

Differential carcinogenicity of cigarette smoke in mice exposed either transplacentally, early in life or in adulthood

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Abstract

Cigarette smoke (CS) plays a dominant role in the epidemiology of human cancer. However, it is difficult to reproduce its carcinogenicity in laboratory animals. Recently, we showed that CS becomes a potent carcinogen in mice when exposure starts soon after birth. In our study, we comparatively evaluated the carcinogenic response to mainstream CS in mice at different ages. Neonatal mice were exposed daily for 4 months to CS, starting within 12 hr after birth, and sacrificed at 8 months. Adult mice were exposed for the same time period (3–7 months) and sacrificed at 11 months. Other mice were exposed transplacentally or both transplacentally and early in life. A total of 351 neonatal mice and 80 adult Swiss H mice were used. With varying intensity depending on age, CS induced pulmonary emphysema, bronchial and alveolar epithelial hyperplasia, blood vessel proliferation and hemangiomas and microadenomas in lung as well as parenchymal degeneration of liver. Histopathological alterations of kidney were only observed in mice exposed to CS early in life. Lung adenomas and malignant tumors of various histopathological nature were detected in neonatally exposed mice but not in adults. Transplacental CS induced the formation of lung adenomas in the offspring 8 months after birth. Previous exposure during pregnancy attenuated CS-related alveolar epithelial hyperplasia induced after birth. In conclusion, the carcinogenic response to CS varies depending on the developmental stage. The early postnatal life and the prenatal life are particularly at risk for the later development of CS-related tumors.

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