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The link between bacterial radiation resistance and cold adaptation

Living creatures depend on somewhat narrow ranges of temperature, pressure, pH, salinity and hydration to sustain their life. The occurrence of bacteria in extreme environments (e.g. hot springs, glacier, deep sea etc.) provokes us to look into the mechanism of their stress tolerance.

1. The mechanisms of adaptation to different stresses overlap

Mechanisms of cold adaptation in bacteria remain by and large ill-defined. Investigations on psychrotrophic strains have provided some clues (Chattopadhyay and Jagannadham 2001). Recent reports reveal that there are specific factors which promote survival of bacteria not only at low temperature, but also under other types of stress conditions. Thus they provide a link between different types of stress adaptations. For example, monounsaturated fatty acids were found to be important for the growth of a deep sea bacterium in cold, high pressure zones of ocean depth (Allen *et al* 1999). Substantial improvement in survival of an *Escherichia coli* strain was noticed during frozen storage following the inductive synthesis of some heat shock proteins which are believed to protect bacteria from thermal stress (Chow and Tung 1998). Recently it has been demonstrated that specific genes are over-expressed during both oxidative stress and cold stress in *E. coli* (Smirnova *et al* 2001). The work highlighted here provides a possible link between cold adaptation and radiation resistance (Mangoli *et al* 2001).

2. Is radiation resistance a side benefit of a tolerance mechanism to other stress(es)?

The ability of some bacteria to withstand high dose of ionizing radiation is baffling because it is difficult to find a niche where bacteria are subjected to high doses of radiation. Studies performed so far indicate that radiation resistance is an offshoot of the capacity of the organism to tolerate other forms of stress conditions which, like radiation, are damaging to DNA (Chattopadhyay 2000). A hint of a possible link between cold adaptation and radiation resistance was obtained earlier from a study in which radiation resistance was demonstrated in an Antarctic isolate (Billi *et al* 2000). The studies reported by Mangoli *et al* (2001) provides stronger evidence for this link.

The protein that appears to have a dual role in low temperature tolerance and radiation resistance of *E. coli* is called GicA. Previous studies by these investigators led to the isolation of a spontaneous mutant characterized by delayed appearance of colonies and loss of the ability to form colonies in streptomycin-containing medium, both at 20°C. Thus, besides being cold-sensitive, the cells were streptomycin-sensitive at low temperature. By mapping, two loci were located at the 14–15 min region of the *E. coli* genome. They were called *gicA* and *gicB* (gic for growth in cold). A mutation in *gicA* was responsible for both growth retardation and streptomycin-sensitivity in cold but that in *gicB* had no detectable role in cold sensitivity. During cell division of bacteria, the single DNA molecule replicates and gives rise to two daughter molecules. Successful segregation of these two DNA molecules in the daughter cells requires not only segregation of chromosomes but also the proper positioning of the chromosomes into the daughter cells. The MukB protein of *E. coli* is believed to play an important role in the process of partitioning. A mutation in the *mukB* gene results in

normal-sized anucleate cells. The GicA protein is known to suppress the effects of this mutation. The gene *gicA* has been found to be identical with *cspE*, one of the members of the family of cold-shock genes in *E. coli* (Yamanaka *et al* 1994).

3. Does the cold shock protein GicA preserve cooled cells from mutagenesis but promote mutagenesis in over-cooled cells?

Mangoli *et al* (2001) have demonstrated that following inactivation of chromosomal *gicA* gene, at low temperature the strain became sensitive to both UV and gamma radiation. When a high intracellular concentration of GicA was artificially created by over-expression using a multicopy plasmid, the strain was sensitive to UV but not gamma radiation. We know that several enzymes are involved in DNA repair following the damage caused by UV radiation. The role of scavengers in protecting the cells from the harmful free radicals, generated by gamma radiation, is also well-documented. The investigators postulate a negative effect of the over-expressed GicA in regulating the expression of genes which encode DNA-repairing enzymes and a positive effect of the same protein on the expression of genes encoding the scavengers. A high concentration of GicA was therefore associated with sensitivity of the cell to UV since synthesis of DNA-repairing enzymes was suppressed by GicA. On the other hand, in the absence of GicA (caused by mutation), expression of genes that encode the protective scavenger molecules was adversely affected. Deficiency of the scavengers in the cell made it sensitive to gamma radiation. The fact that gamma radiation sensitivity was manifested only in aerobically grown cells, strongly suggests the possible involvement of a scavenger of free radicals in this process. Thus sensitivity of the bacteria to UV and gamma radiation could be explained by the differential role of the GicA protein. At normally induced levels *GicA* protects cooled cells from UV and gamma-mutagenesis, but it seems that when *gicA* is over-expressed, it switches off the repair process. Is this switch-off of any physiological relevance or is it an “un-natural response” of the recombinant cells to the “un-natural” over-production of GicA protein? This question remain to be answered.

During starvation stress a special type of mutagenesis (so-called adaptive mutagenesis) is induced in non-growing bacterial cells. In part, this mutagenesis is caused by the switch-off of mismatch repair (activation of mutagenic DNA polymerase IV is also involved) (review Velkov 2001). As the result of such adaptive mutagenesis, a mutation that adapts a non-growing cell to a previously non-utilizable substrate could arise and, consequently, enable the cell to resume growth.

The sensitivity of the bacteria to UV, both in presence of high amount of GicA and in the situation of its total absence seems enigmatic. But it indicates that there may be a critical intracellular level of GicA which is essential for UV tolerance.

This study corroborates earlier reports that there is a link in the mechanism of bacterial adaptation to different types of stress conditions. It is also revealing as regards the subtlety of the cellular regulation of stress response. Further studies on the radiation sensitivity of cold-adapted bacteria are likely to provide more insights.

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