Bat-derived partially cytoplasmic p53 mutant does not increase DNA damage repair rate

p53 is most widely recognized for the role it plays in oncogenesis, as it is the most frequently mutated gene in human cancer. However, the function of p53 remains incompletely understood. p53 has well described stress-activated transcription factor activity but has less well described cytoplasmic functions as well. Bats have evolved non-canonical amino acid changes in the nuclear localization signal (NLS) of p53, which result in partial cytoplasmic location. Bats' intrinsic ability to more rapidly repair various forms of DNA damage suggests that p53 may have unknown and beneficial novel cytoplasmic functions. In order to investigate these functions, our lab generated the p53K316P (PKK) mutant mouse, which mimics the NLS mutation and partial cytoplasmic localization seen in bat p53. We hypothesized that partially cytoplasmic p53 may increase the rate of DNA damage repair in p53K316P mouse embryonic fibroblast (MEF) cells. We tested this hypothesis by treating wild-type and p53K316P MEF cells with DNA damage conditions and measuring the rate of DNA damage repair using immunofluorescent staining of Œ≥H2AX, a marker of DNA damage.