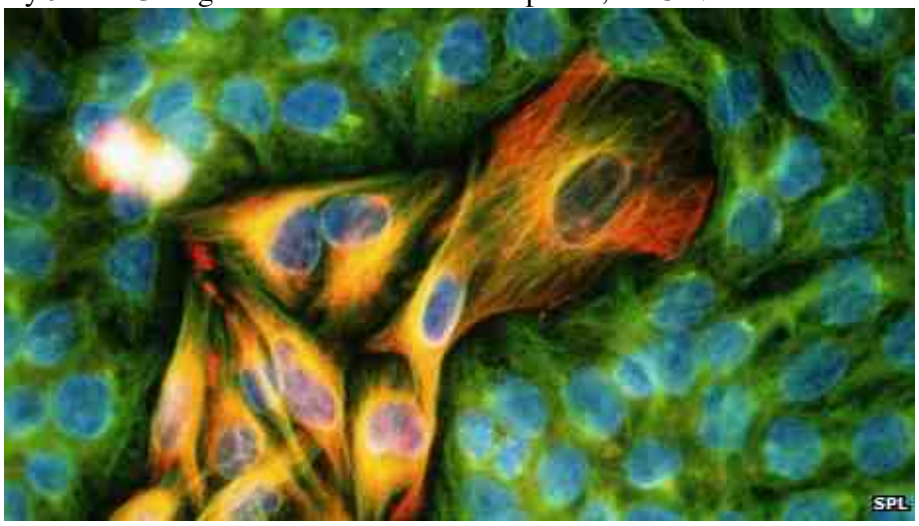
10 January 2013 Last updated at 01:24 GMT

'Drug holidays' beat cancer drug resistance in mice

By James Gallagher Health and science reporter, BBC News



Melanoma cancer cells surrounded by healthy tissue

- Skin cancer drug hopes from study
- Drug hope for advanced melanoma
- New skin cancer treatments emerge

Introducing medication-free spells to some cancer treatments may keep patients alive for longer, studies in mice with skin cancer suggest.

The animals had melanoma, which can rapidly become resistant to treatments.

However, a study in the journal Nature showed tumours also became dependent on the drug to survive. Withdrawing treatment caused tumours to shrink.

Experts said the findings were exciting, but still needed testing in people.

A team of scientists at the University of California, San Francisco and University Hospital Zurich, in Switzerland, were investigating how melanoma cells became resistant to a drug, vemurafenib.

The drug can slow the progress of a tumour in the short-term, but it soon becomes ineffective with deadly consequences.

Addiction

The tumours gain resistance by changing the chemistry of the inside of a cell. However, the researchers showed this process left the cancer cells dependent on the drug - like an addict.

When the mice were no longer given the drug, the tumours began to shrink.

The scientists used this knowledge to test a new way of prescribing the medication. Instead of giving the drug every day, the mice were given drugs for four weeks and then had a two week "drug holiday" before starting the pattern over again.

"It also offers the possibility of more cost effective treatment, with fewer side effects"

Prof Mark Middleton Cancer Research UK
Efim Guzik, professor of cancer biology at University of California, San Francisco, said: "Remarkably, intermittent dosing with vemurafenib prolonged the lives of mice with drug-resistant melanoma tumours.

"By seeking to understand the mechanisms of drug resistance, we have also found a way to enhance the durability of the drug response."

Whether the same effect would be seen in people given the same medication is uncertain.

Prof Mark Middleton, director of Cancer Research UK's Experimental Cancer Medicine Centre in Oxford, said: "We still need to test the idea in the clinic, but these results suggest a way in which this important new treatment might be able to increase the benefit to patients and their families.

"It also offers the possibility of more cost effective treatment, with fewer side effects, because patients would spend some of the time off vemurafenib."

Vemurafenib targets a mutated protein called BRAF. Prof Richard Marais, from the Paterson Institute for Cancer Research in Manchester, was involved in the discovery of the BRAF fault.

He said the findings were "very compelling".

"This new study is exciting because it suggests a way to combat the evolution of drug resistance in melanoma patients using the drugs we already have, rather than having to develop new ones.

"It will be interesting to see if these lab results are mirrored in clinical trials."

Prof Marais said the same effect is possible in other forms of targeted cancer drug treatment.