Lung, liver and bone cancer lifetime risk after intake of ^{239}Pu

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Workers involved in processing of ^{239}Pu are subject to two major intake pathways of the nuclide: inhalation and wounds. While the inhalation intake limit is based in understanding of Pu biokinetics and lung clearance, the intake limits for intake via wounds do not exist.

Three organs in the human body are subject to greatest exposures from incorporated Pu: lung, liver and bone after inhalation intake and liver bone after intake through wounded skin.

Earlier we have calculated excess risk of lung, liver and bone cancer related to inhalation intake of ^{239}Pu in Mayak Worker Cohort. In our current report we use these models to calculate lifetime risk of lung, liver and bone cancer after ^{239}Pu intake via inhalation and wounded skin.

In order to do this, we calculated equivalent doses resulting from ^{239}Pu incorporation in lung, liver and bone according to several scenarios including (among others) chronic inhalation intake at the level of annual limit of intake (ALI). We used Lifetime Excess Risk (LER) and Risk of Exposure Induced Death (REID) and their annual increments as characteristics of excess lifetime detriment caused by incorporated ^{239}Pu.

For the scenario of chronic intake at the ALI level we demonstrate that at age 70 the annual lifetime risk increment of lung cancer is by a factor of 4 higher than that assumed by regulatory bodies as acceptable (0.001 per year).

We discuss the reasons for that and possible ways of avoiding extra carcinogenic risk in workers who contact to ^{239}Pu or other transuranium nuclides with long biological half-life.