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[\[Cancer Research\] Taiwan Researchers Identify Critical Enzyme Class Related to DNA Repair and Carcinogenesis](#)

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([Chinese Version](#))

Liberty Times & Apple Daily (2013/09/13) Current cancer research scholarship suspects a tight relation between carcinogenesis and DNA repair deficiency, for the chaotic orders in DNA sequence in cancer cells. A research team with members from National Taiwan University and Academia Sinica spent three years and succeeded in identifying the critical role of the helicase Pif1 in DNA repair. The findings are supposed to help with new drugs and treatments development. The report about the findings is also published in Nature.

DNA damage can be caused by instances such as free radicals attacking upon normal genomics, and cancer cells are created because of DNA repair deficiency in them. Two years ago, we already had a report proving that the apoptosis rate in cancer cells with Pif1 suppression is ten times than that without Pif1 suppression. But the understanding about the function of Pif1 in cancer cells' DNA repair was still missing. Indicated by Professor Peter Hung-Yuan CHI at NTU's Institute of Biochemical Sciences, the function of Pif1 is to fix the damage of DNA's double helix structure, which is the most serious form of DNA damage.

Addressed by Present NTU President, Pan-Chyr YANG, who is also a famous expert in treating lung cancers, the results of the investigation will be tested via clinical experiments, and it is expectable that targeted therapies controlling the activity of Pif1 against those frequently occurred cancers in Taiwan, like lung cancer, can be developed. MD. Ji-Hong Hong, Chef at Cancer Center, Chang Gung Memorial Hospital, said, because Pif1 exists in all cells, if drugs that can inhibit the activity of Pif1 can be developed, it will be a significant breakthrough in cancer treatment.

Further Information:

[Liberty Times 2013/09/13](#) (Chinese)

[Apple Daily 2013/09/13](#) (Chinese)

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[National Science Council International Cooperation Sci-Tech Newsbrief](#)  
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