CHARACTERIZATION OF *RECK* TRANSCRIPTIONAL REPRESSION MEDIATED BY THE C-*MYC* ONCOGENE PRODUCT

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Deregulated expression of the c-*myc* proto-oncogene contributes to malignant progression of a variety of human tumors, such as breast, colon, and cervical carcinomas, glioblastomas, and myeloid leukemias. Several biological functions of c-Myc have been described, including induction of programmed cell death, inhibition of terminal differentiation and potentiation of cell cycle progression. The *RECK* gene mRNA is abundantly expressed in many human normal tissues, whereas its expression is suppressed in both tumor cell lines and in oncogenically transformed fibroblasts, including fibroblasts transformed by the c-*myc* gene, suggesting that *RECK* is a physiological target for c-Myc. We have previously described that inhibition of the *RECK* gene expression by c-Myc occurs through alterations in the transcriptional activity of this gene and that *RECK* repression parallels c-*myc* induction by serum. The mechanisms by which c-Myc is able to repress *RECK* expression is yet obscure. In order to address this question, we used cycloheximide and trichostatin A in NIH-3T3 cells that were stably transfected with pBPuro-MycER™ (for conditional c-Myc expression) or with the empty vector. The results obtained by Real Time PCR show that *RECK* inhibition by c-Myc is dependent on protein synthesis and chromatin remodelling.

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