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Oxidative stress, tumor microenvironment and metabolic reprogramming: A diabolic liaison

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Conversely to normal cells, where deregulated oxidative stress drives the activation of death pathways, malignant cells exploit oxidative milieu for their advantage. Cancer cells are located in a very complex microenvironment together with stromal components that participate to enhance oxidative stress to promote tumor progression. Tumor cells engage a key relationship with cancer associated fibroblasts (CAFs), behaving as active participants in tumor progression, by eliciting in cancer cells epithelial mesenchymal transition (EMT) and enhancing stem cells traits and metastatic dissemination. This reciprocal interplay between CAFs and cancer cells goes beyond the engagement of EMT, including mutual metabolic reprogramming. Indeed, cancer cells allocate Warburg metabolism to their corrupted stromal CAFs and exploit their byproducts to grow in glucose free environment, symbiotically adapting with its stromal cells to glucose availability. Our studies are aimed at understanding the interplay among tumor microenvironment, oxidative stress and metabolic reprogramming of cancer cells, recently defined by Hanahan and Weinberg as new "hallmarks of cancer". We identified in miR200 family a potential molecular link among these events, and involved this family of miRNAs in oxidants handling by cancer cells, in sirtuins regulation and nutrient sensing, as well as in EMT induced by stromal CAFs.

Biography

Paola Chiarugi is Full Professor of Biochemistry at the Faculty of Medicine and Surgery of Florence, coordinates the Cell Biology Laboratory of the Excellence and Research Center DENOThe'. Her studies on redox regulation of cell motility and cancer spreading, span from redox signalling during epithelial-mesenchymal transition, to the role of oxidative stress in the achievement of a metastatic and invasive phenotype. She contributed more than 100 original manuscripts and reviews in international peer-reviewed journals, dealing on motility, anchorage independence of cancer cells, achievement of anoikis resistance, epithelial mesenchymal transition or mesenchymal amoeboid transition and their regulation by tumor microenvironment.

Correlation between EGFR expression and accelerated proliferation during radiotherapy of head and neck squamous cell carcinoma

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In the recent years there has been a great interest to find factors that predict tumours suitable for accelerated radiotherapy and considerable interest has been given to cell kinetic parameters. Since regeneration and tumour cell proliferation are mechanisms at the cellular level, particular attention has been focused on identifying the specific cellular characteristics, such as variations in EGFR expression. Therefore, a survey of the published papers comparing the local regional control rate for patients treated with conventional and accelerated radiotherapy and with a pretreatment assessment of EGFR expression was made. Different values of doubling time were obtained by a model incorporating the effective dose delivered and the local control rate for high and low EGFR groups of patients, respectively. A higher expression of the EGFR leads to enhanced proliferation and our results indicate a clear reduction of effective doubling time between the two groups, respectively. This reduction tends to reach the minimum value which is represented by potential-doubling-time (the limit where the cell loss fraction is reduced to zero and the proliferation is fastest). In practice clonogens are lost through many possible mechanisms (differentiation, apoptosis, etc) and the net result is that the effective doubling time is longer than the potential doubling. However, poor differentiation and loss of apoptosis are mechanisms accompanied with loss of asymmetric cell division suggesting the involvement of subpopulations of specifically tumorigenic stem cells when irradiated. This has become more than just an academic question in combined treatment of radiation and biological drugs EGFR inhibitors.

Biography

Piernicola Pedicini is a medical physicist expert in mathematical modeling. He applies that knowledge to the possible integration between radiosensitization of drugs, biological and chemotherapy, with radiation therapies. He has authored some important peer-reviewed reports in this area and many others papers are in press. He has served on the editorial boards for the *International Journal of radiation Oncology Biology and Physics, Radiotherapy and Oncology, Medical Physics, Radiation Oncology, Journal of applied Medical Physics* and many other journals. He is currently the coordinator of the radiobiology group of the Italian Association of Medical Physics.