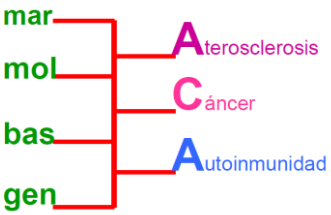
Summary



- Experimental cell model of early atherosclerosis: We have developed a cellular model to study changes in the differentiation state and transdifferentiation of chicken smooth muscle cells (SMC) induced by cholesterol diet. Comparative studies of SMC cultures from control chickens (SMC-C) and SMC cultures from hypercholesterolemic chickens (SMC-Ch), as well as a human SMC stablished cell line (A10), on the genetic regulation and nutritional control of the cellular cholesterol homeostasis by SREBPs, PPARs, LXR, ABC, CD36, as well as its modulation mediated by protein isoprenylation. Transdifferentiation of SMC: Foam cell formation *in vivo* and *in vitro*. Modulation mediated by isoprenylization protein.

- Identification of pancreas adenocarcinoma biomarkers. Genes and proteins expression by microarrays. Study of correlation of clinical answers to gemcitabine, and to erlotinib: Gemcitabina is the standard agent in advanced pancreas cancer disease. The gemcitabina action mechanism implies its intra-cell metabolization to diphosphate nucleoside and threephosphate, both with cell-toxic activity, inhibiting the DNA synthesis both at reductase ribonucleotide as by competition with other nucleosides in the incorporation to DNA. In this way it seems to induce the programmed cellular death known as apoptosis. Erlotinib is an inhibitor of HER1, family of EGFR, key components of the altered trait transduction pathway related to the appearance and growth of many cancer types. Its action mechanism inhibits the HER1 tyrosine-kinase activity and the trait transduction initiated by the activation of these receptors. In this way, Erlotinib blockages the tumour cell growth and proliferation. Our objective is to determine the effect of treatments with gemcitabine and erlotinib about the proliferation and apoptosis in pancreas adenocarcinome tissue cells and peripheral blood by means of the following analyses: 1. Apoptosis marker molecules and proliferation in different pancreas cancer cells, by using the analyses of proteins Bcl-XL, Bcl2, P53, p21, PCNA y Rb and K-ras. 2. Expression of apoptotic and proliferation genes after different treatment.