

Cancer Hallmarks ABM — Quick Reference

Baseline: Cancer cell starts with the same division rate (0.008/step), death rate (0.005/step), and division cap (40) as normal cells. It can grow in any open direction but is blocked from dividing when both lateral neighbors are occupied.

Original 6 Hallmarks

- ① **Sustained Growth Signals** Division rate doubles ($0.008 \rightarrow 0.018/\text{step}$). Cancer no longer waits for external growth factors.
 - ② **Evade Growth Suppression** Contact inhibition removed. Normally a cancer cell cannot divide when squeezed between two lateral neighbors — with H2 on, it divides regardless of crowding.
 - ③ **Resist Apoptosis** Death rate drops $\sim 25\times$ ($0.005 \rightarrow 0.0002/\text{step}$). Cells accumulate rather than turning over.
 - ④ **Replicative Immortality** Division cap removed ($40 \rightarrow \infty$). Without this, growth plateaus once lineages hit the Hayflick limit.
 - ⑤ **Induce Angiogenesis** Blood vessels (purple) spawn near dividing cells. Without them, fully-surrounded cells die from hypoxia and the tumor core hollows out.
 - ⑥ **Invasion & Metastasis** Rare cancer cells stochastically undergo EMT and become migrants (green). Migrants move freely through tissue. Reaching the domain border triggers the metastasis alert.
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Advanced 6 Hallmarks (*always built on top of the original 6*)

- ⑦ **Immune Evasion** Killer T cells (gray) normally patrol toward the tumor and kill adjacent cancer cells with 12% probability per contact. With evasion on, T cells wander aimlessly and slowly, and kill probability drops to 1%.
- ⑧ **Genome Instability** Each cancer cell division has a 4% chance to produce a yellow mutant subclone with a $1.6\times$ division rate boost. Each lineage gets a distinct yellow shade. At 5+ distinct clonal lineages, targeted therapy fails (heterogeneity alert).
- ⑨ **Tumor-Promoting Inflammation** Macrophages (gray) are recruited from domain edges and secrete growth factors. Cancer cells near a macrophage divide $2\times$ faster.
- ⑩ **Metabolic Reprogramming (Warburg)** Cancer cells switch to glycolysis and survive in fully-surrounded, oxygen-poor positions that would otherwise trigger hypoxia death.

⑪ **Senescent Cell Promotion** SASP factors from senescent stromal cells boost division rate $1.8\times$ at the tumor edge (cells with at least one free neighbor). No visual cell — the effect is applied directly to edge cancer cells.

⑫ **Unlocking Phenotypic Plasticity** Cancer cells stochastically dedifferentiate into stem-like cells (purple). Stem-like cells divide $2.2\times$ faster, are nearly apoptosis-resistant, and partially evade T cell killing. When stem-like cells reach 20% of the domain, the treatment resistance alert fires. With H8 also on, each stem cell lineage gets a distinct pink shade; 5+ stem lineages triggers a second heterogeneity alert.