SUPPLEMENTAL INFORMATION

The role of XPB/Ssl2 dsDNA translocation processivity in transcription-start-site scanning.

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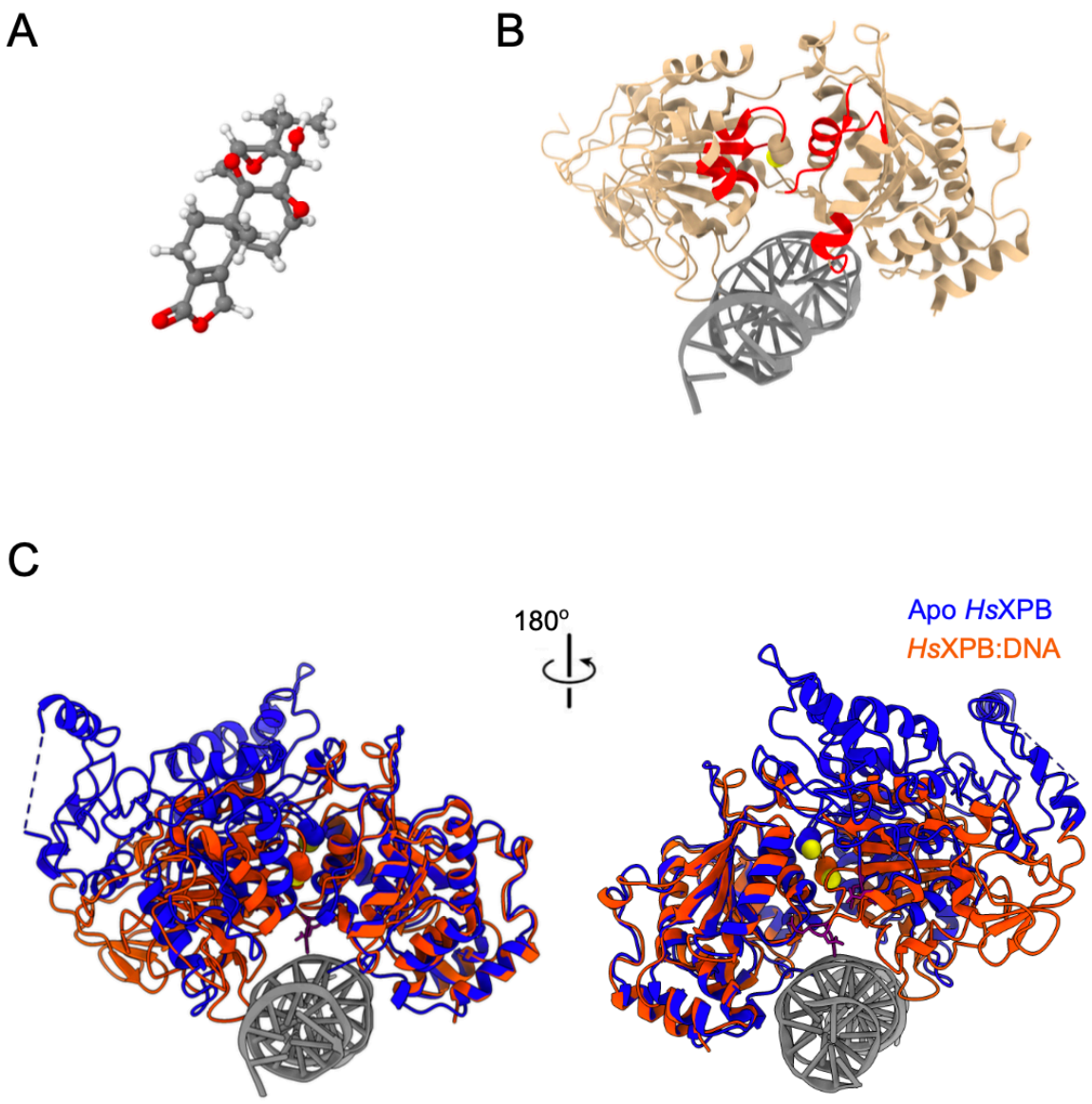

Figure S1. Site of triptolide modification on XPB potentially disrupts ATP binding site and can be blocked by DNA binding. (A) Molecular structure of triptolide. **(B)** *Hs*XBP substructure bound to duplex DNA from the *Hs*PIC structure[17] (PDB: 5ivw). Residue C342 of XBP is shown in space fill and is the site of triptolide modification[26]. XBP structure in red are the conserved motifs found in DNA-stimulated ATPases that generally comprise the ATP binding site[28]. **(C)** Alignment of the *Hs*XBP substructure bound to duplex DNA from the *Hs*PIC structure[17] (PDB: 5ivw) with *Hs*XBP substructure from apo, core-*Hs*TFIIH structure[27] (PDB: 6o9m; blue) using Chimera[42]. Residue C342 is shown space filled and becomes less exposed in the DNA bound structure.

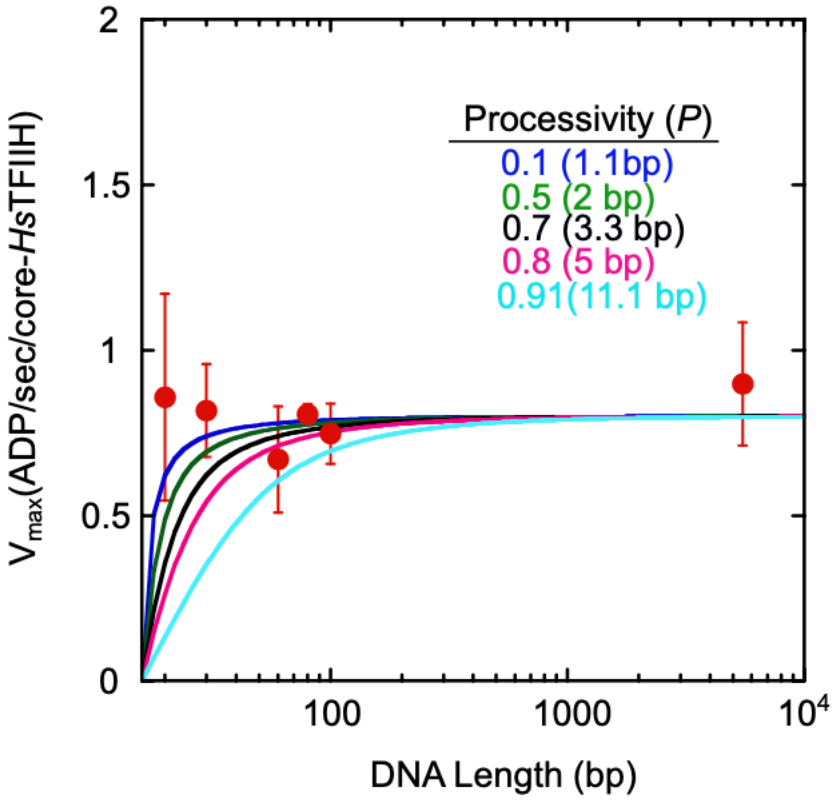

Figure S2. Estimating an upper limit on *Hs*TFIIH dsDNA translocation processivity. A series of V_{max} length dependencies calculated from equation (1) using different processivities (P) are plotted along with the observed core-*Hs*TFIIH V_{max} values. The V_{max} length dependencies for P = 0.1 and 0.5 are within the error of all the observed V_{max} values, indicating both are consistent with the data. Thus, an upper limit on the translocation processivity is 0.5 (i.e. ~2 bp translocated on average per binding event).