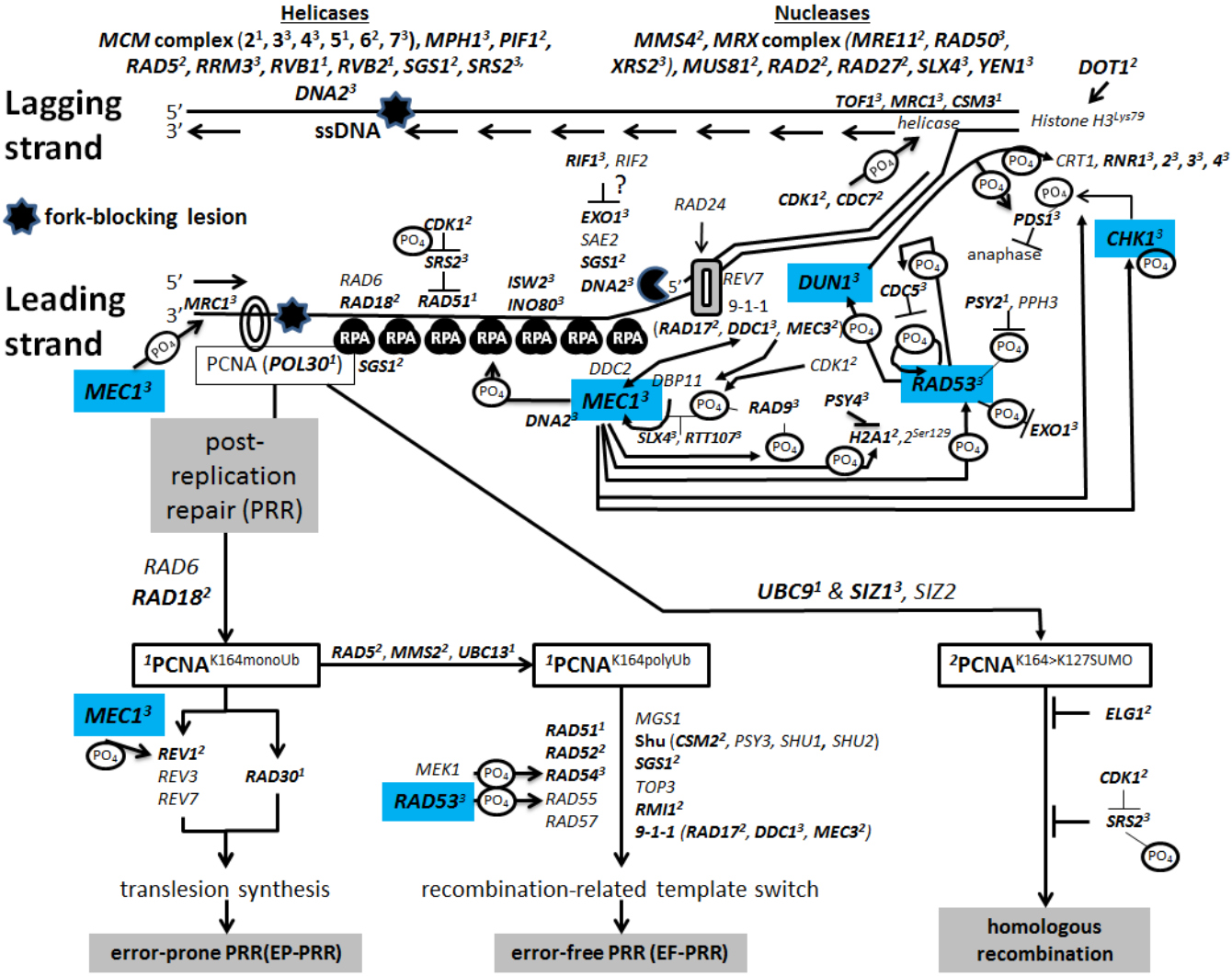
Helicases
MCM complex (2[1], 3[3], 4[3], 5[1], 6[2], 7[3]), MPH1[3], PIF1[2], RAD5[2], RRM3[3], RVB1[1], RVB2[1], SGS1[2], SRS2[3]
Nucleases
MMS4[2], MRX complex (MRE11[2], RAD50[3], XRS2[3]), MUS81[2], RAD2[2], RAD27[2], SLX4[3], YEN1[3]
DOT1[2]
Lagging strand
5′
3′
DNA2[3]
ssDNA
TOF1[3], MRC1[3], CSM3[1]
helicase
Histone H3[Lys79]
fork-blocking lesion
RIF1[3], RIF2
EXO1[3]
SAE2
SGS1[2]
DNA2[3]
RAD24
CDK1[2], CDC7[2]
CRT1, RNR1[3], 2[3], 3[3], 4[3]
PDS1[3]
anaphase
CHK1[3]
Leading strand
MRC1[3]
RAD6
RAD18[2]
RAD51[1]
ISW2[3]
INO80[3]
CDK1[2]
SRS2[3]
REV7
9-1-1
(RAD17[2], DDC1[3], MEC3[2])
DUN1[3]
CDC5[3]
PSY2[1], PPH3
PCNA (POL30[1])
SGS1[2]
RPA
MEC1[3]
DDC2
DBP11
CDK1[2]
RAD53[3]
RAD9[3]
PSY4[3]
SLX4[3], RTT107[3]
H2A1[2], 2[Ser129]
EXO1[3]
post-replication repair (PRR)
RAD6
RAD18[2]
UBC9[1] & SIZ1[3], SIZ2
[1]PCNA[K164monoUb]
RAD5[2], MMS2[2], UBC13[1]
[1]PCNA[K164polyUb]
[2]PCNA[K164>K127SUMO]
MEC1[3]
REV1[2]
REV3
REV7
RAD30[1]
MEK1
RAD53[3]
RAD51[1]
RAD52[2]
RAD54[3]
RAD55
RAD57
MGS1
Shu (CSM2[2], PSY3, SHU1, SHU2)
SGS1[2]
TOP3
RMI1[2]
9-1-1 (RAD17[2], DDC1[3], MEC3[2])
ELG1[2]
CDK1[2]
SRS2[3]
translesion synthesis
recombination-related template switch
homologous recombination
error-prone PRR(EP-PRR)
error-free PRR (EF-PRR)

Figure S1. Nearly all proteins known to be present at the replication fork and/or required for DNA replication stress response in *S. cerevisiae* were detected LC-MS. Many forms of DNA damage block replication fork progression, causing replication stress, and initiate extensive phosphorylation cascades. The top half of the figure shows a replication fork that is stalled by fork-blocking lesions on both the leading and lagging strands. Because the lesions are in ssDNA, they cannot be repaired and must be circumnavigated in order for the replication to resume. The ssDNA also evokes a checkpoint response that activates an extensive phosphorylation cascade to enhance DNA repair and coordinate replication of damaged DNA templates. The bottom half of the figure summarizes the possible PRR and HR mechanisms for circumnavigating the lesions. In the figure, the gene symbols of proteins detected by LC-MS in our experiments are annotated with superscripts. The superscripts indicate the nature of protein detection in our MS experiments: [1]only the unmodified protein form was detected; [2]phosphorylated form was detected; [3]phosphorylated form was detected and found to be MMS-responsive. The blue color-shaded genes encode DNA damage checkpoint kinases.