Cancer

Cancer is a rupture of the social contract engaged by cells of the somatic lineage of multicellular organisms. This defection is caused by a collection of critical failures of the genetic systems evolved to ensure the correct and timely integration of the cellular unit's physiology at the tissue and organism's level.[1]

[1] Homeostasis, is the property of a system in which variables are regulated so that internal conditions remain stable and relatively constant.

Cellular transformation, the process through which the neoplastic phenotype arises, is caused by genetic and epigenetic alterations in somatic cells. Fortunately, most somatic cells exhibiting behaviours beyond physiological ranges end up being singled out and targeted for removal, either by eliciting an immune response or by triggering self-induced cellular death (*note about apoptosis here*). In order for a neoplasm to become clinically relevant, it has thus to acquire a number of alterations that consign it with the capacity to evade its host organisms' regulatory control mechanisms against unicellular defection.

From a functional point of view, the defining attribute of a neoplastic lineage is, arguably, its ability to sustain chronic proliferation, irrespectively of the social cues conveyed by its tissular context. Beyond that, the most conspicuous feature of cancer has to be the diversity of shapes and forms these proliferating masses can take when departing from the neatly organized architectural tissue types they arise from. An integrative framework to grasp this remarkable phenotypic plasticity has been proposed by Hanahan and Weinberg,[2] who proposed six essential and complementary capabilities for tumour growth and metastatic dissemination. These include self-suficiency in growth signals, insensitivity to growth-inhibitory (antigrowth) signals, evasion of programmed cell-death (apoptosis), limitless replicative potential, sustained angiogenesis, and tissue invasion and metastasis.

[2] Hanahan and Weinberg, 2000; and Hanahan and Weinberg, 2011

Specific genes, termed oncogenes, have the potential to induce transformation when disrupted in particular circumstances. Alterations in nearly 500 of such genes have been linked to cancer initiation and progression.[3] The multistep model for the nature of cancer posits that several such alterations are cumulatively required in order to initiate tumorigenesis and to evolve increasingly more aggressive and invasive tumour phenotypes.[4]

[3] Forbes et al., 2008

[4] Vogelstein and Kinzler, 1993

The number and patterns of somatic alterations vary dramatically across cancer types. At one extreme, childhood medulloblastomas can harbour fewer than ten genomic alterations, whereas over 50 000 somatic changes have been observed in primary lung adenocarcinoma samples.

In biological systems the instanciation of information is dna.

Clinical definition of cancer. A vast collection of diseases (almost 100, according to http://medical-dictionary.thefreedictionary.com/Cancer).

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