of the consequences of changes in the biocatalysts themselves was unknown or insufficiently appreciated. Knowledge gained from the industrial use of catalysts, which demonstrated the enormous effects that may result from the addition even of traces of specific promotor substances to catalysts, led the writer to the view[2] that an analogous situation must exist with biocatalysts.

In applying this "promotor" notion to biology, the term "modification" is preferable, since here human choice does not control. We must also recognize that a limiting case of catalyst modification arises where a new active catalyst area is brought into being by union of appropriate substructures, for example, by the fixation of a prosthetic group by a carrier.

There are here epitomized three basic biological events which seem to be clarified by the concept of catalyst modification:

(1) *Differentiation and the Orderly Course of Life:* At definite stages in the development of a zygote, specific molecules or other particles, carried in the zygote cytoplasm or formed by genic or other biocatalysts, apparently modify existing catalysts so that their chemical output is changed in nature or in relative proportions. The fixation of these modifiers may follow, for example, ionic changes which must develop as a consequence of differential diffusion when the blastula mass increases in size, and must become more marked as it assumes the gastrula form. Apart from the heavy responsibilities carried by the genes, we see here a factor that may modify both genic and non-genic catalysts in a specific and orderly manner; and the tiny amounts of the original or "templet" modifier particles could readily be carried in the zygote cytoplasm.

The fact that fully differentiated cells can continue to duplicate themselves, as such, even in tissue culture, proves that a mechanism exists for the heritable continuance of modified catalysts or of catalyst areas produced de novo. This fact has been successfully demonstrated by the work of geneticists with the self-duplicating (autocatalytic) genes, and seems probable with some other biocatalysts.

(2) *Evolution:* Any heritable catalyst change may have as a consequence changes in the biont (plant or animal) which make it structurally and/or functionally different from its progenitors. This would give a physico-chemical basis for evolution, since natural selection, operating on these differences, could result in the establishment of any new species thus originated.

(3) *Cancer:* Here there seems to be a heritable change in cellular catalysts, due to modification or following the introduction of a virus (Rous), with the consequence that the cells continuously duplicate themselves and invade healthy tissue. Non-invasive growth would constitute a benign tumor. Cancers may result from radiation (known also to produce heritable chromosomal or genic changes); or from the introduction of ultrafiltrable particles or viruses, or of definite chemical substances, for example, methylcholanthrene, or 3,4-benzpyrene; or from less defined causes, such as trauma and burns (kangri cancer). Many cancer cells have been grown as such by transplantation or in tissue culture. The main difference between cancerous and other heritable catalyst changes seems, therefore, to lie in the diagnostic consequences of the catalyst change. Other diseases may be considered from this point of view; for it must be stressed that catalyst modification may in some cases be reversible and is not necessarily heritable.

A more extended consideration of these and allied questions will be given in a paper to appear in Volume V of the international series on colloid chemistry, now in preparation.[3]

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Alexander and C. B. Bridges, in Vol. II, "Colloid Chemistry, Theoretical and Applied," N. Y., 1928. Alwin Mittasch, "Ueber katalytische Verurschachen in biologischen Geschehen" (1935); "Ueber Katalyse und Katalysatoren in Chemie und Biologie" (1936); "Katalyse und Determinisimus" (1938), all publications by Julius Springer, Berlin.

[2] J. Alexander, *Protoplasma*, 14: 296–306, 1931; "Colloid Chemistry," 4th ed. D. Van Nostrand Company, 1937; *Biodynamica*, December, 1939.

[3] To appear in 1943, Reinhold Publishing Company, N. Y.

THE INFLUENCE OF SEX ON NUTRITIONAL ACHROMOTRICHIA IN MICE

A DIFFERENCE in the response between male and female mice to nutritional achromotrichia was observed with 39 male and 55 female mice of the C-57 strain kept on a diet similar to that employed by Unna and coworkers.[1] The following observations were made.

On the 30th day of the experiment 16 of the 29 or 55 per cent. of the surviving males showed various degrees of graying, while but 3 out of 50 or 6 per cent. of the living females grayed and this was not so marked. Within 75 days all but one of the males had become gray, while 13 per cent. of the surviving females only revealed slight changes.

Three additional groups each of 8 male and 7 female C-57 mice were fed the same basic diet, but each mouse of the first group also received 0.75 mg para-aminobenzoic acid daily and each of the second group 100 gamma calcium pantothenate daily and

[1] K. Unna, G. V. Richards and W. L. Sampson, *Jour. Nutrit.*, 22: 553, 1941.

each in the third group the same daily quantities of both of the above compounds. Achromotrichia occurred in these three groups as well as it did in the larger basic experiment with the same difference in the reaction of males and females.

These observations imply that the achromotrichia produced in C-57 mice by certain dietary deficiencies may depend also on hormonal factors.

The report of Forbes[2] that a pellet containing estrogenic substances implanted subcutaneously produced local pigmentation of the fur in albino rats, while testosterone diproprionate failed to do so, is of interest in connection with these results.

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CONGENITAL AND ACQUIRED ANOMALIES OF COLOR VISION

APROPOS of recent criticism in these columns of the ambiguous concept of "color blindness,"[1] subsequently endorsed and extended by Loken and Dunlap,[2] I find after careful consideration nothing in common between the assumptions and deductions of the latter and my own.[3]

Controlled experimentation over a period of months and years will be necessary before the existence of congenital as distinct from acquired types of color anomaly can be contested.[4] Such experimentation should be carried on by responsible trained workers, combining medical with psychological techniques. Adequate insight into the uses and shortcomings of the various color tests available, based on five or more years of experience with a variety of cases, is indispensable. Ability to distinguish minor from major anomalies, a knowledge of the relations of day and night vision, of the effects on the color sense of fatigue, of excessive use of nicotine, alcohol and other drugs, and of various infections are also essential.

Promotion of the use of drugs or vitamins to enable an applicant for aviation or naval service to "pass a test," where knowledge of the permanence of the "cure" is not yet available in the opinion of the writer is little better than coaching students to cheat in a qualifying examination. In the present emergency, when perfection of vision is vital in submarine, aviation and naval branches, and when one of our opponents is undoubtedly possessed of unusual visual equipment, the ill effects of such a line of action are incalculable.

There is nothing in common between the Loken-Dunlap position and my own.[5]

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SPROUTING OF SUMAC IN DRY STORAGE

SPROUTING of adventitious buds in logs or twigs of woody species freshly cut and left in contact with moist earth is said to be common in tropical regions and not rare in temperate climates. The remarkable case of a sprout on a mulberry log in England after six years of dry storage has been recorded by the late Sir A. W. Hill,[1] but in that instance the sprout appeared after the log had been used as a prop with one end in contact with moist earth. The scriptural record[2] of the sprouting of the rod of Aaron has, of course, been familiar to many generations of men, but comparable cases in North America are not so well known. A recent development of sprouts on a stored log of staghorn sumac (*Rhus typhina* L.) although a slower proceeding than that of the biblical account is so striking as to seem noteworthy:

A sumac tree 5 inches in diameter at the base and reaching over 20 feet in height growing in the writer's yard (elevation, 320 feet) in Arlington County, Va., was cut down on September 1, 1941, and the log 12 feet long stored for curing as lumber in a dry unheated shed, where it had no direct contact with moisture for the next eleven weeks. After two weeks a number of buds and small sprouts were observed, chiefly on the basal half of the log. Four weeks later all except two of the sprouts had aborted. The larger sprout, then reaching eleven inches in length, originated at a point fifteen inches from the base of the log; the other, but 3 inches long, was at a point five feet from the base. After five more weeks, *i.e.*, eleven weeks after the log had been cut, but one sprout, the lower one, remained, it having by that time reached sixteen inches in length with a maximum diameter of seven sixteenths of an inch. By this time, November 17, some of the leaves had begun to wither at the tips, but whether from dryness or from the effect of cold was not readily determinable.

It does not seem profitable here to discuss at length the means whereby the sumac stored sufficient water for the eleven weeks' growth and transpiration. But it may be pertinent to point out that the much split

2 T. R. Forbes, *Endocrinology*, 30: 465, 1942.

1 E. Murray, SCIENCE, 96: 2484, 133–5, August 7, 1942.

2 K. Dunlap and R. D. Loken, SCIENCE, 96: 2489, 251–2, September 11, 1942.

3 *Idem.*, SCIENCE, 95: 2474, 554 *ff.*, May 29, 1942.

4 See Köllner, "Die Störungen des Farbensinnes," 1912; or other ophthalmological texts.

5 E. Murray, *Psychol. Bull.*, 39: 165–72, March, 1942.

1 A. W. Hill, *Ann. Bot.*, 39: 210–211, ill., 1925.

2 Numbers, Chapter 17.