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Commentary

Senescent Cell Derived Hepatocyte Growth Factor Reduces the Apical Elimination of Carcinogenic Cells

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DESCRIPTION

Cellular senescence and cell competition are important tumour suppression mechanisms that limit the growth of cells with oncogenic mutations early in the cancer development process. However, the relationship between cellular senescence and cell competition is still unknown. Senescent cells that accumulate during the ageing process contribute to age related cancers through the development of a Senescence Associated Secretory Phenotype (SASP). Furthermore, high-fat diet induced cellular senescence promotes the survival of cells with oncogenic mutations and an inhibitor of Hepatocyte Growth Factor (HGF) signalling causes the removal of mutated cells from mouse livers and intestines.

Cancer is caused by somatic mutations in some oncogenes or tumour suppressor genes. Several tumor suppressive mechanisms, such as cell competition, cellular senescence and various types of cell death pathways, selectively remove mutated damaged cells at the earliest stage of cancer development. Although *Drosophila* developmental studies were the first to report on cell competition, recent research has revealed that cells with oncogenic mutations compete with surrounding normal cells and are apically excluded from the epithelial layer in mammalian organs. Cellular senescence, on the other hand, is a phenomenon in which somatic cells exhibit a finite replicative lifespan *in vitro*, inhibiting the proliferation of cancer-prone cells in precancerous lesions and benign tumours.

Aging causes the functional decline of various tissues and organs, which leads to the development of a variety of diseases. The state of cell fitness also changes as we age. Skin homeostasis is maintained by epidermal stem cell competition, whereas epidermal stem cell ageing results in decreased cell competition, which leads to skin ageing. Furthermore, ageing is a significant risk factor for cancer incidence. The effects of ageing on cell competition as a tumor suppressive mechanism, on the other hand, remain unknown. Because most premalignant mutant cells were eliminated by cell competition in an esophageal carcinogenesis mouse model, cell competition may play an important role in tumour suppression during the early stages of carcinogenesis in mammals.

Furthermore, the incidence of esophageal or other cancers increases dramatically with age, indicating that ageing is a critical risk factor for cancer development. Cancer is caused by changes in key regulatory genes that control cell proliferation, differentiation and survival. Tumor virus studies revealed that specific genes can induce cell transformation, providing the first insights into the molecular basis of cancer. However, the vast majority of human cancers are not caused by viruses, but rather by other factors such as radiation and chemical carcinogens. As a result, it has been critical to our overall understanding of cancer that studies of viral oncogenes have also led to the identification of cellular oncogenes, which are involved in the development of non-virus-induced cancer. Many nuclear oncoproteins are transcription factors that regulate the expression of specific target genes. Although a thorough understanding of the mechanisms by which nuclear oncoproteins regulate transcription is still lacking, homo and heterod imerization of these proteins *via* well-defined motifs plays an important role in the process. Other domains provide DNA binding specificity, which is important in the regulation of cell cycle control genes. A retrovirus acquiring a cellular oncogene is a rare event that occurs during viral passage in an animal but is rarely seen in cell culture. Proto oncogenes are naturally present within the genomes of a wide range of vertebrate animals, including humans and have DNA sequences that are similar to, but not identical to, their viral equivalents. However, they do not result in cellular transformation. Although the proto usefulness oncogene's was not immediately evident and it was first thought that it was "silent" or not expressed until it was "turned on" to induce uncontrolled growth, its significance in cell regulation was quickly discovered.

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