Lung cancer is the leading cause of cancer-related death in the western world. At the time of presentation, most patients are at an advance stage of disease and have a poor prognosis. Lung cancer comprises two major pathologic categories, which are treated differently: small-cell (SCLC) and non small-cell lung cancer (NSCLC). SCLCs display neuroendocrine features and have a propensity for rapid growth and early metastasis. NSCLCs include adenocarcinoma, squamous, bronchio-alveolar and large-cell histologies, although the latter often has neuroendocrine features. Associated with an increase in female smokers and the use of filtered cigarettes, the proportion of adenocarcinomas has increased, while squamous carcinomas and SCLCs have decreased. SCLCs account for less than 20% of lung tumors. Because of its propensity for early metastases, it is treated with chemo- and radiation therapy, but not surgery. While initially very responsive, nearly all patients relapse and die of their disease. In contrast, NSCLCs are potentially curable by surgery if discovered early. Thus, attempts at earlier diagnosis are ongoing and are discussed by Drs. Tanner and Silvestri. Two important new developments in NSCLC have been the discovery of activating mutations involving EGFR and ALK in distinct subsets, which respond to specific inhibitors. Lung cancers are characteristic of complex cytogenetic abnormalities that include EGFR and ALK alterations, described in the review by Dr. Varella Garcia. In addition to cytogenetic abnormalities, an increasing number of chromatin epigenetic and microRNA alterations are being recognized, together with dysregulation of the cell cycle. These are described in two articles by Dr. Gazzeri’s group and colleagues (Van Den Broeck et al.; Eymin and Gazzeri). Knowledge of the functional role of these recurrent genetic and epigenetic changes should lead to the development of novel therapeutics, which is discussed by Drs. Varella Garcia and Gazzeri. Poor prognosis generally implies invasion and metastasis. The role of integrins in cell adhesion, migration, proliferation, lung 

**Letter from the guest editors**

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**About Dr. Joëlle Roche**

Dr. Joëlle Roche is Professor of Biochemistry and Molecular Biology at the University of Poitiers (France) and is currently a visiting Professor of Medicine at the Medical University of South Carolina in Charleston (USA). Dr. Roche received her Ph.D. (Doctorat-ès-Sciences) in 1985 at the University of Grenoble (France) under the supervision of Dr. Jean Jacques Lawrence. This was followed by a position of Associate Professor at the University of Lyon (France) and a sabbatical at the University of Colorado in Denver, where she developed her interest in lung cancer after discovering the semaphorin, SEMA3F, from a recurring 3p deletion. Dr. Roche is interested in gene expression, chromatin remodeling and guidance molecules. During the last fifteen years, her work has focused on semaphorin expression and function in lung cancer.

**About Dr. Harry Drabkin**

Dr. Harry Drabkin is Head of the Division of Hematology-Oncology at the Medical University of South Carolina (MUSC) in Charleston and holds the Gilbreth Chair in Clinical Oncology. He is known for his work in leukemia, having discovered the AML1-ETO fusion gene resulting from the t(8;21). His studies with his colleague, Dr. Robert Gemmill, involve the hereditary kidney cancer gene, TRC8, and their early genomic work led to the establishment of a yeast artificial chromosome (YAC) contig of human chromosome 3. It was the genomic work that provided many of the reagents that led to the discovery of the SEMA3F gene with Dr. Joëlle Roche, when she was a visiting Professor in Denver. Before coming to MUSC in 2007, Dr. Drabkin spent most of his professional career at the University of Colorado, where he worked with Dr. Paul Bunn. Dr. Drabkin has continued his involvement in the Colorado Lung Cancer SPORE grant, which has led to increasing ties and collaborations between the two universities.
cancer cell survival, activation of growth factors and trafficking of their cognate receptors is described by Dr. Caccavari et al. Integrin activation is triggered by extracellular guidance cues, chemokines and growth factors. Nasarre et al. review the repertoire of guidance molecules involved in lung cancer. Although initially described in the nervous system to control axon outgrowth, these molecules are now well recognized as contributors to tumor growth, angiogenesis and metastasis as well regulators of the tumor microenvironment. MET, the receptor for hepatocyte growth factor (HGF), is often upregulated in lung tumors and its tyrosine kinase signaling pathway is involved in cell adhesion, growth and motility. Drs. Lawrence and Salgia describe MET signaling in lung cancer and corresponding therapeutic approaches.

Another form of lung cancer, malignant pleural mesothelioma (MPM), is associated with exposure to asbestos. This disease, described by Dr. Grégoire, has a very poor prognosis although immunotherapy strategies may prove useful.

Together, these reviews provide a current view of many aspects of lung cancer biology and new potential therapeutic targets.