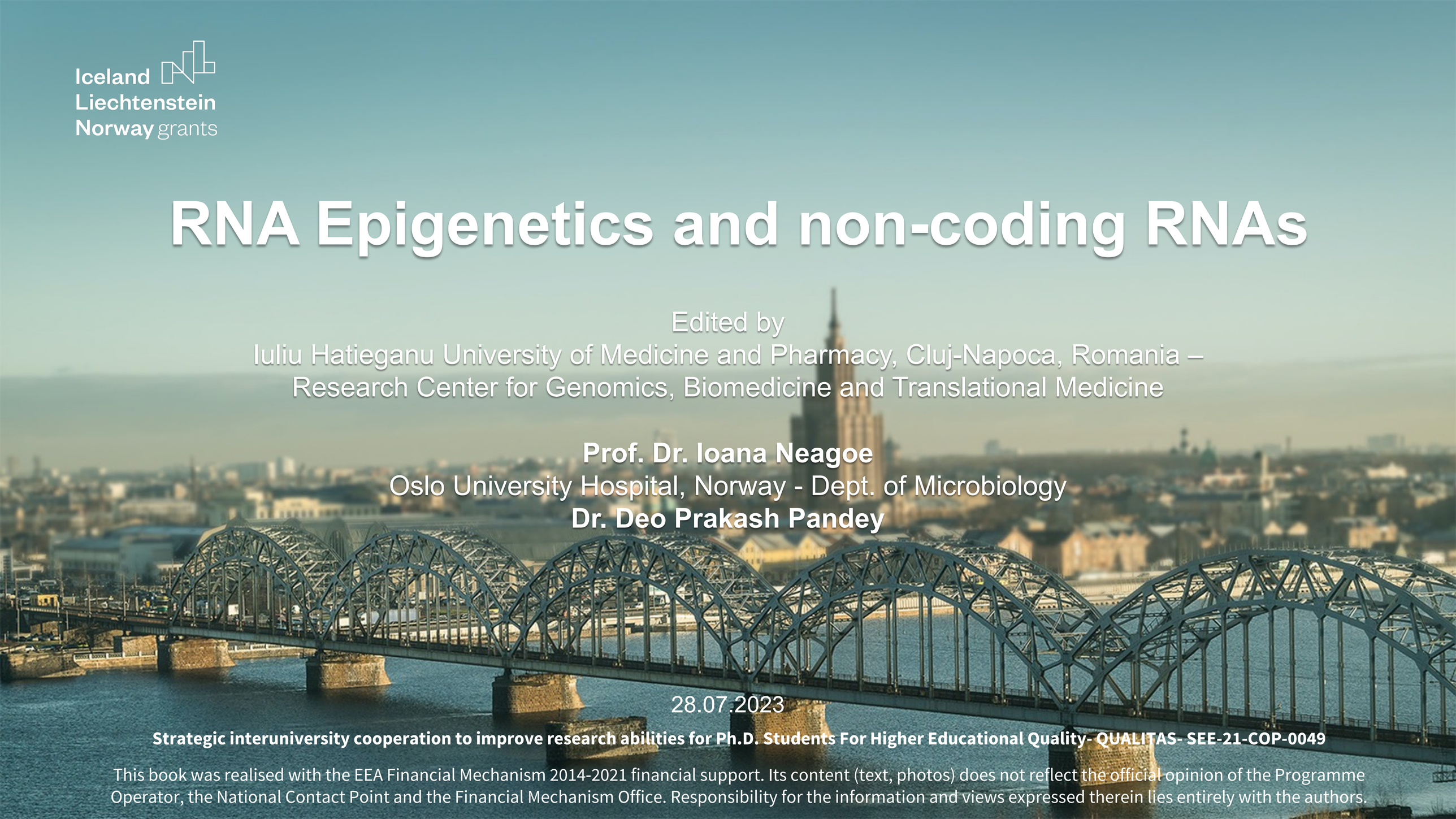

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RNA Epigenetics and non-coding RNAs

Edited by

Iuliu Hatieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania –
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Strategic interuniversity cooperation to improve research abilities for Ph.D. Students For Higher Educational Quality- QUALITAS- SEE-21-COP-0049

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RNA EPIGENETICS AND NONCODING RNAs book comes as a small compendium for a new generation of researchers in Health and Life Sciences, opening access to a field not only of great actuality but also of complex research and technologies that can decipher the human genome, state-of-the-art therapeutic solutions and understanding the heterogeneity of all cellular entities that make up the human body.

The book is written for all specialists in molecular biology and genomics, as well as for PhD students working in the field of tranlational medicine or intending to deepen their knowledge.

An excellent initiative was realized with the support of two institutions, the Genomics Center from Iuliu Hatieganu University of Medicine and Pharmacy and Oslo University Hospital, through the SEE programme.

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RNA EPIGENETICS & NON-CODING RNAs

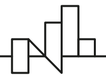
UMF

Oslo University Hospital

GENOMICS

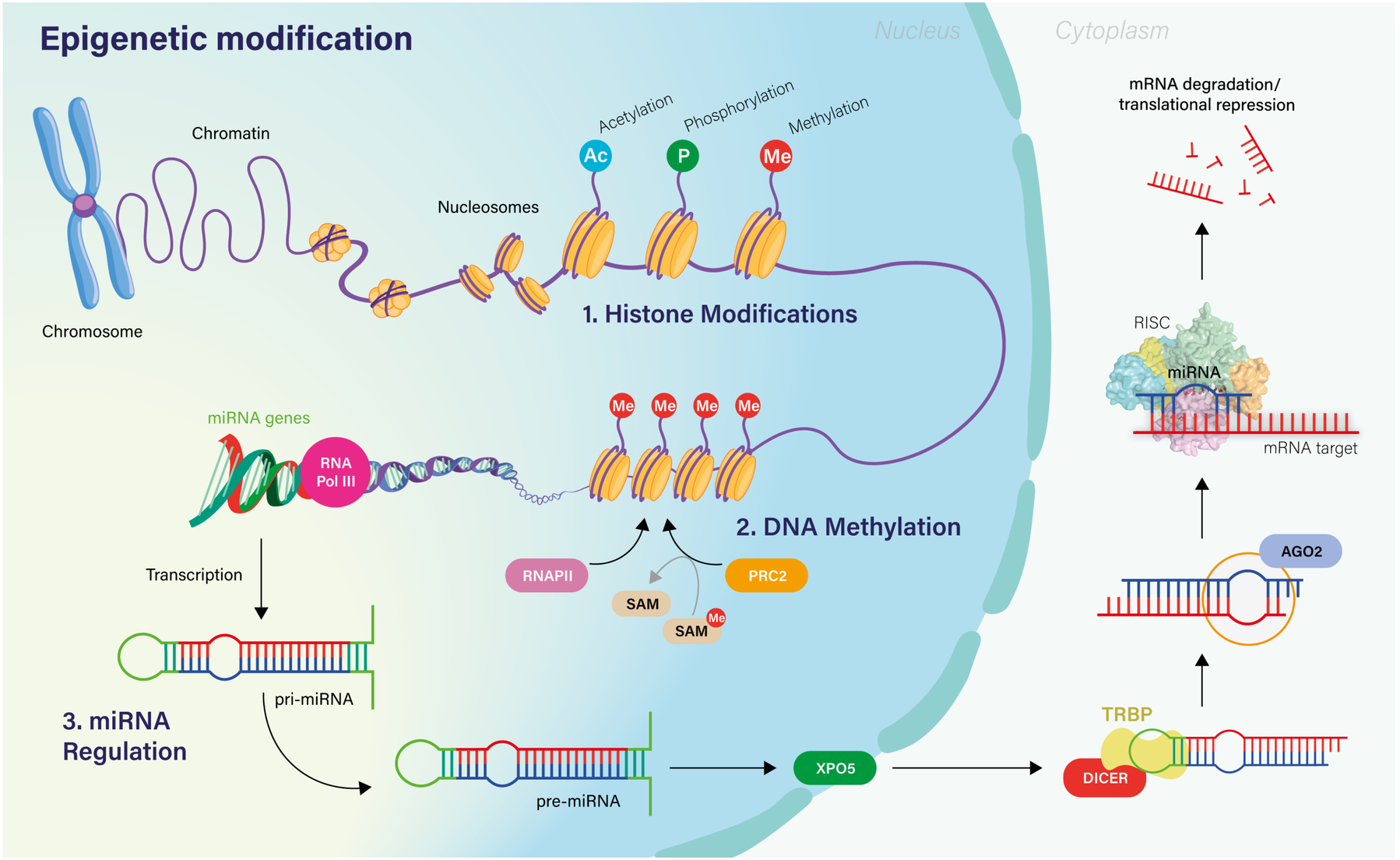
Chapters

1. Introduction to Epigenetic Control
2. Overview of epigenetic inheritance “coded by” proteins, DNA, and RNA
3. Genomic Imprinting and Epigenetic Reprogramming
4. The Influence of the Environment on Epigenetic Control
5. Reversible modifications on protein, DNA, and RNA and their role in cancer
6. Non-coding genome. Pervasive transcription
7. Long non-coding RNAs
8. Centromeres & telomeres & Genome integrity
9. Mobile Elements
10. MicroRNAs in therapeutical clinical applications



2. Overview of epigenetic inheritance “coded by” proteins, DNA, and RNA

Figure 1. Epigenetic modifications involved in the regulation of gene expression. 1. Histone Modifications; 2. DNA Methylation; 3. miRNA Regulation.



4. The Influence of the Environment on Epigenetic Control

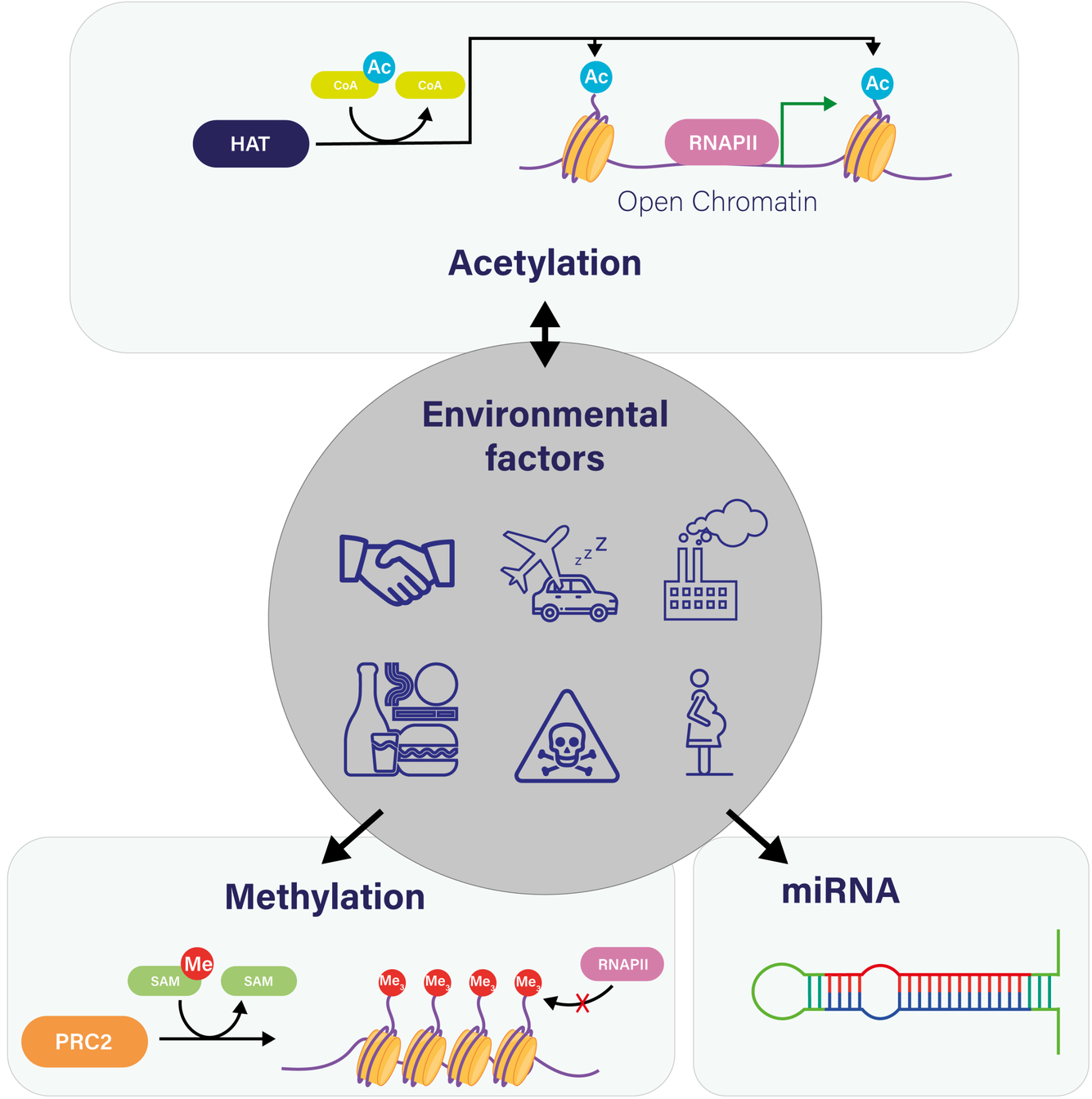


Figure 1. The main environmental factors that influence epigenetic changes.

5. Reversible modifications on protein, DNA, and RNA and their role in cancer

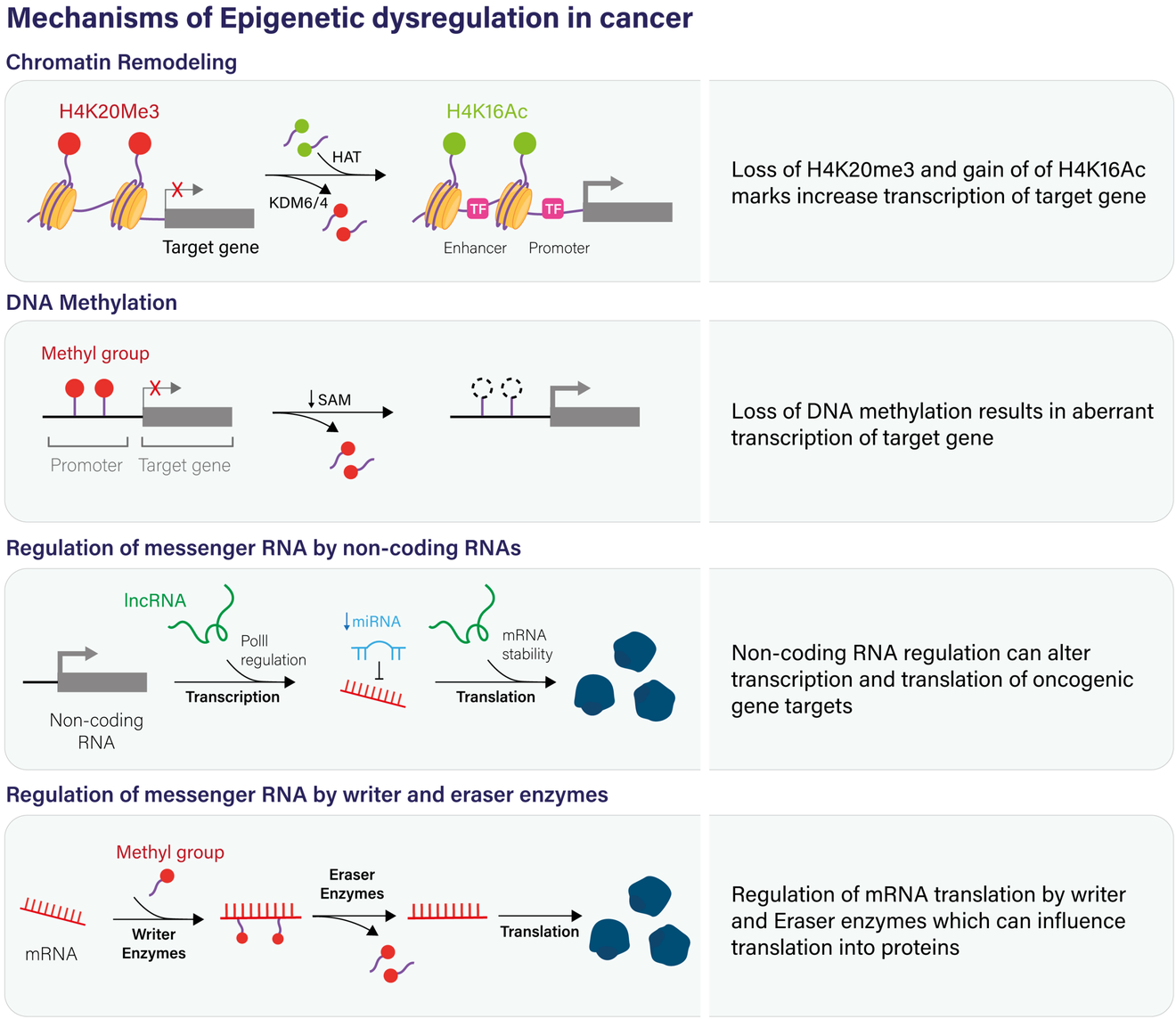


Figure 1. The principal mechanisms of epigenetic regulation via reversible modifications in cancer

8. Centromeres & telomeres & Genome integrity

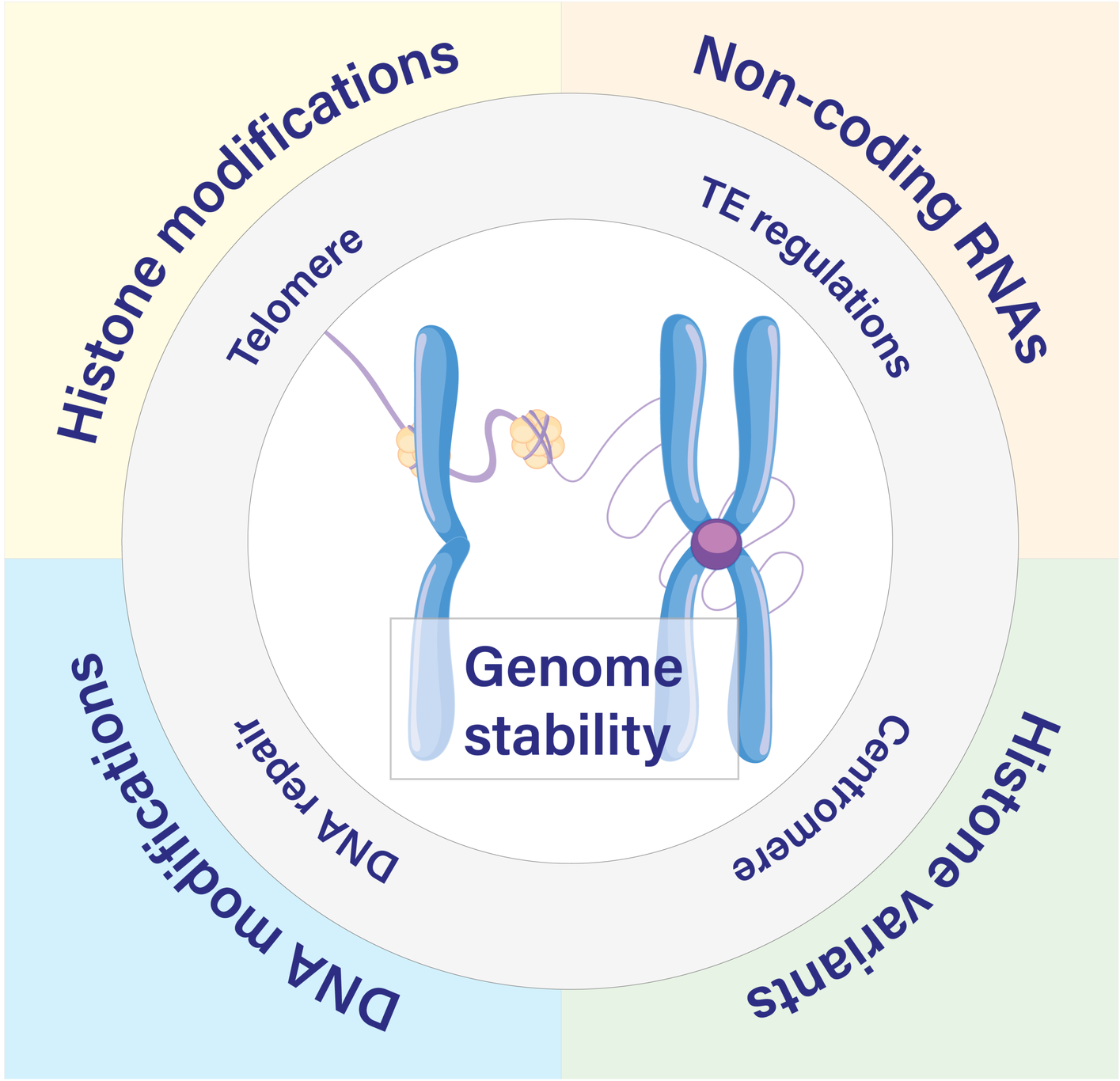
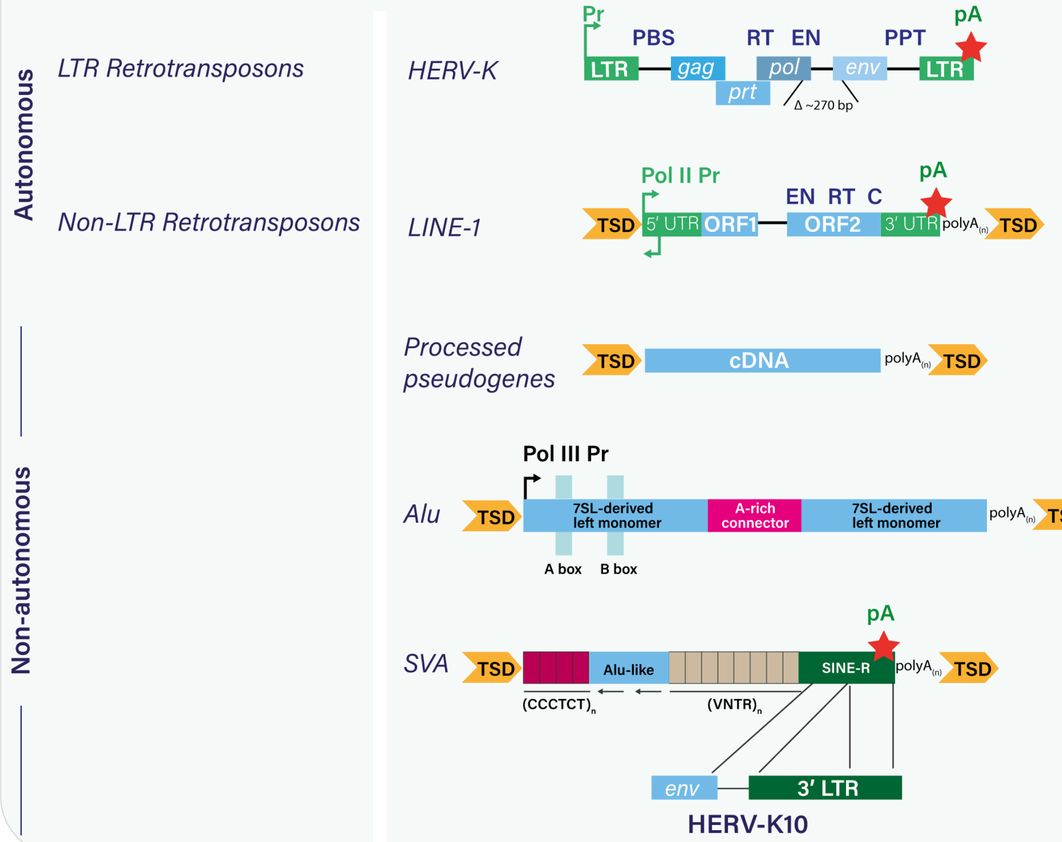


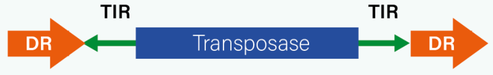
Figure 1. The interaction between epigenetics and genome stability. In the middle, is shown the critical significance of genome stability for the survival of organisms and the intricate interplay between epigenetics and this fundamental process. The focus lies on four major aspects of genome stability, namely telomeres, centromeres, TE regulation, and DNA repair, alongside the epigenetic mechanisms that actively contribute to these pathways.

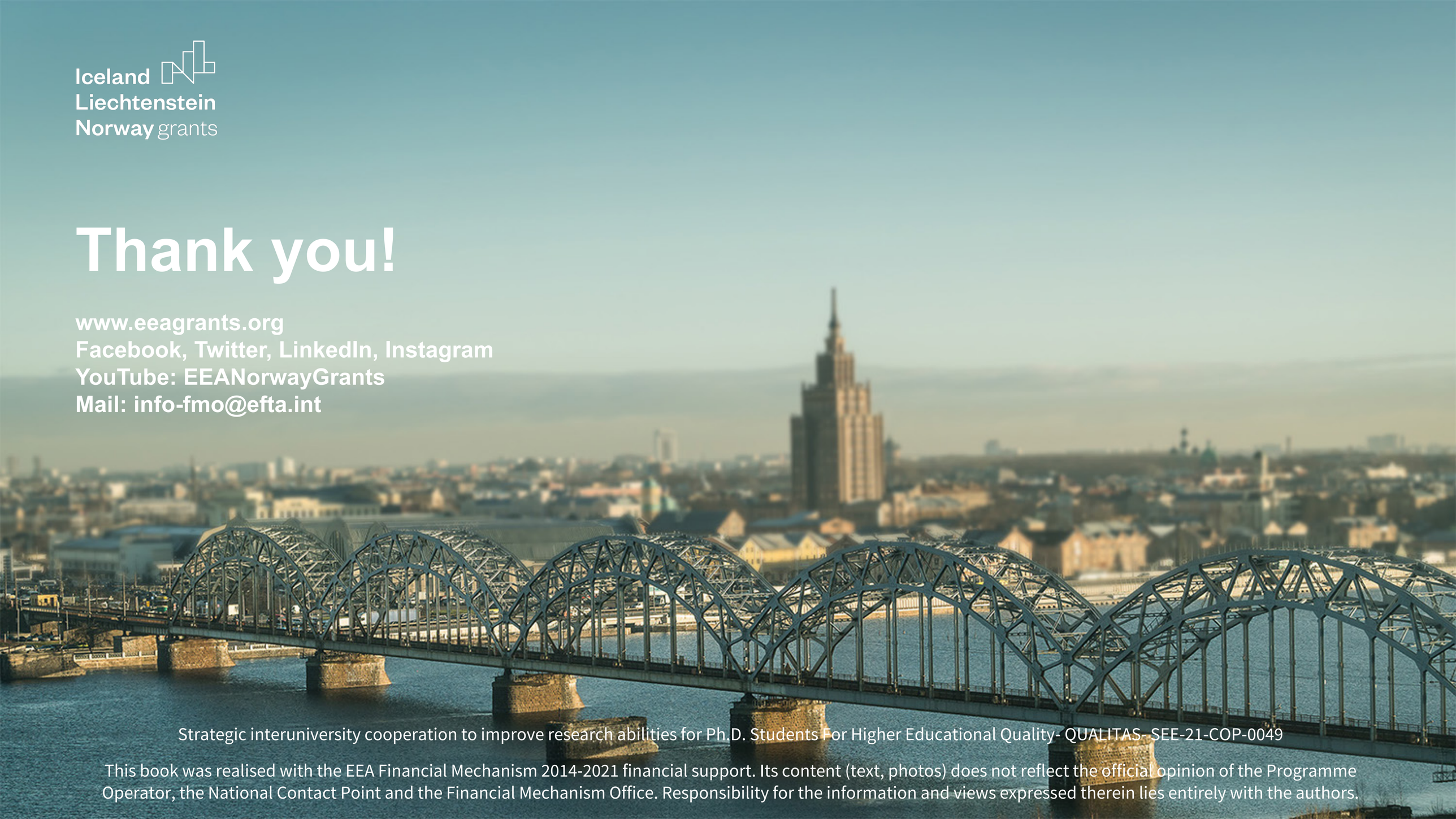
9. MOBILE ELEMENTS

Figure 1. Classification of mobile elements. Based on the mechanism of mobilization, mobile elements or transposable elements are classified in Class I or Retrotransposons which are mobilized by a “copy and paste” mechanism, and Class II or DNA transposons which use a “cut and paste” mechanism for mobilization. Class I retrotransposons are further divided into autonomous, that encode the factors needed for mobilization, and non-autonomous retrotransposons that rely on the factors encoded by autonomous retrotransposons for mobilization. The autonomous retrotransposons include the long terminal repeats (LTR) such as the human endogenous retrovirus K (HERV-K) that resemble retroviruses in structure, and the non-LTR retrotransposons with long interspersed nuclear element 1 (LINE-1) being the most representative. The non-autonomous non-LTR retrotransposons comprise the processed pseudogenes, and the Alu and SVA elements. These types of retrotransposons feature the hallmarks of LINE-1 transposition, namely the poly(A) n tail, and the target site duplications (TSDs) at both ends. Class II DNA transposons elements, such as the TC1/Mariner encodes the transposase gene, two inverted repeats (ITR) and the structure is flanked by two direct repeats (DR). The copy number and percentage in the human genome (HG), and evolutionary age for each type of mobile elements are indicated. Promoters (P) of autonomous retrotransposons are depicted as bent arrows. PBS – primer binding site; gag – capsid polyprotein; prt – protease; pol – polymerase; RT – reverse transcriptase domain; EN – endonuclease domain; env – envelope; Δ ~270 bp – deletion of approximately 270 bp; PPT – polypurine tract; pA – polyadenylation signal; UTR – untranslated region; ORF – open reading frame; C – cysteine rich domain; VNTR – variable number tandem repeats; SINE – short interspersed nuclear elements; cDNA – complementary DNA; MYA - million years ago

Transposon classification		Structure	Copy number	% in HG	Evolutionary age
Class I Transposons - “copy and paste” mechanism					
Autonomous	*LTR Retrotransposons*	*HERV-K*	~ 203000	~ 8 %	~ 45 MYA
Autonomous	*Non-LTR Retrotransposons*	*LINE-1*	~ 520000	~ 17 %	~ 170 MYA
Non-autonomous		*Processed pseudogenes*	~ 8000-200000		~ 75 MYA
Non-autonomous		*Alu*	~ 1.1 mil.	~ 11 %	~ 65 MYA
Non-autonomous		*SVA*	~ 2800	~ 0.1 %	~ 18-20 MYA
Class II Transposons - “cut and paste” mechanism					
DNA Transposons	*TC1/ Mariner*		~ 100000	~ 3 %	~ 81-150 MYA







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