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Genetic and environmental interaction in variation of skinfold thickness in children. C. G. D. Brook, R. M. C. Huntley, and J. Slack. Department of Paediatrics, Middlesex Hospital, and Institute of Child Health, London.

The complexity of determining the relative importance of genetic and environmental influences in the variations observed in body build is considerable. The availability of triceps and subscapular skinfold measurements collected on identical and fraternal twins during childhood, together with suitable standard measurements, has provided an opportunity to do this. The proportion of variance for a characteristic which is due to genetic variation can be calculated from the difference between correlation coefficients of the characteristic observed in monozygotic and dizygotic twins, if it is assumed that the within-pair differences due to environmental factors would be similar for both.

Triceps and subscapular skinfold thickness have been measured on 78 pairs of identical twins (38 male, 40 female), on 144 pairs of fraternal twins (67 male, 77 female), and on 117 pairs of fraternal twins of unlike sex. There were marked differences between the two sexes and between the two sites: age also considerably affected the results. In children under the age of 10 years environmental influences accounted for a large proportion of the variation of limb fat in both sexes and body fat in girls. In older children genetic factors accounted for a very large proportion of the variation in both types of fat in both sexes.

Pancreatic glucagon in diabetic ketoacidosis. D. I. Johnston, S. R. Bloom, and D. O'Brien. Department of Child Health, King's College Hospital; Middlesex Hospital, London; and University of Colorado Medical Center, Denver.

Pancreatic α and β cell inter-relations in health and disease have received recent emphasis with the development of sensitive radioimmunoassays for glucagon. Glucagon opposes the actions of insulin and may have a significant role in the pathogenesis of diabetes.

We studied 10 cases (age range, 11–18 years) of diabetic ketoacidosis requiring intravenous fluid therapy. Intravenous and intramuscular insulin was administered at intervals as indicated by clinical and biochemical parameters. Plasma glucose, acetone, carbon dioxide, free fatty acids, and glucagon were measured at admission, during therapy, and after an overnight fast following recovery. Plasma glucagon at admission,

250 ± 37 pg/ml (mean $\pm$ SEM), was significantly greater than that after recovery, 105 ± 5 pg/ml, and that of overnight fasted nondiabetic controls, 80 ± 4 pg/ml. There was a significant correlation between the initial glucagon value and plasma acetone ($r = +0.752$, $P < 0.05$), and carbon dioxide ($r = -0.771$, $P < 0.01$), but not with glucose or fatty acids. Plasma glucagon fell after initiation of therapy.

The 5 patients admitted with plasma glucagon > 200 pg/ml had predisposing illness of greater severity and required significantly more insulin/kg body weight (2.90 ± 0.25 units) for control of ketoacidosis than those with lower glucagon values (1.25 ± 0.45 units).

Clinical and experimental studies have shown that pancreatic glucagon is secreted in response to physiological stress, such as pyrexia and hypovolaemia. The high glucagon levels associated with illness resulting in ketoacidosis may have an adverse effect on its course and subsequent management.

α -L-Iduronidase deficiency associated with chondroitin sulphate mucopolysaccharidosis. A. Babarik, P. F. Benson, M. F. Dean, and H. Muir. Paediatric Research Unit, Guy's Hospital Medical School, and Kennedy Institute of Rheumatology, Bute Gardens, London.

Chondroitin sulphate is the principal glycosaminoglycan of normal cartilage, liver, and urine. A mild rise in the urine has been reported in one of 2 unrelated children with β -glucuronidase deficiency and in other forms of mucopolysaccharidosis (MPS). In MPS I–III, however, the principal urinary glycosaminoglycans are dermatan sulphate and heparan sulphate. There are also published reports of 5 patients with MPS in whom the predominant urinary glycosaminoglycan is chondroitin sulphate. One of these patients (Benson, Dean, and Muir, 1972) had features of Hurler's syndrome and died aged 6 years 10 months with bronchopneumonia and hydrocephalus due to basal cistern block. We now report that cultured fibroblasts from this boy had accelerated incorporation of ^{35}S -sulphate into cellular glycosaminoglycan which could be corrected by mixing his fibroblasts with Hunter cells but not with Hurler's cells; and absent α -L-iduronidase activity. Activities of 8 other fibroblast lysosomal hydrolases (β -D-glucuronidase, β -D-galactosidase, β -D-hexosaminidase A and B, α -D-glucosidase, β -D-xylosidase, β -D-glucosidase, and α -D-galactosidase) were normal, but that of α -D-mannosidase was moderately raised. These