Gene fault could predict ovarian cancer drug success

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Faults in a gene commonly inactivated in many different types of cancer could be used to predict which drug combination ovarian cancer patients are most likely to benefit from, according to research at Newcastle University.

The study presented at the American Society for Clinical Oncology found that women with faults in a gene known as p53 were 50 per cent less likely to survive, but tended to be more likely to respond to the drug paclitaxel in addition to the standard treatment of carboplatin.

Those without such mutations had better survival rates but did not benefit from having paclitaxel added to their treatment, meaning patients like this could be spared unnecessary side effects in the future.

Dr Hilary Calvert, who led the study, said: "These results show that ovarian cancer patients whose tumour had a faulty p53 gene survive longer if given paclitaxel. Although survival rates have improved dramatically in recent years, ovarian cancer remains one of the most deadly cancers in women and efforts to improve survival by targeting treatments at those most likely to benefit are urgently needed."

The work, led by researchers from Newcastle University including Professor John Lunec, the UCL Cancer Institute and the MRC, was funded by Cancer Research UK and the Medical Research Council (MRC).

The scientists examined tumour samples from 265 patients who had taken part in the MRC's ICON 3 study, which found no significant benefit in giving patients paxlitaxel in addition to the standard treatment of carboplatin.

But this contradicted earlier studies, which showed that adding paxlitaxel to treatment improved survival leading to this drug combination being adopted as a standard treatment for ovarian cancer.

The researchers believed that this difference in response to paxlitaxel could be related to the genetic makeup of the tumour and suspected that p53 faults may be behind this.

So to put their theory to the test they analysed all the tumour samples to see which had p53 faults. This revealed that p53 was inactive or faulty in around half of the samples (130/265) and also that patients only benefited from paxlitaxel if p53 was faulty in their tumour.

Dr Lesley Walker, Cancer Research UK's director of cancer information, said: "Our scientists discovered p53 over thirty years ago and it's good to see this now being used as a biomarker to improve treatment for patients.

"There's no such thing as a one-size-fits-all drug and increasingly scientists are developing ways to identify groups of patients that are most likely to respond to a particular drug. This approach is called stratified medicine and many scientists now believe it could transform cancer treatment in the future."

Provided by Newcastle University