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**ABSTRACT
BOOK**

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*.

Study of chromatin structural changes during DNA Double Strand Break repair

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The DNA repair pathways are essential to minimize the chance of cancerous malformations. During the DNA repair processes histone post-translational modifications are important to open chromatin structure. The goal of our team is to set up a novel experimental system where we can follow the chromatin changes during DNA repair.

In eukaryotic cells the DNA is packed onto a protein backbone to form chromatin. When different process which uses the DNA as a template (transcription, replication) takes place the tight chromatin structure opens then close again. The chromatin structural changes affects the probability of DNA damage occurrence and also influences the recruitment of DNA repair proteins. Slower repair kinetics or improper repair increase the probability of chromosome rearrangement leading to genome instability and cancer development. In order to understand the regulatory role of the chromatin structure during DNA damage response and repair we set up a novel system where we can study histone modifications around the damaged DNA. We also try to identify the novel histone modifiers implicated in DNA repair and we plan to follow how they are regulated in space and time at the break site. Understanding the regulation of chromatin structural changes during DNA repair will help to identify how DNA damage influences genome stability. These result would also help identifying new key targets in DNA damage repair and the final goal of the project is to find potential biomarkers which could be used in anti-cancer therapies.