Austin, R., and MacLeod, C. M. (1949) J. Exp. Med. 89: 451-460

Acquisition of M protein by pneumococci through transformation

\[ \begin{align*}
\text{I - SVr} & \quad \text{I - SVr} \\
\text{III - Ab6 recruits} & \quad \text{III - Ab6 recruits} \\
\end{align*} \]

The "Dawson Rough" seems to correspond to Taylor's ER.

\[ \text{III - R36NC} \quad (\text{II; 2'}M) \]

When II - R36NC is (II; 2'M) was transformed with

III - Ab6 TP, III 2'M was obtained.

do. \( \text{II TP transformed} \).

Dawson Roughs were obtained from R36NC.

Some of these were transformed to III 3'M.

III 2'M. These may arise

from cells which still had some 2'M (antiglobulin detectable).

Their transformation does not take place so regularly. Griffield Roughs

not tested for II TP.

Avivo: ER + vaccine I \( \times 10^4 \)

+ vaccine III \( 2'M \)

Consistent acquisition of M3 antigen noted in

one class each.

I

II

Extracts of chromogenic S. aureus (strain??) transformed white strain to colored. Transformant strain retained for characterization.
Bennett, F.M. & McKeir, M. (1929) Type differences amongst
SF: ML+ lact- gel-

Phage B gave thick plaques of SF1B: opaque white; colorless or
translucent; thready appearance. 1/3 was also resistant to C.
SF1B was non-lysozyme, but after being kept on agar for some
weeks gave rise to papillae some of which were of the chalky white
otype, others feebly opaque. Either in this way, or directly
... 51/12 ... the areenotype of SF1B could be obtained.

Non-competitive:

\[ E + I \rightleftharpoons EI \]

\[ K_I = \frac{(E_0)(I_0)}{(EI)} = \frac{(E_0)(I_0 - EI)}{(EI)} \]

\[ E = E_0 + EI \]

\[ = aE + EI \]

Let \( I' = \frac{I}{K_I} \); \( E' = \frac{E}{K_I} \)

"specific concentrations"

\[ I' = \frac{1-a}{a} + (1-a)E' \]

\[ \text{free} \quad \text{(combined)} \]

\[ \text{(i.e. } I = I') \]

\[ \text{2me A: } I' = \frac{1-a}{a} \quad \text{2me B: } I' \neq I' \neq EI \]

\[ \text{2me C: } I' = (1-a)E' \quad (I' = EI) \]

\[ E + S \rightleftharpoons ES \rightarrow E + P \]

\[ a = \frac{V}{V_{\text{max}}} \quad V = k_0(ES) \]

\[ V_{\text{max}} = k_0(E) \]

\[ S' = \frac{a}{1-a} + aE' \]

Most enzyme systems operate in zone A, i.e. \( S' = \frac{a}{1-a} \) (Michaelis-Menten)

They prefer to plot \(\frac{V}{V_{\text{max}}} / \log_{10} S \)

Consider \(1.1 \times 10^{-5}, 1.25 \times 10^{-5}, 1.7 \times 10^{-5} \)

The Sauerbruch equation is fitted as follows:

\[ \frac{S}{a} = K_S \frac{1}{1-a} + E \quad \text{and} \quad \frac{I}{1-a} = K_I \frac{1}{a} + E \]

s.a. 26, 559.
\[
\frac{V_{\text{max}}}{V} = 1 + \left( K_s + \frac{I}{K_I} \right) \frac{1}{s}.
\]

For \( I = 0 \), \( \frac{V_{\text{max}}}{V} = 2 \) where \( \frac{K_s}{S} = 1 \).

Otherwise, for a given, constant activity:

\[
\frac{K_s}{S} + \frac{I}{SK_I} = C.
\]

\[
C = \frac{1}{S} K_s + \frac{I}{SK_I}.
\]

\[
SC = K_s + \frac{I}{K_I}.
\]

\[
S\alpha = 1 + \frac{I}{K_s K_I}.
\]

\[
\alpha S - bI = 1.
\]
Competitive equilibrium.

\[ \frac{E_s I_s}{(EI)} = K_I \]
\[ \frac{E + S +}{(ES)} = K_S \]

\[ \frac{(E S)}{E} = a. \quad ES = q E \]

\[ E = ES + EI + E_+ \]

\[ EI + E_+ \]
\[ E = 1 - a \]

\[ EI = (1 - a)E - E_+ \]
\[ = (1 - a)E - K_S a E \]
\[ = S - a E \]

\[ I' = \left[ (s' - a E_s) \left( \frac{1 - a}{a} \right) - 1 \right] + \left[ 1 - a \left( 1 + \frac{1}{s' - a E_s} \right) \right] E_+ \]

\[ \text{If } I_0 \approx I \]
\[ I' = (s' - a E_s) \left( \frac{1 - a}{a} \right) - 1 \]

\[ \text{or if } I_T \approx I \]
\[ I' = \left[ 1 - a \left( 1 + \frac{1}{s' - a E_s} \right) \right] E_+ \]

He finds \( \frac{I'}{s'} = 1 - \frac{a}{a} \)
\[ i.e. \text{ for } a = \frac{1}{2}, \quad \frac{I'}{S'} = \frac{K_I}{K_S}. \]

\[ \frac{1 - a}{I'} = \frac{a}{S'} \]

\[ \frac{E I}{E} = \frac{E S}{E} \quad \text{and} \quad \frac{E I}{I'} = \frac{E S}{S} = \frac{K_S}{K_I}. \]
Hoder, F. + Miano, R., Z. Immunf. 85:423 - (1935)
Some observations on staphylococci dependent strain of Staphylococcus aureus.


Breth & vania P inoculated with varying autumos (10^{-1} to 10^{-6}) of a 24hr. broth culture. Later plated loopful (ca. 0.02 ml) on agar.

With large inocula, secondary growth found up to 1/4 ou/ml;
with initial bacteria $< 10^3$, no sec. gr. but eventually comes up.

"Dose rate is not correct and that the resistant bacteria appear only after contact with penicillin for some time. Hence, if dose?"

Reasoning? Note that with ca. 1/8 ou/ml and perhaps 10^{-5} ml, any secondary growth was delayed 48-72 hours.

In 5-7 days it appeared only after 6 days. "In these cases where the secondary growth appears, in the late portion, possibly it can be taken for granted that the growth does not originate from resistant bacteria present in the original culture."

(Some confusion about isolation of pure resistant cultures, in last line for clarity.)

Poured scintill in nutrient medium only in 3rd culture. Not 3rd ml cultures.
Treatment of recombination in cells since 1948

1930 Clifton Introduction to the bacteria, pp 73-75
"Possibility of recombination of genes by other than sexual mechanisms may exist; and the original definition of bacteria as "apparently sexless organisms" is still valid." Fair statement of facts, Tet 1947

1944 Barcroft et al. p. 187 for sexual analysis of variations
Streus, B.A.D. (1949) Measurement of age of mutation of Mycobacterium
plague in mice. J. Hyg. 47: 598-641.

We need to estimate the infection rate and the infection rate at this time.

Streus observed a mutation rate of approximately 1 in 10^6 for the plague.

occasional or rare occurrence in a population.

unusual. Some populations have a mutation rate of 1 in 10^4 in one
rate of 3.5 x 10^-4/generation for E. coli. Noted

K R

Michael, E. (1973) Genetic stability. The genetics of the

in a cell. J. Mol. Biol. 10: 35.
Himite flies (H/m) show m spots. Originally interpreted as elimination of H carrying deficient chromosome. By use of t. translocation, it was shown that the H phenotype (not merely deficiency, covered by duplication) was necessary for spotting. Double (bb) spots not found: interpreted as partial elimination.

Autosomal H also cause x-mosaic (\(s^{n^3}\) spigid). However, the Blk \(\times\) Himite causes x-spots, but not H-spots!!!!

Effect of autosomal H on Notch. If was studied:

\[ N^8/y \times s^{n^3}; H/m + s^3 \]

Among females:

\[ \frac{N^8/s^{n^3} y/280}{y/s^{n^3} 15/381} \]

No difference.

\(s^{n^3}\) sets (elim? N^8)

\[ 2 \] 2 y spots

11 twin spots (y.y)


You+/+ flies \(\rightarrow\) 110 y sp, 43 y. 75 m spots. \(y\) and m simply somatic crossing-over as well as segregation. But no y-sin twin spots were found, ruling out two-strand crossing-over. Complete reduction is ruled out by absence of you-sin-y- (+) triple spots.

\[ \frac{Y}{m} \times \frac{X}{\_} \frac{y}{\_} \quad \frac{1}{2} \]

Segregation patterns:

\[ \frac{y}{\_} \frac{m}{\_} \frac{X}{\_} \frac{Y}{\_} \]

\[ \frac{\text{equational}}{} \quad \frac{\text{reductional}}{} \]

Regions of crossing-over varies with spot size (developmental stage). Crossing-over to the right \(\times\) in you spots suggested by spots with 2 translocation.

Segregation is probably nearly always equational.
bb fails to show segregation in +/bb flies. Assumption of phenotype
swallowing seemed unlikely. Crossing over to the right of bb considered
my case.

Determine X-block of spots by color of 5-6th abdom. segments.
Most spots in jirds come XX by color.

Autosomal mosaics

Under influence of autosomal M.
Secondary Sources:

1. Sorsby "Clinical Genetics"; pp 337-40; 313-15
2. Kallmann and Sander 1947. in Hoch & Knight, "Epilepsy". Chap. 3

Acc. (3) 25-30% of propositi have family history (5-6x as frequent in parents, sibs and children of propositi). monozygotic twin correlation 70%. Quotes Lennox extensively on cerebral dysrhythmia. In 24% of families both parents showed dysr. Obvious complexity.

Examples in animals; also audiogenic seizures. From Conrad; (incidence figures)

<table>
<thead>
<tr>
<th>gen. pop.</th>
<th>childr. sibs</th>
<th>neph &amp; nieces</th>
<th>dizyg.</th>
<th>monozyg.</th>
</tr>
</thead>
<tbody>
<tr>
<td>.3</td>
<td>6.3</td>
<td>4</td>
<td>1.2</td>
<td>3.1</td>
</tr>
<tr>
<td>66.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

Concordance in twins:

diz

<table>
<thead>
<tr>
<th>idipath.</th>
<th>symptom.</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.3</td>
<td>86.3</td>
</tr>
<tr>
<td>0</td>
<td>12.5</td>
</tr>
</tbody>
</table>

Thus even sympt. epilepsy has a genetic component. Index twins were restricted to severe hospital cases.

Also found consanguinity correlations with mental deficiency, but not with schizophrenia.

From Lennox: dysrhythmia

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<tbody>
<tr>
<td>general pop</td>
</tr>
<tr>
<td>epileptics</td>
</tr>
<tr>
<td>par and sibs</td>
</tr>
</tbody>
</table>

In twins, 85% show concordance of encephalo. of monozyg; 5% if dizyg.

Similar to 2, but emphasizes consanguinity correl. with psychopathy.

Conclusions: Inheritance not simple (probably several different mechanisms). Certainly a very large genetic component in severe cases, from Condrad's twin studies. Most frequent suggestion is dominant with low penetrance, but high incidence of dysrhythmia in both parents of propositi (Lennox) suggests recessive factors also.

(Lennox '47 is Res Pub Ass Res nerv ment dis 26:11)

CC: Dr. Javid
Conjugation in yeast.
Fowell 1951: Lymphocytes divide: mating of cells gives \( \text{\textbullet} \) from which either haploid or diploid
or diploid (i.e., \( \text{\textbullet} \) and \( \text{\textbullet} \))
spores may be generated. To be sure to remove pseudospores.

Paried 250+/− cells; 30 zygotes formed.

\( \text{\textbullet} \) and \( \text{\textbullet} \) zyg. =
only haploid. Other zygotes = mit. 2n. "An investigation of
spore fusion revealed that nuclear fusion apparently always occurs
in zyg. formed by this proc." Paried 1946 also suggest die.
Also discussed by Stainman 16; (Bosherland 75, Bot Rev 1940 6:1) Lab. Hapl.
J. Finzi 1928.

Paried may give hybrid cells before fusion.

W. L. Sineine \( \text{\textbullet} \) spores. But 6'35 also shows
substantially complete expectation of diploid spores: some variation.

Bunte analogy of Fowell's hic. i ergie formation.

S. para B + G+ rodvext → significant antigenic variation
in Salmonella → antitoxins; Breslau.
20. Andrews 75 Ac. 62 R (Ed 32 Dec 37)
18. Bundt Acet (Exp BM 6: 27)

Delbruck 1943 Adsorption, etc. lysis = loss of
22, 365 -
2 2,365 -

Temperature same as for cell decision

Receptor: 63. Levine + Fredech, JEM 59: 213
See Bundt 9, AJEMY 15: 227
J. Experm. 76: 281.

(decrease glucose in virus media)

Tryptone 20% glucose 1% NaCl 1% pH 7.4

AD: Hickray 8

5 ml plague
2 ml 12-24 hours 10^8/ml
3.5 ml mixture 0.7% agar

pour on plate

Handwritten notes.
Recherches sur la Paraglutination. Différenciation des antigènes H et O.

They had shown that P. exhibits a different endotoxin specificity from the "agglutinins" composite de Schütze. But the strains do contain an antigen related to the preceding strains.

The paraglutination of strains are homogeneous; repeated resolution indicates that the modification is heritable. Only some strains are capable of paraglutination.

coli-typhoid paraglutination.

The P. coli absorb H-antigens from anti-typhoid sera. The original coli does not.

anti-H was removed by adsorption as Stanley. There was little further agglutination adsorption. However, this was still considerable aggl. of coli. Paraggl. coli has all H antibodies, and a fraction of the O of typhii. Anti-P colisum has a nowtiter on heated typhii. Typhii phagocytose large (P) coli.


Baug mit Typhusserum.
Using para A and the tripels, P1 is also obtained with cross-
reactivity, but very little in para B. Could not transform stable.
Relevant paragptsuration to the
for transformation.

Wahlen + Almender. JID 65:147-55 (1977)
Aggelony, J. C.  J. Back 38: 611-57 (1937) Cytology and methods of reproduction of two cocci and the possible relation of these organs to a unique family need.

Cocci appeared in a culture of the bacterios.
J. Bart 50
Chum. + Agronomy Illinois

Some morphological characteristics of nodule beet as shown by the electron microscope, if. I See Soil Sci Soc Am. Proc. 7: 269-71 (1942)

4-5 granules/cell untreated + E. 0.2% NaHCO₃ 2½ hrs. Attempted staining w.g. M saline lift mottef cells. (several transparent, corresponding to nuclei?) After NaHCO₃ saline did not remove granules acetone removed granules, also HNO₃, HCl

low pH etc.

Sporoae are not found until onset of phytoplanktonic assemblage + other the autotrophic group.

"heating cells, facial structures, coordinate with ..."

Geo.
Fusca H. L. J. Biol. 35: 161
Koayai, I. + M. M. / Bericht 45: 391-57 (1973)

The internal structure of carbonic lactate

Apparent nucleic acid material organisation in C. Seegawa
Most adjacent cells were in the same early phase
B. coli (N4) dependent keratinocystinagin appeared as filaments from a "many very large concave like forms" were encountered developing from the filaments.

Kisito, proteose peptone, 5% NaCl broth + 1% Na2HPO4, autoclaved at pH 6.8, autoclaved, filtered + reautoclaved. Pgl reduced inoculum.

Single cell isolate inoculated in broth 37° 72h. Then, in broth, streaked out 1 end (with peptone 10% 0.8) were inoculated at 37° 18-24 hours, periphery of colonies under fungus & pycnospore formation.

" attempted has been made to study the fate of these spore like bodies."

Similar forms were found in small cells.

No convincing evidence of origin from 1 cell.

Assumes that cell-division has taken place. Citing Heringst.

"made it necessary to rule out the purely symbiotic influence of the accompanying strain."

10: 574-88 (1925)