terior, there is much data[7] which suggests that von Hann's concept holds to the extent that the functional presence of the pars anterior normally intensifies the severity of the d.i. resulting from a given deficit in the antidiuretic principle. However, White and Heinbecker[4c] report that in certain instances hypophysectomy did not reduce the magnitude of the d.i. induced by hypothalamic lesions. This may indicate that the above relationship does not hold when the animal is entirely antidiuretic-free.

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Are the Groups of Beta Hemolytic Streptococci Inter-Transformable?

RALPH R. MELLON AND PHILIP B. HADLEY.

From the Western Pennsylvania Hospital Institute of Pathology, Pittsburgh, Penn.

It has been reported[1] that fibrinolysis by Group A hemolytic streptococci was a character of diminishing intensity as one proceeded from the S-culture phase through the R and finally to non-hemolytic colonies showing an exclusively diphtheroid cell-morphology. Moreover, these feebly fibrinolytic diphtheroids possessed an unusual lability, as indicated by their occasional reversion to the identical S-colony type of hemolytic streptococcus (Stoddard strain) from which they had originally dissociated. Such a "reverted" diphtheroid culture agglutinated significantly in the antistreptococcal S-serum and absorbed from this serum the antibodies specific for it.

Subsequently the cultures of this diphtheroid, started from a single cell, have undergone further changes that appear to determine the Lancefield group into which the streptococcus redissociated from this diphtheroid shall fall. These changes have occurred under the following conditions. When this strain was kept for several months in our desiccated stocks and then transferred to semi-solid blood agar at room temperature, 4 transfers being made at monthly intervals, it lost practically all of its fibrinolytic ability. The same strain

[7] (a) Barnes, B. O., Regan, J. F., and Bueno, F. G., *Am. J. Physiol.*, 1933, **105**, 559; (b) Biasotti, A., *Compt. rend. Soc. de biol.*, 1934, **115**, 329; (c) Mahoney, W., and Sheeban, D., *Am. J. Physiol.*, 1933, **112**, 250; and (d) Keller, A. D., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 787; 1938, **38**, 31; and *Am. J. Physiol.*, 1938, **123**, 111.

[1] Mellon, R. R., and Cooper, F. B., PROC. SOC. EXP. BIOL. AND MED., 1935, **33**, 451.

kept under the same conditions, except that it had been stocked on blood-agar slants in the cold room instead of being desiccated, retained its original fibrinolytic capacity. Inasmuch as Group C strains are usually non-fibrinolytic, the loss of this character might be viewed as the precursor of additional changes that resulted eventually in a strain having the serological reactions of Group C.

The non-fibrinolytic diphtheroid was then transferred to ordinary blood-agar slants which were sealed and put in the cold room (10°C). Transfers were made to the same medium under the same conditions at monthly intervals. In a future paper it will be shown that under these conditions after 5 transfers this strain underwent changes that resulted in its reversion to a hemolytic streptococcus (NF), the characters of which were obviously different from those of the streptococcus (F) that reverted under identical conditions from the fibrinolytic diphtheroid. That is to say, the F streptococcus had a 4-plus hemolysis of *beta* type in contrast with a 1-plus of *alpha* prime type for the NF strain. Moreover, the colonies were mucoid and smooth-mucoid and possessed high virulence for mice in contrast to a slight or negligible virulence of the original Group A strain (Stoddard). Fermentatively the strain had become trehalose-negative and sorbitol-positive. Thus according to this criterion the strain had assumed the status of an intermediate between Groups A and C. Antigenically it produced in animals precipitating antibodies for Group A, and was precipitated by known Group A sera. No cross-reactions were observed with Group C sera at this time.

Some 6 months later the progressive trend of this intermediate strain toward Group C became virtually complete in the sense that it acquired some antigenic characters of this group at the same time that it lost those of Group A. This change came about spontaneously while the strain was being kept on sealed blood-agar slants in the cold room. One reversion from the highly virulent intermediate C to the non-virulent Group A has been observed. This occurred *in vivo* in 2 mice that had been injected intraäbdominally with culture dilutions of 10^{-6} cc, which were lethal for them. In the peritoneal exudate of both the mice 2 types of colonies were obtained in about equal numbers: (a) the mucoid colony of the inoculated strain; and (b) the small SR type of colony which proved to be indistinguishable culturally and serologically from the original, slightly virulent, Group A strain.

Employing another Group A strain it has been possible to anticipate the possibility for transformations between Group A and Group C strains. This consists of broadening the range of group-antigens

in a type-specific Group A strain so that its antigen agglutinates not only in several Group A serums, but also to a lesser degree in sera of Group C. This change has occurred as the result of transforming cultures of the S-phase of the type-specific culture to its corresponding R-phase and subsequently reconverting the latter to an S phase. It would thus appear that the R-phase culture, like the diphtheroid, may be the seat for reorganizations of antigenic constitution.

Both may be viewed as having something in common in respect of their proclivity for reorganization of biologic characters. The two mechanisms appear to differ in degree however, since passing through the diphtheroid state may lead to a frank recombination of characters that finds expression in a group-transformation. Further evidence for similarity between the diphtheroid and the R-culture-phase will be given fuller consideration in a subsequent publication.

The reorganizational significance of special cell-types and culture-phases was first pointed out in 1920,[2] and the special bearing of such variability phenomena on the origin of group and specific agglutinogens was indicated in 1926.[3] In 1931,[4] the same variation principles found application to the much discussed filtrable forms of bacteria—the G types, whose filtrability and marked variability have recently been confirmed by Haddow.[5]

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A Pressor Substance is Not Present in the Perfusate of Ischemic Kidneys.

G. A. BOYLSTON, E. G. MCEWEN AND A. C. IVY.

From the Department of Physiology and Pharmacology, Northwestern University Medical School, Chicago.

The physiological mechanism by which increased arterial pressure is produced and maintained following the establishment of renal ischemia is unknown. There is, however, much evidence indicating that the kidney plays a specific rôle. There are several studies which seem to eliminate the possibility that reflex constriction of the peripheral vascular bed is responsible for the increased pressure observed. Since the rise in pressure may occur in the presence of one

2 Mellon, R. R., *J. Med. Res.*, 1920, **42**, 61.

3 Mellon, R. R., and Jost, E. L., *J. Immun.*, 1926, **11**, 139.

4 Hadley, P. B., Delves, E., and Klimek, J., *J. Inf. Dis.*, 1931, **48**, 1.

5 Haddow, A., *J. Inf. Dis.*, 1938, **63**, 129.