# Lamin B locks up Oct-1

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A large fraction of the transcription factor Oct-1 is associated with the inner nuclear envelope, but how and why it is retained there was unknown.

As for how, Malhas et al. show—in the January 12, 2009 issue of the *Journal of Cell Biology* (www.jcb.org)—that Oct-1 binds to lamin B1, a prominent intermediate filament that lines the nuclear envelope, and in cells expressing a drastically truncated mutant of lamin B1, Oct-1 was disassociated from the nuclear envelope.

This left the question, why? The authors asked whether disrupting lamin B1-Oct-1 interactions could affect the expression of genes regulated by Oct-1. Indeed, in cells with truncated lamin B1, they found that expression of several Oct-1-regulated genes was altered because more Oct-1 could bind at these genes' promoters. Among the genes was a group involved in the oxidative stress response. As a result, these mutant cells accumulated higher levels of reactive oxygen species than wild-type cells.

It remains to be seen whether and how lamin B1-Oct-1 interactions are actively regulated in cells to help control gene expression. But, it is evident from these results that perturbation of lamin B1-Oct-1 interactions can make cells more vulnerable to oxidative stress. This could be particularly important in aging cells, where nuclear envelope integrity (and lamin B1 localization) is often perturbed, says author David Vaux. Lamins support the structure of the nucleus, and compromised nuclear structure has been a suspected cause of aging;

another type of lamin, lamin A, is known to cause a premature aging disease when faulty. Increased production of reactive oxygen species—due to the perturbation of lamin B1 in mature cells—could be another way in which lamins contribute to the aging process.

Source: Rockefeller University