Estimation of residential radon exposure and definition of Radon Priority Areas based on expected lung cancer incidence

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A B S T R A C T
Radon is a naturally occurring gas, classified as a Class 1 human carcinogen, being the second most significant cause of lung cancer after tobacco smoking. A robust spatial definition of radon distribution in the built environment is therefore essential for understanding the relationship between radon exposure and its adverse health effects on the general population. Using Ireland as a case study, we present a methodology to estimate an average indoor radon concentration and calculate the expected radon-related lung cancer incidence. We use this approach to define Radon Priority Areas at the administrative level of Electoral Divisions (EDs).

Geostatistical methods were applied to a data set of almost 32,000 indoor radon measurements, sampled in Ireland between 1992 and 2013. Average indoor radon concentrations by ED range from 21 to 338 Bq m$^{-3}$, corresponding to an effective dose ranging from 0.8 to 13.3 mSv y$^{-1}$ respectively. Radon-related lung cancer incidence by ED was calculated using a dose-effect model giving between 15 and 239 cases per million people per year, depending on the ED. Based on these calculations, together with the population density, we estimate that of the approximately 2,300 lung cancer cases currently diagnosed in Ireland annually, about 280 may be directly linked to radon exposure. This figure does not account for the synergistic effect of radon exposure with other factors (e.g. tobacco smoking), so likely represents a minimum estimate. Our approach spatially defines areas with the expected highest incidence of radon-related lung cancer, even though indoor radon concentrations for these areas may be moderate or low. We therefore recommend that both indoor radon concentration and population density by small area are considered when establishing national radon action plans.

1. Introduction
Radon is a natural radioactive gas present in all soils (Cothern and Smith, 1987), representing a significant source of ionizing radiation (WHO, 2009). When radon reaches the outdoor atmosphere it is diluted to lower concentrations. In confined places (e.g. dwellings, workplaces, caves and underground mines) however, radon may accumulate to higher concentrations where it may pose a substantial health risk (e.g. UNSCEAR, 2000a; US-EPA, 2003; WHO, 2009). Radon exposure is principally linked to lung cancer (e.g. Catelinois et al., 2006; ICRP, 1987; Pérez-Ríos et al., 2010), but may also be a contributing factor for other diseases such as skin cancer, Non-Hodgkin’s Lymphoma, stomach cancer and brain cancer (Ha et al., 2017; López-Abente et al., 2018; Vienneau et al., 2017).

Globally, lung cancer is one of the most common cancer types, resulting in approximately 1.6 million annual deaths worldwide (Sugawara and Nikaido, 2014). In Ireland, lung cancer is the fourth most common cancer type, and with approximately 2,300 new cases every year is currently the primary cause of cancer deaths (about 20% of total cancer deaths; NCRI, 2015). Survival rates are normally low, < 20% after five years (e.g. Drolet and Martel, 2015; Jemal et al., 2010; NCRI, 2015), indicating that any reduction in the number of lung cancer cases will therefore have a significant positive impact on the health of the general population. Smoking has an overwhelming influence on lung cancer (i.e. up to 90% may be attributed directly to smoking; NCRI, 2011), and it may be difficult to identify any increase in the risk due to other factors (e.g. radon, occupational exposure, air pollution). Darby et al. (2005), however, estimated that the risk of lung cancer increases by about 16% for each 100 Bq m$^{-3}$ increase of radon exposure, and that between 3% to 14% of lung cancer fatalities globally may be attributed to inhalation exposure of radon (WHO, 2009). Radon is therefore the second cause of lung cancer after tobacco smoking (WHO, 2009).

The number of annual lung cancer fatalities related to radon...
exposure (e.g. around 18,000 in Europe, Gray et al., 2009; 21,000 in the USA, Casey et al., 2015) indicates that radon is an important health issue globally. Radon exposure can however be mitigated if appropriate measures are implemented. In this context, for example, the EU developed Council Directive 2013/59/EURATOM, in which the strategies to reduce exposure to ionizing radiation are defined. The EU Directive especially defines radon as one of the risks that Member States should address, and mentions that each Member State must identify areas (i.e. Radon Priority Areas) where a high radon exposure is probable (i.e. “areas where the radon concentration (as an annual average) in a significant number of buildings is expected to exceed the relevant national reference level”). The efficacy of national strategies has been demonstrated in Ireland where the national average indoor radon concentration has been reduced from 89 Bq m$^{-3}$ to 77 Bq m$^{-3}$ since the introduction of building regulations in 1997 (Dowdall et al., 2017a, 2017b).

Although in this definition of priority areas the health effects are not tacitly mentioned, the EU Directive clearly remarks that the long-term goal is to reduce the lung cancer risk attributed to radon exposure, and therefore the main objective of a National Radon Action Plan is to reduce the adverse health effects resulting from radon exposure. It is therefore beneficial to include the expected number of lung cancer cases linked to radon exposure in the definition of Radon Priority Areas. Such a classification will have significant impact on the implementation of a cost-effective and spatially targeted Radon Action Plan (e.g. Bochicchio et al., 2017). Population density is consequently an important consideration in a Linear No-Threshold scenario as described by Darby et al. (2005), since it is understood that there is no lower limit for a safe radon exposure and that exposure to low indoor radon concentrations may also cause lung cancer. In this sense, for example, in terms of lung cancer prevention it could be more effective to reduce the average indoor radon concentration in a highly populated area by 5–10 Bq m$^{-3}$ than attempting to do so by 15–20 Bq m$^{-3}$ in areas with a lower population density. Estimation of the number of lung cancer cases due to radon exposure by administrative level, as proposed in this study, will therefore allow a focussed effort in reduction of indoor radon concentrations in areas where more inhabitants are exposed to radon and, consequently, more lung cancer cases would be expected.

Data pertaining to adverse health effects are often aggregated at administrative levels (e.g. Electoral Divisions, Municipalities; e.g. Bivand et al., 2008; Hansell et al., 2014), hence analysis of possible adverse health effects of a contaminant requires estimation of its concentration at the same domain. In this regard, for radon risk assessment the objective is to estimate an average indoor radon concentration in an administrative area, based on indoor radon concentrations measured from individual houses (Borgoni et al., 2010). Different methods have been proposed in the literature, including geostatistical methods (e.g. Borgoni et al., 2010; Ha et al., 2017; Hauri et al., 2012; Vienneau et al., 2017). The advantage of geostatistical methods is that they take into account the spatial correlation between the data (modelled by the variogram) to predict a value in a non-sample point.

In this paper, we apply a novel and robust method to define Radon Priority Areas, using Ireland as a case study. We estimate an average indoor radon concentration by Electoral Division (3,409 EDs, with total population ranged from 66 to 38,900 and an average of 1,400 inhabitants, and areas ranged from 0.04 km$^2$ to 162 km$^2$; Census 2016 Central Statistics Office Ireland), and then estimate the number of annual lung cancer cases attributed to this carcinogen. Data collected by the National Cancer Registry are also at this level (NCR/NICR, 2011). The methodology described in this study represents an important advance in the field of radiological protection, helping to define Radon Priority Areas based on a combination of indoor radon concentration and human population distribution, thereby contributing to a cost-effective national radon action plan.
2. Material and methods

2.1. Indoor radon measurements

In total 31,910 dwellings (Fig. 1) were sampled between 1992 and 2013 by the Environmental Protection Agency of Ireland (EPA), and then geo-referenced by the Geological Survey Ireland and Ireland’s Health Service Executive (Hodgson et al., 2014; HSE, 2013). The same dataset has been used as one of several input parameters for developing a new geogenic radon risk map of Ireland (Elío et al., 2017). Indoor radon (InRn) concentrations were measured by installing two alpha track detectors (CR-39), one in the main living area and another in an occupied bedroom (Fennell et al., 2002), for a minimum of three months and seasonally adjusted to give an annual value (Burke et al., 2010). The average concentration was assigned as the indoor radon concentration, and the average outdoor radon concentration (i.e. 5.6 Bq m\(^{-3}\); Gunning et al., 2014) was subtracted from the data (Hodgson et al., 2014), which improved log-normality (Fennell et al., 2002).

A low number of indoor radon measurements in some EDs hampers estimation of radon exposure in Ireland. In this regard, many EDs (i.e. 266 EDs; approximately 8%) have no data or very few indoor radon measurements for representative summary statistics (i.e. 2,710 EDs, approximately 80%, have between 1 and 15 indoor radon data measurements; Fig. 1b). Geostatistical method overcome this difficulty by estimating an indoor radon concentration in non-sampled points, taking into account the spatial correlation between observations.

2.2. Geostatistical analysis

The average indoor radon concentration was estimated over larger areas (Electoral Divisions - EDs) by ordinary kriging applying a trans-Gaussian kriging with Box-Cox transformation (Elío et al., 2016). An indoor radon concentration was then estimated over a grid of 100 × 100 m, and the arithmetic mean and standard deviation of the points within each ED were calculated. The analysis was carried out on R software (R Core Team, 2016) using package “gstat” and function “krigeTg” (Pebesma, 2004). A variogram was derived from the entire dataset, with a maximum distance between pair of points (i.e. 50,000 m). The indoor radon concentration was predicted with a maximum number of nearest points (“nmax”) of 50.

2.3. Lung cancer cases attributed to radon exposure

The number of lung cancer cases attributed to radon exposure were estimated based on a dose-effect model and the average indoor radon concentration at ED level. The indoor radon concentration was used to calculate an annual effective dose (mSv \(y^{-1}\)) in each ED, and knowing the adverse health effects of this radiation dose, the number of lung cancer cases can therefore be estimated. The following equation was used (Quarto et al., 2015):

\[
D (\text{mSv} \cdot \text{y}^{-1}) = C_{\text{air}} \cdot F_{\text{E}} \cdot T \cdot F_{\text{O}} \cdot F_{\text{D}}
\]

where \(C_{\text{air}}\) is the average indoor radon concentration (Bq m\(^{-3}\)), \(F_{\text{E}}\) is the equilibrium factor (\(F_{\text{E}} = 0.4\)), \(F_{\text{O}}\) is the occupancy factor (\(F_{\text{O}} = 0.8\)), \(T\) is time in hours in a year (\(T = 8760 \text{h} \cdot \text{y}^{-1}\)), and \(F_{\text{D}}\) is the dose conversion factor (\(F_{\text{D}} = 14 \times 10^{-6} \text{mSv per Bq m}^{-3}\)).

The number of lung cancer cases per year per million people in each ED was calculated using a conversion factor of 18 mSv \(y^{-1}\), as recommended by the International Commission on Radiological Protection - ICRP (Quarto et al., 2015). Then knowing the population in each ED, the total number of lung cancer cases linked to radon exposure were estimated. The total lung cancer cases in Ireland is the sum of the lung cancer cases in each ED. These were calculated at the 95% confidence interval, estimated by Monte Carlo simulation (n = 10,000; US-EPA, 2003), assuming a normal distribution of the average indoor radon concentration in each ED, with a mean of the average kriging predictions, and the standard deviation of these predictions; a log-normal distribution of the equilibrium factor (\(F_{\text{E}} \sim \text{LN}(0.40,1.15,\text{US-EPA}, 2003)\); a normal distribution of the occupancy factor (\(F_{\text{O}} \sim \text{N}(0.8,0.03)\)), i.e. ranged approximately from 0.7 to 0.9; e.g. Colgan et al., 2008; UNSCEAR, 2000b); a normal distribution of the dose conversion factor (\(F_{\text{D}} \sim \text{N}(14 \times 10^{-6}, 3 \times 10^{-6})\), i.e. ranged approximately from 3 \times 10^{-6} to 24 \times 10^{-6}, e.g. EURATOM, 1996; Portendorfer, 2001; UNSCEAR, 2000a); and a normal distribution of the conversion factor with a mean of 18 and a standard deviation of 2 (i.e. ranged from approximately 12 to 24).

3. Results

3.1. Indoor radon measurements

Indoor radon concentration data (n = 31,910) ranged from 0.1 (< limit of detection) to 9,708 Bq m\(^{-3}\), with median and mean values from the entire data set of 53.4 and 109.7 Bq m\(^{-3}\) respectively (Fig. 1). A log-normal distribution of indoor radon concentration was assumed, although the histogram and normal Q-Q plot (Fig. 2) show a slight deviation from log-normality, with a left tail of low values (even with some data lower than the detection limit of 10 Bq m\(^{-3}\), where an indoor radon concentration of 0.01 Bq m\(^{-3}\) were assigned) and some high values on the right tail, probably caused by a higher density of dwellings sampled in high radon risk areas, and as a consequence, with higher indoor radon concentrations (Burke and Murphy, 2011). The Box-Cox transformation (Box and Cox, 1964) confirms that data are not log-normally distributed (the optimal lambda, and 95% confidence interval, was −0.011 (−0.017 to −0.005)), however, the optimal lambda is very close to 0 and a log-normal distribution may be assumed (i.e. \(\lambda = 0\) in the trans-Gaussian kriging with the Box-Cox transformation).

Geostatistical analysis indicates the existence of a spatial correlation in the data; the sample variogram being outside the envelope of all variograms generated by random permutations of the data (Bivand et al., 2008). This spatial correlation was modelled by an exponential model with a sill of 0.606, a range of 1,420 m, and a nugget of 0.567 (Fig. 2). The high nugget effect confirms that indoor radon concentrations have a high variability at a small spatial scale.
3.2. Average indoor radon concentration and expected numbers of lung cancer cases

The average indoor radon concentrations by ED ranges from 20 to 338 Bq m\(^{-3}\) (Table 1 and Fig. 3a), with a Relative Standard Deviation (RSD) mostly between 5% and 20% (Fig. 3b). These average concentrations correspond to an effective dose ranging from 0.8 to 13.3 mSv y\(^{-1}\). The expected number of lung cancer cases per year per million people due to radon exposure vary therefore from a minimum value of 15 and maximum of 239, depending on the ED (Fig. 4a).

Knowing the population density in Ireland by ED (e.g. population in 2016, census data available from the Central Statistics Office - CSO; Fig. 4b), the expected number of lung cancer cases for each ED attributed to indoor radon exposure may be calculated (e.g. number of lung cancer case in 2016; Fig. 4c). It should be noted that there are likely large uncertainties when calculating lung cancer incidence at low dose rates (ICRP, 2007), however assuming a Linear-No-Threshold for radon-related lung cancer (Darby et al., 2005), this is still a valid approach. The number of lung cancer cases by ED linked to radon exposure in 2016 ranged from 0.001 to 2.262, making a total in Ireland of 286 (CI95%: 150–474). If the same analysis is carried out for 2011 (2011 and 2016 are the last two census data sets available from the CSO in Ireland), then the number of expected lung cancer cases would be 276 (CI95%: 144–457).

4. Discussion

4.1. Average indoor radon concentration

The predicted average (PA) values obtained with geostatistical methods (i.e. ordinary kriging) were cross-validated with the arithmetic mean (AM) of the observations for EDs with > 25 observations (Fig. 5). In general, the PA and the AM in these EDs are in agreement. The AM ranges from 20 to 587 Bq m\(^{-3}\), while the average estimations (PA) range from 24 to 289 Bq m\(^{-3}\) (Fig. 5a), and the differences between PA and AM (in %) are normally lower than ± 25% (Fig. 5b). These results are even better for EDs where > 25 dwellings were sampled and the surface area is < 2 km\(^2\) (Fig. 5c and Fig. 5d). In these small EDs the number of observations are spatially well distributed and the AM may represent a true value of the average radon concentration, hence the similarities between PA and AM indicate that PA also gives a realistic average radon concentration in the EDs.

Indoor radon concentration may also have high spatial variability, even at a small scale (e.g. Friedmann et al., 2017; US-EPA, 2001). This
The effect was corroborated by the high variability of the Standard Deviation (SD) in the EDs with > 25 observations (from 3.47 to 157), and the high nugget effect in the variogram (Fig. 2). In this regard the Confidence Interval (CI) of the AM may be large, even with high number of observations, and geostatistical methods improve the predictions.

Uncertainties on the estimation of radon exposure may also be produced when indoor radon measurements are clustered around zones with high (or low) radon concentrations, and therefore the AM would be not be spatially representative of the radon exposure in an ED, although the number of data points are high. This effect may cause discrepancies between PA and GM estimates, however the geostatistical methods are preferable because these estimates are carried out by modelling the spatial correlation between observations, and therefore the effect of data clustering is minimised. The limitation of geostatistical methods in this case is when data clustering occurs because houses are built only in a small area of the EDs, where people principally live in restricted zones. In these cases smoothing the radon concentration by EDs may underestimate, or overestimated, radon exposure. Avoiding
this effect would require further investigation of the dwelling distribution at a local scale, potentially allowing for a better assessment of radon exposure within some EDs. At a national scale level, however, such detail is currently not feasible.

4.2. Lung cancer cases attributed to radon exposure

It has been estimated that the average national indoor radon concentration in Ireland has been reduced from 89 Bq m$^{-3}$ ($\text{CI}_{95\%} = 88–91$ Bq m$^{-3}$) to 77 Bq m$^{-3}$ ($\text{CI}_{95\%} = 71–83$ Bq m$^{-3}$) between 2002 and 2015 (Dowdall et al., 2017a, 2017b). If the effective dose and lung cancer incidence per million are estimated (Table 2), and the total population is around 3.916 and 4.584 million for the periods 2000–2004 and 2010–2014, respectively (CSO, 2017, 2016), then the total annual radon-related lung cancer incidence is estimated to be 246 in 2000–2004 and 249 in 2010–2014 (Table 2). These estimates are in agreement with previous calculations in Ireland (i.e. 250; NCRI, 2014). The slight increase may correspond to an increase of the population by almost 1 million (20%), even though the national average indoor radon concentration was actually reduced.

The above estimates are also in agreement with the linear model of the dose-response relationship (Darby et al., 2005). In this case, an increase in the risk of lung cancer is correlated to the radon concentration by the expression $\text{IR} = 1 + \beta C_{\text{Rad}}$ (where $C_{\text{Rad}}$ is the radon concentration and $\beta$ is the increase in risk per unit increase in radon; 0.16 ($\text{CI}_{95\%}$: 0.05–0.31) per 100 Bq m$^{-3}$; Darby et al., 2005). By simulations ($n = 10,000$; $C_{\text{Rad}2002}\sim\text{N}(89,0.75)$; $C_{\text{Rad}2004}\sim\text{N}(77,3)$; and $\beta$ a normal square root distribution with an expected value of $\sqrt{0.16}$, Hassjell et al., 2017), the increase in risk of lung cancer in Ireland in the periods between 2000–2004 and 2010–2014 were found to be 1.14 (1.05–1.27) and 1.12 (1.05–1.24), respectively. This corresponds to a proportion of the number of lung cancer cases ($P = (\text{IR} – 1)/\text{IR}$) attributed to radon of 12.5% (5.0%–21.3%) for 2000–2004 and 11% (4.4%–19.4%) for 2010–2014. With the annual average lung cancer incidence in Ireland (i.e. 1,726 in 2000–2004, and 2,332 in 2010–2014; National Cancer Registry Ireland, www.ncri.ie) the expected number of annual lung cancer cases linked to radon may be calculated as 216 (86–368) between 2000 and 2004 and 257 (100–452) in 2010–2014. Comparable to those estimated by the dose-effect model (Table 2).

A population-weighted average for indoor radon may be also used for estimating the number of lung cancer cases linked to radon exposure. In this case, we have calculated the average based on the average indoor radon concentration by ED and the inhabitants of each ED ($AM_{\text{pW}} = \Sigma ED C_{\text{Rad}(ED)} / \Sigma ED_{\text{pW}(ED)}$), resulting in a population-weighted indoor radon average of 85 Bq m$^{-3}$ ($\text{CI}_{95\%}$: 83–87). The annual radon lung cancer incidence may therefore be calculated as 279 or 275 using either the linear model or the dose-effect model, respectively (Table 2). If data obtained by the national radon surveys carried out by the Environmental Protection Agency of Ireland are used (i.e. 91 and 98 Bq m$^{-3}$ for 2002 and 2016 respectively; Fennell et al., 2002; Walsh; Murphy P., pers.comm., 29 Feb.), then the estimated annual number of lung cancer cases in the period 2000–2004 would be 220 and 251, for the linear model and the dose-effect model respectively, and 316 and 317 in the period 2010–2014 respectively (Table 2). The estimates using both models are therefore in agreement, showing that they can be equally used to estimate radon-related lung cancer incidence.

Our calculations of the expected number of lung cancer cases linked to radon exposure by ED (i.e. 276 [CI$_{95\%}$:144–457] for 2011 and 286 [CI$_{95\%}$:150–474] for 2016) are similar to the estimates obtained based on population-weighted averages (around 280–315 for 2010–2014; Table 2) but slightly higher than those obtained using the geographic means (around 250; Table 2). These results suggest that estimates based on the geographic national average underestimate radon related lung cancers, and techniques which take into account the population density are more accurate to evaluate the health effect of radon exposure. The estimates by ED also have the advantage that they allow definition of a spatially targeted radon action plan.

A potential limitation of lung cancer estimates at administrative level (i.e. ED) with respect to calculations based on population-weighted arithmetic means is however that they are time and resource consuming, and therefore it may not be feasible apply this approach regularly to monitor the efficiency of a national radon action plan. In this sense, calculations of an average indoor radon concentration by ED requires a high number of indoor measurements around the country (e.g. in this study we used around 32,000 measurements), while an update on the arithmetic mean of the indoor radon concentration can be made with a considerable lower number of houses tested (i.e. in Ireland an update was carried out using around 700 randomly selected houses, and in less than one year; Dowdall et al., 2017a, 2017b).

On the contrary, although annual surveys represent the actual situation in a country, these surveys may be influenced by the dwellings sampled and by temporal (seasonal) variations on indoor radon concentration. The AM may therefore fluctuate and may not properly represent radon exposure to the general population. In order to minimize these effects the annual surveys may be aggregated in several years (e.g. 5 years; minimum period to observe a change of lung cancer risk due to radon exposure, NRC, 1988; Tomášek et al., 2001) and thus the data will be more representative of radon exposure over long periods. Furthermore, since lung cancer has a long latency period (Cheng et al., 2016; Field et al., 2002; NCRI, 2005), aggregating the data over several

<table>
<thead>
<tr>
<th>Period</th>
<th>AM</th>
<th>SD</th>
<th>Linear Model Proportion</th>
<th>Average Annual Cases</th>
<th>Annual Cases</th>
<th>Dose-Effect Cases per million</th>
<th>Average Population (Million)</th>
<th>Annual Cases</th>
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<td>Geographic indoor radon average based on national surveys</td>
<td>2000–2004</td>
<td>89</td>
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<td>3</td>
<td>257</td>
<td>54</td>
<td>4.584</td>
<td>249</td>
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<td>Population-weighted indoor radon average based on national surveys</td>
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<td>91</td>
<td>4</td>
<td>220</td>
<td>64</td>
<td>3.916</td>
<td>251</td>
<td>(139–404)</td>
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<td></td>
<td>2010–2014</td>
<td>98</td>
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<td>316</td>
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<td>4.584</td>
<td>317</td>
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<td>(86–383)</td>
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<td>(107–490)</td>
<td>(36–115)</td>
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* The confidence interval was not provided in Fennell et al. (2002), so the same standard deviation (SD) as the 2010–2014 period was assumed.
years is likely a better approach to more accurately estimate the possible adverse health effects of radon.

The annual lung cancer cases in Ireland linked to indoor radon exposure alone, are therefore estimated to be 270–280 (= 12% of the annual lung cancer cases). In the different estimates we have considered, we see a temporal increase of radon-related lung cancer cases, despite a reduction of the national average indoor radon concentration (Table 1 and Table 2). This supports the hypothesis that the increase in absolute numbers of lung cancer cases is due to an increase of the population, and further argues for including population data in such studies. It is also important to mention that although the geographic average has been reduced from an AM of 89 to 77 Bq m\(^{-3}\), the population-weighted average has increased from 91 to 98 Bq m\(^{-3}\), and together with a population growth of almost 1 million, the expected number of lung cancer cases has apparently increased from around 250 to 300 (Table 2). Although the ED-based predictions suggest this increase in radon-related lung cancer incidence may not be so severe (around 280), it indicates that radon prevention measurements could be improved if demographic data are included. These results have to be taken with caution since other lung cancer causes (e.g. tobacco smoking) were not taken into account in this study, however it shows the importance of considering differential population growth as part of the evaluation for long-term effects of radon exposure to the general population, and in defining a rigorous national action plan.

5. Conclusion

In Ireland, the current annual numbers of lung cancer cases linked to indoor radon exposure are estimated to be approximately 270–280 (= 12% of the annual lung cancer incidence). Although the health results shown in this study are preliminary and more detailed epidemiological studies should be carried out to better understand the effects of radon exposure in Ireland (especially to reduce the uncertainties associated with the effective dose received by inhabitants, and to evaluate the synergistic effect of radon exposure and smoking), they illustrate the importance of including the possible adverse health effects of radon exposure in the definition of Radon Priority Areas. Additionally, we have demonstrated that carrying out such studies at an administrative scale level (e.g. ED), rather than at a national level, improves national estimates of radon-related lung cancer incidence and allows defining and prioritising health protection in certain districts to promote radon awareness policies.

A strategy of estimating the probability of having an indoor radon concentration higher than a reference level (e.g. Elío et al., 2017) is a useful approach in order to estimate the expected number of houses in a country with an indoor radon concentration higher than this reference level. However, as there is no safe level of radon exposure, and low radon concentration may also cause adverse health effects, only focusing radon actions on reducing the indoor radon concentration which are higher than a reference level (e.g. 200 Bq m\(^{-3}\) in Ireland) may not be sufficient (e.g. Gray et al., 2009). Estimation of an average indoor radon concentration over large areas and the possible adverse health effects on the population as described in this study will therefore overcome some of the limitations of the probability maps, and together with them will help to define a spatially targeted national radon action plan.

A national radon action plan based solely on radon concentration, or probability maps using geogenic information, will inherently have a different focus to one which takes population distribution into account. We do not view the different approaches as mutually exclusive, in that a national policy can be designed to use a combination of all methodologies. Radiation protection has essentially two targets, protection of individuals and protection of the collective. The first implies reducing extremes, even if only one person is affected; this is where the probability maps have an important contribution. The second implies a reduction policy spatially focused where the majority of the collective dose occurs. Such areas are where the highest radon-related lung cancer incidence is expected, even if indoor radon concentrations are relatively low; this is where the methodologies as described in this study, are essential.

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