To protect genetic information of DNA from damage, all organisms have DNA repair systems. Nucleotide excision repair (NER), one of such repair systems, can recognize a lot of species of damage such as AP site, oxidative damages, (6-4) photoproducts, and other bulky DNA adducts. In prokaryotes, the enzymes named UvrA/B/C, plays key roles in the system: UvrAB recognition complex binds to the lesion of DNA, followed by dual incision around the lesion made by UvrC. We have studied NER system in <I>*Thermus thermophilus*</I> HB8 with structural and functional approaches. We have determined the crystal structure of <I>*T. thermophilus* </I>HB8 UvrB (ttUvrB) by X-ray crystal crystallographic analysis, and also studied interaction between DNA and UvrA/B. However, there are still many questions about mechanisms of NER, e.g., recognition of such a wide range of lesions, interaction between UvrB and UvrC, and cleavage reaction by UvrC. To further analyze the molecular mechanisms of the NER, we prepared the enzyme UvrC and other proteins involved in this repair system of <I>*T. thermophilus* </I>HB8. UvrA alone promiscuously bound to DNA along with concentration. When UvrB existed, however, they bound to DNA with a different affinity. The bulkier the lesion on DNA was, the stronger UvrAB bound to it. For further analysis we are trying to produce several mutants of UvrA.