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

Acquisition of M protein by pneumococci through transformation reactions.

I - SVI
III - A66 } used. { I -
III - 3M

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

When II - R36NC (or - 36A) (II; 2'M) was transformed with
III - A66 TP , III 2'M was obtained.

do, ∈ TPI transformation.

Dawson Roughs (= ER) were obtained from R36NC.

Some of these were transformed to III 3M.
from cells which (altho) still had some 2'M (to III 2'M. These may arise
(serologically detectable)

This transformation does not take place so regularly. Griffith Roughs not tested for TP.

In vivo: ER + vaccine I 2/10
+ vaccine III 2/10

Concomitant acquisition of M3 protein noted in one case each.

↓
R
↓
II.

Bryatt, Pamela H., Jann, D. J. & Salle, A. J. (1948) Variation in pigment production in Staphylococcus aureus.

Extracts of chromogenic S. aureus (strain??) transformed white streaks to colored. transformed strains retained lac- character.

Burnet, FM + McKie, M. (1929) Type differences amongst staphylococcal bacteriophages. Austr. J. EBMS. 6: 21-21.

SF: Mle- lac+ gel-.

Phage B gave three kinds of SF/B: opaque white; colorless + translucent; frank aureus. /B was also resistant to C.

SF/B was non-lysogenic, but after being kept on agar for some weeks gave rise to papillae some of which were of the chalky white type, others frankly aureus. Either in this way, or directly ... SF/B ... the aureus type of SF/B could be obtained.

Goldstein, Aronson (1944) The mechanism of enzyme-inhibitor substrate reactions. J Gen Physiol. 27:529-580

Non-competitive.

$E + I \rightleftarrows EI$. a = fractional activity = $\frac{E_f}{E}$. E = total enzyme, f = free

(1) $K_I = \frac{(E_f)(I_f)}{(EI)} = \frac{(E_f)(I-EI)}{(EI)}$ $E = E_f + EI$

$= aE + EI$

(2) $I = K_I \frac{(1-a)}{a} + (1-a)E$. Let $I' \equiv \frac{I}{K_I}$; $E_I' = \frac{E}{K_I}$

= "specific concentrations"

(3) $I' = \frac{1-a}{a} + (1-a)E_I'$ (2me B).

(free) (combined) 2me A = $I' = \frac{1-a}{a}$ ($E < \frac{K_S}{10}$, i.e. $I \simeq I_f'$)

2me B: $I' \neq I_f' \neq EI$.

2me C: $I' = (1-a)E_I'$ ($I' \simeq EI$)

$E + S \rightleftarrows ES \rightarrow E + P$. $a = \frac{v}{V_{max}}$ $v = k_0(ES)$, $V_{max} = k_0(E)$

(3B) (4A) and $S' = \frac{a}{1-a} + aE_S'$

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

They prefer to plot $\frac{v}{V_{max}}$ / $\log_{10} S$. Considers 1.1×10^{-3}; 1.25×10^{-3}; 1.7×10^{-3} as good fits for K_S.

The zone B equation is fitted as follows:

$\frac{S}{a} = K_S \frac{1}{1-a} + E$ and $\frac{I}{1-a} = K_I \frac{1}{a} + E$.

S.A. 26:559.

$$\frac{V_{max}}{v} = 1 + \left[K_s + \frac{I}{K_I} \right] \frac{1}{S}.$$

For $I = 0$, $\frac{V_{max}}{v} = 2$ when $\frac{K_s}{S} = 1$.

otherwise, for a given, constant activity:

$$\frac{K_s}{S} + \frac{I}{S K_I} = C.$$

$$C = \frac{1}{S} K_s + \frac{I}{S} \cdot \frac{1}{K_I}$$

$$SC = K_s + \frac{I}{K_I}$$

$$Sa = 1 + \frac{I}{K_s K_I}.$$

$$aS - bI = 1.$$

Competitive equilibrium.

$$\frac{E_f I_f}{(EI)} = K_I \qquad \frac{E_f S_f}{(ES)} = K_S$$

$$\frac{(ES)}{E} = a. \quad ES = aE. \qquad E = ES + EI + E_f.$$

$$\frac{EI + E_f}{E} = 1 - a \qquad EI = (1-a)E - E_f$$

$$= (1-a)E - K_S aE$$

$$S - aE$$

$$I' = \underbrace{\left[(S' - aE_S')\left(\frac{1-a}{a}\right) - 1\right]}_{\text{Free}} + \underbrace{\left[1 - a\left(1 + \frac{1}{S' - aE_S'}\right)\right]}_{\text{Contrif}\ (= (EI)')} E_I'$$

If $I_f \simeq I$ $\qquad I' = (S' - aE_S')\left(\frac{1-a}{a}\right) - 1$ $\qquad$ GAIBS

or if $EI \simeq I$ $\qquad I' = \left[1 - a\left(1 + \frac{1}{S' - aE_S'}\right)\right] E_I'$

Hefrids $\frac{I'}{S'} = \frac{1-a}{a}$ i.e. for $a = 1/2$, $\frac{I}{S} = \frac{K_I}{K_S}$.

$$\frac{1-a}{I'} = \frac{a}{S'}$$

$$\frac{\frac{EI}{E}}{I'} = \frac{\frac{ES}{E}}{S'} \quad \text{and} \quad \frac{\frac{EI}{I}}{\frac{ES}{S}} = \frac{K_S}{K_I}.$$

Hoder, F. + Akano, R., Z. Immunf. 85:423- (1935)

Foley, G.E. and Schwachman, H. (1950) J. ~~Exptl.~~ Clin. Micr. 4: 141-149 Some observations on a streptomycin-dependent strain of Staphylococcus aureus. RR

Bawden, F.C., Kassanis, B., and Nixon, H.L. (1950) The mechanical transmission and some properties of RR potato paracrinkle virus. JGM 4: 210-219.

Fleming, A., Voureka, A., Kramer, IRH, + Hughes, V.H. (1950) The morphology and motility of Proteus vulgaris and other organisms cultured in the presence of penicillin. JGM 4: 257-269.

RR

Eriksen, K.R. (1949) Studies on the mode of origin of penicillin resistant staphylococci. Acta path 26: 269-279. From Univ Inst General Path. Copenhagen.

Broth & various P inoculated with varying amounts (10^{-1} to 10^{-6}) of a 24 hr. broth culture. Later plated loopful (ca. .002 ml) on ~~ag~~ agar. With large inocula, secondary growth is found up to 1/4 OU/ml; with initial bacteria of 10^{-3}, no sec. gr, but eventually comes up. "Demerec is not correct and that the resistant bacteria appear only after contact with penicillin for some ~~time~~ length of time." Reasoning ?? Noted that with ca 1/8 OU/ml and perhaps 10^{-5} ml, any secondary growth was delayed 24-48 hours. In 6 ~~cups~~ tubes, it appeared only after 6 days. "In these cases where the secondary growth appears at such a late juncture, presently it can be taken ~~that~~ 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 testing for stability.)
Found variance in mutant numbers only in 3 ml cultures, ~~not~~ in 15 ml cultures.

Treatment of recombination in texts since 1948

1950 Clifton Introduction to the bacteria pp 73-75

"Possibilities of recombination of genes by other than sexual mechanisms may exist, and our original definition of bacteria as "apparently sexless" organisms is still valid." Fair statement of expts. T+L 1947

1949 Burrows et al.
extensive ~~treatment~~ discussion of genetical analysis of bacteria p. 184 passing reference T+L 1947.

Stocker, B.A.D. (1949) Measurements of rate of mutation of flagellar antigenic phase in [illegible]. J. Hyg. 47: 398–413.

[Dept. of Bacteriology [illegible] [illegible] of Hygiene & Tropical Medicine.]

[illegible] [illegible] [illegible], [illegible] [illegible] [illegible] [illegible] [illegible].
Occasional [illegible] [illegible] [illegible]. Some non-motile [illegible] (< 2%) were found. Some populations at mutational equilibrium were noted.
Rate of 3.5×10^{-4} / generation found by [illegible]. Noted [illegible] phenomenon. p. 410

KR

Klieneberger-Nobel, E. (1949) [illegible] of the [illegible] of [illegible]. J. [illegible]. 11. [illegible].

L-forms filterable [illegible], filterability of [illegible] [illegible] [illegible] possible.

Stern, C. 1936 Somatic crossing over and segregation in Drosophila melanogaster
Genetics 21: 625-730.

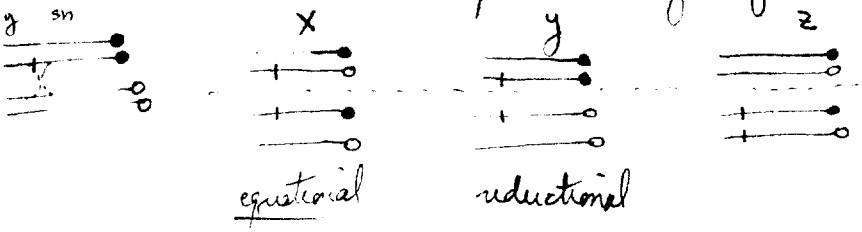
Minute flies (M/m) show m spots. Originally interpreted as elimination of M carrying (deficient chromosome). By use of ⊙ translocations, it was shown that the M phenotype (not merely deficiency, covered by duplications) was necessary for spotting. Bobbed (bb) spots not found: interpreted as partial elimination.

Autosomal M also cause X-mosaics (~~sn~~ sn^3 (singed)
However, the Bld ~~=~~ Minute causes X-spots, but not III-spots!!!!
Effect of autosomal Mw on $Notch^8$ Df. was studied:

N^8/y ♀ × sn^3; Mw/+ ♂ Among females:

N^8/sn^3 9/280 y/sn^3 15/381 No difference.
↓ sn seta (elim? N^8) ↓ 2 y spots
2 sn^3 spots
11 twin spots (y)(sn) suggesting segregation of X-chromosome.
No y sn spots. ∴ 2-strand and no reduction.

y sn/++ flies → 110 y sn 43 y 7 sn ~~spots~~. y and sn imply somatic crossing-over as well as segregation. But no y-sn twin spots were found, ruling out two-strand crossing over. Complete reduction is ruled out by absence of y sn - sn - y - (+) triple spots.



Segregation patterns.

Region of crossing-over varies with spot size (developmental stage). Crossing-over to the right of sn in y sn spots supported by expts. with ⊙ translocations.
Segregation is probably nearly always equational.

bb fails to show segregation in +/bb flies. Assumption of phenotypic masking seemed unlikely. ∴ Crossing-over to the right of bb considered very rare.

Determined X-ploidy of spots by color of 5-6th abd. segments. Most spots in females were XX by color.

Autosomal mosaics

Under influence of autosomal M.

Notes on genetics of epilepsy 3/3/55

Secondary Sources:

1. Sorsby "Clinical Genetics"; pp/ 337-40;313-15
2. Kallmann and Sander 1947. in Hoch & Knight, "Epilepsy". Chap. 3
3. Neel 1947 Medicine 26:115. at 123-125

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.

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

gen. pop.	childr.	sibs	neph&nieces	dizyg. motwins	monozyg cotwins
.3	6.3	4	1.2	3.1	66.6

concordance in twins:

	diz	monoz
idipath.	4.3	86.3
symptoma.	0	12.5

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
general pop	.10
epileptics	.9
par and sibs	.6

in twins, 85% whow concordance of encephalo. of monozyg; 5% if dizyg.

1. 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 patents of propositi (Lennox) suggests recessive factors also.

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

CC: Dr. Javid

1957

1/21/54

Conjugation in yeast.

Fowell 1951 emphasizes dicaryon: mating of cells gives  from which either haploid or diploid buds may be generated. Tools care to remove prefusion buds.
or dicaryotic (i.e. → + and − haploid)

Paired 250+/− cells; 30 zygotes formed. 50% Zyg. → only haploid. Often zygotes → only 2n. "An investigation of spore fusion revealed that nuclear fusion apparently always occurs in zyg formed by this proc." Renaud 1946 also suggest dic. Also discussed by Däumann 16; (Buchheimond 25. Bot Rev 1940 6:1.) Comp. Morph. of Fungi 1928.

Wingé & Roberts 1948. Unsuccessful crossing: spores may give haploid cells "before fusing"

W & L figure  spores. But W'35 also shows substantially complete copulation and diploid buds. ∴ some variation.

But note analogy of Fowell's dic. & oogen formation.

Kawada, H. Zbl. Bakt. I 118: 304-16 (1930)

S. para B + G+ soil bact → frequent antigenic variations in Salmonella → enteritidis; breslau.

JPB 35:851

19 Burnet 1932 Lysogen. Delaweiten 69. J Bact 34:285
[Andrewes 7. Pr. Roy Soc Med 33 Dec 39]
5 Krueger Physiol Rev 16:129
18 Burnet Aust J Exp BM 6:277

~~Delbruck JGP 23:443 Adsorption rate.~~
22,365 — Expt. lysis -> loss of virus.
temperature same as for cell division

Receptors: 63 - Levine + Frisch JEM 59:213 ✓

See Burnet 9. AJEBM 15:227

J Immun 46: 281.

(leave out glucose in virus media)

Tryptose 2% glucose .1% NaCl 1% pH 7 [.6% agar smaller.]

AD Hershey. 1/8

,5 ml phage

2 ml 12-24 h. bact. 10^8/ml

3 mins later, .5 ml mixture + 3.5 ml .7% agar

pour on plate:

harden agar!!!

Frendzel, J. + Z. Szymanowski, CRSB 117:543–546 (1934)

Recherches sur la Paragglutination: Différenciation des antigènes H et O.

They had shown that P. exhibits a different serological specificity from the "agglutinine complete de Schütze". But the R strains do contain an antigen related to the parenteral strains. ~~His~~ paragglutinable strains are homogeneous + repeated resolution indicates that the modification is heritable. Only some E. coli are capable of paragg.

coli-typhoid paragglutination:

The P. coli absorb H-agglutinins for anti-typhoid serum. The original coli does not. anti-H was removed by absorption on Stanley. There was little further agglutinins absorption. However, there was still considerable agglut. of "coli". ∴ Paragl. coli has all H antigens, and a fraction of the O of typhi. anti-P coli serum has a low titer on heated typhi. Typhi phages do not lyse (P) coli.

Z. Balat (I, 121:448–451 (1931) Paragglutination des Bact. Bang mit Typhusserum. —

2 strains - ctd.

Using para A and ~~the~~ typhi, (P) is also obtained with cross-reactivity, but very little ← para B.

Could not transform staphs. Related paragglutination to the pn. transformations

Smith WE, J Bact 47:417-418 (1944)

Wahlen + Almadin JID 65:147-55 (1975)

Appleby, J. C. J Bact 38:641-51 (1939) Cytology and methods of reproduction of two cocci and the possible relation of these organisms to a spore forming rod.

Cocci appeared in a culture of the bacillus.

Agr Bact Dept, Univ Reading England

Sex in Bacteria. Literature:

J. Bact 50

Nuclei - El. M. cr. (R)

Baylor, M.B., M.O. Appleman, O.H. Sears + G.L. Clark, J. Bact 50: 249-56 (1945)
Chem. + Agronomy Illinois

Some morphological characteristics of nodule bact as shown by the electron microscope II. [See Soil Sci Soc Am. Pr. 7: 269-71 (1942)

4-5 granules/cell untreated + ē .02% $NaHCO_3$ 2¼ hrs. Attempts at staining e.g. M saline left mottled cells. (several transparencies; corresponding to nuclei? After 15 min. $NaHCO_3$ saline did not remove granules. acetone removed granules. also HNO_3, HCl

Knaysi, G. J Bact 49: 473- 1945. A study of ... factors .. in production of Polysaccharides.

low p 11 n.g.

Spores are not formed until sugar + hydrolytic products are used up + also the nitrogenous comp.

"healthy cells, facing starvation, sporulate with ..."

See: Reed HC J Bact 35: 261

Kuaysi, G + S Mudd J Bact 45: 347-57 (1943)
Emull.

The internal structure of certain bacteria.

Apparent DR nucleic ac. material in granular form in S. Stolonifera.

Most dividing cells exist in 4's and 8's.

R.R. Mellon, J. Bact. 10:481-501 (1925) Studies in Microbic Heredity I Observations on a primitive form of sexuality (zygospore formation) in the colon-typhoid group.

B. coli (Nx) In patient bringing urine origin appeared as filamentous form & "many very large cocci like forms were encountered developing from the filaments."

In vitro, peptone-veal-5% NaCl broth + 1% Na_2 glycerophosphate at pH 6.8 autoclaved; ~~ppt redissolved~~ ~~filtered~~ + reautoclaved. ppt redissolved in serum. Single cell isolate inoculated into broth 37° 72h. Then at R.T. streaked out on Endo. (with broth-glyc base p. 8) & incubated at 37° 18-24 hours, periphery of colonies wide fungoid & zygospore formation.

"no attempt has been made to study the fate of these spore like bodies".

Similar forms are found in small cells.

No convincing evidence of origin from > 1 cell.

Mystic on sexuality + variability. Does not understand bases of relationship.

Assumes that cell-fusion has taken place. Criticizes Klinquist.

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

10:579-88 (1925)