The optimum NaCl concentration in formiate buffer was 0.25 ± 0.05 M. These features indicate that a lysosomal-like hyaluronidase is secreted by hepatoma cell lines.

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RSV-INDUCED CELL TRANSFORMATION : EFFECT OF PROTEASE INHIBITORS ON FIBRONECTIN AND PLASMINOGEN ACTIVATORS

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RSV-induced cell transformation is promoted by fibronectin fragments (FNdp), tissue-type plasminogen activator (t-PA) and by 12-0-tetradecanoylphorbol-13 acetate (TPA). It is known that high level of plasminogen activator (PA) activity is present in the conditioned medium (CM) of RSV-transformed cells which are also depleted of an organized extracellular matrix (ECM): this loss might be due to the direct catalytic action of PA on ECM proteins. In this study we report that: 1) RSV-transformed chicken embryo fibroblasts (CEF) release in the CM, in the absence of serum, FN peptides with MW between 230 and 110 kD and different molecular forms of PAs (MW ranging between 180 and 43 kD); 2) TPA induces an increased secretion of PAs and FN fragments: PAs and FN fragment release is suppressed by 2mM benzamidine but not by 100 IU/ml trasylol; 3) benzamidine is able to inhibit the transformed phenotype; some protease inhibitors exert a differential effect on the quantitative release of FN in RSV-transformed CEF and in uninfected CEF.

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MODULATION OF P53 EXPRESSION DURING CELLULAR TRANSFORMATION WITH SV40

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We recently demonstrated that SV40 transformed cells harbour non-complexed p53 (free p53) which is metabolically stable in addition to p53 complexed with the large T antigen. These findings suggested that a mechanism for p53 stabilization independent from large T/p53 complex formation also operates in cellular transformation by SV40. To explore this hypothesis further, we have analyzed p53 epression in mouse BALB/c 3T3 cells abortively infected with SV40. These cells transiently express SV40 large T, but are not stably transformed. We have shown that in these cells neither p53 complexed to large T nor free p53 is metabolically stable. However, if stably transformed cells are selected from abortively infected cells by a focus assay and analyzed for p53 expression, both complexed and free p53 are metabolically stable. Our experiments demonstate (1) that complex formation of p53 with large T *per se* does not stabilize p53 and (2) that p53 stabilization is a transformation specific event which seems to be a second step in cellular transformation by SV40.

CHOLESTEROL ESTERS AND CELLULAR PROLIFERATION IN THE LIVER

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Several investigators have attempted to correlate the induction of cholesterol synthesis with cellular proliferation. This has been repeatedly evaluated *in vitro* through the inhibition of HMGCoA reductase. *In vivo*, the inhibition of cholesterol synthesis is not easily achieved. We have studied cellular proliferation induced by an hepatic mitogen, lead nitrate, during fasting, a condition associated with very low levels of cholesterol synthesis. The accumulation and synthesis of cholesterol esters under such conditions has been investigated in relation to DNA synthesis.

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PROTEINS PHOSPHORYLATED ON TYROSINE AS MARKERS OF HUMAN MALIGNANCIES

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Previous work has shown that proteins phosphorylated on tyrosine are selectively detectable by antibodies against phosphotyrosine (P-Tyr) in cells transformed by retroviral class 1 oncogene-encoded kinases endowed with non regulated activity (Di Renzo *et al*, Eur. J. Biochem., 1986).