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Opinion

The SOS Response, why is there for?

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The discovery of the SOS response originates from the experiments done by Weigle, who reported that when UV-irradiated *E. coli* bacteria were infected with lambda phages the total amount of the phage raised dramatically (the so called "Weigle's reactivation"). Later on, Radman and co-workers reported that, along with Weigle's reactivation, there were some other events happening such as cell filamentation or an increase in mutation frequency. Based on that, this group proposed the existence of some kind of inducible DNA repair system related to mutagenicity that was triggered by stress situations and that activated the expression of several proteins. They called this phenomenon SOS response (same as the international call for help "save our souls") because it was thought to be the last chance for the cell to survive. Initially, by using the Mud(lacZ) fusions, Kenion and Walker identified 17 SOS genes which were generically called "din" (for damage inducible). Later on and thanks to the advances in molecular biology technics, it was possible to identify (by means of microarrays) up to 43 genes belonging to the SOS system. The relatively large amount of genes implicated and the delicate regulation of the response suppose a huge spend of energy, yet the SOS response is present in many bacterial species, thus a question arises: what is its evolutive importance?

In this pathway, there are genes involved in DNA repair (such as uvrA) or tolerance (sulA for example), and there are also a few genes of polymerases (polB, umuC and dinB, which codes for Pol II, Pol IV and Pol V respectively) in charge of the so called translesion synthesis. However, while the number of mispairing events by Pol II is low, those for Pol IV and V are very high, which explains why the DNA synthesis occurring when the SOS response has been activated leads to a dramatic raise in the amount of mutations.

It is known now that bacterial species such as Vibrio cholera or Staphylococcus aureus possess systems similar to the SOS response of *E. coli* and controlled by RecA/LexA as well, that can be triggered by several antibiotics such as ciprofloxacin, a fluoroquinolone that acts upon topoisomerases causing DNA breakage. As part of these SOS systems there are proteins similar to Pol IV and Pol V

In *E. coli*, the SOS genes related to DNA repair are the first to be overexpressed and only when the damage is too extensive those genes responsible of mutations appearance or horizontal character transference are activated. Conversely, in *S. aureus* has been observed that are precisely the genes associated mainly with the acquisition of antibiotic resistance and the horizontal transfer of virulence factors the first to be transcribed. Apparently in *S. aureus* the SOS response involves mainly changes in metabolism and activation of prophages that are possibly responsible for the horizontal transfer mentioned above, as well as the increase in the rate of mutation by the polymerases responsible of the error-prone repair.

On the other hand, in bacteria such as Vibrio cholerae has been identified Integrating conjugative elements (ICEs) that are transferred by cell-cell contact and are incorporated into the chromosome of the new host. Some of these, such as the ICE SXT of *V. cholerae*, carry the genes that confer resistance to several antibiotics (chloramphenicol, sulfamethoxazole, streptomycin or Trimethoprim). The genes necessary for the transfer of these carry out ICE are regulated by the SOS response.

All this suggests that, even though in the SOS response are genes involved in the repair of lesions in the DNA, the important part of this system is the appearance (and transmission) of fortuitous mutations that allow bacteria to cope with an adverse environment. Some

interesting issues concerning the evolutionary and mechanistic significance of the SOS response remain unresolved. For example, why the classic SOS response system regulated by LexA is lacking in archaea, many of which live also in extreme environments? and why despite eukaryotes possess translesion synthesis similar to that of bacteria, it is error-free? It is very important then to continue with the study of the SOS response, which still may have more surprises reserved for us.

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