Gene assay to help to predict lung cancer treatment resistance

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The genes that may contribute to drug resistance in non-small cell lung cancer (NSCLC) can be predicted. Researchers writing in the open access journal *BMC Cancer* found good correlation between genes believed to be involved in drug sensitivity and resistance and actual in vitro chemosensitivity.

Ian Cree, from Queen Alexandra Hospital, Portsmouth, UK, led a team of researchers who assessed the chemosensitivity of a series of 49 NSCLC tumors and compared this with quantitative expression of putative resistance genes measured by RT-PCR. He said, "There was considerable heterogeneity between tumors, and while this showed no direct correlation with individual gene expression, there was strong correlation of multi-gene signatures for many of the single chemotherapy agents and combinations tested. This may allow the definition of predictive signatures to guide individualized chemotherapy in lung cancer".

The researchers tested docetaxel, cisplatin, gemcitabine and combinations of the agents on tumour cells taken from 49 fresh NSCLC samples. There were considerable differences between tumors in their sensitivity to individual agents and combinations, though the combination of cisplatin + gemcitabine was usually the most active.

When these results were compared to the gene expression in the tumors, Cree and his colleagues were able to identify a number of patterns, especially in chemosensitivity to combinations of treatments. Cree said,

"The genes identified in this study fall into several categories, linked with much studied mechanisms such as metabolism within the cell, membrane drug pumps, and DNA repair, but also with apoptosis, suggesting that the general susceptibility of the cell to undergo this process may be an important determinant of tumor chemosensitivity, outweighing more specific mechanisms".

More information: Resistance gene expression determines the in vitro chemosensitivity of non-small cell lung cancer (NSCLC); Sharon Glaysher, Dennis Yiannakis, Francis G Gabriel, Penny Johnson, Marta E Polak, Louise A Knight, Zoe Goldthorpe, Katharine Peregrin, Mya Gyi, Paul Modi, Joe Rahamim, Mark E Smith, Khalid Amer, Bruce Addis, Matthew Poole, Ajit Narayanan, Tim J Gulliford, Peter E Andreotti and Ian A Cree; *BMC Cancer* (in press); www.biomedcentral.com/bmccancer/

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