

# Cloning of an engineered histone cluster in *Drosophila melanogaster*

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Unrepaired DNA damages could lead to cancer formation. To allow repair of these damages, the repair proteins must assess the damage site, so the condensed DNA needs to be looser. Our group is interested in to understand chromatin changes during DNA repair processes by using novel experimental system.

In eukaryotic cells the nucleus contains highly condensed DNA. The base of the chromatin structure is the evolutionarily conserved histone protein family. The histone proteins necessary for nucleosome assembly by forming a heterooctamer with two copies of H2A, H2B, H3 and H4. Finally the linker histone H1 requires for the proper chromatin condensation. All of the histone proteins can be post-translationally modified. These PTMs are the main rulers of epigenetic regulation. Processes using DNA as a template (transcription, replication and DNA repair) are greatly affected by the chromatin structure. Unimproved breaks lead genome instability or translocations which easily results tumor formation. Our aim is to understand how does the chromatin structure change around the break during the DNA repair and what is the link between unique histone PTMs and the mechanisms of the repair. We plan to set up an experimental system by which we will be able to study how do unique histone modifications affect the DSB repair. The advantage of this new experimental system is the clone of the histone cluster which contains all of the canonic (H2A, H2B, H3, H4) and linker (H1) histone genes in *Drosophila melanogaster*.