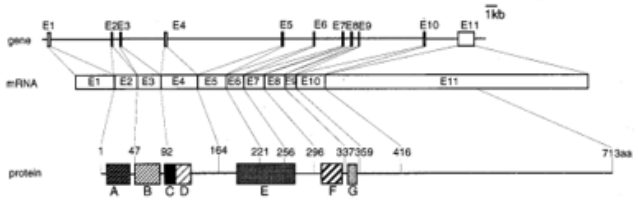
1.

First, sequencing the protein and searching sequence databases for homologous proteins identified several proteins from both eukaryotes and prokaryotes that are involved in lesion-bypass DNA synthesis, including two genes from *Saccharomyces cerevisiae* involved in error-free lesion-bypass activity. Additionally, an *in vitro* damage bypass replication assay showed that XP-V cells could not undergo translesion replication, but the addition of HeLa DNA Pol η restored this activity.

2.



Exon-Intron organization of the human XPV gene and schematic drawing of the mRNA and encoded protein. Source: Mayumi Yuasa et al. Genomic structure, chromosomal localization and identification of mutations in the xeroderma pigmentosum variant (XPV) gene, Oncogene (2000) 19, 4721-4728

The 4 bands in the northern blot correspond to 4 different alternatively spliced isoforms of the gene.

3.

The inclusion of DNA Pol α enabled comparison of this polymerase's activity against that of DNA Pol η. On a 30 bp oligonucleotide containing a T-T dimer, the recombinant protein successfully bypassed the dimer, whereas the DNA Pol α stopped short and only synthesized 16-mers.

4.

XP-V carry a mutant form of the XPV gene, which codes for DNA Pol η. The mutant polymerase cannot bypass DNA damage caused by UV radiation, affecting its ability to replicate and therefore causing a lower sensitivity to UV radiation.