When a cut makes the difference
DNA damage incision from human cells to *C. elegans*

1. ERCC1-XPF displays tissue-specific activity which may explain why in humans hereditary ERCC1-XPF deficiency affects tissues differently. (This thesis)

2. Persistent targeting of core NER proteins to DNA lesions is associated with additional Cockayne syndrome features present in a sub-group of xeroderma pigmentosum patients. (This thesis)

3. Differential engagement of ERCC1-XPF in NER or ICLR is controlled by pathway-specific interactions. (This thesis)

4. Functional analysis of single XPF mutations carried as heterozygous compound by XP group F patients is vital to dissect the contribution of each allele to the disease. (This thesis)

5. Despite the fact that TTDN1 is implicated in non-photosensitive trichothiodystrophy, TTDN1 is involved in the DNA damage response. (This thesis)

6. The common practice of freely sharing genetic and cellular information within the *C. elegans* research community should be a golden standard for all scientific disciplines.

7. Learning more about patients with cancer predisposition syndromes is fundamental to improve cancer prevention, surveillance, treatment and follow-up of affected individuals and their families.

8. Flawless genome maintenance is essential to avoid mutagenesis, but precludes evolution.

9. Identifying somatic mutation profiles of cancer cells does not only provide a historical record of their mutational experiences, but is also essential to predict its development and responses to therapeutics.

10. 60-80% of human genes and 40% of those associated with human diseases have clear orthologs in the *C. elegans* genome. *C. elegans* is not “just a worm”!

11. To achieve great things, two things are needed: a plan and not quite enough time. (Leonard Bernstein)

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