ative results. It would appear then that the demethoxylation of the lignin takes place in the stomach of the animal and that this is not brought about by bacteria, but rather by some other agent, possibly in the nature of an enzyme which is present in the gastric juice of the animal.

In connection with our animal experiments, we observed that when lignin was fed in larger doses than 2.0 gm. per kilo weight, toxic symptoms were developed. In 2 instances we found increased amounts of non-protein nitrogen in the blood, pointing to an impaired renal function.

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The Possible Significance of d-Xyloketose (Urine Pentose) in Normal Metabolism.

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The author[1] has pointed out that pentosuria is hardly likely to be a disorder of carbohydrate metabolism. The amount excreted is always small and is, apparently, unaffected by the administration of large quantities of glucose or galactose. Since the excretion of pentose is diminished in fasting,[2] it would seem that the most likely source is protein. The pentose may be the result of an abnormal metabolic sequence or an intermediate in normal metabolism, which is not further oxidized.

The experiment reported in this paper was designed to throw some light upon this question. It is intended to repeat and to extend the work in various directions; but circumstances preclude the immediate continuance of the work.

The *p*-bromphenylhydrazone of the pentose was prepared by a modification of the method employed by Levene and LaForge.[3] It melted at 127-8° and decomposed at 165°. In a 1% solution in alcohol, the rotation was −1.87° shortly after preparation and +2.43° after 24 hours. 7.20 gm. of this compound were decomposed with benzaldehyde, as described by Levene and La Forge. The filtrate from the benzaldehyde *p*-bromphenylhydrazone was ex-

[1] Greenwald, I. In Endocrinology and Metabolism, ed. Barker, Hoskins and Mosenthal, New York and London, 1922, iv, 289.

[2] Klercker, K. O., *Deutsches Arch. f. klin. Med.*, 1912, cviii, 277.

[3] Levene, P. A., and LaForge, F. B., *J. Biol. Chem.*, 1914, xviii, 319.

tracted with ether and then evaporated, *in vacuo,* to 100 cc. In a 1 dm. tube, the rotation was 1.18°. Since the calculated yield of pentose was 3.39 gm, $[\alpha]_D^{22} = 34.8°$ The glucose equivalent of 1 gm. of pentose. calculated from the *p*-bromphenylhydrazone was 1.25 gm. by Sumner's method,[5] 1.45 gm. by the NaOH-picrate-acetone method[6] and 1.18 gm. by the Na_2CO_3-picrate method.[7]

This solution was injected into a young female dog, weighing 5.6 k., kept on a diet of 100 gm. beef heart, 16 gm. cracker meal, 16 cc. maize oil, 6 gm. bone-ash and 200 cc. water. There were 4 injections of 20 cc. and a fifth one of 10 cc., at intervals of approximately 2 hours. Calculated from the amount of hydrazone taken, the total amount of pentose injected was 3.05 gm. By Sumner's method, this showed a reducing action equivalent to that of 3.80 gm. glucose. The increase in the sugar content of the urine was equivalent to about 6% of the amount injected. A few days later, the animal received a similar series of injections of *l*-xylose. About 60% appeared in the urine. Another control experiment with glucose, gave, as was expected, no increase in the excretion of sugar.

d-Xyloketose is, therefore, not a substance that cannot be further metabolized by the organism. Whether or not it is a product of normal metabolism and what its action in insulin hypoglycemia, phlorhizin diabetes, etc., may be, are yet to be determined.

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Influence of Whole Wheat upon Hemoglobin Regeneration in Albino Rats.

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Young rats from mothers on a uniform diet (Sherman's Diet 13, consisting of one-third whole milk powder and two-thirds ground whole wheat plus common salt equal to 2% of the weight of the wheat with meat and lettuce 3 times a week) have been made anemic by exclusive feeding of fluid milk (pasteurized) from time of wean-

[4] Sumner, J. B., *J. Biol. Chem.*, 1925, lxv, 395.

[5] Benedict, S. R., *J. Biol. Chem.*, 1925, lxiv, 207.

[6] Benedict, S. R., and Osterberg, E., *J. Biol. Chem.*, 1921, xlviii, 51.

[7] Benedict, S. R., and Osterberg, E., *J. Biol. Chem.*, 1918, xxxiv, 194.