In the only figure in **"The Origin of Species"**, Darwin illustrated his theory stating that species arise from existing ones, with extinction events occurring continuously, while simultaneously other species persist or give rise to new taxa. Since the publication of his breakthrough theory, we have gained a much deeper understanding of many processes driving evolution. The simplest ones, involving changing of a nucleotide pair, small insertions or deletions, can often immediately alter the phenotype, especially if they occur at coding position or in a **regulatory sequence**.

Beyond these relatively straightforward mutations, there are more complex mechanisms by which evolution is propelled. These encompass creating **structural variants** like duplications, inversions, deletions, or translocations. Such genetic alterations drive evolution by introducing large-scale changes to the genome, which can have significant impact on phenotype and adaptation. They can alter gene dosage, create novel gene fusions, and modify chromatin structure perturbing the regulatory landscape of genes. Moreover, gross chromosomal rearrangements, such as **fusion and fission**, can contribute to reproductive isolation between populations, promoting speciation, as incompatibilities arising from rearranged chromosomes can lead to reduced fertility or viability.

In our research conducted for over a decade with Baylor College of Medicine, we have been focusing on the **genome architecture** that mediates these kinds of changes. By developing a series of algorithms for chromosome rearrangements identification and analysis, we have demonstrated a **drastic instability of the human genome**. The research in which we participated has changed the way we think about the impact of evolution on the formation of **genomic disorders**.

Key to the formation of structural mutations are **repetitive elements** of the genome such as segmental duplications. While these repetitive elements make analysis more difficult, they also present an opportunity to understand evolution. A significant success in this field was the **reconstruction of the chromosome 2 fusion**, which introduced a reproductive barrier between great apes and human ancestors. Our improved method of dating the fusion demonstrated a correlation between the fusion's timing and a **bottleneck period** in the population size of our ancestors.

Drawing inferences from gigabytes of data detailing genomic architecture, dynamics, and regulatory processes necessitates **mathematical models and efficient computational methods** capable of drawing meaningful conclusions about the evolutionary forces shaping the genome of species. Our prior experience in analyzing **sequencing data**, research concerning the stability of the human genome perturbed by structural variants and **assembling most difficult regions of genome** will enable us to propose new procedures for extracting biological and medical insights from the analysis of differences in genome architecture, at both intra- and inter- species level and its role in the evolution and to the development novel algorithms.

The main research tasks will include: analysis of **mutations in regulatory elements**, exploration of **repetitive elements**, development of new algorithms for **dating major evolutionary events**, and understanding the **genome architecture underlying complex genetic syndromes**.