Arsenic (As) is a highly toxic element which causes several types of cancer in humans. Unfortunately, at least 140 million people all over the world drink every day water containing high concentrations of arsenic. Moreover, some staple foods, like rice, accumulate high amounts of As becoming important sources of the poisonous element for humans and stock animals. On the other hand, As-containing drugs are used in the treatment of acute promyelocytic leukemia and human African trypanosomiasis. Despite many years of research conducted on variety of organisms precise mechanisms of arsenic carcinogenesis remain elusive and are still a matter of debate. Several in vitro and epidemiological studies performed on different human populations suggest that As may promote cancerogenesis through induction of DNA damage. If not repaired or repaired by a mutagenic, low-fidelity mechanisms, these DNA lesions may cause accumulation of point mutations and/or generation of chromosomal rearrangements that drive oncogenesis. The goal of this project is to create a genome-wide map of As-induced DNA breaks. In this regard, we will take advantage of chromatin immunoprecipitation coupled to next generation sequencing (NGS) to analyze global distribution of several proteins, that signal and repair DNA breaks induced by arsenic using both yeast and human cell lines as models. Moreover, we will find out whether DNA breaks caused by As are formed randomly or at particular genomic locations. To this end, we will use bioinformatic tools and publicly available ChIP-seq datasets to define characteristic properties of As hot spots. Finally, by utilizing NGS and PCR-HRM, we will also verify whether As binds to DNA and if so to what sequences. The results of this project will not only generate novel and fundamental insight into the molecular mechanisms of arsenic genotoxicity but also may help to understand how As promotes cancerogenesis in humans.