not react with its homologous immune-rabbit serum absorbed by the new polysaccharide (Table I). A quantitative precipitin-titration showed that the new polysaccharide precipitated almost 3 times more antibody-protein than did the acetyl polysaccharide.

Although an antipneumococcal serum absorbed with our polysaccharide was still agglutinative and specifically protected mice from an otherwise fatal dose of pneumococci (Type I), the titre was greatly reduced.

The new polysaccharide in small doses produced active immunity in mice. Rats given intravenous injections of the polysaccharide-solution produced sera which protected mice from a fatal dose.

When the polysaccharide solution was heated at 100°C. in 0.05 N NaOH or 0.5 N acetic acid, the hydrolytic product still produced active immunity in mice, but failed to precipitate the immune serum absorbed with the acetyl polysaccharide.

It appears, therefore, that our polysaccharide may be the parent substance from which the acetyl polysaccharide could be obtained by appropriate treatment.

8744 P

Metabolism of Ethyl Esters of Fatty Acids.

H. J. DEUEL, JR., L. HALLMAN, J. S. BUTTS AND S. MURRAY.

From the Department of Biochemistry, University of Southern California School of Medicine, Los Angeles.

It has been demonstrated[1] that the conversion of caproic, butyric, and beta-hydroxy butyric acids to acetone bodies in fasting rats by beta oxidation is quantitative, whereas greater amounts of ketone bodies originate after sodium caprylate than after isomolecular quantities of sodium acetoacetate are fed. The latter phenomenon suggests that delta oxidation occurs in the latter case. No ketone bodies are formed when the sodium salts of the fatty acids with an odd carbon chain, as propionic, valeric, heptoic or nonylic acids were fed. It was later demonstrated[2] that the conversion of the odd chain fatty acids into glycogen must represent an approximately quantitative transformation by beta oxidation into propionic acid.

1 Butts, J. S., Cutler, C. H., Hallman, L., and Deuel, H. J., Jr., *J. Biol. Chem.*, 1935, **109**, 597.

2 Deuel, H. J., Jr., Butts, J. S., Hallman, L. F., and Cutler, C. H., *J. Biol. Chem.*, 1935, **112**, 15.

The fatty acids with an even number of carbon atoms were entirely ineffective as glycogen formers. However, it was impossible to feed the soaps of the fatty acids having a greater number of carbon atoms than 9 in an equimolecular dose to that found effective with the shorter chain fatty acids because of the relatively large quantities of solution which must be administered.

In the present experiments, by administering the fatty acids as their ethyl esters it has been possible to feed isomolecular quantities of all of the fatty acids up to stearic acid in similar doses to those employed in the earlier work—namely, 15 gm. per square meter of body surface per day, calculated as acetone.

The average excretion of acetone bodies in fasting male rats, second to fourth days, calculated as acetone in grams per square meter of body surface per day after feeding the ethyl esters was as follows: acetoacetate, 2.01 (57)*; butyrate, 1.37 (27); caproate, 1.95 (31); caprylate, 7.29 (12); caprate, 6.74 (16); laurate, 6.75 (17); myristate, 5.21 (9); palmitate, 2.94 (10); stearate, 2.40 (11); oleate, 5.07 (11). This indicates that the excretion of acetone bodies after the caproate and butyrate is an approximately quantitative one, whereas the acetonuria after the administration of the ethyl esters of the fatty acids with 8 or more carbon atoms is greater than that of the acetoacetate controls. This would indicate that more than one acetoacetate residue originates from the oxidation of one molecule of fatty acid having 8 or more carbon atoms.

The relatively low values with ethyl palmitate and ethyl stearate are traceable to the lack of absorption as evidenced by the large excretion of stools in these animals. It has been demonstrated that the administration of the palmitate and stearate in Wesson oil very much increased the absorption of these esters. No acetone bodies were formed after the administration of ethyl palmitate intraperitoneally. The relatively large excretion of acetone bodies following the administration of ethyl oleate indicates that the cleavage of this molecule at the double bond into 2 molecules of nonylic acid cannot be the chief course of oxidation or glycogen should result as the end-product instead of the acetone bodies.

When the ethyl esters of the fatty acids having an odd carbon chain were fed, only insignificant amounts of acetone bodies were excreted in the urine. The average acetone body excretion for the ethyl esters of the fatty acids having odd carbon chains were as follows: propionate, 0.22 (4); valerate, 0.37 (20); heptylate, 0.22

*The figures in parentheses represent the number of experiments of which this is the average.

(21); undecylate, 0.15 (25); sodium chloride controls, 0.19 (70).

Methyl esters of the fatty acids were found to be somewhat toxic. Experiments are in progress to demonstrate the comparative ketogenic effect of the ethyl esters of palmitic and stearic acids with that of acetoacetate when administered in much smaller doses.

8745 P

Initiation of Lactation in the Hypophysectomized Guinea Pig.

WARREN O. NELSON AND ROBERT GAUNT.

From the Department of Anatomy, Yale University Medical School, and the Department of Biology, Washington Square College, New York University.

The anterior pituitary secretes a hormone concerned in the initiation of lactation. Male or immature female guinea pigs whose mammary glands are developed under the influence of ovarian grafts or oestrin injections lactate when the source of oestrin is removed, but will not do so if the pituitary is simultaneously removed.[1] The following experiments indicate that the adrenal cortex, as well as the pituitary, may be involved in the initiation of lactation in the guinea pig.

Series I. Fourteen guinea pigs were prepared for lactation by prolonged oestrin injections or, in some of the males, by functional ovarian grafts. At the time the ovarian grafts were removed or oestrin injections discontinued hypophysectomy was performed. Injections of a purified preparation of lactogenic hormone* were begun immediately. This treatment in no instance initiated lactation, although continued from 5 to 15 days. In 3 partially hypophysectomized animals lactation was initiated.

Series II. Nine animals similarly prepared for lactation were hypophysectomized and treated with a crude alkaline extract of sheep pituitaries.* This extract contains, in addition to the lactogenic hormone, most if not all of the other known pituitary hormones.

The crude extract, in contrast to the purified lactogenic preparations initiated lactation in the hypophysectomized guinea pigs in every case. Six animals from Group I, failing to lactate on puri-

1 Nelson, W. O., PROC. SOC. EXP. BIOL. AND MED., 1935, **33**, 222.

* For the crude pituitary extract, Theelin, and purified lactogenic hormone used here the authors are indebted to Dr. Oliver Kamm of Parke, Davis & Co.