Surprising new insights into the repair strategies of DNA

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(Physorg.com) -- A microscopic single-celled organism, adapted to survive in some of the harshest environments on earth, could help scientists gain a better understanding of how cancer cells behave.

Experts at The University of Nottingham were astonished to discover that the archaeon Haloferax volcanii was better at repairing DNA damage if enzymes, that are widely considered to be critically important in coordinating the repair of DNA, were mutated.

Dr Thorsten Allers, from the Institute of Genetics, said: "These results surprised us. It is the first time, as far as we know, that anybody has found such resistance to DNA damage in mutant cells. Normally, cells that are missing enzymes for DNA repair become more sensitive to DNA damage."

Like cancer cells, archaea are polyploid — which means they contain more than two sets of chromosomes. Although similar in structure and appearance to bacteria, archaea share a common ancestor with eukaryotes, which include plant and animals. This kinship is at its closest in the way archaea process DNA. Although Dr Allers's discovery is at the basic biological level, it is the similarities with cancer cells that make him convinced that scientists have much more to learn from archaea.

Discovered just 32 years ago, there are less then 200 experts around the world studying archaea. On the other hand, the mechanisms by which cells perform the repair of DNA breaks has been the subject of decades

of research using bacterial and eukaryotic cells. We are only just beginning to learn how this process works in archaea.

DNA breaks can be caused by, among other things, radiation, UV rays and chemotherapy. Dr Allers said: "All organisms can use enzymes to simply glue the broken strands of DNA back together, but this is prone to error and can give rise to mutations which cause cancer. The alternative is to perform a kind of molecular gymnastics called recombination, where healthy strands of matching DNA are used to repair the broken ends. This is a complicated and time-consuming strategy to mend DNA, but avoids mutations. When the enzymes that carry out recombination are defective, cancer can develop more easily. This is what happens in patients with mutations in the BRCA breast cancer genes."

Dr Allers's research, published in the journal *PLoS Genetics*, shows how, unlike other organisms, *Haloferax volcanii* deliberately avoids using recombination to repair DNA breaks. His results suggest that other polyploid organisms, such as cancer cells, might work in much the same way. What scientists need to know now is why.

Source: University of Nottingham (news : web)