Propositions belonging to the thesis

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)

Mariangela Sabatella, 2019