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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

results are compatible with the patient having MPS I (Hurler's syndrome) but do not explain why chondroitin sulphate was the principal urinary and hepatic glycosaminoglycan.

REFERENCE

- Benson, P. F., Dean, M. F., and Muir, H. (1972). A form of mucopolysaccharidosis with visceral storage and excessive urinary excretion of chondroitin sulphate. *Developmental Medicine and Child Neurology*, **14**, 69.

Blood pressure and angiotensin II in the newborn. F. B. Pipkin and O. R. C. Smales (introduced by D. Hull). City Hospital, Hucknall Road, Nottingham.

Angiotensin II (AII) is the most potent naturally occurring pressor agent known. It has been measured by radioimmunoassay on 1–2 ml plasma obtained from infants in the first week of life at the time of sampling for routine investigations. Systolic blood pressure was measured before sampling, using the Doppler ultrasound technique. Venous samples were obtained from 25 infants in whom physiological jaundice or prematurity was the only abnormality. 20 infants were sampled in whom there were additional clinical complications, such as respiratory distress or vomiting. AII levels fell from a mean of 178.4 ± 26.2 SE pg/ml in cord venous blood at birth to a mean of 60.3 ± 9.2 pg/ml during the first 6 days of life. Mean adult values were 28.8 ± 4.2 pg/ml as compared with 97.5 ± 9.0 in pregnant women at delivery. AII levels were higher in preterm infants than in term infants (mean 75.6 ± 11.4 pg/ml against 54.6 ± 12.1 pg/ml), but this was not statistically significant. Systolic blood pressure in 70 infants increased significantly during the first week of life ($P < 0.001$, $r = 0.5490$), but was more closely related to birthweight ($P < 0.001$, $r = 0.7411$, no. = 66) and gestational age ($P < 0.001$, $r = 0.7313$, no. = 67). There was a significant inverse relation between the mean arterial blood pressure and venous AII in the 45 infants in which both were measured ($P < 0.01$).

Plasma renin activity and aldosterone concentration in children: results in salt-wasting states. M. J. Dillon and J. Ryness. The Hospital for Sick Children and Institute of Child Health, London.

Semimicro methods for the measurement of plasma renin activity (PRA) and plasma aldosterone concentration (PALdo) by radioimmunoassay have been developed using 0.25 ml plasma and between 0.5 ml and 1.0 ml plasma, respectively. Normal ranges for healthy children on free diets have been established and it was found that values of PRA and PALdo varied inversely with age. In infants, the mean PRA value was 1404 pgAI/ml per h (range 472–3130) with a progressive decrease through childhood to the mean adult value of 85 pgAI/ml per h (range 22–311). In children under 1 year of age the mean value for PALdo was 24 ng/100 ml (range 8–75). There was a similar decrease with age, such that mean value between 5 and 9 years of age was 4.5 ng/100 ml (range 1.0–15), but this was followed by a slight rise to the adult mean of 8.2 ng/100 ml.

PRA and PALdo values were considerably greater in children with evidence of saline depletion than in healthy children of equivalent age. Children with hypernatraemic diarrhoeal dehydration were found to have lower values of PRA and PALdo than children with gastroenteritis but no evidence of hypernatraemia. In children with chronic saline depletion PRA values were markedly increased with a mean figure of 25 000 pgAI/ml per h. However, PALdo values were not uniformly raised and those from children with adrenal insufficiency were within the normal range compared with the very high values from the other salt-wasters. The relation shown between PRA and PALdo in individuals with no abnormality of the aldosterone response to renin/angiotensin stimulation permit identification of situations in which inappropriate responses occur, e.g. congenital adrenal hyperplasia and Conn's syndrome.

Is human milk the best food for preterm infants? D. P. Davies. Department of Child Health, University Hospital of Wales, Cardiff.

Since the early days of caring for preterm infants it has been widely held that human milk is the food of choice for these infants. This belief, however, has not prevented some paediatricians from suggesting that human milk might not in fact be the ideal food on the grounds that its low protein content is insufficient for growth requirements. Adequate protein intake in the early weeks of life is necessary if growth is to proceed normally. Failure to grow satisfactorily at this stage might result in permanent detrimental effects on body growth. The question of optimum protein requirements for preterm infants is therefore an important one. The present study investigates the adequacy of human milk for the growth of preterm infants. 106 preterm infants were fed one of three isocaloric milks for a period of 2 months. Milk A: high protein milk (21% calories as protein); milk B: medium protein milk (15% calories as protein); milk C: human breast milk (7% calories as protein). Changes in weight, length, head circumference, and triceps skinfold thickness were evaluated. The results suggest that though human milk is adequate for the growth needs of the more mature preterm infants (33–36 weeks' gestation), less mature infants (28–32 weeks' gestation) fed human milk failed to achieve adequate growth rates compared with infants on higher protein intakes.

Fat absorption and weight gain of small babies fed two filled milk formulae. R. D. G. Milner, Y. Deodhar, C. R. Chard, and R. M. Grout. St. Mary's Hospital, Hathersage Road, Manchester M13 0JH. To be published in full in the *Archives*.

Calorific cost of activity in neonates. J. Meyer (introduced by J. Scopes). St. Thomas's Hospital, London S.E.1.

The data on calorific expenditure on activity in neonates are incomplete. An experimental situation was devised where total calorie balance studies could be