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Well water radioactivity and risk of cancers of the urinary organs

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Abstract

Water from bedrock frequently contains higher concentrations of natural radionuclides than water from other sources. Bladder and kidneys receive a radiation dose when radioactive isotopes are excreted into urine. The subjects for this case–cohort study were selected from all drilled wells users in Finland. The study comprised 61 bladder cancer and 51 kidney cancer cases diagnosed between 1981 and 1995, as well as a random sample of 274 reference persons, stratified by age and sex. The median activity concentrations of radon in drilled wells used by bladder and kidney cancer cases and the reference cohort were 170, 140, and 130 Bq/L, respectively. The radium concentration was 0.01 Bq/L for all groups and the uranium concentrations were 0.08, 0.07, and 0.06 Bq/L, respectively. The bladder cancer risks associated with radon, radium, and uranium activity concentrations in drinking water were 1.02 (0.68–1.54) per log(100 Bq of radon/L), 0.73 (0.21–2.50) per log(0.1 Bq of radium/L), and 0.77 (0.32–1.89) per log(1 Bq of uranium/L). The corresponding figures for kidney cancer were 0.81 (0.47–1.37), 0.12 (0.01–1.10), and 0.92 (0.36–2.35), respectively. In conclusion, even though ingested radionuclides from drilled wells are a source of radiation exposure, they are not associated with a substantially increased risk of bladder or kidney cancers in concentrations occurring in drilled wells.

Keywords: Bladder cancer; Kidney cancer; Radium; Radon; Uranium; Drinking water; Drilled wells; Ionizing radiation

1. Introduction

Ionizing radiation is one of the best established human carcinogens (IARC, 2001). Natural radiation is the major component of radiation exposure for the general population. Due to granitoid composition combined with soft and slightly alkaline bicarbonate waters typical for Finnish bedrock, concentrations of several natural radionuclides, such as radon and uranium, can be high in wells drilled into bedrock, whereas radium levels usually remain low. The radionuclide levels in drilled wells are clearly higher than in shallow wells dug in soil or in public water supplies (Salonen, 1994).

Uranium-238 (^{238}U) series radionuclides are the major contributors to the radiation dose caused by natural radionuclides ingested with drinking water (UNSCEAR, 2000). The most important of these nuclides is ^{222}Rn (referred to as radon in the rest of this paper), which is also released from water to indoor air. Other long-lived nuclides include ^{238}U, ^{234}U, ^{226}Ra (radium), ^{210}Pb, and ^{210}Po. These nuclides are mostly alpha emitters but they or their short-lived progeny also emit beta and gamma radiation. In addition, a minor fraction of natural uranium consists of ^{235}U. Alpha radiation and beta radiation have a short range and are unable to penetrate deep into tissue. Therefore, they can irradiate cells of internal organs only through internal exposure, i.e., if they enter the body through ingestion of water containing radionuclides. Cells in kidneys and bladder are potentially irradiated by radionuclides excreted in the urine. Gamma radiation, on the other hand, is able to penetrate tissue and hence damage cells even when the source remains outside the body.

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Most of the ingested radon is removed through exhalation, but the longer-lived nuclides are eliminated also by urinary excretion (National Research Council, 1999). The majority of the effective dose from ingested radon is directed to the stomach, but bladder and kidneys also receive some dose (Kendall and Smith, 2002). Approximately, 20% of ingested radium is absorbed, is taken into the bloodstream, and accumulates mainly in the bones (ICRP, 1993). Radium is excreted slowly through the urine and feces. For the ingested uranium, the main target organ of toxicity is the kidney (Kurttio et al., 2002; Zamora et al., 1998). In continuous uranium exposure through drinking water, urinary uranium excretion is approximately 0.3% of the ingested uranium (Karpas et al., 2005).

An increase of kidney cancers in male rats exposed to radon has been reported (Masse et al., 1992). A correlation study on country-specific indoor air radon levels and cancer rates suggested that radon is associated with increased kidney cancer incidence (Henshaw et al., 1990). However, no evidence between inhaled radon and urinary organ cancers was found in a large combined miner cohort (Darby et al., 1995). These studies, however, concerned primarily exposure through inhalation rather than ingestion.

As far as we know, no earlier studies have been conducted at an individual level evaluating the relationship between radon, radium, or uranium in drinking water and risks of bladder or kidney cancer in humans. The aim of the present study was to examine the association between exposure to naturally occurring radionuclides in drinking water and risks of bladder or kidney cancers.

2. Materials and methods

2.1. Study population

The base population was defined from the Population Register Centre as the 144,627 persons born between 1900 and 1930 who had lived outside the municipal water supply from 1967 to 1980. All the 884 bladder and 644 kidney cancer cases diagnosed during 1981–1995 within the base cohort were identified from the Finnish Cancer Registry. A base reference cohort ($n = 4590$) was selected randomly from the base population, with the number of persons in each stratum (defined by sex and 5-year age category) set as four times the highest number of the bladder, kidney, or stomach cancers or leukemia (same base reference cohort was used earlier for analysis of risk of stomach cancers (Auvinen et al., 2005) and leukemia (Auvinen et al., 2002)). Information on residential history and potential confounders was collected using mailed questionnaires. Only those persons who had lived in residences with drinking water from drilled wells before 1981 were eligible for the study: 371 (8%) reference cohort members, 79 (9%) bladder cancer cases, and 65 (10%) kidney cancer cases. The final study population for whom both informed consent and activity concentrations of water samples were available consisted of 274 (74% of the eligible) reference cohort members, 61 (77%) bladder cancer cases, and 51 (78%) kidney cancer cases. The selection of the cancer cases and the reference cohort as well as the description of the questionnaires has been described in more detail elsewhere (Kurttio et al., 1999).

2.2. Exposure assessment

Water samples of cases and controls were collected and analyzed in a blind fashion. The procedures of water sampling, sample conserving, and preparation have been described in more detail elsewhere (Auvinen et al., 2002; Kurttio et al., 1999). Radon (^{222}Rn) was measured using liquid scintillation counting (Salonen, 1993a). The occurrence of radium (^{226}Ra) and total uranium (^{234}U + ^{235}U + ^{238}U) in water samples was first screened by measuring the gross alpha and gross beta activities using low-background liquid scintillation spectrometry (Salonen, 1993b). Radium was determined from the alpha spectrum of the liquid scintillation spectrometer using the separate peak of ^{214}Po. Radiochemical alpha spectrometry was conducted for uranium isotopes if the radon concentration exceeded 500 Bq/L or if the gross alpha or gross beta spectrum indicated the presence of these nuclides. If concentrations of uranium isotopes were low according to the alpha spectrum, the total uranium concentration was calculated by subtracting the radium and ^{210}Po concentrations from the gross alpha concentration (total uranium = gross alpha—(radium + ^{210}Po)). From the total uranium, the concentrations of ^{234}U and ^{238}U were calculated using the average activity ratio (^{234}U/^{238}U) of 2.

Radionuclides from thorium decay series do not contribute to the result of gross alpha because thorium isotopes occur generally at low concentrations in ground waters and ^{224}Ra decays off during the month that passes from sample preparation to its measurement.

Minimum detectable activity concentrations were 1 Bq/L for radon, 0.01 Bq/L for radium, and 0.0005 Bq/L for uranium isotopes (by alpha spectrometry) and 0.02 Bq/L for total uranium (by liquid scintillation spectrometry).

To evaluate the validity of the water sampling technique, two water samples were taken from 49 randomly selected wells at two different times (median 34 days apart). There was good correspondence of the levels of radon, radium, and gross alpha activities in the original samples and in the duplicate samples (r^2 and regression coefficients from log-transformed values were 0.91 and 0.87 (95% confidence interval 0.78–0.95) for radon, 0.95 and 1.38 (1.29–1.47) for radium, and 0.98 and 1.11 (1.06–1.16) for gross alpha).

To calculate the equivalent organ doses (i.e., a measure that indicates the degree of biological damage caused by different types of absorbed radiation) a daily water consumption of 2 L was assumed. It was also assumed that 75% of radioactivity attributable to radon gas is released from water before ingestion. The ingestion dose coefficient (Sievert/Becquerel (Sv/Bq)) of radon, radium, ^{234}U, and ^{238}U were taken from different sources (National Research Council, 1999; ICRP, 1993, 1994). The total equivalent bladder or kidney doses were calculated as the sum of organ doses from each nuclide.

The effective dose takes into account the equivalent doses of all organs and reflects the different radiosensitivities of various tissues. The effective dose coefficients were taken from the same sources as the ingestion dose coefficients. The total annual effective radiation doses were calculated as the sum of doses from each nuclide.

Cumulative equivalent organ doses and effective doses were calculated using the duration of well water consumption and annual doses.

2.3. Data analysis

In a case–cohort study, the analysis is based on a modified proportional hazards model, with comparison of each case at the date of diagnosis with the members in the reference cohort who are at risk at that time (Barlow, 1994; Gail and Benichou, 2000; Langholz, 2000; Prentice, 1986). The closing date of follow-up was cancer diagnosis, death, or January 1, 1996, whichever was earliest. Hazard ratios (HRs) were estimated using the PHREG procedure of SAS (SAS Institute, Cary, NC). The calculation of 95% confidence intervals (95% CI) was carried out using a robust variance matrix (Barlow, 1994) obtained with the SAS interactive matrix programming language.

To allow for the latency period of cancer, we took into account only exposure occurring up to 10 years prior to the cancer diagnosis and the respective year for referent persons. The exposure variables were log-transformed (log(value + 1)) to limit the influence of a few very high values on the regressions. Indicators of exposure were also analyzed using