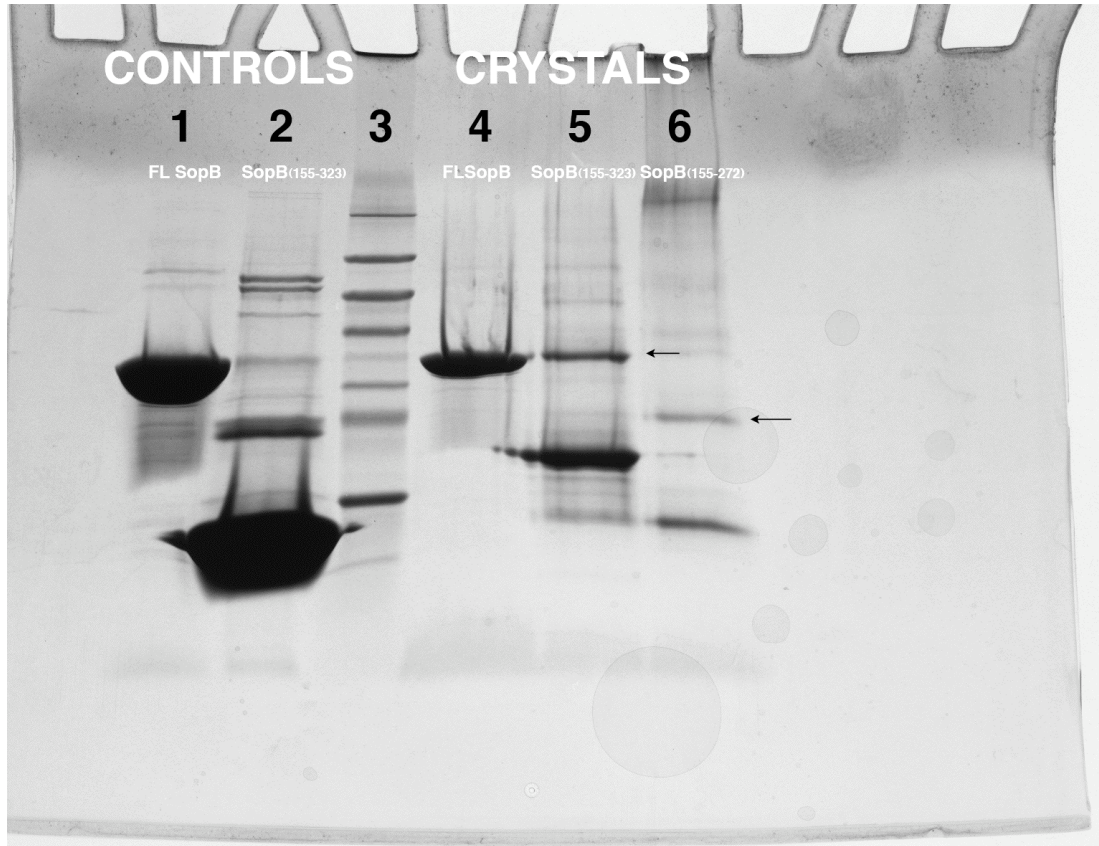


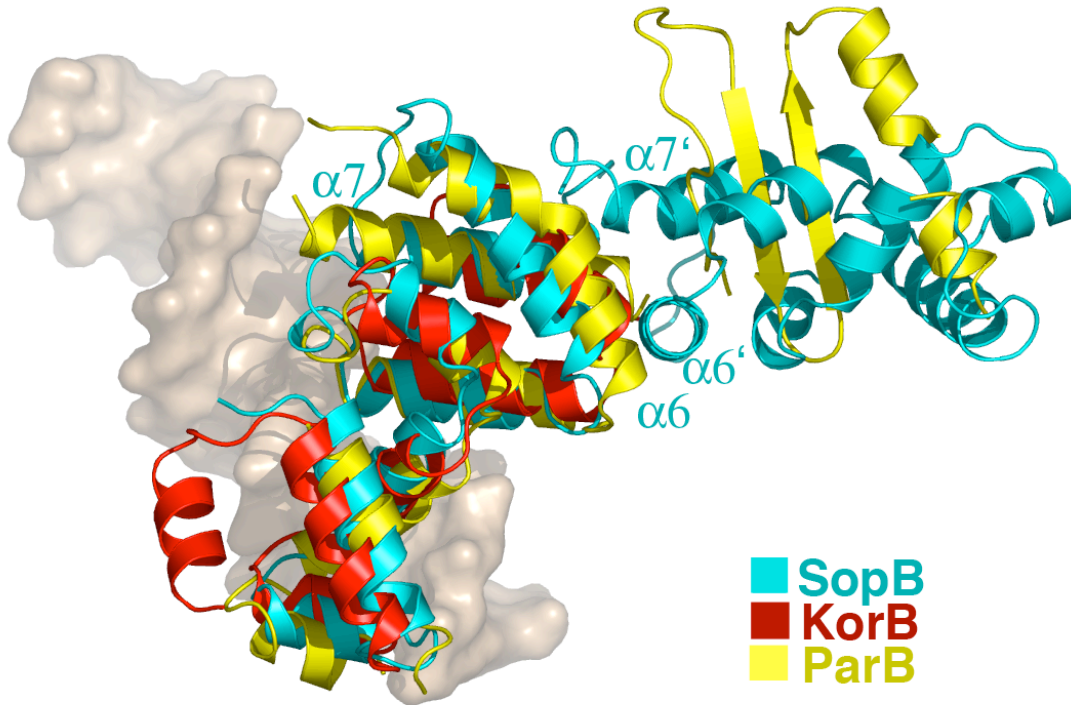
Supplementary Data

Supplementary Figure 1



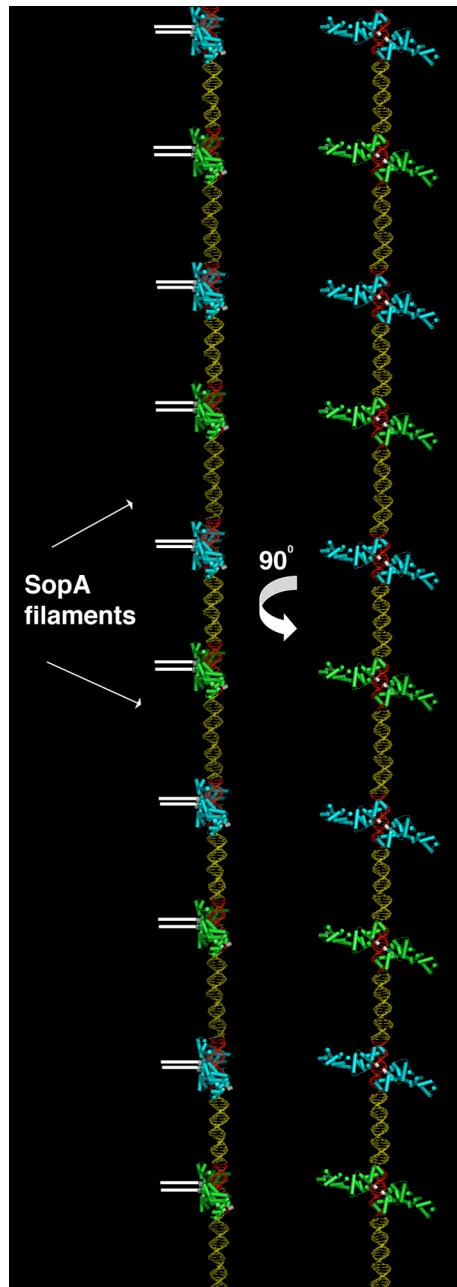
Supplementary Figure 1: SDS PAGE of washed SopB-18mer crystals. Lanes (1) Control showing purified FL SopB (2) purified SopB(155-272) (3) Molecular weight marker (4) Washed FL SopB-18mer crystals (5) Washed SopB(155-323)-18mer crystals (6) Washed SopB(155-272)-18mer crystals. The arrows indicate the location of dimers in the respective proteins that are often observed on SDS gels.

Supplementary Figure 2



Supplementary Figure 2: Superimposition of SopB(cyan), KorB DNA-binding domain (red) and P1 ParB(142-333) (yellow). The 18mer DNA from the SopB structure is shown as a grey surface representation for reference. Helices 6 and 7 from SopB are labeled. These helices contribute to the formation of the SopB secondary dimer.

Supplementary Figure 4



Supplementary Figure 4: Stylized model of F plasmid SopB-*sopC* partition complex. Model of the FL SopB-*sopC* partition complex. SopB does not cause any DNA distortions. Hence the intervening DNA present in the FL centromere were modeled as straight B-DNA (colored yellow) and used to connect each SopB-18mer complex (in which the 18mer DNA is red) providing an extended partition complex model. Notably, all the SopA interacting regions (shown as white lines emanating from SopB) lie on one face in the extended model. On the opposite side (90° rotation) are the secondary dimer contacts that function to link SopB subunits and thus DNA *in trans*. Notably, the DNA is likely flexible *in vivo* and this represents a highly stylized view of the partition complex, making the point that without extensive DNA distortion, the N-terminal NTPase interaction regions will tend to all align on one major face creating a high local concentration of NTPase interacting surfaces for recruitment and stabilization of SopA polymers.