ABSTRACT
We find that MCM2 is detectable in premalignant lung cells that are 2-3 times more proliferating than Ki-67. The fact that it is present in cells at the surface of metaplastic lung lesions, which are more likely to be exfoliated into sputum, lends support to its use as an early marker for significantly enhancing lung cancer survival rates through anti-MCM2 therapy.

INTRODUCTION

Introduction The lung is a major organ of the body and major organ of the immune system. The lung is a central organ of the immune system and plays a significant role in a wide range of diseases, including asthma, allergies, chronic obstructive pulmonary disease (COPD), chronic obstructive pulmonary disease (COPD), and various types of cancer, and is often the first line of defense against many of these diseases. Lung cancer is the second most common cause of death in men, and lung cancer is the most common cause of death in women. Lung cancer is the second leading cause of cancer mortality in the world. Lung cancer is a leading cause of lung cancer-related deaths globally.1,2

Cancer is a chronic disease in which the cancer cells in the body proliferate and grow Therefore, it is necessary to develop methods for detecting bronchial mucosal abnormalities that are precursors to lung cancer before systemic shedding of tumor cells occurs (these precursor lesion types may also appear after tumour growth and after the removal of some other potentially benign lesions), such as metaplasia, dyspnea or carcinoma in situ (CIS), which are thought to represent progressive histologic causes of squamous cell carcinoma. According to current data, 23% of current and former smokers have metaplastic lesions, while 2% have dysplastic deseases. However, not all cesions develop lung cancer. Smoking cessation, which can be viewed as an active intervention, appears to decrease metaplasia rates from 27% to 7% in former patients. It is estimated that 50% of CIS will progress to non-invasive cancer over 6 months, but 4 patients who underwent regular bronchoscopy at 6-month intervals developed lung tumors One potential solution to these questions is the development of specific immunohistochemical markers that can enhance the sensitivity and reliability of current methods used to detect precursor lesions in histologic and cytologic specimens. As a result, proliferation is essential for lung cancer development, and the use of markers specific to cell proliferation should be considered. Proliferating cell nuclear antigen (PCNA) and Ki-67 have been extensively researched in this context. PCNA is a homotrimeric protein that binds to DNA and proteins involved in DNA replication and repair, and is essential for DNA proliferation. However, due to its importance as an epitope of nascent nuclear protein recognized by the MIB-1 monoclonal antibody, Ki-609 is frequently expressed throughout the cell cycle of proliferous cells. Immunohistochemical studies suggest that increased lung tumor marker markers can indicate decreased survival. We have presented results from our comparison of Ki-67 and a new proliferation marker, MCM2, along with evidence that the latter is also detectable in proliferating cells. MCFs are responsible for limiting DNA replication to one round per cell cycle and can act as 'licensing factors' For example, immunoglobulin A (IBSA) may be used by certain cancer models to detect human breast cancer, while others have found evidence similar to this.

CONCLUSION

We have demonstrated that MCM2 antibodies can provide consistent,

reliable staining in fixed tissues without the need for antigen retrieval. The results obtained are easy to interpret as there is a significant difference between normal bronchoepithelium and premalignant lesions, making it an easy-to-use marker that has great potential for assessment of the progression and regression of morphologically abnormal lesIONS in future primary lung cancer prevention studies and for early detection of lung Cancer screening studies.