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> Signal transduction pathways are typically controlled by protein-protein interactions, which are mediated by specific modular domains. One hypothetical use of such interaction domains is to generate new signaling pathways and networks during eukaryotic evolution, through the joining of distinct binding modules in novel combinations. In this manner, new polypeptides may be formed that make innovative connections among preexisting proteins. Adaptor proteins are specialized signaling molecules composed exclusively of interaction domains, that frequently link activated cell surface receptors to their intracellular targets. Receptor tyrosine kinases (RTKs) recruit adaptors, such as Grb2 and ShcA, that activate signaling pathways involved in growth and survival, whereas death receptors bind adaptors, such as Fadd, that promote apoptosis. To test the ability of interaction domains to create new signaling pathways, we have fused the phosphotyrosine recognition domains of Grb2 (Scr homology 2 domain) or ShcA (phosphotyrosine-binding domain) to the death effector domain of Fadd. We find that these chimeric adaptors can reroute mitogenic or transforming RTK signals to induce caspase activation and cell death. These hybrid adaptors can be used to selectively kill oncogenic cells in which RTK activity is deregulated.

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