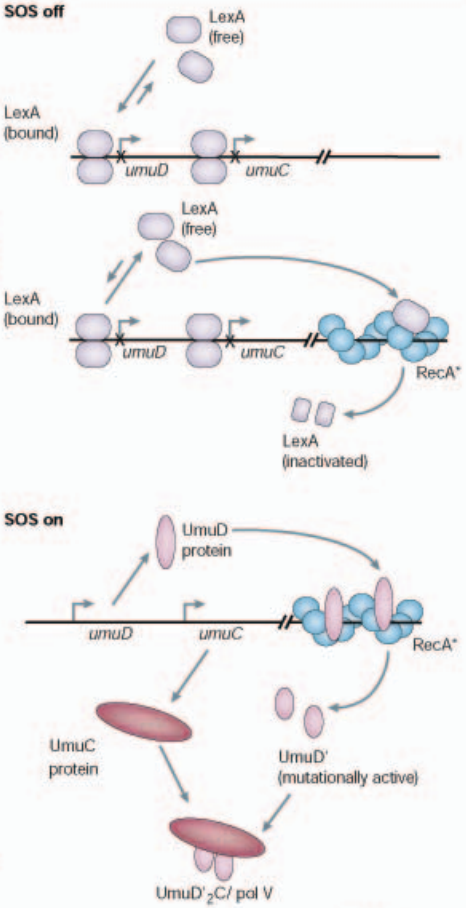
## Box 1 | **SOS mutagenesis**

*Escherichia coli* keeps an arsenal of regulated pathways that help it to survive when under stress[31]. One of these is the 'SOS' regulon, which is thought to be induced in response to regions of single-stranded DNA — presumably a hallmark of large-scale DNA damage. Normally, the LexA repressor binds to the operators of more than 30 SOS genes and keeps them repressed. But in the presence of single-stranded DNA, the RecA protein forms a close-packed 'activated' RecA filament, RecA*, which acts as a co-protease to cleave any LexA released from low-affinity operators. Further cleavage of LexA frees up the more weakly bound operators, and the SOS genes are relieved from repression (see figure).




The SOS proteins are mainly involved in nucleotide-excision and recombination-repair pathways to remove the DNA damage. However, the two 'UV mutagenesis' (umu) genes, ***umuC*** and ***umuD***, are instead required for replication past unrepaired lesions in the DNA template. They leave behind mutations targeted to sites of DNA damage. To be active, UmuD must be post-translationally cleaved to UmuD′ on the RecA* filament[31,35,97,98] (see figure). UmuC and UmuD′ then form a tight complex, $UmuD'_2C$, which has an intrinsic, low-fidelity DNA polymerase activity[43].

A replication fork blocked by DNA damage is dealt with by two SOS-induced DNA polymerases — pol II and pol V ($UmuD'_2C$). About two minutes after SOS induction, pol II reinitiates replication downstream from the lesion, leaving a gapped structure that is resolved by homologous recombination[43]. Replication restart is an error-free repair process. Pol V appears 30–45 minutes later. It binds at the 3′-OH adjacent to the lesion, then copies past the lesion, often inserting the wrong base opposite it. This process also requires RecA*, single-stranded DNA binding protein (SSB), and the β/γ processivity proteins (FIG. 3).