

Flipping The Cancer 'Off' Switch

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Cancer is the result of abnormal, uncontrolled cell growth. While every case of cancer is different, they all share some main features. In order for normal cells to become cancerous, several genes must begin to malfunction or not function at all. These genes can be categorized into two major groups: tumor suppressors and oncogenes. Tumor suppressor genes contain the information needed to make protein products that directly or indirectly block cell division or lead to cell death. Proto-oncogenes contain the information to make protein products that drive cell growth and promote cell survival. Abnormal proto-oncogenes are known as oncogenes, and their protein products are known as oncoproteins. Having too many oncoproteins and not enough functional tumor suppressors result in uncontrolled cell growth - a balance between the groups is necessary for normal cell activity.

Many cancer treatments are designed to target abnormal tumor suppressor proteins and oncoproteins. Generally, if a drug blocks an oncoprotein, cancer cells no longer receive that abnormal signal to keep growing, so these cancer cells die. If a drug activates a tumor suppressor protein, cancer cells are given the signal to stop growing, so these cells also die.

A recent study using mice found that a protein known as DT-061 can stimulate a tumor suppressor called PP2A to block key oncoproteins, including c-Myc. The ability of DT-061 to selectively target oncoproteins involved in cancer growth opens up possibilities for the development of new types of cancer treatments.

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