New study bolsters beliefs about DNA repair

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Aucott et al. report the first in vivo experiments on the heterochromatin protein 1 (HP1) family, which sidles up to silent DNA. The results, to be published in the Nov. 17 issue of the *Journal of Cell Biology*, add to the evidence that the different versions of the proteins help cells fix broken DNA.

The function of HP1 proteins has puzzled researchers. The proteins, which come in three forms in mammals, cozy up to heterochromatin—the tightly wound sections of DNA where genes are usually inactive. Early studies indicated that the proteins' job was to turn genes off. But recent work suggested that the proteins are essential for repairing damaged DNA. These results came from in vitro studies, however, and the proteins' powers in vivo remained uncertain.

Aucott et al. created the first mouse strain missing one of the HP1 versions, HP1b. The animals die shortly after birth because their lungs don't inflate. The rodents show brain defects as well. Large numbers of neurons die, for example, and the neural stem cells in the cortex divide sluggishly. Both effects could arise from unfixed DNA. When the researchers grew brain cells from HP1b-lacking mice in culture, they saw clear indications of genomic instability that can result from faulty DNA repair, including unpaired sister chromatids that separated prematurely and even extra sets of chromosomes. The HP1 proteins latch onto the methylated version of the H3 histone, but how this interaction promotes repair is an unanswered question.

Source: Rockefeller University