A longer lasting tumor blocker

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On the heels of dismaying reports that a promising antitumor drug could, in theory, shorten patients' long-term survival, comes a promising study by a Japanese team of researchers that suggests a potentially better option. The study appears in the May 11 issue of the *Journal of Experimental Medicine* (online April 27).

Many cancer treatments work by disrupting the formation of new blood vessels that feed growing tumors. Agents that block a vessel-promoting factor called VEGF have shown promise in human clinical trials. But recent studies in mice show that when treatment stops, tumor growth rapidly resumes. Now, Yoshiaki Kubota and colleagues find that blocking a different molecule, called M-CSF, suppressed tumor growth even after treatment was stopped.

Kubota and his team compared the efficacy of inhibitors against M-CSF and VEGF in mice with a certain kind of bone tumor. Three weeks of anti-VEGF treatment suppressed tumor growth but, similar to other recent reports, the tumors bounced back when the drug treatment was curtailed. Tumor growth in mice on a similar regiment of an M-CSF inhibitor remained suppressed in the absence of drug.

Another distinction between the two inhibitors was the type of vessel growth that was blocked. Blocking VEGF prevented dangerous vessels from growing such as those that feed tumors. But it also stopped beneficial vessels from growing, such as those that help injured tissues heal. Blocking M-CSF, on the other hand, only impeded bad vessel growth.

Most likely, the anti-M-CSF treatment had a lasting effect because it resulted in damage to the scaffolding that surrounds cancerous vessels, robbing the tumors of the structural support they need to grow. Meanwhile, the scaffold of mice treated with anti-VEGF remained intact.

M-CSF levels soar in patients with osteosarcoma (a malignant bone cancer), breast cancer and prostate cancer, making these cancers potentially the most responsive to M-CSF-blocking drugs Whether or not other types of cancer rely more on M-CSF than on VEGF for their blood supply remains unknown.

Source: Rockefeller University (news : web)

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