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**Elucidating the role of miRNAs expression as a post-transcriptional mechanism in the carcinogenesis of air pollution-related lung cancer**

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**ABSTRACT**

Previous epidemiological studies have reported positive associations between ambient air pollution and lung cancer, and henceforth, air pollution has been classified as group 1 carcinogen in humans. There are limited studies examining the molecular pathogenesis in the tumorigenesis of air-pollution lung cancer, including the role of noncoding RNA (ncRNA) and micro RNA (miRNAs). Dysregulation of miRNAs expression are shown to be correlated with cancer, suggesting their potentials as the biomarker and therapeutic agents in cancer. To determine the differentially expressed miRNAs in air pollution-related lung cancer, we performed mature miRNAs expression profiling using real-time PCR in 10 lung adenocarcinomas with low (n=5) and high (n=5) exposure of air pollution which were defined based on residential address, annual particulate matter 10 and anthropometric activities such as occupation, transportation and daily ambient exposure of less than one or more than four hours. We identified 27 upregulated miRNAs in the high exposure group. miR-15b-5p (p=0.0269), let-7a-5p (p=0.0426), miR-151a-5p (p=0.0449), miR-222-3p (p=0.0446), and let-7f-5p (p=0.0333) were the most significantly upregulated miRNAs. All the miRNAs, except miR-151a-5p, were reported to be involve in non-small cell lung cancers (NSCLCs), and showed the highest number of targets, including the proto-oncogene such as epidermal growth factor receptor (EGFR), RAF family member (BRAF), and RAS GTPase family member (KRAS, NRAS and HRAS), and tumour suppressors Retinoblastoma 1 (RB1) gene, whereas miR-151a-5p revealed the lowest number of targets. Pathway enrichment analysis showed that all the significantly upregulated miRNAs were most frequently associated with adherens junction pathway, which play a major role in cell-cell adhesion in tumour as well as in normal tissues. These preliminary results suggest that upregulation of miR-15b-5p, let-7a-5p, miR-151a-5p, miR-222-3p, and let-7f-5p may be crucial in the tumorigenesis of air pollution-related lung cancer via the dysregulation of adherens junction pathway.