Bacterial FtsK is one of the fastest-known dsDNA translocases in nature (>5 kb/s, stall force ~60 pN). It coordinates circular chromosome segregation with cell division by clearing trapped DNA away from the closing septum, and by activating a tyrosine recombinase machine, XerCD-dif, to convert chromosome dimers to monomers. The directionality of translocation is imposed by interaction of its [gamma]-subdomain with ‘KOPS’ (FtsK Orienting / Polarising Sequences). Single-molecule work suggested that FtsK reverses direction on DNA at non-permissive KOPS, but doing so would require a double-hexameric motor, which is inconsistent with the biochemistry of XerCD activation. We show, using bulk assays, that a simple loading hypothesis is sufficient to explain the interaction of FtsK with KOPS. Given FtsK’s stall force, one would expect it to strip most proteins from DNA but cease translocation when it encounters XerCD-dif. The distribution of chromosomal KOPS suggests that FtsK should be able to translocate towards either side of dif. We used triplex displacement to investigate the role of XerCD-dif as roadblock. XerCD bound in tandem and encountered in either orientation caused robust stoppage of translocation. The mechanism for this stoppage is unlikely to be a specific recognition of XerCD by FtsK. We rule out the formation of synaptic complexes, and transient covalent XerCD-dif intermediates. We present evidence for these assertions and present a possible model for XerCD activation by FtsK.