C212 can kill both dormant and active leukemia cells

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Video Byte

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Abstract

Leukemia relapse after conventional treatments is difficult to treat and has high patient mortality. Relapse is driven by the reawakening of previously quiescent cancer cells. Traditional treatments are ineffective against quiescent cells, as they instead target rapidly dividing cells. A new study investigated a potential dual-target treatment, C212, in cell culture. C212 is a derivative of curcumin, a compound that has shown anticancer properties in previous research. C212 was effective against both growing and quiescent leukemia cells. C212 drives quiescent leukemia cells deeper into dormancy by increasing their exit threshold, and then kills these deep-quiescent cells. This differs from previous strategies that awaken quiescent cells to kill, which runs the risk of wakening treatment-resistant subpopulation of cells. Molecular docking and experimental analyses showed that C212 could bind to Hsp90 and interferes with its function. Hsp90 is a chaperone protein that stabilizes proteins critical to cellular survival and growth and administration of C212 led to the degradation of Hsp90 clients and aggregation of misfolded proteins in leukemia cells. Further study of the unique dual-effect of C212 in eliminating both growing and quiescent cells could one day lead to new therapies for treating leukemia and preventing relapse.