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Cancer stem cells and biological behavior of head and neck squamous cell cancer

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Head and neck squamous cell cancers (HNSCC) account for approximately 3 percent of all cancers in the United States. Among them, oral cavity is the most frequent affected site. The natural history of HNSCC is variable and difficult to predict. Overall, HNSCCs frequently metastasize, and metastases are frequently unresponsive to radio- and/or chemotherapy.

For several solid human tumors, it has been hypothesized that subsets of self-renewing, quiescent tumor cells with stem-like properties (cancer stem cells, CSCs), could account for the resistance to DNA-damaging agents, such radio- and chemotherapies, which in turn target only the hierarchically organized, rapidly dividing "differentiated" tumor cells, which constitute the bulk of the tumor mass.

We provide evidences for the existence of a definite inter-relationship between the constant overexpression of several stem cell markers (nestin, CD166, CD44 and CD44v6) and a history of node- or distant metastases of our HNSCC patients.

In particular, we found the highest immunohistochemical levels of nestin always coupled to the overexpression of both PARP-1 and the molecular chaperone chromatin assembly factor-1 (CAF-1). CAF-1 comprises a complex of three subunits (p150, p60, and p48) and drives the incorporation and assembly of H3K56-acetylated histones into chromatin in response to oxidative stress, DNA damage, and mismatch-containing strands, restoring chromatin structure on the completion of double-strand break repair. Similarly, PARP-1 contributes to the DNA-repair process of normal cells, and its expression has been found frequently deregulated in aggressive malignant tumors.

Our findings suggest that all these molecules, when overexpressed, may be used for the generation of a novel predictive immunohistochemical panel, to screen the chances of chemo/radiotherapy responsiveness and overall prognosis of these lethal cancers, shedding new light on the very complex molecular events underlying the neoplastic progression of HNSCCs.

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