

## Hallmarks of Cancer

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### Background

It is considered that cancer's physio-pathology is fully based on 6 basic hallmarks along with two emerging hallmarks. N.B. Recognizing, understanding and widely accepting these concepts will increasingly affect the development of new means to treat cancer. Two emerging hallmarks are being updated which include; "development of genomic instability" and "inflammatory state of premalignant and frankly malignant lesions" Objectives to explain efficacy and importance of these hallmarks are:

1) *Sustaining proliferative signaling*: Most fundamental trait of cancer cells involve their ability to sustain chronic proliferation. Enabling signals are conveyed by growth factors that bind cell surface receptors, typically having tyrosine kinase domains. Cancer cells can acquire the capability to sustain proliferating signalling in a number of alternative ways.

2) *Evading Growth Suppressors*: Dozens of tumor suppressors that operate in various ways to limit cell growth and proliferation have been discovered through their characteristic inactivation in one or another form.

3) *Resisting Cell Death*: The apoptotic machinery is composed of both upstream regulators and downstream effector components.

4) *Enabling Replicative Immortality*: Non-cancer cells die after a certain number of divisions. Cancer cells escape this limit and are apparently capable of indefinite growth and division (immortality).

5) *Inducing Angiogenesis*: Angiogenesis is an important hallmark based on inducers and inhibitors. Inducers include vascular endothelial growth factor (VEGF) and acidic and basic fibroblast growth factor (FGF 1/2), which bind to transmembrane tyrosine kinase receptors displayed on endothelial cells.

6) *Activating Invasion and Metastasis*: Cancer cells can break away from their site or organ of origin to invade surrounding tissue and spread (metastasize) to distant body parts. The newly formed metastasis arise as amalgams of cancer cells and normal supporting cells conscripted from the host tissue.

### Aims & Objectives

To study the future aspects of emerging hallmarks and their influence on cancer treatment.

### Material & Methods

However, the utility of such attempts has been limited

because tumor cells have demonstrated an ability to develop resistance to drugs that disrupt a single pathway. This adaptability of cancer cells suggests to Hanahan and Weinberg that simultaneous targeting of two or more hallmark pathways may be a more effective approach to therapy.

### Results

Inflammation can contribute to multiple hallmark capabilities by supplying bioactive molecules to the tumor microenvironment, including growth factors that sustain proliferative signaling, survival factors that limit cell death, proangiogenic factors, extracellular matrix-modifying enzymes that facilitate angiogenesis, invasion, and meta-stasis, and inductive signals that lead to activation of EMT and other hallmark-facilitating programs. Additionally, inflammatory cells can release chemicals, notably reactive oxygen species, that are actively mutagenic for nearby cancer cells, accelerating their genetic evolution toward states of heightened malignancy. In addition to providing a solid basis for cancer research, the hallmarks have served to identify certain cell functions that have become therapeutic targets.

### Conclusions

What does it mean for patients? New ideas will undoubtedly influence future cancer treatment or not? How then let's take a look: Let's take the blocking of new blood vessel growth, or angiogenesis, as an example. This is an important hallmark and subsequently a key focus of recent drug development research. Several anti angiogenesis drugs have now been developed (for example, bevacizumab but even though they seem to work as they're supposed to in the lab, they haven't had the clinical impact that many predicted. Hanahan thinks that their 'hallmarks' approach could explain why. In the face of it this is a rather worrying prospect not only might the drugs not work, they could even make things worse. But Hanahan pointed to the positive prospect that, once we've developed drugs (or drug combinations) that can target multiple hallmarks at the same time, this would bring these new treatments back into play. Key words hallmarks, proliferation signaling, growth suppressors, Resisting cell death, replicating immortality, angiogenesis, invasion and metastasis.