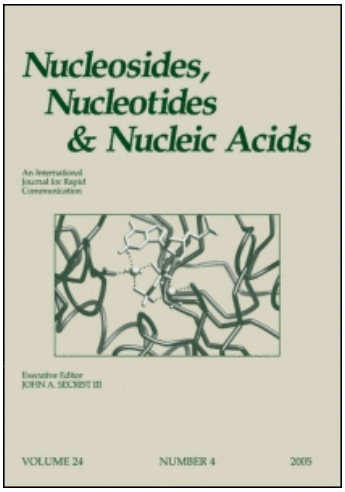

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INVESTIGATION OF THE C-MYC AND C-FOS GENES FUNCTIONS WITH RETROVIRAL VECTORS PRODUCING ANTISENSE RNA

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Abstract.Highly tumorogenic mouse fibroblast cell line loses its capacity to grow in singene animals after infection with a retroviral vector, producing antisense RNA to gene c-fos.

Sequences from the coding regions of the human c-fos and mouse c-myc genes were cloned as tandem repeats in antisense orientations in the pPS3 shuttle vector (Figure 1).

These DNA constructs were introduced into Psi-2 cells by the calcium phosphate precipitation procedure using neo gene as selectable marker. Viruses generated from Psi-2 cells were used for infectioning of cells FC3H 3v7 spontaneously transformed in vitro. The parent FC3H 3v7 cells grow fast in the singene mice causing solid sarcoma which kills the animals in about 40 days. The cells transformed with the

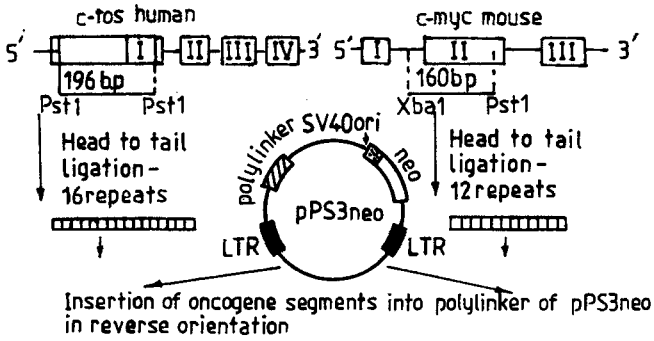

FIG.1. Outline of construction of the retroviral vectors.

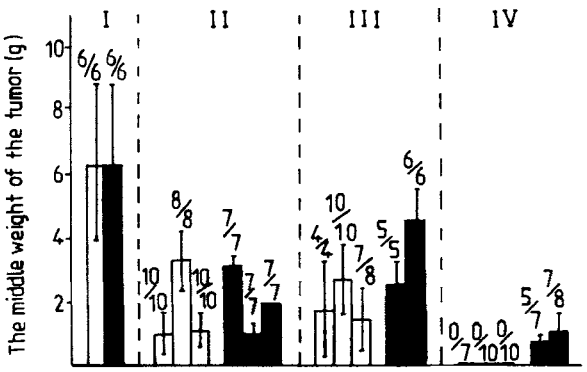

FIG.2. Tumorigenicity of the cells.

I. Parental cells FC3H 3v7; II. The cells transfected with pPS3neo; III. The cells trasfected with pPS3(antimyc); IV. The cells transfected with pPS3(antifos).
□ Tumor growth in nonirradiated mice
■ Tumor growth in irradiated mice
Over the line - number of mice with tumor
Under the line - number of mice in experiment

plasmid pPS3(antifos) show a lower level of p53 fos protein and lose the ability to grow in animals. They become sensitive to the serum factors and require fetal serum in contrast to the parent cells growing on the calf serum. The parent FC3H 3v7 cells infected with the other vectors retained the essential features of the parent cells (Figure 2).

It is interesting to note decrease in the rate of in vivo growth of all transfected cells (Fig.2). What we know for sure is that the decrease is not connected with the host immune response because the growth of transplantats was the same in immunodeficient and intact animals. We hypothesize that the loss of tumorogenicity of cells bearing antifos constract may be due to the expression of some antigene determinants (for example fetal) causing immonogenity of cells.