

**F805** Yeast two-hybrid screens for proteins interacting with cell death protein Reaper in *Drosophila melanogaster*

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Reaper plays an important role in programmed cell death throughout development of *Drosophila melanogaster*. We are using reaper in yeast two-hybrid assay for detection of protein-protein interactions *in vivo* to identify other genes involved in the programmed cell death pathway and to further understanding of how Reaper might function. The GAL4 DNA-binding domain fused to *reaper* was used as bait to screen a *Drosophila* embryonic cDNA library in which the cDNA was fused to the GAL4 activation domain. In this study, we isolated several positive clones that interact with Reaper and obtained cDNA sequences. Now, we are in the process of determining the chromosomal map positions and the expression patterns of these positive clones.

**F806** Cytosolic Calcium Alteration and Cell Injury by Silica in Rat Hepatocytes

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The purpose of this study was to clarify the effect of silica on cytosolic free calcium mobilization and cell injury in primary cultured rat hepatocytes. Cytosolic free calcium concentration ( $[Ca^{2+}]_i$ ) was measured employing calcium sensitive fluorescent dye, Fura-2/AM, and cell injury was evaluated by determination of cellular ATP contents. Silica increased  $[Ca^{2+}]_i$  in a concentration-dependent manner in hepatocytes ( $10^{-5}$  -  $10^{-2}$  M). Silica caused a biphasic increase in  $[Ca^{2+}]_i$  which was composed of an initial rapid rise and following sustained phase.  $Ca^{2+}$  removal from the medium resulted in abolishment of initial and sustained phase of silica ( $10^{-2}$  M)-induced  $[Ca^{2+}]_i$  in hepatocytes. The pretreatment with nifedipine (1  $\mu$ M) attenuated silica-induced  $[Ca^{2+}]_i$  increases. Silica decreased cellular ATP contents in a dose-dependent manner. This silica-induced cell injury was attenuated by the pretreatment with EGTA (100  $\mu$ M) and nifedipine (1  $\mu$ M). This study suggests that the elevation of  $[Ca^{2+}]_i$  caused by silica may be due mainly to influx through a plasma membrane  $Ca^{2+}$  channel and hepatotoxicity by silica relate with alteration of calcium homeostasis.