TECHNICAL PAPERS

Pentavalent Manganese[1]

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In fused alkaline melts, the lower oxides of manganese react with oxygen until an oxygen-to-manganese ratio of about 2.5 is established (*5*). For aqueous media, reliable evidence pointing to the existence of pentavalent manganese has only recently been reported (*4*). The present paper will show that such a valence state can be detected polarographically in strongly alkaline solutions.

A solution containing 1.00×10^{-3} M potassium permanganate and 0.10 M sodium hydroxide was deaerated with nitrogen and then polarographed, using a stationary platinum electrode (*6*) and an outside saturated calomel electrode (S.C.E.). The resulting curve is shown in Fig. 1.

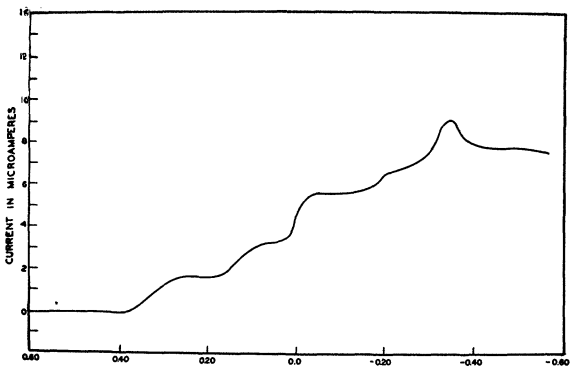

FIG. 1.

The first step of the reduction ($E_{\frac{1}{2}} = +0.33$) has a diffusion current corresponding to a one-electron change (for the particular electrode which was employed) and a formal oxidation potential of -0.58 v, versus the normal hydrogen electrode. Since the permanganate reaction has a normal potential of -0.54 v (*2*), the first wave can definitely be assigned to this reaction. The second ($E_{\frac{1}{2}} = +0.13$ v) and third steps ($E_{\frac{1}{2}} = +0.01$ v) also have diffusion currents corresponding to one-electron changes. Hence the reactions taking place must be $Mn^{VI} \rightarrow {}^{V}$ and $Mn^{V} \rightarrow {}^{IV}$. The fourth (and last) step beginning close to -0.2 v must be due to the reduction of Mn^{IV}. The irregularity of the step can easily be explained by the fact that the electrode is covered with a visible layer of precipitated manganese dioxide.

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There is indirect evidence that the lifetime of pentavalent manganese in 0.1 M hydroxide is not more than a few minutes. The polarographic half-wave potentials and and diffusion currents appeared to be independent of the rate at which the motor-driven slide-wire changed the voltage. On the other hand, a current-voltage curve obtained by a manual method (*1*) resulted in a single broad wave. It was not surprising, therefore, that an attempt to produce pentavalent manganese by electrolysis of manganate at a suitable potential (*3*) produced a large amount of manganese dioxide. The solution had a bluish color (similar to chromous sulfate) which agrees with the work of Lux (*4*).

Preliminary studies have also been made in other concentrations of sodium hydroxide. In a 1.0 M solution, the polarogram is very similar to the one described for a 0.10 M solution; but in a 0.010 M solution, the manganate is reduced directly to manganese dioxide in a single two-electron step. A more complete study of changes in half-wave potentials with the concentration of hydroxide will be necessary before the reactions of the Mn^{V} ion can be described accurately. The results of such a study will help in predicting the behavior of technetium ions.

References

1. LAITINEN, H. A., and KOLTHOFF, I. M. *J. phys. Chem.*, 1941, **45**, 1061.
2. LATIMER, W. M. *Oxidation potentials.* New York: Prentice-Hall, 1938. P. 223.
3. LINGANE, J. J. *Ind. eng. Chem.* (Anal. Ed.), 1944, **16**, 147.
4. LUX, H. *Z. Naturforsch.*, 1946, **1**, 281.
5. MELLOR, J. W. *A comprehensive treatise on inorganic and theoretical chemistry.* New York: Longmans, Green, 1932. P. 284.
6. ROGERS, L. B., MILLER, H. H., GOODRICH, R. B., and STEHNEY, A. F. Paper presented before the 113th meeting of the American Chemical Society, Chicago, April 1948.

Note on the Genetics of Hypercholesterolemia

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An important aspect of the expanding interest in human genetics is the detection of carriers of hereditary diseases, both in the sense of normal heterozygotes of recessive defects, as in some forms of epilepsy (*4*), and in the sense of those individuals who exhibit some predisposing condition without showing the characteristic symptoms of the disease, e.g. hyperuricemia in gout (*7*). It is elementary in all cases to determine on the basis of numerical tests the mode of inheritance, the gene frequency, penetrates, and so on. Boas, *et al.* (*1*) have

recently published data on hypercholesterolemia, a disturbance of cholesterol metabolism which "may be the common denominator in most patients who have coronary artery disease." At Boas' suggestion and with his kind permission, the following analysis of their data was undertaken. These authors studied the families of patients chosen because they had proved coronary artery disease, the symptoms of which began before the age of 50. Fifty families yielded 37 families which could be used for a numerical test of the mode of inheritance, because they included more than one child in each family and at least one sibling per family was affected with hypercholesterolemia. Study of these 37 families revealed 11 families of 2 children, with 14 affected; 10 families of 3 children, with 22 affected; 10 families of 4 children, with 19 affected; 2 families of 5 children, with 6 affected; and 4 families of 6 children, with 6 affected; giving a total of 67 affected out of 126 children. When the number expected to be affected for these 37 families is calculated on the basis of a 1: 1 ratio, using the corrective factors for small family size given by Hogben (*3*), for hereditary characters with complete penetrance, the result is 70.5 expected affected, with a standard deviation of 4.8.

This is clearly an excellent fit to a 1: 1 Mendelian ratio, which is obtained in the case of a dominant trait when one parent is heterozygous for a dominant defective gene and the other parent is homozygous for the recessive normal allele, or which is obtained in the case of a recessive trait when one parent is homozygous for the recessive defective allele and the other is heterozygous normal. Since the data do not include the parents, a decision can be reached only tentatively as to which one of these two possibilities is a priori more probable. Data somewhat similar to those of Boas, *et al.* have been obtained and analyzed by us for the inheritance of Heberden's nodes (*6*) and hyperuricemia (*8*), in comparison with similar cases in the literature which were shown more conclusively to be due to autosomal dominance. We are thus led to the conclusion that hypercholesterolemia is an autosomal dominant trait with complete or nearly complete penetrance. As in research on many other hereditary conditions, the gene frequency, linkage relations with other genes, and confirmation of its mode of inheritance as a dominant with complete penetrance require further investigation.

Boas, *et al.* arbitrarily selected a concentration of 300 mg/100 ml as indicative of hypercholesterolemia, purposely choosing a high level to eliminate the influence of minor elevations. This happy choice has been entirely justified by the data of Peters and Man (*5*), who, in a study of 174 determinations in normal individuals, found an average serum cholesterol of 194.1 mg $\pm$ 35.6 mg/100 ml. Three times this standard deviation above the mean gives a limit of normality of 300.9 mg/100 ml. Such a figure, that is, would allow only 1 or 2 persons in 1,000 to be above 300 mg and still be classified as normal in cholesterol concentration in the blood serum.

Certain people with hypercholesterolemia may be considered to exhibit a genetic trait characterized by this chemical abnormality. It is not at all certain that the genetic form of this abnormality can be identified by one chemical determination without regard to age, sex, diet, or other conditions of metabolism which may elevate the level temporarily—any more than one blood sugar determination identifies unqualifiedly a diabetic, or one uric acid determination identifies a gouty individual. Further investigation may reveal hypercholesterolemia as an inborn error of metabolism (*2*) similar to gout, albinism, cystinuria, or pentosuria, which are definitely dependent upon genetic factors. Constitutional hypercholesterolemia may offer an organic explanation for some cases of familial angina pectoris and coronary artery disease, as well as familial xanthelasma and xanthomatosis.

References

1. Boas, E. P., Parets, A. D., and Adlersberg, David. *Amer. Heart J.*, 1948, **35**, 611.
2. Garrod, A. E. *The inborn factors in disease.* Oxford: Clarendon Press, 1931.
3. Hogben, L. *Nature and nurture.* New York: W. W. Norton, 1933.
4. Lennox, W. G., Gibbs, E. L., and Gibbs, F. A. *Arch. Neurol. Psychiat.*, 1940, **44**, 1155.
5. Peters, John P., and Man, E. B. *J. clin. Invest.*, 1943. **22**, 707.
6. Stecher, R. M., and Hersh, A. H. *J. clin. Invest.*, 1941, **23**, 699.
7. Stecher, R. M., and Hersh, A. H. *Genetics*, 1945, **30**, 24.
8. Stecher, R. M., Hersh, A. H., and Solomon, Walter M. *Ann. int. Med.*, in press.

The Flowering and Seed-Setting of Sweet Potatoes in Puerto Rico

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The work of Hartman (*3*) reporting complete failure of flower formation in Jersey-type sweet potatoes and the more recent work of Mikell, Miller, and Edmond (*4*) have prompted us to report certain observations on the flowering of sweet potatoes in Puerto Rico.

Thirteen of 16 varieties grown in field plots at the Federal Experiment Station flowered during the fall and winter of 1947–48. These were grown from sprouts, set in the field in July. Plants were trained up on 6′ chicken-wire trellises and were kept thinned by constant pruning, following the methods of Miller (*5*). It was not found necessary to girdle the plants.

Among the varieties which flowered was one of the difficult-to-flower Jersey types, Orange Little Stem. Two out of three plants of this variety included in the trials began to flower early in December and continued through the middle of January.[2] These flowers opened at the same hour and were very similar in appearance to the flowers of other varieties. Microscopic examination of

[1] Administered by the Office of Experiment Stations, Agricultural Research Administration, U. S. Department of Agriculture.

[2] It may be reported that this variety again started flowering in August 1948.