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CITY

Study could shed light on causes of epilepsy

Lack of a protein might be root of certain types of the disorder, HKUST researchers believe

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Lack of a particular protein in early brain development might be the root of certain types of epilepsy.

A study by researchers on embryonic mice at the Hong Kong University of Science and Technology found that with less of the a2-chimaerin protein, brain cells in early development would not migrate to the correct areas of the brain, and also become hyperactive in firing neurons.

"The brain's neurons form circuitry and fire electric transmissions. You need precise wiring for daily activities ... the consequence if they fail to migrate, if they are impaired, is Lissencephaly, or smooth brain which causes epileptic seizures and mental retardation," said Jacque Ip Pak-kan, one of the main researchers.

Epilepsy is caused when the brain releases excessive electrical signals causing its normal wiring to go awry. It can be brought on by deformations during early development or by injuries in later life.

In Hong Kong, there are an estimated 40,000 to 70,000 epileptics based on studies by Queen Mary Hospital, and global rates of epilepsy. Around 50 million people worldwide are thought to have the disorder.

"The next step is to think about gene therapy," said Professor Nancy Ip Yuk-yu who supervised the study, though she declined to say when such treatment could be viable for the public.

The discovery would most probably affect research into focal epilepsy where the seizures start from one part of the brain and spread to others, said Gardian Fong Chung-yan, a clinical neurologist and member of the Hong Kong Neurological Society.

He said there were possibly thousands of causes of the disorder, not just the improper migration of brain cells.

"I can't comment on the study ... but with epilepsy, and with neurology in general, how diseases occur is kind of a mystery. Anything that will help us to learn how a disease develops, if the discovery shows the key processes of the disease, is good," he said.

The HKUST findings were published in *Nature Neuroscience*, a well-respected journal in the neuro-science field.

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有助研究治療癲癇藥物

科大破解蛋白功能

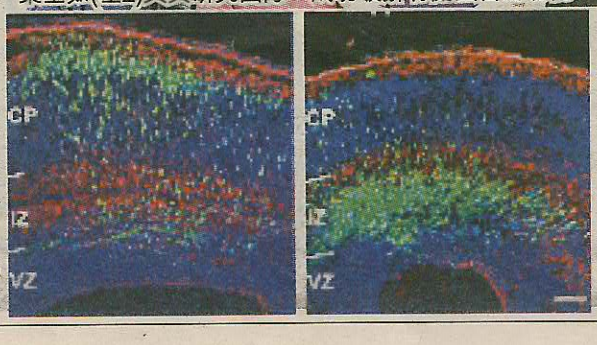
目前醫學界已知精神科疾病與腦內神經細胞異常有密切關係，但對大腦早期發育缺陷所引致的神經系統疾病，仍缺乏了解。科技大學的分子神經科團隊，便全球首次成功解開一種名為「α2-chimaerin」的蛋白，與癲癇發作的神經訊號傳遞機制有關，研究有助日後研發治療癲癇症的藥物。

科大理學院院長及分子神經科學國家重點實驗室主任葉玉如表示，腦是人體內最複雜的器官，其發育過程受到精密調控。新生的神經細胞必須在特定時間，遷移到正確的位置，才可以發揮正常功能。而神經細胞遷移紊亂便會導致大腦皮質發育異常，有可能引致精神分裂症、自閉症等神經系統疾病。

葉玉如與其研究隊伍，最近成功發現「α2-chimaerin」蛋白在調節神經細胞遷移和大腦功能方面有一定的作用。研究人員利用老鼠進行實驗，透過子宮內電穿孔技術，抑制老鼠胚胎大腦皮質中「α2-chimaerin」蛋白的功能，阻礙神經細胞的遷移過程，從而導致神經細胞在錯誤的區域堆積；有關老鼠出世後，因「α2-chimaerin」基因缺陷，出現自發性癲癇發作等異常行為。有關研究已在國際神經科學期刊《自然神經科學》(Nature Neuroscience)上發表。

研究結果證明，神經細胞的遷移過程一旦受到干擾，將會破壞神經迴路，並導致癲癇等病症發作。葉玉如指出，由於許多神經系統疾病與神經細胞的遷移異常有關，因此今次發現「α2-chimaerin」的功能，有助研究神經系統疾病的機制，以及日後研究治療藥物。

葉玉如(左2)與其研究團隊，成功破解有關蛋白的功能。



螢光綠色分為神經細胞，左圖大腦神經細胞的正常遷移；右則是受α chimaer抑制，導細胞遷移常。