

tolerance, did not differ from the controls. — It is concluded that increased albumin excretion is associated with diabetes or impaired glucose tolerance, but is not associated with genetic predisposition to diabetes.

#### Epidemiology of Diabetes Mellitus in a Country District.

A. Kaeding and coworkers. Medical Polyclinic, The University of Rostock, Rostock, DDR.

In a country district with a population of 38 000 prevalence of the diabetes has been investigated by carrying out two screening surveys in 1960 and 1970, using the same methods. The influence of the two surveys on the prevalence of spontaneous diabetes is reported for the period between the two screening surveys. Resulting from the first screening in 1960 the diabetes "prevalence" doubled in this district. Ten years later, in 1970, the second screening revealed an increase of only 15% in the apparent prevalence of diabetes. — Clinical data, including secondary diabetic complications of the newly discovered diabetics from the two surveys are discussed and compared. — The possible reasons for the significant differences between the two surveys are discussed, and therapeutic and epidemiological conclusions are drawn.

#### Comparison of the Results of an Analysis of the i.v. Tolbutamide Test According to Lange and Krick as well as by the Method of Unger and Madison.

H. Kaffarnik, U. Heink, W.D. Gassel, P. Zöfel, K. Mylarch. Medical Polyclinic, University of Marburg/Lahn, W.-Germany.

In order to improve the assessment of the diabetic metabolic state, Lange and Krick have applied a modification of the method of Unger and Madison, suggesting extension of the Tolbutamide test from 30 to 60 min. In routine diagnosis a 1 h test, including additional blood sugar measurements, means considerable additional workload and material. The authors have therefore tested the reliability of the two methods in 150 subjects. A close correlation was observed between the  $T_2$  value and the blood sugar assessment 20 and 30 min after the injection of Tolbutamide. Only slight correlations were found between the  $T_2$  value and the increase in blood sugar 40 and 60 min after the injection. — In comparing the above values with the glucose assimilation constant K (according to Conard), we found sufficient correlation between the values at 20 and 30 min, but no correlation between the 40 and 60 min values. — These results demonstrate that the Tolbutamide test according to Unger and Madison is really sufficient in routine diagnosis.

#### Peripheral Insulin and Glucose Utilization in Tolbutamide-Induced Hypoglycaemia.

B. Karamanos, W.J.H. Butterfield, A.C. Asmal, M.J. Whichelow, B.D. Cox. Department of Medicine, Guy's Hospital Medical School, London.

This study was designed to investigate (a) the early ar-

terial insulin response to i.v. tolbutamide, (b) the relationship between insulin and glucose uptake in the periphery, (c) the role of peripheral insulin uptake and glucose metabolism in the development of tolbutamide induced hypoglycaemia. — Five normal subjects were studied before and for 60 min after 1 g i.v. tolbutamide. Continuous half minute arterial and venous samples were taken for glucose and insulin estimations over the first 10 min and thereafter at regular intervals for the rest of the test. Tissue uptake was measured using the forearm technique. — Our results indicate that there is a well marked insulinaemic response in the arterial plasma within half a minute of commencing the tolbutamide injection and this reaches a peak by the second minute. The arterial response is reflected in the venous plasma with a time lag of less than half a minute but the venous insulin levels are lower. — The arterial insulin response is paralleled by changes in the peripheral insulin uptake. The correlation between the two is highly significant ( $p < 0.001$ ). — The increase in insulin uptake is accompanied by a rise in glucose uptake but the changes are not concomitant and there is no correlation between the two. However, there is a good correlation between the total one hour glucose uptake and the total one hour insulin uptake. — The results suggest that the magnitude of the peripheral glucose uptake after tolbutamide cannot in itself account for the degree of fall in the blood sugar.

#### Utilization and Exhalation as $^{14}\text{CO}_2$ of I.V. Administered Loads of $^{14}\text{C}$ Labeled Glucose, Xylitol, Fructose and Sorbitol in the Fasted and in the Streptozotocin-Diabetic Rat.

U. Keller, E.R. Froesch. Metabolic Unit, Department of Medicine, University of Zurich, Switzerland.

35–37% of equivalent loads of glucose, xylitol and fructose and 20% of sorbitol were exhaled as  $^{14}\text{CO}_2$  by 24 h fasted rats within six hours. The peak of  $^{14}\text{CO}_2$  was 20–30 min after administration. Streptozotocin-diabetic rats exhaled 11–18% of the administered dose of each substrate. Maximal  $^{14}\text{CO}_2$ -production was registered 20 min after fructose and 40–60 min after glucose, xylitol and sorbitol. After six hours, the amounts of carbon-14 recovered in serum, serum osazones, glycogen and total lipids of liver and diaphragm glycogen were similar after each of the four substrates in either of the two groups of animals. — Diabetic rats incorporated only minimal quantities of carbon-14 into diaphragm glycogen. 40–55% of the given dose of any one of the substrates was lost in the urine, mostly as glucose- $^{14}\text{C}$ . Only one clear cut difference between glucose and the other substrates was apparent. Much less  $^{14}\text{C}$ -glucose was incorporated into glyceride-glycerol of liver total lipids, particularly in diabetic rats. The results suggest that all three glucose substitutes are rapidly converted to glucose and that their utilization is, therefore, highly dependent on insulin. — Thus, the value of the application of these glucose substitutes to diabetics is presently reassessed in our laboratory.

### Corrigendum Notice

Diabetologia, 7, 349–356 (1971)

U. Keller and E. R. Froesch: Metabolism of U- $^{14}\text{C}$ -Glucose, Xylitol, Fructose and Sorbitol in the Fasted and in the Streptozotocin-Diabetic Rat.

Unfortunately the figures in this article have been changed around by mistake. The following order would have been right:

Fig. 4 should be Fig. 1;

Fig. 5 should be Fig. 3;

Fig. 2 should be Fig. 5;

Fig. 6 should be Fig. 2;

Fig. 1 should be Fig. 4;

Fig. 3 should be Fig. 6.

Legends are correct as printed.

Requests for correct reprints to:

Professor E. R. Froesch  
Kantonsspital Zürich  
Medizinische Universitätsklinik  
Stoffwechsellabor  
CH-8006 Zürich, Rämistraße 11