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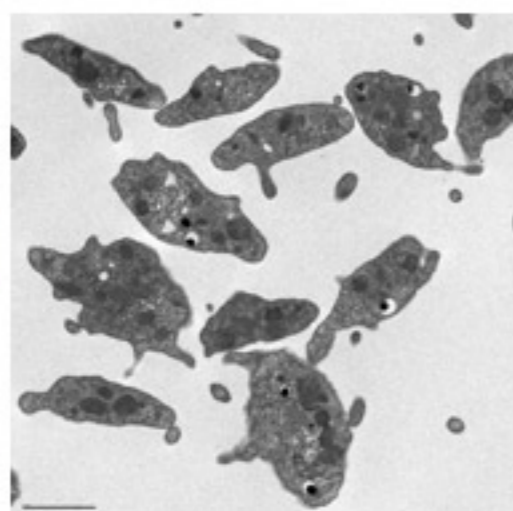
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## Defusing the Platelet Time Bomb

By Dave Moshier  
*ScienceNOW* Daily News  
23 March 2007

Cut your finger, and specialized blood cells called platelets rush in and plug the leak. Major wounds can overwhelm this rescue mission, however, and the donated platelets hospitals use are scarce and short-lived. Now, a team of researchers has isolated the proteins that spur platelets to kill themselves--a discovery that could double the cells' donated shelf life and boost their numbers in weakened patients.

### Ticking away.

Platelets patiently wait to clot your next wound.

Credit: Ben Kile / WEHI

All cells with a nucleus carry a self-destruct program, but platelets lack this genetic control center. Nevertheless, the cells survive just 10 days in the bloodstream--and 5 days in donated plasma--so they must contain some sort of "ticking time bomb," says Ben Kile, a molecular geneticist at the Walter and Eliza Hall Institute of Medical Research (WEHI) in Victoria, Australia.

Kile and colleagues located this auto-destruct mechanism by noticing that the anticancer drug ABT-737 slashes platelet counts. "This chemical really turns on the death switch," says co-author and WEHI molecular biologist David Huang. The team found that the drug blocked a protein called Bcl-x<sub>L</sub> in platelets. When the researchers knocked out the genes responsible for making Bcl-x<sub>L</sub> in mice, platelet counts dropped about 70 percent.

The killer appears to be a protein called Bak, whose activity increased when Bcl-x<sub>L</sub> was off the scene. Conversely, removing the gene for Bak in mice boosted platelet numbers by roughly 30 percent, the team reports today in *Cell*. Giving the cancer drug to Bak-lacking mice had almost no effect on their platelet numbers. In essence, says Kile, Bak is the "bomb" and Bcl-x<sub>L</sub> is the "clock" that tells the bomb when to go off. Kile says finding a way to turn back this clock "could be an entry point to a lot of breakthroughs" in diagnosing and treating platelet disorders caused by genetics and chemotherapy drugs.

"This gives us new angles for approaching platelet problems," agrees Amy Geddis, a pediatric hematologist at the University of California, San Diego. "If platelets could last twice as long, I might give one blood transfusion to some patients each day instead of three," she says.

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