The chemical elements in lung tissue and lung cancer subtypes

Ryosuke Chiba[1], Naoto Morikawa[1], Koichiro Sera[2], Takako Hosokawa[3], Satoshi Moriguchi[1], Wataru Shigeeda[4], Hiroyuki Deguchi[4], Makoto Tomoyasu[4], Hajime Saito[4], Kazuyuki Ishida[5], Tamotsu Sugai[5] and Makoto Maemondo[1]

[1]Division of Pulmonary Medicine, Allergy and Rheumatology, Department of Internal Medicine, School of Medicine, Iwate Medical University
19-1 Uchimaru, Morioka, Iwate 020-8505, Japan

[2]Cyclotron Research Center, Iwate Medical University
348-58 Tomegamori, Takizawa, Iwate 020-0603, Japan

[3]Nishima Memorial Cyclotron Center, Japan Radioisotope Association
348-58 Tomegamori, Takizawa, Iwate 020-0603, Japan

[4]Department of Respiratory Surgery, Iwate Medical University School of Medicine
19-1 Uchimaru, Morioka, Iwate 020-8505, Japan

[5]Department of Molecular Diagnostic Pathology, School of Medicine, Iwate Medical University
19-1 Uchimaru, Morioka, Iwate 020-8505, Japan

Abstract

Background: Several studies have suggested that some trace elements may affect the onset of lung cancer. However, the effect of trace elements on lung cancer carcinogenesis is poorly understood. The aim of this study was to assess if trace elements may be the cause of carcinogenesis in lung cancer tissues of patients with lung cancer with a non-smoking history, driver mutations, or histology.

Methods: The study included patients with non-small cell lung cancer who had undergone surgical resection. For the measurement of trace elements, surgically resected samples were studied using particle induced X-ray emission analysis. In total, 54 elements were investigated in each sample. Based on the pathology and driver mutation status, samples were classified into the following groups: lung adenocarcinoma (LADC) with *EGFR* mutation (LADC EGFRm+); LADC with *KRAS* mutation (LADC KRASm+); LADC without *EGFR* mutation, *KRAS* mutation, and *ALK* rearrangement (LADC wt); and lung squamous cell carcinoma (SCC). Tissues from 20 patients with a non-malignant disease were also analyzed for trace elements as controls.

Results: The levels of 6 trace elements were increased in the LADC wt group. Copper was increased in the LADC EGFRm+ group. Cobalt and zinc were increased in the LADC KRASm+ group. There were no differences in trace element levels between the SCC group and the control group.

Conclusion: Trace elements may play a role in the pathology and molecular signature of lung cancer.