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Letter to Editor

Radon risk in carcinogenesis what prevention

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In environments, exposure to 222radon is to be considered chronic and the effects are based on cellular, subcellular and molecular radiotoxicological mechanisms [1]; We start from the alpha particles and their decay products, but the action continues through the access into the pulmonary tract with aerosols and environmental polluting particles (size 0.0005-100 μm in diameter) [2].

The action consists of an immunological reaction based on macrophages and involvement of the lymphatic system. Alpha particles react with DNA, create oxidative stress and radiolysis, but the radioactive effects also continue due to the gamma energy of 14 Bi [3,4]. Many studies prove to be aimed at clinical evidence deriving from the action of radon and in predicting diseases such as lung cancer [5-7], as well as to indicate which carcinogenetic theory is applicable, it is conceivable that 222radon alters communication between cells and their microenvironment, because the biophysical and molecular messages of induction processes are no longer recognized [8,9]; US-EPA [7] indicates 13% of lung cancers to be attributed to radon in the general population, but if the exposure is in the work environment this disease becomes occupational. Preventive action must certainly identify environments with objectives of improving ventilation and useful and necessary building rules and regular monitoring. The concomitance of COPD in the exposure to radon and the decay product cannot be excluded [10]. The most exposed subjects must be considered with particular attention, at the same time discouraging the habit of active and passive smoking, activating where necessary high-resolution CTA, periodic cytopathological examination, spirometry and alveolar-capillary diffusion, detection of allergic diseases. The environment requires adequate environmental changes, continuous ventilation, environmental remediation of fungi and bacteria. The analysis of lung histological types also predicts that in the case of primary lung tumors, there is a higher incidence of the histological type of NSLC (Non small cell Lung Cancer) "Lung adenocarcinomas" of the order of 43% in smokers and 45% in non-smokers, compared to "squamous epidermoid carcinoma" equal to 42% in smokers and 33% in non-smokers. What remains about 20% includes small cell lung cancer (17% SCLC), carcinoid, sarcoma and other histoptypes [11].

Many studies in monitoring of miRNA in pulmonary cancer are ongoing and bode well, miRNAs profile were changed when exposed to radon in cellular culture [12].

The latency time is very long necessary for the appearance of these respiratory pathologies determines an etiological uncertainty about the origin of the pulmonary pathology. Some studies on the respiratory system show pulmonary hypertension, hypoxigenation, cor pulmonale [4] arouse attention even if a true association between residential 222radon and mortality from non-malignant respiratory diseases needs to be further investigated (Cancer Prevention Study-II) indicates a positive linear trend in COPD mortality by increasing categories of radon concentrations ($p < 0.05$) [13-16].

Recommendation to maintain the 300 Bq m^{-3} levels in the indoor phase, which were reviewed in 2014 at the lower level reasonably achievable in the range 100 - 300 Bq m^{-3} [17].

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