**The SOS response in *Zymomonas mobilis***

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## Abstract

The SOS response in bacteria is a stress-inducible mechanism that engages genes involved in DNA repair, cell-cycle arrest and error-prone replication. At a molecular level, the response is governed by the proteins RecA and LexA. LexA is a global repressor that binds to operator regions – the SOS boxes – located upstream from SOS-regulon genes, maintaining the genes transcriptionally repressed. RecA is the major homologous recombination protein, which, upon DNA damage and SOS activation, facilitates the auto-cleavage of LexA, bringing about SOS-gene derepression and expression.

*Zymomonas mobilis* is a bioethanol producer studied in our labotarory in basic and applied directions. Its tolerance to mutagenic stress is of interest in terms of understanding the genetics underlying the organism's performance and also creating useful strains. One such strain has been a *recA*$^-$ CP4 derivative that exhibits all features of homologous recombination knock-outs and can be used as a reliable host for gene introduction purposes.

In computational WGS searches in *Z. mobilis*, typical α-proteobacterial SOS-box motifs precede several genes, including the archetypical SOS genes *recA* and *lexA*, which indicates the presence of an SOS regulon. The *Z. mobilis* RecA and LexA proteins bear functional sequence signatures met in the well-characterized *E. coli* orthologs. Many DNA-repair genes, usual members of SOS networks, can be found in the *Z. mobilis* genomes. The *Z. mobilis recA* gene is functional in *E. coli* and complements an *E. coli recA*$^-$ strain. Lastly, in *in vivo* expression studies in *E. coli,* the *Z. mobilis* LexA protein appears to bind to the cognate *recA* operator.

Due to such evidence regarding an operational SOS network in *Z. mobilis*, we analyzed the transcriptomes of *Z. mobilis* strains CP4 and UA1, the CP4 *recA*$^-$ mutant, in order to reveal the extent of the network. At increasing SOS-inducing agent concentrations, an impressive amount of genes was found to be differentially expressed. Of these, no less than 650 genes, preceded by SOS boxes of varying stringency, comprised the sensu stricto SOS regulon. SOS genes were found to reside in both the chromosome and plasmids, and govern various functions. The lack of gene inductions in UA1 verified its *recA*-null phenotype.

## Brief Biography

Katherine M. Pappas is Associate Professor of Molecular Microbial Genetics in the Department of Genetics & Biotechnology, Faculty of Biology, School of Sciences, University of Athens. Her research interests lie in the fields of genomics, transcriptomics, transcriptional

regulation, bacterial cell-cell signaling, plasmid biology, synthetic biology, and strain engineering. She has collaborated with US and European academic and research institutions, as well as US DOE federal laboratories. She has been a primary investigator in competitive national and international research programs, evaluator in research programs and institutions in Greece and the EU, member of academic and governmental councils, as well as editor and reviewer in high-IF journals. She is Secretary of the Hellenic scientific society 'Mikrobiokosmos', member of eight national and international scientific societies including the American Society of Microbiology (ASM), and serves as ASM international mentor (2010-) and ASM country ambassador (2018 -).

**Brief CV**

**Katherine M. Pappas, PhD**

Faculty of Biology, National and Kapodistrian University of Athens (NKUA), Greece

**Education:**

BSc, PhD: Faculty of Biology, NKUA, Greece

**Professional Career:**

1999-2002: Department of Microbiology, College of Agriculture and Life Sciences, Cornell University – Post-doctoral Associate

2003-2010: Department of Genetics & Biotechnology, Faculty of Biology, NKUA, Greece – Lecturer

2010-2018: as above – Assistant Professor (tenured in 2014)

2019 - : as above – Associate Professor

**Research Interests:**

1. Genome Biology
2. Metabolic Engineering and Synthetic Biology
3. Transcriptional Regulation and cell-cell signaling

**Selected publications**

1. Yang *et al*., ***Biotechnol. Biofuels***, 2018, 11: 125
2. Pinto et al., ***Nature Rev. Biotechnol***., 2012, 10: 755
3. Pappas *et al*., ***J. Bacteriol***. 2011, 193: 5051
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5. Yang *et al.,* ***Nature Biotechnol***., 2009, 27: 893
6. Pappas *et al*., ***Mol. Microbiol***., 2004, 53: 755
7. Zhang *et al*., ***Nature***, 2002, 417: 971