The RecQ protein family are 3'-5' helicases and involved in recombination. Mutations in three of the human orthologs, BLM, WRN, and RTS cause Bloom's, Werner's and Rothmund-Thomson syndromes, respectively, and are characterized by genetic instability, due to hyperrecombination, and cancer.

Studies reveal that BLM forms foci upon HU treatment, which co-localize with its heterodimeric partner TOPO IIIα and also with RAD51, PCNA, γ-H2AX and ATR (by which it can be phosphorylated). Also, BLM deficient cells fail to recover from S-phase arrest (88) and BLM is required for HU induced co-localization and association of p53 with RAD51 (391).

Equally, the WRN mutation leads to sensitivity to DNA damage. Suppression of RAD51-dependent recombination leads to increased survival of the WRN mutant following DNA damage (380). Furthermore, the mutant could be rescued by over-expression of RusA, suggesting that WRN functions in resolving recombination structures (HJs).

The yeast RecQ homologues are called Rqh1 in S. pombe and Sgs1 in S. cerevisiae. Both mutants display genomic instability and are deficient in DNA repair (128). Sgs1 is involved in proper S-phase checkpoint functioning upon perturbed DNA replication via interaction with Rad53 (116) and forms a heterodimer with topoisomerase Top3.

Another 3'-5' helicase in budding yeast is Srs2. The double mutant Sgs1-Srs2 is synthetically lethal, but can be rescued by an additional deletion of RAD51 (or RAD55, RAD57). Both Sgs1 and Srs2 are involved in DNA replication (and transcription) and suppress crossovers during DSB repair (128, 238, 485). Srs2 is also involved in proper S-phase checkpoint functioning (467). As the synthetic lethality suggests, both helicases may (partially) substitute for each other when a cell is challenged with compounds interfering with DNA replication, but there are also differences.

Srs2 disrupts the pre-synaptic Rad51 nucleoprotein filament by removing Rad51 (218, 468). On the other hand, Sgs1 will probably function as its mammalian homologue BLM, acting at a later stage of recombination. BLM can resolve recombination intermediates with dHJs by reversing the branch migration and its partner TOPO IIIα resolves the junction by strand passage (485).