Understanding Brain Tumor Growth Opens Door For Non-Surgical Treatment

One in 25,000 people worldwide is affected by neurofibromatosis type 2 (NF2), a condition where the loss of a tumour suppressor called Merlin results in multiple tumours in the brain and nervous system.

Sufferers may experience 20 to 30 tumours at any one time and such numbers often lead to hearing loss, disability and eventually death. Currently, the only available effective therapies are repeated invasive surgery or radiotherapy aimed at one tumour at a time and which are unlikely to eradicate all the tumours in one go. NF2 can affect any family, regardless of past history, through gene mutation. There is no cure.

However, a research team from Plymouth University Peninsula Schools of Medicine and Dentistry have moved one step nearer a non-surgical therapy, by identifying for the first time a new group of growth factor receptors that signal to brain tumours.

The study, which is published today (14th January 2013) in *Oncogene* (one of the world's leading cancer journals), shows that such receptors are over-expressed and activated in certain brain tumours. The study also identifies the mechanism that causes this activity and shows that, by interfering with the activation, tumour cells can be corrected.

Such growth factors are known to play a role in the development of other cancers, but this is the first time that the link has been made to cancer tumours in the brain and nervous system.

The breakthrough is key to the development of non-surgical therapies for NF2: there are drugs already available that target these growth factor receptors in other cancers and the Plymouth research team shows that there is scope for adapting such drugs for the treatment of NF2.

The research was led by Professor C. Oliver Hanemann, Director of the Institute of Translational and Stratified Medicine and Chair of Clinical Neurobiology at Plymouth University Peninsula Schools of Medicine and Dentistry, and Consultant in Neurology and Plymouth Hospitals NHS Trust. He said: "At present the only treatment available to NF2 sufferers is repeated surgery to remove tumours. This is only partially effective, because in some cases tumours are in areas where it is impossible to reach with surgery, and because eradicating a tumour from a part of the brain or nervous system does not mean that another one will not grow in its place. Chemotherapy is not an option, because in most cases NF2 tumours are slow growing – it is their sheer number that causes risk to the patient.

"Our study in *Oncogene* offers real hope to patients, because it identifies how the

growth of NF2 tumours works and shows that existing drugs could be modified to help stop and even reverse the rate of tumour growth. This is good news for patients for two reasons: it shows that there could be a valid alternative to surgery; and because the answer may be the adaptation of existing drugs, therapies could be developed relatively quickly because the process of clinical trials and drug registration has already taken place. Also the mechanism causing tumours in NF2 is also causing many spontaneous brain cancers and is found in other cancers. So what we found has potential relevance for other cancers."

The study comes hard on the heels of news of funding from the Medical Research Council for a study headed by Professor David Parkinson and Cancer Research UK for the same research team. The funding is being used to investigate why the mechanisms that suppress the growth and multiplication of tumours in the brain and nervous system do not work in some people, and to show how a new drug could be used as an alternative treatment to surgery.

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