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Battling defiant leukemia cells

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Two gene alterations pair up to promote the growth of leukemia cells and their escape from anti-cancer drugs, according to a study in *The Journal of Experimental Medicine*.

Chronic myeloid leukemia (CML) is an aggressive cancer of the blood that is often treated with a drug called Imatinib (a.k.a. Gleevec). Although Gleevec is highly effective, some cancer cells can develop resistance to the drug. The mechanism that drives this resistance is not completely understood, but there is evidence that cancerous stem-like cells are particularly resistant and help to perpetuate disease.

CML stem-like cells rely on a protein called ?-catenin for their survival and maintenance, and deletion of this protein in mice delays cancer growth. Another protein, IRF8, inhibits the survival of cells and is often reduced in CML cells, particularly in patients with advanced disease.

A group of scientists from the Max Delbrück Center for Molecular Medicine in Berlin now show that CML cells with activating ?-catenin mutations are particularly resistant to Imatinib. Resistance is boosted further when this defect teams up with those that abolish IRF8 expression, rendering CML cells highly aggressive in mice. In fact, in CML cells lacking IRF8, ?-catenin activation was essential for the cell's growth and survival.

Combined treatment with drugs that restore the expression of IRF8 and those that inactivate ?-catenin may force these cells to surrender to treatment, preventing fatal leukemia progression.

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