p16INK4A inactivation mechanisms in non-small-cell lung cancer patients occupationally exposed to asbestos.

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Figure 1. Immunodetection of $p16^{INK4A}$ by immunohistochemistry in NSCLC patients

A: Positive control of $p16^{INK4A}$

B: Positive immunostaining of $p16^{INK4A}$ in a lung adenocarcinoma case (× 200)

C: Lack of $p16^{INK4A}$ expression in a lung adenocarcinoma case (× 200)

Figure 2. Representative patterns of aberrant methylation promoter of *P16/CDKN2A* gene in NSCLC patients.

Primer sets used for amplification are designated as unmethylated (UM) or methylated (M). Twenty µl of PCR product were run on 2.5% agarose gel stained with ethidium bromide, and visualized under UV illumination. T: tumor tissue; N: normal lung tissue; M: DNA 50 bp ladder; N_M: negative control with methylated sequence specific primer; N_{UM} negative control with unmethylated sequence specific primer.

Figure 3. FISH hybridization. Dual-color FISH with PONC0921 probe. (*P16/CDKN2A* specific DNA probe is direct-labeled with rhodamine and the chromosome 9 classical satellite probe is direct-labeled with fluorescein).

A: Normal lung tissue used as positive control showed two red signals and two green signals.

B: NSCLC case showing two green signals and one red signal indicating a loss of heterozygosity of *P16/CDKN2A* gene.

C: NSCLC case showing two green signals and no red signal, indicating a

homozygous deletion of *P16/CDKN2A* gene.