

New research confirms need for lung cancer testing

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Different kinds of lung cancer behave in different ways, suggesting they are fundamentally different diseases. According to a University of Colorado Cancer Center study published in *Cancer*, the official journal of the American Cancer Society, different subgroups of non-small cell lung cancer (NSCLC) show distinct patterns of spread in the body.

The study looked at 209 patients diagnosed with stage IV [non-small cell lung cancer](#) separated into four different molecular subgroups using testing performed by the University of Colorado Molecular Correlates Laboratory (CMOCO): those with epidermal growth factor receptor (EGFR) mutations, v-Ki-ras2 Kirsten rat sarcoma viral oncogene homolog (KRAS) mutations, anaplastic lymphoma receptor tyrosine kinase (ALK) gene rearrangements or a group without any of these abnormalities.

ALK positive lung cancer was strongly associated with cancers that spread to the linings around the heart and lungs (pericardial and pleural disease). Patients with ALK positive NSCLC were also predisposed to develop [liver metastases](#) as were those with an EGFR mutation when the different subgroups were compared.

"In the last few years we have been able to separate lung cancer into different molecular subtypes to help improve outcomes from specific targeted therapies. This study really confirms that these molecular subtypes are manifesting as different diseases in patients," said Robert Doebele, MD, PhD, lead author of the study and CU Cancer Center

investigator.

Another University of Colorado study published in the same edition of Cancer drills down on how some of these different molecular subtypes of lung cancer are detected in the first place. Specifically, the study examined the companion diagnostic test for detecting the ALK positive lung cancer cases, called a fluorescence in situ hybridization (FISH) test. This test is used to select patients for treatment with crizotinib, an ALK inhibitor drug. Now University of Colorado researchers have solved an unanswered question as to why patients who respond to crizotinib only appear to have the ALK change in a fraction of the cells in their cancers.

By looking in detail at the genetic changes present in the cancer cells they came to two major conclusions. First- cell counts below 100 percent in ALK positive tumors reflect the fact the assay misses a proportion of cells and not that the cells are truly missing the ALK change.

Second, the ALK change happens early in the development of the cancer and is likely to be a fundamental driver of the cancer's growth. The study was conducted by D. Ross Camidge, MD, PhD, the director of the lung cancer clinical program at the CU Cancer Center and University of Colorado Hospital and Marileila Varella Garcia, PhD, professor of medicine, medical oncology and pathology at University of Colorado School of Medicine.

"Knowing that ALK changes are driving the cancer and that they are not actually missing from a significant proportion of the cancer reinforces our understanding of ALK positive lung cancer. It now makes sense why targeting these changes is going to affect a very large proportion of the tumor and explains the dramatic clinical responses seen with crizotinib."

"In order to treat any disease successfully, you have to know what you are really treating," said Camidge. "It is only by understanding [lung](#)

[cancer](#) better that we can hope to improve outcomes in the long term."

Provided by University of Colorado Denver

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