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Transport of folate compounds through
the membrane of normal and transport-
deficient lymphoid cells.

The active transport of folic acid
(FA) 5-methyltetrahydrofolate (5-MeFH4)
and methotrexate (MTX) was investigated
in mouse leukemia L 1210 and in a human
lymphoid cultured cell line. In both
cell types exist two different trans-
port systems, one for 5-MeFH4 which
transports also MTX and one for FA which
is much less effective. The first system
can be inhibited by blocking sulfhydryl
groups in the membrane with organic
mercurial compounds. But no SH-groups
seem to be involved in the transport of
FA. Iodoacetate, however, leads to an
increased total uptake of MTX. Resistan-
ce to MTX can be due to decreased up-
take of the folate antagonist. A cell
strain with impaired transport of MTX
takes also up less 5-MeFH4, where as
the transport of FA is not decreased.
Competition experiments show that foli-
nic acid shares the transport system
with MTX and 5-MeFH4. This system seems
to be the important pathway for the up-
take of folate and antifolate compounds
into these cells, while FA is probably
taken up by a much less specific system.

C. LOIRAT, M. BROYER, Hôpital des Enfants
Malades, Paris, France. Renal transplantation
in children. First year experience.

14 children, 5 to 14 years old, received ca-
daveric renal kidneys during 1973. Donors were
2 to 41 years old. Kidney size was greater than
normal for the recipient in 9 cases, less in 3
cases, and equivalent in 2 cases. When too lar-
ge to be placed in the pelvis, the kidney was
placed in a paravertebral position. Uretero-
ureteral anastomosis was performed in most ca-
ses. Treatment was Azathioprine and corticothe-
rapy. High doses of Furosemide (10 mg/kg) du-
ring operation prevented ischemic acute renal
failure, despite cold ischemia as long 23 hours.
One child died after 1 week from intracerebral
hemorrhage due to severe hypertension. A second
died after 5 weeks from a diffuse hemorrhagic
syndrome. Primary thrombosis of the kidney ve-
ssels led to transplantectomy in 1 case. Kidney
function is normal in 9 cases, chronic rejec-
tion is present in 2. No urologic complications
were observed. Hypertension developed in half
of the children. One case was associated with
renal artery stenosis, another with chronic re-
jection. Hypertension was more frequent in tho-
se who had severe hypertension, leading to bine-
phrectomy, before transplantation. No correla-
tion was found between occurrence of hyperten-
sion and kidney size. Although follow-up is too
short, growth appears possible in prepubertal
children, when steroid doses are low. Rehabili-
tation was total within 3 months in 10 cases.
Cadaveric kidney transplantation seems to be a
valuable therapeutic procedure in children with
chronic renal failure.

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The concentrations of immunoglobulins and
complement components in the sera of children
with glomerular disease.

Various complement components are estimated
in the sera of patients with renal disease for
the purposes of diagnosis and investigation.
Deviation from normality may reflect involve-
ment in the pathogenetic process, or may be a
secondary consequence of proteinuria. We have
therefore systematically measured plasma C1q,
C4, C3, GBG, IgG (and specifically subclass 3),
IgA and IgM, and have related them to the
plasma concentration of albumin and to the
urine albumin/creatinine concentration ratio.

GBG correlates positively with the plasma
albumin concentration, and negatively with the
urine albumin/creatinine ratio, indicating that
its serum concentration is affected by urinary
loss. Plasma IgG, but not IgG subclass 3,
correlates positively with plasma albumin,
indicating a different clearance and/or
turnover rate of IgG3 from other IgG
subclasses.

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Complement studies in nephrotoxic
serum nephritis (NSN) and aminonucleo-
side nephrosis (AN) of the rat.

Total complement (CH50) and C1-C9
have been determined in sera and urines
of rats with NSN and AN. In NSN, 3 hrs.
after injection of antiserum, a signifi-
cant drop of CH50 and C1-C8 in serum
was observed, whereas C9 remained nor-
mal. At day 12 CH50 and the complement
factors were normal except C1, which
was still slightly diminished. In urine,
at day 1 only C8 and C9, at day 12
small amounts of C1, C4, C8 and C9 and,
less frequently, of C3-C7 could be mea-
sured. In AN, at day 12 CH50 and C1-C9
were strikingly diminished in serum. In
urine, at day 12 C3, C5, C7, C8 and C9
could be determined regularly, whereas
urinary excretion of C1, C4, C2 and C6
was observed in about 50% of the ani-
mals. In NSN, the consumption of comple-
ment in serum reflects the ongoing im-
munological process. The low clearances
of complement components, compared to
AN, may be due to their fixation and in-
activation in the kidney. In AN, the re-
duction of complement in serum may be
the result of loss of complement factors
in oedema, ascites and urine, and/or of
impaired synthesis of these proteins.