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HP1 dynamics

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Gene silencing can be achieved by packaging of genes into repressive **heterochromatin** domains. In the January 31 *Science* two papers describe the dynamics of mammalian heterochromatin and the associated **heterochromatin protein 1 (HP1)**. Festenstein *et al.* generated transgenic mice that express a chimeric protein of HP1 β fused to green fluorescent protein (GFP) in T lymphocytes (*Science* 2003, **299**:719-721). HP1 β -GFP was seen in heterochromatic foci in T-cell nuclei. Using fluorescence recovery after photobleaching (FRAP) in living T cells, Festenstein *et al.* show that HP1 is highly mobile suggesting that heterochromatin maintenance is dynamic. HP1 mobility was increased upon T cell activation. In an accompanying paper Cheutin *et al.* describe a similar approach using GFP fusions of HP1- α , - β and - γ in hamster cells (*Science* 2003, **299**:721-725). The GFP-HP1- α and - β forms were localized in larger heterochromatin domains. Experiments in fibroblasts from mice lacking the Suv39h histone methyltransferases demonstrated that HP1 mobility in heterochromatin is due to its binding to sites created by Suv39h. HP1 binding appears to correlate with global chromatin organization. Both the chromodomain and the chromoshadow domain are required for HP1 to bind to chromatin *in vivo*.

References

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3. The HP1 protein family: getting a grip on chromatin.