Homologous recombination (HR) is one of the main pathways to repair DNA double strand breaks (DSB) occurring either spontaneously or upon induction by DNA damaging agents. In eukaryotes, the recombinase Rad51 plays a central role in HR by forming nucleoprotein filaments (NPF) on single-stranded DNA (ssDNA) to scan the genome and invade a homologous sequence used as a template for DNA repair synthesis. HR is essential to maintain genome stability and to generate genetic diversity, but it can also lead to genome instability and cell death. Particularly, excessive, or unscheduled HR can generate toxic Rad51 filaments. Also, finding a homology through HR is risky and could easily be prone to errors, especially in the absence of a sister chromatid. In mammalian cells, it is a known fact that HR is less prevalent than NHEJ pathway (Non-Homologous End Joining) yet it is essential for cell viability. Furthermore, evidence has shown that it also plays a fundamental role in carcinogenesis and the progression of cancer. It is thus essential to **decipher the mechanisms regulating the formation and stability of Rad51 filaments, which is the main goal of my PhD project.**

Although Rad51 has been extensively studied by *in vitro*, molecular, and genetic approaches, its dynamics *in vivo* could not be assessed due to the lack of functional tagged versions of this protein. The Taddei team has recently developed and characterized the first fully functional version of Rad51 in budding yeast allowing to monitor the formation of micrometer long filaments following the induction of a unique DSB. These filaments adopt different shapes and are highly dynamic with frequent collapse events followed by re-extension, allowing an efficient exploration of the nuclear space in quest of a homologous sequence. This unveils a new mechanism of homology search to ensure robust and efficient search. These observations leave many open questions some of which I will address including the characterization of the structures formed by Rad51 *in vivo*, the study of the dynamics and shapes of the filament upon homology search/strand invasion and identifying the factors regulating them.