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the specific mechanisms underlying the regulation of the traf2-bound and nontraf2-bound ciap1 and ciap2 by birinapant are unknown. it was recently demonstrated that a bivalent smac mimetic, bv6, induced the autoubiquitylation and degradation of the single bir domain-containing protein, ml-iap, whereas a related monovalent smac mimetic had no effect. this result suggested that bir domain crosslinking may have a mechanistic role in bivalent smac-mimeticinduced e3 ligase activity (42). it is therefore possible that a similar ciap bir3 domain crosslinking may be responsible for the selective degradation of traf2-bound ciaps by birinapant. in this article, i thought it might be interesting to look at the trajectory of these evolutions in it to see what smac may bring over the next several years. one of the key changes over time, throughout all these evolutions, has been the exponentially increasing processing power of computers, and the steady growth in the number of computing devices, applications and users. the table below shows the rough magnitude of these changes across the various computing eras dates and numbers are approximate just to give a sense of the order of magnitude. formation of the smac/diablo complex upon cellular apoptosis is mediated by the tripartite interaction of the bir2 domain of diablo with its cognate bir2 binding partner, smac. binding of smac to the bir2 domain of diablo disrupts the binding of ubiquitin ligase e3 ligase, ciap1, and procaspase-8 at the diablo/ciap1 binding site, preventing the ubiquitination and activation of procaspase-8. activated caspase-8 then cleaves and activates effector caspases such as caspase-3, -6, and -7, which in turn degrade critical cellular proteins required for cell survival.

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this report, together with a recent report, suggests that cytotoxic activity is a common feature of gem-b and sn-38-mediated activity (18). unlike gem-b, however, sn-38 has been shown to kill only cells containing wild-type tp53 (18). since tp53 is the most common tumor-suppressing gene mutated in human cancer, it was of interest that gem-b, sn-38, and sn-38 plus smac-mimetic efficiently killed only p53-mutant cell lines. the ability of gem-b and sn-38 to kill only tumor cells with wild-type tp53 suggests that gem-b and sn-38 may have additional proapoptotic functions in addition to their previously established roles as genotoxic agents. p53-independent cytotoxicity of sn-38 and gem-b has been observed in other contexts. for example, sn-38 was shown to exhibit differential cytotoxicity in primary versus cell line-derived cell populations with tp53 mutations (54, 55). cytotoxic activity of gemcitabine and sn-38 has been observed in nonmalignant cells. for example, sn-38 plus gemcitabine induces cytotoxicity in normal human gastric epithelial (hugo) cells, whereas it causes no apparent cell death in chinese hamster ovary (cho) cells (18). ciap1 knockdown led to increased trail sensitivity in colorectal cancer cells, further supporting an important role for ciaps in cancer cell survival. however, the ciap knockdown studies were performed with trail as the inducer of caspase activation, but this could be circumvented with birinapant because of the high degree of overlap between the smac mimetic and tnf signaling pathways. since tnf is a constitutively expressed cytokine, it provides a physiological setting for the testing of this ciap e3 ligase inhibitor. birinapant did not have direct antiproliferative effects in colorectal cancer cell lines, but inhibited colony formation, an assay that correlates with the potential for metastasis. birinapant did not seem to have any effect on the survival of normal primary cells with the exception of the melanocytes, possibly due to their reduced proliferative potential in comparison with cancer cells. birinapant showed minimal to no toxicity in mice and had a favorable pharmacokinetic profile. 5ec8ef588b

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