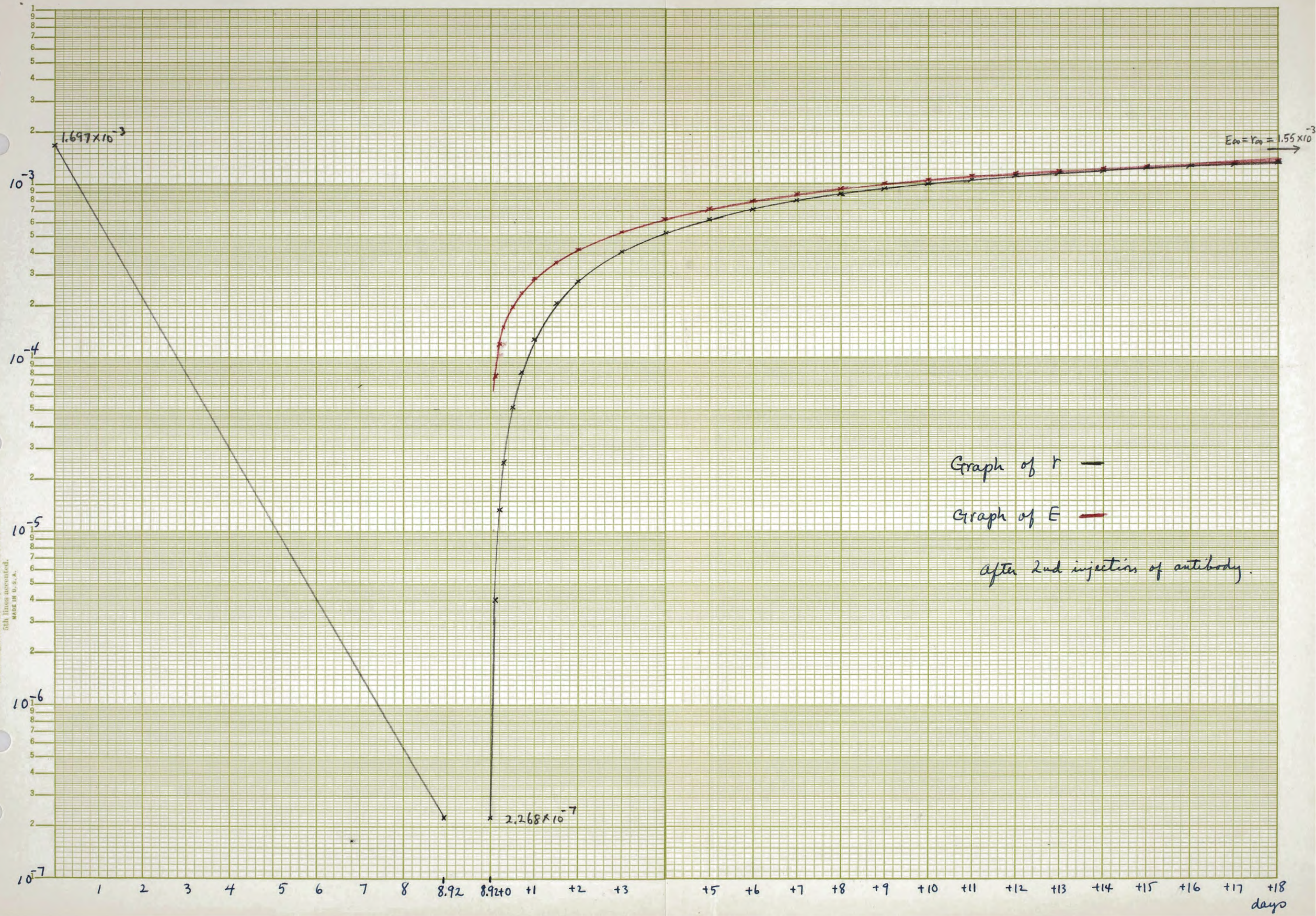
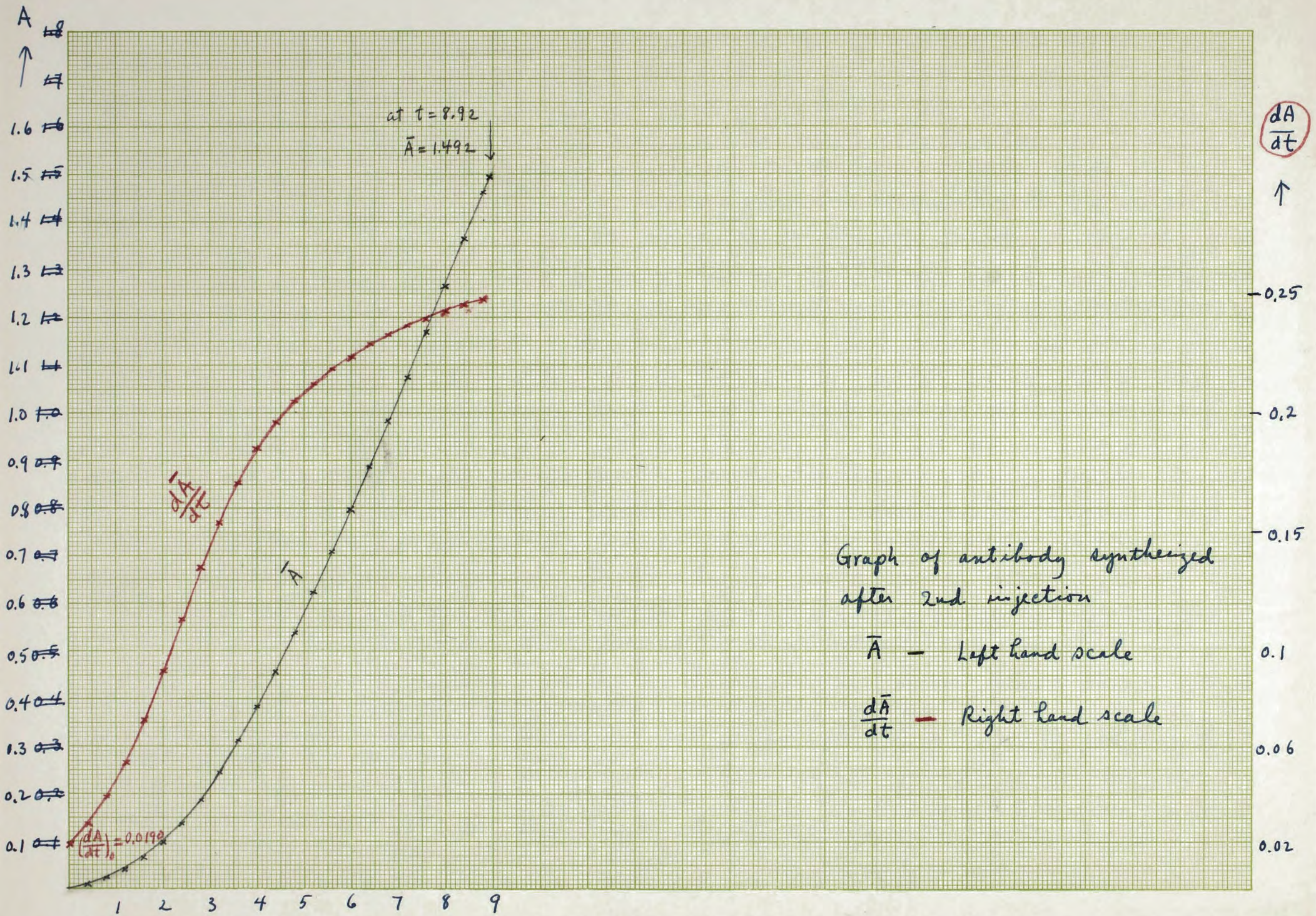
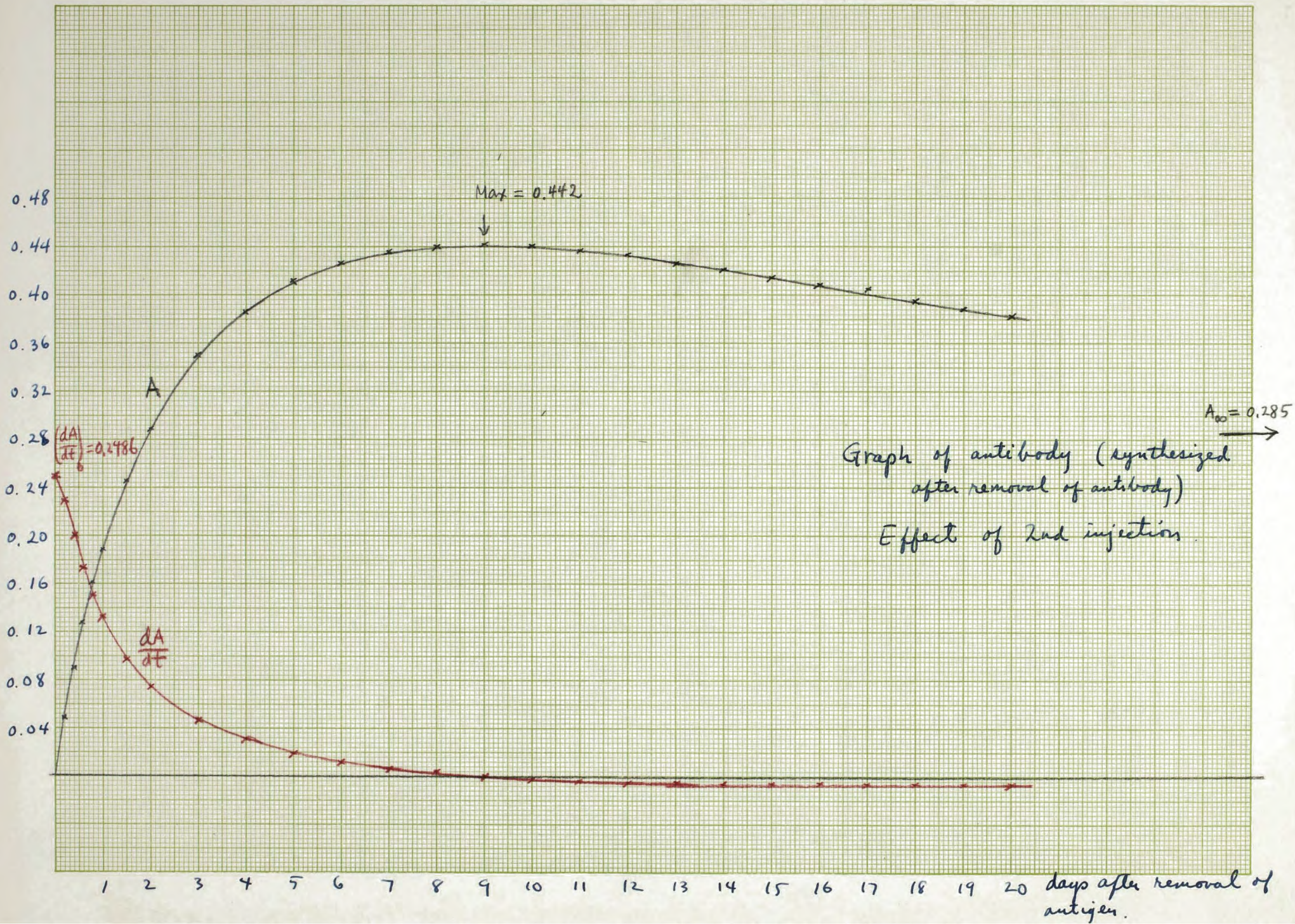

1.697×10^-3
E∞ = r∞ = 1.55×10^-3
10^-3
10^-4
10^-5
10^-6
10^-7
2.268×10^-7
Graph of r
Graph of E
after 2nd injection of antibody.
1
2
3
4
5
6
7
8
8.92
8.92+0
+1
+2
+3
+5
+6
+7
+8
+9
+10
+11
+12
+13
+14
+15
+16
+17
+18
days
358-91 KEUFFEL & ESSER CO.
Semi-Logarithmic, 5 Cycles X 10 to the inch,
5th lines accented.
MADE IN U. S. A.





Graph of antibody (synthesized after removal of antibody)

Effect of 2nd injection.

IInd attempt with $\frac{r_0}{K_2} = 100$. (II-b)

IInd attempt $\frac{r_0}{K_2} = 100$

For the basic equations and notations, see IInd attempt (a).

With given constants, we get following equation and final values.

Case of 1st injection.

For $0 \leqq t \leqq 16$. $E = 0$, $r = 2 \times 10^{-3} e^{-t}$

$$\frac{d\bar{A}}{dt} = \frac{(1 - e^{-0.01395t})\, e^{-0.006585t}}{1 + 100\, e^{-t}} \quad \text{with } \bar{A}_{t=0} = 0.$$

numerical integration gives $\bar{A}_{t=16} = 1.386$.

For $t \geqq 16$. We have identical formula for r and E as in IInd attempt (a).

$$E_\infty = r_\infty = 1.697 \times 10^{-3}.$$

$$\frac{dA}{dt} = \frac{0.18}{1 + \frac{r}{2 \cdot 10^{-5}}} - \frac{A}{10}. \qquad \text{We get } A_\infty = 0.0210.$$

Case of 2nd injection.

For $0 \leqq t \leqq t_1$. $E = 0$, $r = 1.697 \times 10^{-3} e^{-t}$.

$$\frac{d\bar{A}}{dt} = \frac{(1 - 0.8 \cdot e^{-0.01395t}) \times 0.9 \times e^{-0.006585t}}{1 + \frac{r}{2 \cdot 10^{-5}}} \quad \text{with } \bar{A}_{t=0} = 0.$$

In this case we have $\left(\frac{d\bar{A}}{dt}\right)_{t=0} = 0.0021$.

Numerical integration gives $A_{t=10.32} = 1.386$.

Hence we take $t_1 = 10.32$.

Then $r_{t=t_1} = 5.62 \times 10^{-8}$, $p(t_1) = 0.307$, $q(t_1) = 0.160$.

For $t \geqq 10.32$.

$$\frac{dE}{dt} = \frac{0.5815\,(1 - e^{-\frac{t - 10.32}{5}})}{1 + \frac{r}{2 \cdot 10^{-7}}} - \frac{E}{20}.$$

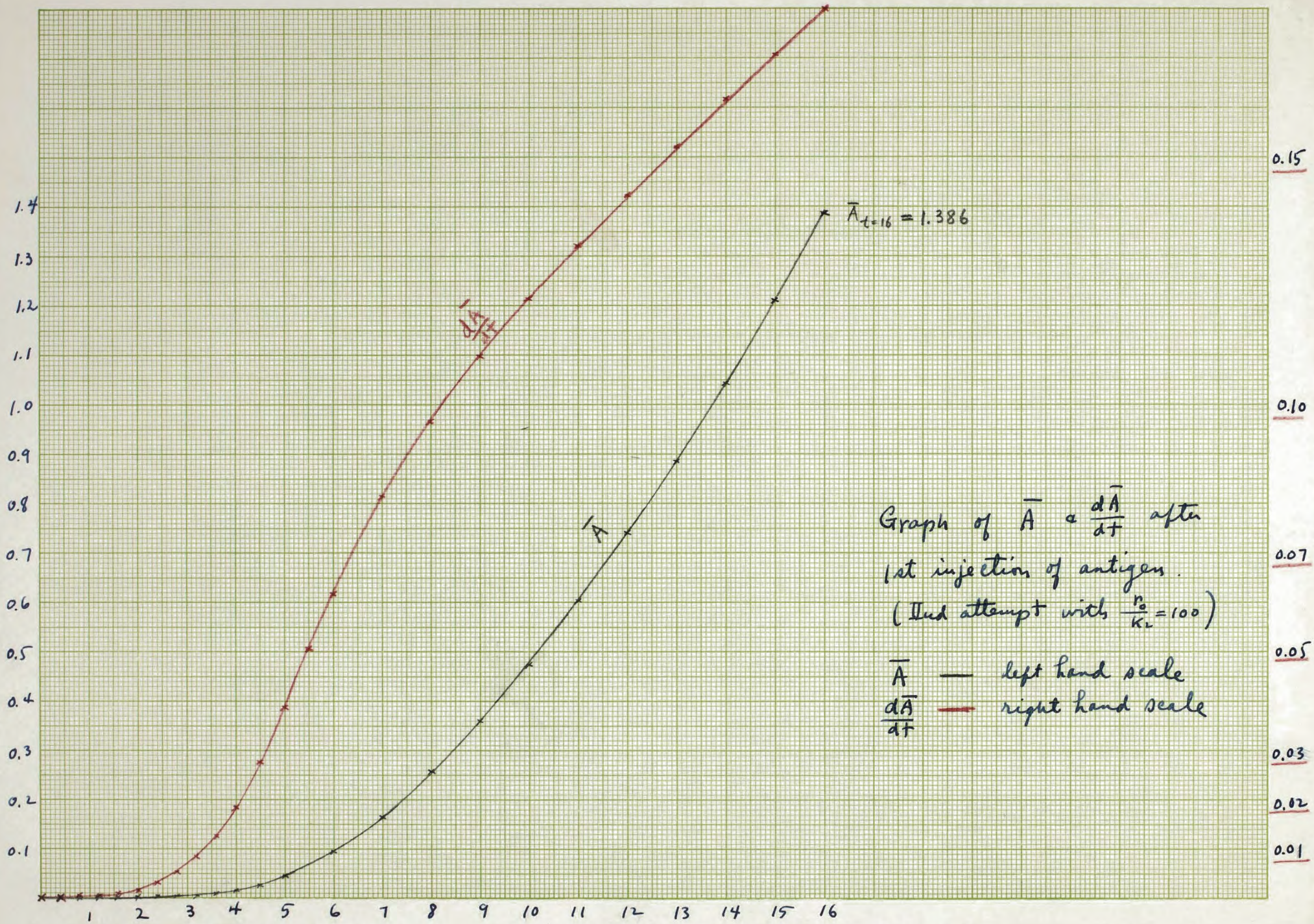
$$\frac{dr}{dt} = E - r$$

$$\frac{dA}{dt} = \frac{0.258}{1 + \frac{r}{2 \cdot 10^{-5}}} - \frac{A}{10}$$

As steady state values, we get $r_\infty = E_\infty = 1.525 \times 10^{-3}$

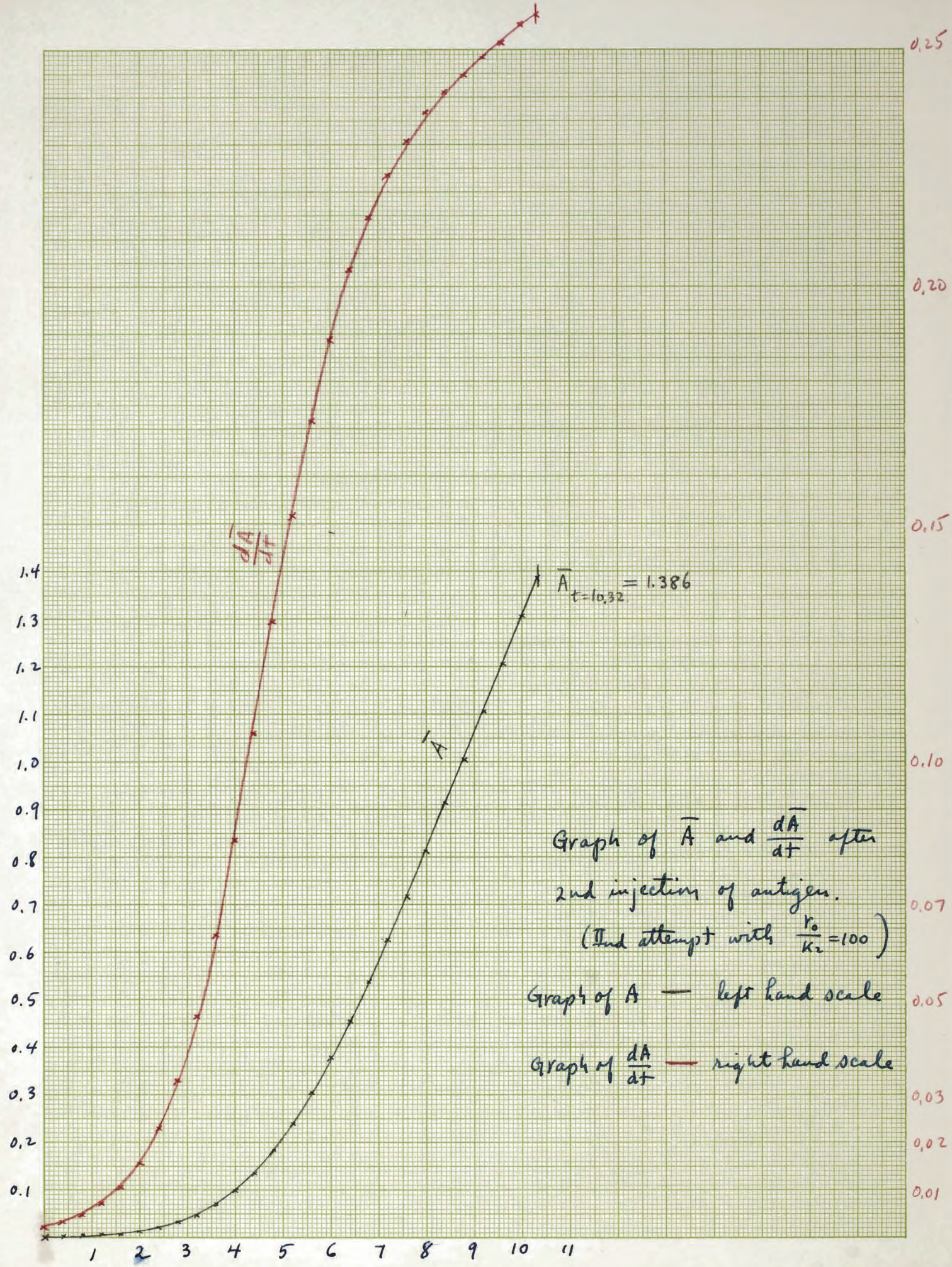
$A_\infty = 0.0332$.

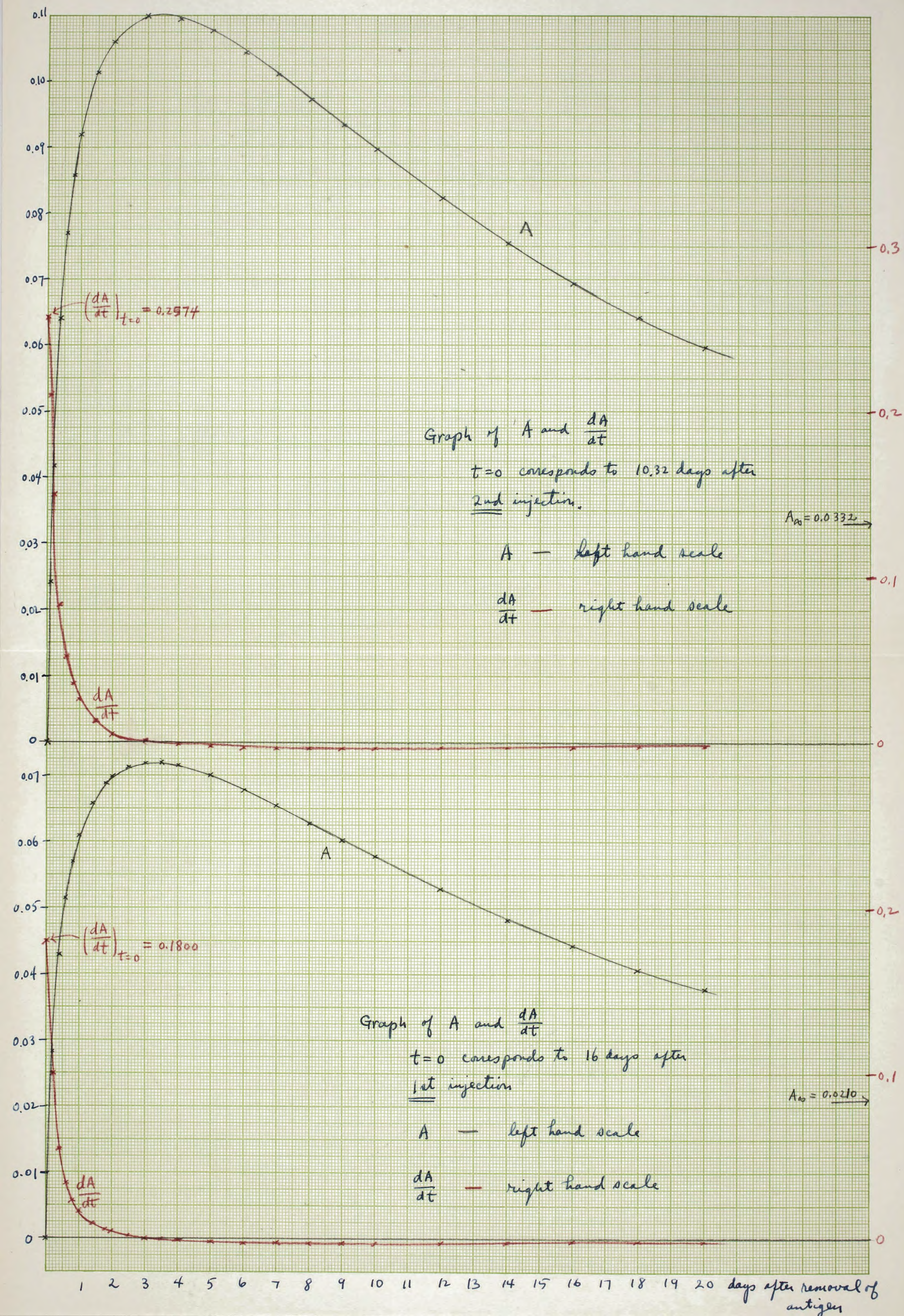
Primary



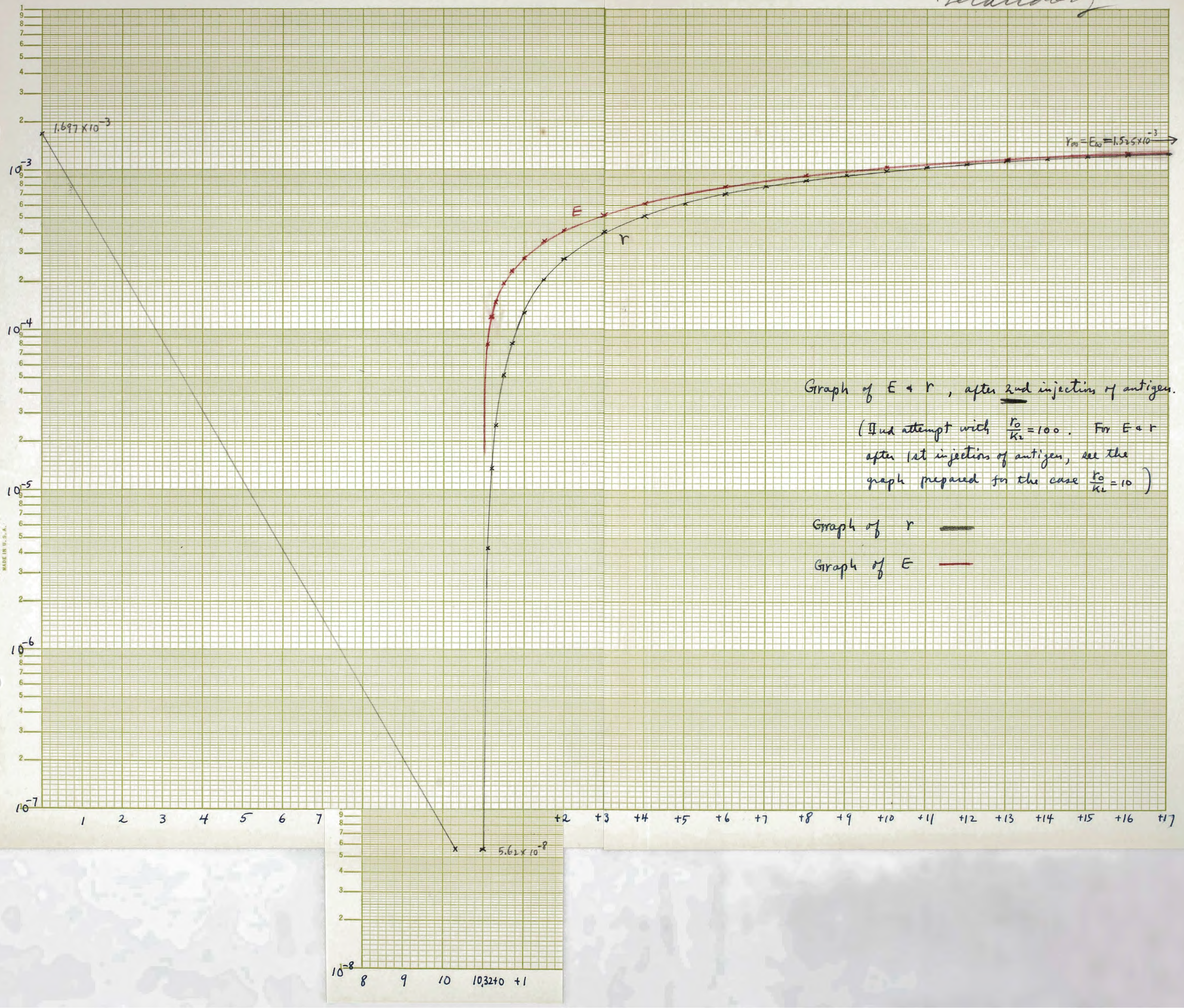
Graph of $\bar{A}$ & $\frac{d\bar{A}}{dt}$ after 1st injection of antigen.
(IInd attempt with $\frac{n_0}{K_2} = 100$)

$\bar{A}$ — left hand scale
$\frac{d\bar{A}}{dt}$ — right hand scale

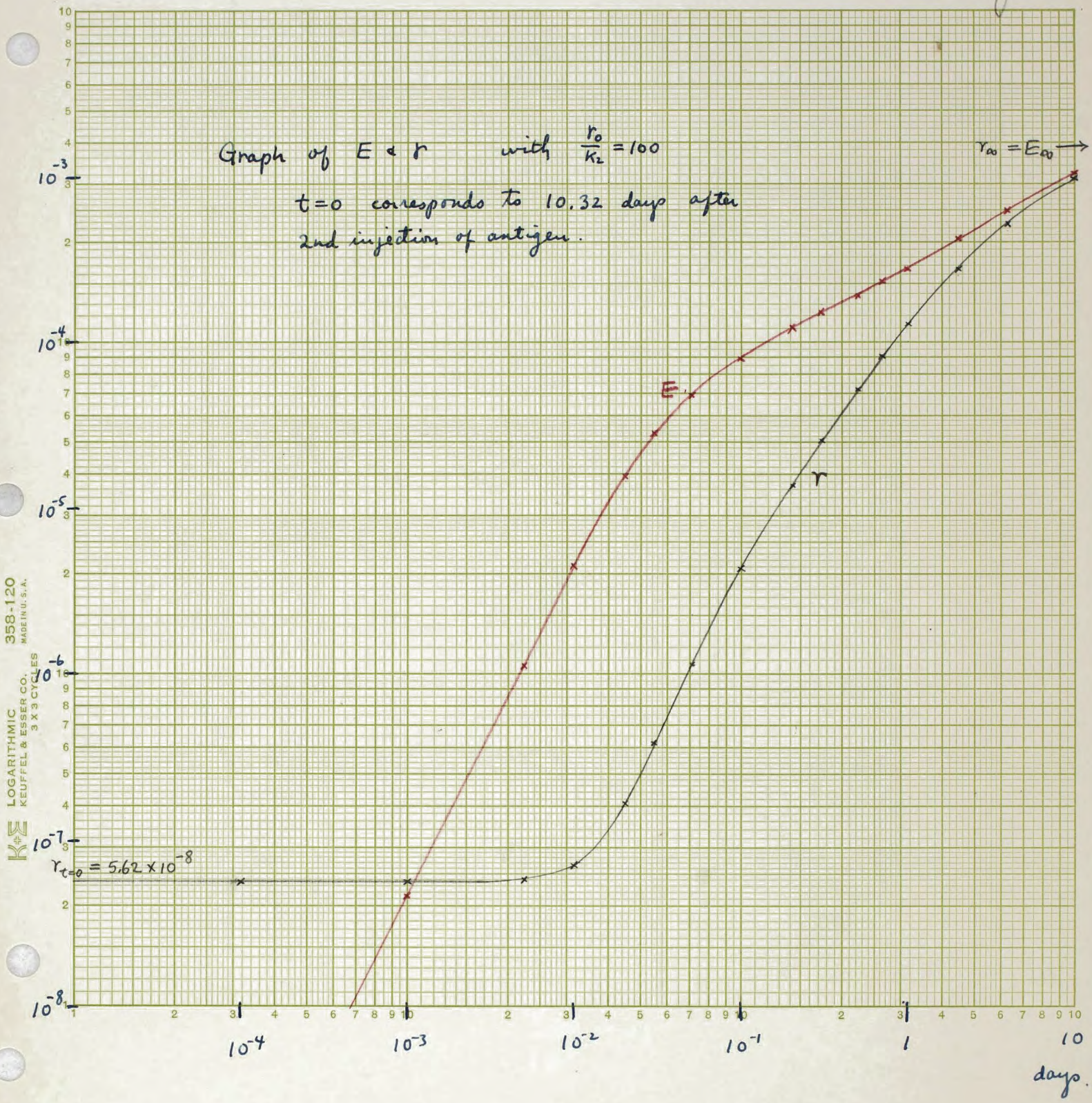




secondary



Secondary


Graph of E & r with $\frac{r_0}{K_2} = 100$
t=0 corresponds to 10.32 days after 2nd injection of antigen.
$r_\infty = E_\infty$
E
r
$r_{t=0} = 5.62 \times 10^{-8}$
10^{-3}
10^{-4}
10^{-5}
10^{-6}
10^{-7}
10^{-8}
10^{-4}
10^{-3}
10^{-2}
10^{-1}
1
10
days.
LOGARITHMIC
KEUFFEL & ESSER CO.
3 X 3 CYCLES
358-120
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