

IN BRIEF

DEVELOPMENT

Generation of a functional mammary gland from a single stem cell.

Shackleton, M. *et al. Nature* **439**, 84–88 (2005)

Although the existence of mammary stem cells (MaSCs) had been postulated, the identity and purification of MaSCs has proved difficult owing to the lack of defined markers. Shackleton *et al.* now describe the potential isolation of mouse MaSCs using specific cell-surface markers and show that a single cell can reconstitute a complete mammary gland *in vivo*.

DNA DAMAGE

Multifactorial contributions to an acute DNA damage response by BRCA1/BARD1-containing complexes.

Greenberg, R.A. *et al. Genes Dev.* **20**, 34–46 (2005)

This study describes a new model for how genotoxic stress enables BRCA1 (breast cancer-1) to execute a diverse set of DNA-damage responses. Greenberg *et al.* examined the biochemical and cellular localization activities of BRCA1–BARD1 (BRCA1-associated RING domain-1) heterodimers and showed that after genotoxic stress, two distinct DNA-damage-dependent complexes emerge. BRCA1–BARD1 specifically interacts with the DNA-damage-response proteins TopBP1 and Mre11/Rad50/NBS1, and these interactions depend on the activity of specific DNA-damage-activated protein kinases.

CELL CYCLE

Caspase-mediated specific cleavage of BubR1 is a determinant of mitotic progression.

Kim, M. *et al. Mol. Cell. Biol.* **25**, 9232–9248 (2005)

These authors found that treating HeLa cells with spindle-disrupting agents caused caspase activation and subsequent cleavage of the mitotic checkpoint protein BubR1, which removed the mitotic block. Two specific, evolutionarily conserved caspase cleavage sites were identified in BubR1, and expressing BubR1 that was mutated at both sites increased the mitotic delay induced by spindle disruption. This links the control of a mitotic checkpoint protein to caspase activation for the first time, and shows that this pathway might be involved in the elimination of defective cells.

RNA METABOLISM

Ge-1 is a central component of the mammalian cytoplasmic mRNA processing body.

Yu, J. H. *et al. RNA* **11**, 1795–1802 (2005)

Multiple processing body factors and the ARE binding protein TTP activate mRNA decapping.

Fenger-Grøn, M. *et al. Mol. Cell* **20**, 905–915 (2005)

P-bodies — mRNA-processing bodies — regulate gene expression by degrading cytoplasmic mRNA. To further our understanding of P-bodies, these two studies identified and characterized novel components of these cellular structures in mammals. Both papers identified the protein Ge-1/Hedls as a central component of P-bodies, and showed that it is important for the decapping step of mRNA degradation.