Research & Reviews: Medical and Clinical Oncology

Somatic Mutation Theory of Cancer and Tumor Clonality

Sterling Kain*

Department of Oncology, University of Cambridge, Cambridge, United Kingdom

Commentary

Received: 29-May-2023, Manuscript No. MCO-23-101732; **Editor assigned:** 02-Jun-2023, PreQC No. MCO-23-101732(PQ); **Reviewed:** 16-Jun-2023, QC No. MCO-23-101732; **Revised:** 23-Jun-2023, Manuscript No. MCO-23-101732(R); **Published:** 30-Jun-2023, DOI: 10.4172/medclinoncol.7.2.003

***For Correspondence:** Sterling Kain, Department of Oncology, University of Cambridge, Cambridge, United Kingdom

E-mail: kainsterl21@gmail.com

Citation: Kain S. Somatic Mutation Theory of Cancer and Tumor Clonality. Med Clin Oncol. 2023;7:003.

DESCRIPTION

A clone is defined as a population of cells derived through mitotic division from single somatic cells of origin. This definition neither implies that a clonal population of cells must necessarily be genetically or phenotypically homogenous nor does it infer that a cell clone must be cancerous. Initially, cancer arises from a single somatic cell present in normal tissue, which during mitosis acquires gene mutations suitable for turning it into an early founder cell of a clonal tumor. Such mutations need to provide a biological advantage to that cell and its progeny as otherwise clonal expansion would not occur. A mutation knocking out a gene that is absolutely essential for the function of the cell of a cancer clone. A totally innocuous mutation in a gene of no interest to a particular cell would fail to alter its biological behaviour, and thus, it provides no basis for neoplastic transformation. It turns out that most gene mutations that succeed in driving a cell into a neo-plastic transformation severely alter cell proliferation, cell differentiation, cell survival, and other vital functions.

A single gene mutation is not enough to found a tumor. Rather, molecular carcinogenesis requires a sequential series of different yet linked mutational hits in at least two or more critical in the same cell. As that cell and progency continue to proliferate in a clonal fashion, daughter cells will subsequently acquire additional mutations,

and will grab yet new selection advantages such a proliferative drive, escape from cell death programs, etc. Such cells would establish sub-clones which may in turn become parent clones themselves. Therefore clonality assessment provides a snapshot of the clonal composition of a tumor at the time of analysis, but the clonal composition of neoplasm may change over time as tumor progress.

The recent concept of cancer stem cells added significant information to the established principles of the origin of a clonal tuomr. One assumes that mutation leading to cancer would occur in rare cells that are long-term residents in the respective tissues, for example in clonal mucosa or in the bone marrow. To become a “mother cell” of a tumor, a muted cell would not only have to proliferate and expand clonally, but it also needs to self-renew and acquire additional mutations. Cancer would thus originate from long-lived uncommon tissue stem cells with the ability for self-renewal that is a typical feature of a stem cell. When mutated, such cells become a cancer stem cell. The quiescence of these cells and their inherent resistance to drugs may account for a consideration amount of treatment failure in clinical oncology. Stem cells including cancer stem cells tend to be more resistant to cytostatic drugs, radiation, and perhaps to some of the “newer” targeted agents than more mature cells form the same clone or tissue. Ideally, cancer treatment would have to target cancer stem cells while spacig normal stem cells within the body’s tissues. Normal colonic mucosa and clonal cancer provides a good example of our yet incomplete notation of how cancer stem cells undergo self-renewal and generate cells that migrate up the crypt and differentiate to form specific cell types in clonal mucosa. Colon stem cells therefor must harbor the genetic programs that determine the structure and function of the specific regions or sections in the gut. There is evidence to support the notion that these cells, when they develop critical gene mutations, may multiple differentiated cell types. Mutations in the adenomatous polyposis coil gene placed on chromosome 5 may be an even in this chain of events.