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The Demethoxylation of Lignin in the Animal Body.

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We recently reported[1] that lignin when fed to a dog or cow causes a substantial increase in the quantity of benzoic acid eliminated in the urine. Since this fact is at variance with results published by others, we undertook to obtain more direct evidence bearing on the metabolism of lignin.

The OCH_3, or methoxyl group, has been definitely shown to be present in lignin. The Kirpal and Bühn[2] modification of the Zeisel method was employed by us for the quantitative estimation of the methoxyl group.

Knowing the methoxyl content of the lignin and also that of the feces before and after the feeding of lignin, it was possible to follow up the demethoxylation in the animal organism. Experiments conducted with a dog showed that over 15% of the methoxyl initially present in the lignin was liberated and, in the case of the cow, 37% loss of methoxyl was observed. We believe that in these experiments we have direct evidence that lignin is metabolized in the animal organism.

We have also conducted experiments *in vitro* in which known quantities of lignin and fresh material taken from the 4 compartments of the cow's stomach were incubated at 38° C. for 8 days. It was found that demethoxylation took place in all our experiments, irrespective of whether or not toluene had been added to the medium. Experiments conducted in a similar manner, using fresh material taken from the large and small intestine of a cow gave entirely neg-

[1] Csonka, F. A., Phillips, M., and Jones, D. B., *Proc. Am. Soc. Biol. Chem.*, 1928, vii, 24.

[2] Kirpal, A., and Bühn, T., *Ber.*, 1914, xlvii, 1084.

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.