

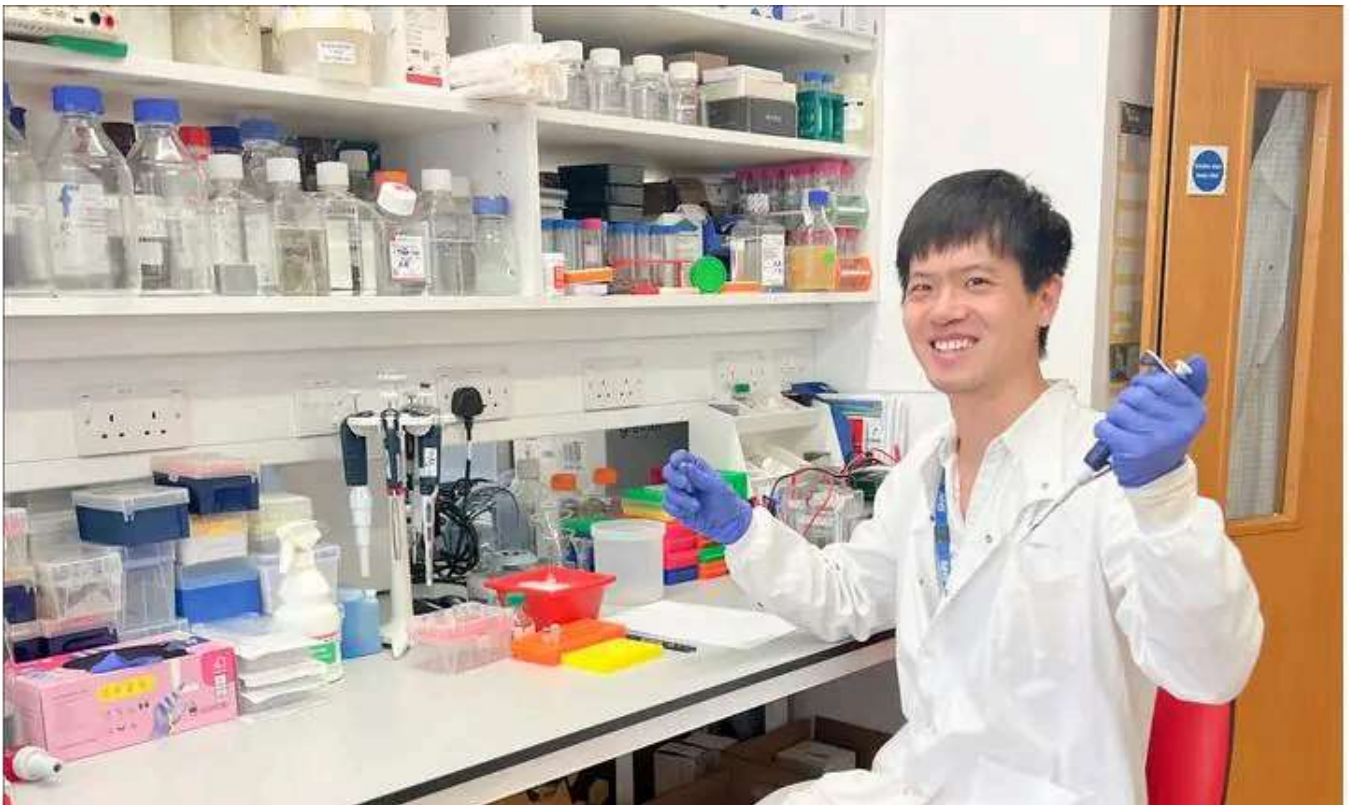
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Healthcare

NTU and Oxford uncover DNA repair mechanism

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Scientists from Nanyang Technological University, Singapore (NTU Singapore) and the University of Oxford have identified how cells repair toxic DNA damage, which is linked to cancer and premature ageing. The study, published in “Nucleic Acids Research”, highlights the role of the enzyme SPRTN in recognising and repairing DNA-protein crosslinks (DPCs), lesions caused by chemotherapy, UV exposure, and formaldehyde.

The research team discovered a new region within SPRTN that targets DPC lesions, increasing its repair efficiency by 67 times without harming other cellular proteins. This discovery is significant for cancer therapy and healthy ageing, as DPCs, if left unrepaired, can lead to neurodegeneration, premature ageing, and cancer.

DPCs occur during normal cellular processes and through exposure to harmful agents. SPRTN acts by degrading proteins in these lesions, allowing DNA replication to continue. The study found that SPRTN's activity is enhanced by ubiquitin chains, which guide the enzyme to DPCs, accelerating the repair process.

Kristijan Ramadan, leading the study, noted the importance of ubiquitin chains in SPRTN's rapid activation. This understanding could lead to new therapeutic strategies, especially for patients resistant to chemotherapy. Dr Wei Song from Oxford emphasised the potential to strengthen the body's defences against age-related diseases.

Future research aims to validate these findings in animal models and explore therapeutic interventions, potentially revolutionising the understanding of ageing and cancer.

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