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Genetic and environmental interaction in
variation of skinfold thickness in children.
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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 subcapular skinfold measurements
collected on identical and fraternal twins during child-
hood, 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 subcapular 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 pro-
portion 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.
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sex 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 develop-
ment 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 adminis-
tration. Intravenous and intramuscular insulin was adminis-
tered at intervals as indicated by clinical and biochemical
parameters. Plasma glucose, acetone, carbon dioxide,
free fatty acids, and glucagon were measured at ad-
mission, during therapy, and after an overnight fast
following recovery. Plasma glucagon at admission,
250±37 pg/ml (mean±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 physio-
logical 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 chon-
droitin sulphate mucopolysaccharidosis. A. Bab-
srik, P. F. Benson, M. F. Dean, and H. Muir.
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London.

Chondroitin sulphate is the principal glycosaminogly-
can 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 glycosaminogly-
cans 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 syn-
drome and died aged 6 years 10 months with broncho-
pneumonia and hydrocephalus due to basal cistern
block. We now report that cultured fibroblasts from
this boy had accelerated incorporation of 35S-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-β-glucuronidase, β-D-galactosidase, β-D-hexosami-
dase 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