Cancer Mortality and Proximity to Hinkley Point Nuclear Power Station in Somerset.1995-1998. Part 2 Prostate Cancer

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1 Introduction and Background

Since the discovery of the Sellafield leukemia cluster in 1983 there has been significant debate around the question of childhood cancer clusters near nuclear sites in the UK and elsewhere (Beral *et al.*, 1993). In the last fifteen years, such clusters have been confirmed in the vicinity of all three nuclear reprocessing plants, Sellafield, Dounreay and La Hague with Relative Risks in the 0-4 age group ranging from 8 to 15. In addition, child leukemia incidence clusters have been confirmed at the Atomic Weapons Establishment (AWE) at Aldermaston and Burghfield in West Berkshire. In 1997 we examined childhood leukemia mortality in South Oxfordshire and showed that there was a significant excess risk only in those County Districts which contained nuclear sites, namely South Oxfordshire (Atomic Energy Research Establishment, Harwell) and Newbury (AWE Aldermaston and Burghfield). Excess leukemia risk has also been reported near nuclear power stations in the UK (Cook Mozzafari *et al.*) in Germany (Hoffmann *et al.*, 1998) and in Sweden (Andersen and Moeller, 1997).

Although the existence of these clusters is no longer disputed, the cause of the effect has been the subject of considerable argument. Official bodies, like the Committee on Medical Aspects of Radiation in the Environment (COMARE), set up at the time of the enquiry into the Sellafield child leukemia cluster, continue to argue that the doses likely to be received by the children are too small, on the basis of the risk models presently used to undepin legal restraints on radiation exposure. On the other hand, an increasingly large body of opinion holds that the risk models used by COMARE and others, which are based on the cancer yield in survivors of the Hiroshima bomb, cannot safely be used to predict or explain cancers caused by chronic internal irradiation from ingested or incorporated radionuclides like Strontium-90, Plutonium-239, Caesium-137 and so forth. These and other man-made radioactive isotopes have not existed on earth prior to 1945 and the combination of their ability to substitute for biologically important elements and then irradiate local tissue in novel ways makes them a potentially serious mutagenic hazard whose magnitude is not informed by studies of external acute exposure, like the Hiroshima study. (Nussbaum and Koehnlein 1994, Busby 1995, 1998, 2000).

All the nuclear sites where childhood leukemia and cancer clusters have been discovered have in common that they routinely discharge significant quantities of these man-made radioisotopes to the air, river or sea under licenses provided by the government. These licenses assume that the levels of exposure are too low to be of radiological significance. The existence of the leukemia and cancer clusters suggests otherwise.

In 1988, Somerset Health Authority conducted a study of leukemia in parishes inside a radius of 15km from Hinkley Point, using data provided by the local large hospital, Musgrove Park (Bowie and Ewings, 1989). The study looked at data from 1959-1986 and concluded that following the commissioning and operation of the Hinkley 'A' station in 1964 there was a significant increase in the rates of leukemia and non-Hodgkin lymphoma among under 25 year-olds living inside a 12.5km radius of Hinkley Point. A statistically significant Relative Risk of 2 to 2.5 times the National average was driven by cases in the 5-year period following the operation of the plant. This seemed to support the conclusion that, like Sellafield and Dounreay, it was the latter that was somehow causing it. Thus there is some prior evidence to suggest that the operation of Hinkley Point may have caused increases in leukemia in its vicinity.

It is now universally believed that ionizing radiation causes leukemia and also cancer through its properties as a general mutagen. It causes mutations in cellular genes. Although

Leukemia has been the outcome most associated with exposure, all other cancers are caused by ionizing radiation (BEIR V) and indeed, the Seascale child leukemia excess was also accompanied by a more modest, though significant, cancer excess. Between 1998 and 2000, our group has studied both leukemia and cancer incidence in small areas of Wales using data provided by Wales Cancer Registry for the period of peak discharge from Sellafield, 1974-1989. This work was funded by the government of the Irish Republic and addressed the question of the possible effects on cancer risk produced by discharges of man-made radioactive isotopes to the Irish Sea by BNFL, Sellafield. What we were able to show was that there was a profound, and statistically significant excess risk of leukemia and most other cancer sites in both adults and children living in areas whose population centroids averaged less that 800m from the Irish sea. The effect was significant for prostate cancer but the fall-off with distance from the coast was less abrupt that it was in the case of breast cancer (Busby *et al*, 2000a).

The overall risk was driven by cases in those seaside towns on the north Wales coast located near large areas of intertidal sediment and mud which were known to contain radioactive material from Sellafield. Examples were Bangor, Llandudno, Conwy, Prestatyn, Colwyn Bay, Rhyl and Abergele. The effect also occurred near the mudflats of the Dee estuary in town of Flint and Holywell. Owing to tidal energy conditions there, the coast has accumulated radioactive mud and silt contaminated by discharges from Sellafield.

Prior to this study, in May 1996, we had attempted to examine cancer risk in small areas around Hinkley Point and we approached Dr Pheby of the Bristol Cancer Registry for data. The letter was referred to Dr Jenifer Smith, the director of a new, Cancer Intelligence Unit based in Winchester. Dr Smith refused to release the small area data on the basis that it was confidential. This was the first in a series of refusals to release data for small areas near nuclear sites. Since our acquisition of the Wales Cancer Registry data in 1995, and 1996, we have applied for and have been refused small area incidence data by all of the regional cancer registries we have written to including the Oxford Cancer Intelligence Unit (Harwell, Aldermaston) and South and West (Hinkley, Channel Islands) and the new Wales Cancer Intelligence and Surveillance Unit which had replaced the old Wales Cancer Registry from whom we obtained our original figures. Further independent examination of areas near nuclear sites remained impossible until the recent release by the new Office for National Statistics, in late 1999, of census ward level mortality data for selected causes of death for the four-year period 1995-1998. We purchased this data from ONS in November 1999 and began looking at cancer risk in adults in wards around Harwell in Oxfordshire and Aldermaston in Berkshire. These results will be published in due course.

In February we were asked to examine cancer risk near Hinkley Point. This first results, published in April 2000, (Busby, Dorfman and Rowe, 2000) examined relative mortality risk from female breast cancer ICD9: 174 in wards in Somerset distant up to 25km and more from Hinkley Point.

Our prior hypothesis is that breast cancer risk is highest in wards which are proximal to large drying offshore mudbanks that have become repositories for man-made radioactive isotopes released from Hinkley Point and accumulated over the period of its operation. We also expect *a priori* that the trend with distance will be highest in the coastal wards with large population density but will fall steeply over the first 5km and thereafter flatten out. We further expect that wards in low-lying areas with high density of river valleys will show a higher relative risk than areas of higher ground.

These prior expectations are based on the findings in north Wales and other considerations

The results of our examination of breast cancer mortality supported the hypotheses. Analysis showed that there was a statistically significant excess risk of dying of breast cancer in the aggregate wards within 5km of the centre of the offshore mud banks near Hinkley Point (RR=1.43;p=0.02). The risk fell off with increasing distance from a point source taken to be the centre of the mud bank with Relative Risks of 1.43, 1.33, 1.24, 1.16 and 1.13 in wards contained within 5, 10,15,10 and 25km rings around the point source. The overall risk in the study area was 1.09 (relative to England and Wales rates for the same period).

The most significant high risk ward was Burnham North with 8.7 deaths expected, 17 observed (RR=1.95; p=0.02).

This report outlines the results for mortality from prostate cancer ICD185 in the same study period 1995-1998.

2.

Prostate Cancer and Ionizing Radiation

Prostate cancer is one of the most common cancers in males and currently represents about 15% of all male registrations.(ONS, 1990) There has been a sharp increase in prostate cancer since 1980: nationally, this increase is about 30% after standardisation for age.(OPCS, 1989) Cancer is a genetic disease expressed at the cellular level. It is almost wholly environmental in origin, being caused initially by a genetic mutation in a single cell. Since the time lag between initial mutation and final clinical expression of the disease is about 15 to 20 years and since the disease began to increase in the UK in the period 1975-1985, we are looking for a mutagen or carcinogen which entered the environment in the period 1955-1965. Although there were changes in a number of environmental stresses in this period, there is another clue to the identity of this carcinogen. The incidence of cancer, including prostate cancer is significantly higher in Wales than in England. Wales is the wettest part of the United Kingdom. These two pieces of evidence strongly suggest that the cause of the prostate cancer increase, and indeed the general cancer epidemic, is exposure to ionizing radiation from global weapons-testing fallout. This mixture of novel man-made radioisotopes fell to earth in the period 1955-1965. The measured concentrations of the main radioactive pollutants, Strontium-90 and Caesium-137 were two to three times higher in Wales that in England. The fallout peaked in 1963 and began to fall following the Atmospheric Test Ban Treaty signed in that year.

The models which are presently used by the risk agencies like the International Commission on Radiological Protection (ICRP) accept that ionizing radiation is a cause of the disease. Increased rates are seen in the Hiroshima survivors cohort. However, these models assume that exposure to doses at the levels provided by the weapons fallout exposures cannot produce the increases in prostate cancer seen. This is an area of dispute. The difference between the ICRP view and that which holds that exposure risks of internal man-made radioisotopes represents a significant cancer hazard essentially arises due to different methods of estimating exposure dose. The averaging methods employed by the early physics-based models which underpin the ICRP risk factors are only applicable to external irradiation, where each cell in the irradiated tissue gets equal chance of being 'hit'. For ingested radioisotopes, particularly those which are in the form of aggregate particles or sequentially decaying isotopes, cells which are close to the atom or particle will get many sequential 'hits' in a short time. A theory has been developed to examine the excess hazard from such processes, the Second Event Theory (Busby 1995, 1998, 2000, Edwards and Cox, 2000) It predicts very large excess risk from certain man-made radioisotopes, notably Strontium-90. It also predicts anomalously high cancer risks from inhalation of micron-sized

aggregate particles of alpha-emitters like Plutonium-239 and Americium-241. Because of large amounts of such man-made radioisotopes have been released from nuclear reprocessing plants and nuclear power stations since the 1960s, it believed likely that these sites will be point sources for breast cancer clusters. Discharge these substances to the sea results in their becoming attached to fine silt particles. These particles are preferentially precipitated in low tidal energy conditions in estuaries or on coastal intertidal banks (Assinder 1994, Baxter, 1989). These banks become repositories of radioactive particles which are re-suspended by wave action and blown ashore. (Eakins, 1987). The particles have their highest concentration in the 0-1km coastal region. The concentration trend for Plutonium with distance from the coast in Cumbria is shown in Fig 1 (Eakins and Lally, 1984) but the trend generally follows the penetration of sea salt inland and in Somerset the trend would be expected to be of the same type, although the concentrations would be lower. Once suspended and blown ashore, the radioactive particulate material is inhaled and transferred through the lung to the lymphatic system where it may become transferred to any organ. Measurements made on autopsy specimens from all parts of England and Wales showed that Plutonium concentrations were highest in the tracheobronchial lymph nodes. (Popplewell 1985). The mechanism we proposed for the cancer increases we found in north Wales near the Irish Sea was that radiation doses from these particles both harmed the immune system and by relocation harmed local cells in organs to which they had been relocated by lymphatic circulation.

Unlike the case of breast cancer, in the case of prostate cancer there have been prior studies which have suggested strongly that internal contamination by man-made radioactive isotopes carry significantly high risk. Although the external radiation exposure, Hiroshima-based models do not consider the prostate to be a radiosensitive organ (BEIR V, 1990), in 1993, Rooney *et al.* made a study of nuclear workers to examine prostate cancer risk in those workers who had been monitored for internal contamination by man-made radioactive isotopes. This followed an earlier examination which showed that nuclear workers were at risk from prostate cancer but this risk did not follow the external film-badge recorded dose. They found significant and high relative risks of prostate cancer in the group of workers who had been monitored for internal contamination by a range of man-made radioisotopes.

Busby (1994) pointed out that these findings of Rooney *et al.* also suggested a cause for the sharply increasing prostate cancer rates in Wales which had begun to rise in 1978. Wales was known to have suffered between twice to three times the English levels of contamination by global weapons testing fallout in the period 1959-63 owing to its higher rainfall. The components of the fallout, e.g. Strontium-90, Caesium-137, Plutonium-239, Tritium etc were all implicated in causing prostate cancer in the nuclear workers and were probably also therefore responsible for the increased prostate cancer in Wales, and probably other cancers also. In order for this to be the case, risk factors for such internal exposure would have to be about 500 times higher, dose-for-dose than those which were being used. This calculation was supported also by the nuclear industry (Atkinson *et al*, 1994).

Besides the supporting evidence for our hypothesis given above, a link between leukemia and radioactive particles is supported by a study carried out in 1990 by workers from the Leukemia Research Fund (Alexander *et al*, 1990). These workers studies leukemia risk in estuaries in England and Wales and established that there was a modest and statistically significant excess risk of leukemia in children living in wards which were adjacent to estuaries.

In addition to the discharge to sea to person route hypothesised above there is , in the case of Hinkley, the direct exposure route in those populations living immediately downwind of the aerial discharge stacks. Thus we would predict *a priori* that the inhabitants of

Burnham on Sea would be at risk following exposure to aerial emissions of Tritium, Carbon-14, noble reactor gases etc. In general, the releases from Hinkley Point would be expected to result in increased levels of radioisotopic contamination in the river valleys also, since the rivers in the area are tidal for a considerable fraction of their length. In addition, any aerial discharges would result in deposition in the basin defined by the Mendip and Quantock Hills and deposited radioactive material will be washed back into the rivers by rain. Thus we would expect a higher risk of cancer in the low-land areas near the rivers, an expectation which was supported by the results for breast cancer.

3 Method

1991 Census Population data by sex and 5-year age group was purchased from ONS for 103 census wards in Somerset. The area studied extended from Porlock and Exmoor in the West along the coast to Berrow, just south of Weston-super-Mare, and extended to Blackdown in the south-east and Wells in the north-east. We chose to exclude the large town of Weston-Super-Mare and its immediate environs since its large population would have swamped any of the distance effects we were examining. Using these ward populations, the number of deaths that should be expected from prostate cancer were calculated for each ward using the England and Wales national aggregated data published annually by ONS for the period 1995-1998. The observed numbers of cases in each ward over the four years 1995-1998 were then divided by the appropriate expected number of cases to give the value of Relative Risk for the ward. In addition, we obtained details Social Class of households in each ward in order to adjust for this parameter when accounting for variations in risk. The relative risk of prostate cancer mortality in each social class was determined by OPCS in 1981 for the years 1970-72. The population weighted mean social class prostate cancer mortality fraction in each ward was calculated and this value was used as a multiplier of risk to establish social class standardised expectation in each ward. With prostate cancer the variation in risk associated with social class was less than 2 percent and so the unadjusted results are reported in Appendix A.

For our main distance analysis we took as our point source the centroid of the offshore mud banks formed by coastal and tidal conditions at the mouth of the River Parrett. The Ordnance Survey Grid reference of this point is ST260480 (this is a more accurate reference for the position than that given in the earlier report on breast cancer mortality). It is these mud banks that we believe to be the main source of radiation exposure, through sea-to-land transfer of radioactive particles. However, we have also analysed the data using a point source 1.5km east of the Hinkey Point outfall pipe, (ST240460) a location used by MAFF for taking samples of mud for analysis. For each point used as centre we constructed concentric rings at 5km radii up to 25km. Wards which were cut by these rings we partitioned according to the fractions of their area which appeared to be in each 5-km annulus. For example, if 30 percent of the area of a ward is in the 0-5km ring and the remaining 70 percent is in the 5-10km annulus we add 30 percent of the observed and expected numbers for this ward to the aggregate expected and observed numbers in the 5-km ring and the remaining 70 percent to the aggregate numbers in the 5-10 annulus. This assumes a uniform density of population.

Statistical significance of Relative Risk was calculated using Poisson Cumulative Probability for less than 50 cases observed and the Chi-squared statistic if the numbers were higher than 50. Statistical significance of the trend with distance from the source was examined by calculating the Chi squared statistic for linear trend in proportions

Results

Relative Risk of prostate cancer mortality in wards in Somerset near Hinkley Point is shown in the coloured map Fig 3. The values in individual wards are given in Appendix A which gives the results uncorrected for social class. The Relative Risk in aggregated populations of increasingly large concentric rings centered at 5km intervals on the point source at the centre of the offshore mud bank (see map) is given in Table 1. In Fig 2 we show the relation between Relative Risk in these increasingly large areas and the radius of the area.

Fig 4 shows the values of Relative Risk in each annular 5km ring and compares the prostate result with the earlier one for breast cancer. Also given is the polynomial best fit to the two sets of data. Figure 4 is a colour map of the study region which includes an overlay which shows the River Parrett catchment area. Table 3 compares Relative Risk in the low land areas near the rivers with that in the higher land.

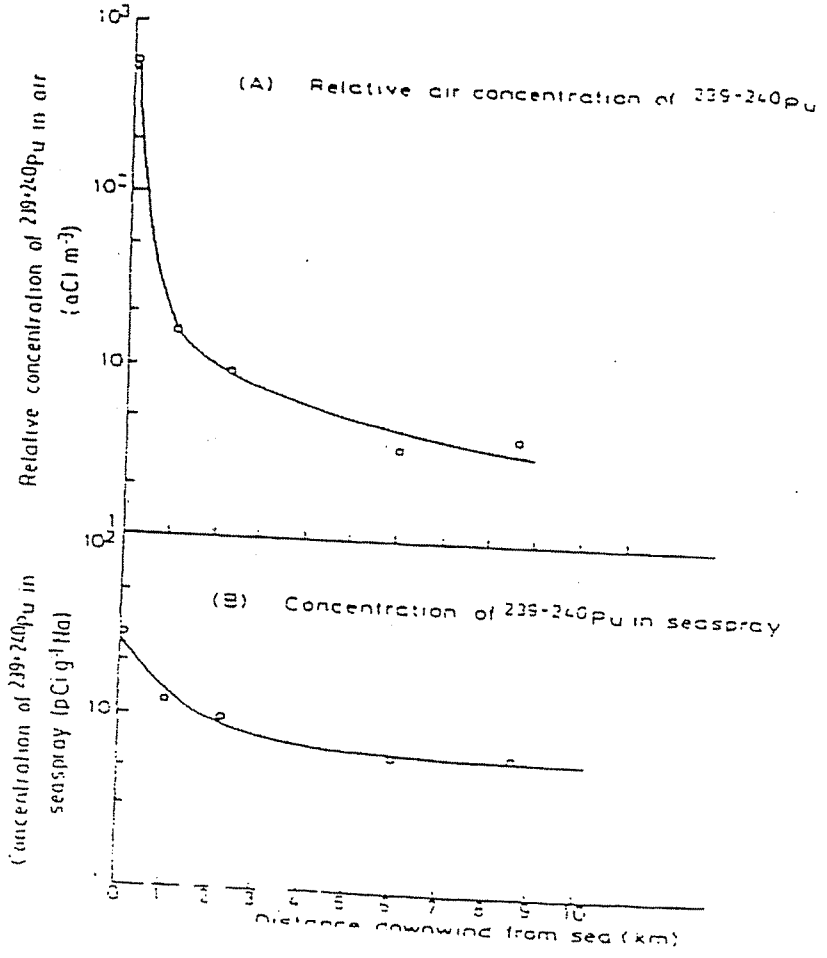


Fig 1. Inland penetration of plutonium particles in seaspray.
(*Source*: Eakins and Lally, 1984)

Radius (km)	Observed cases[a]	Expected cases	Relative Risk	χ^2 (p-value)[b]
0-5	25.7	18.3	1.4	(0.05)
0-10	46.7	32.3	1.44	(0.01)
0-15	85.2	62.5	1.36	8.2 (<.05)
0-20	105.1	78.9	1.33	8.6 (<.05)
0-25	168.9	126.3	1.33	14.4(<.05)
0-all	280	235.1	1.19	8.6(<.05)

[a]Observed cases may be fractional due to splitting of wards by concentric rings.
[b] For the first of these rings the Poisson p-value was 0.02

Table 1. Relative risk of prostate cancer mortality 1995-1998 in ward populations in 5, 10, 15, 20 and 25km radius rings around the centre of the offshore banks near Hinkley Point

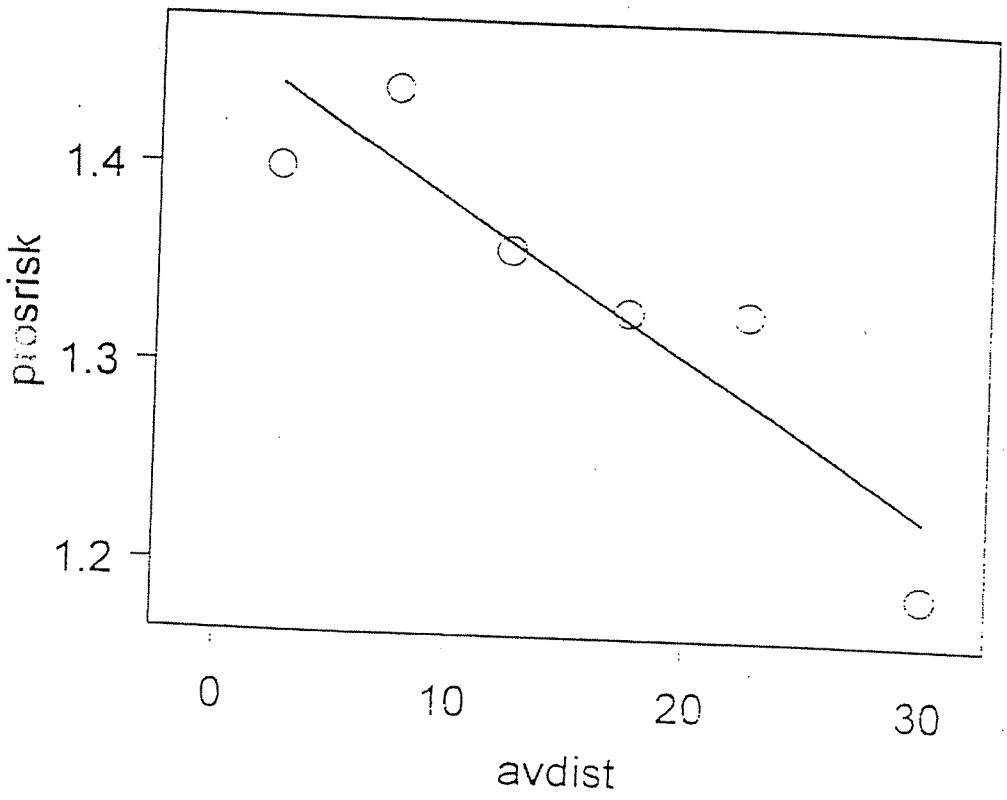
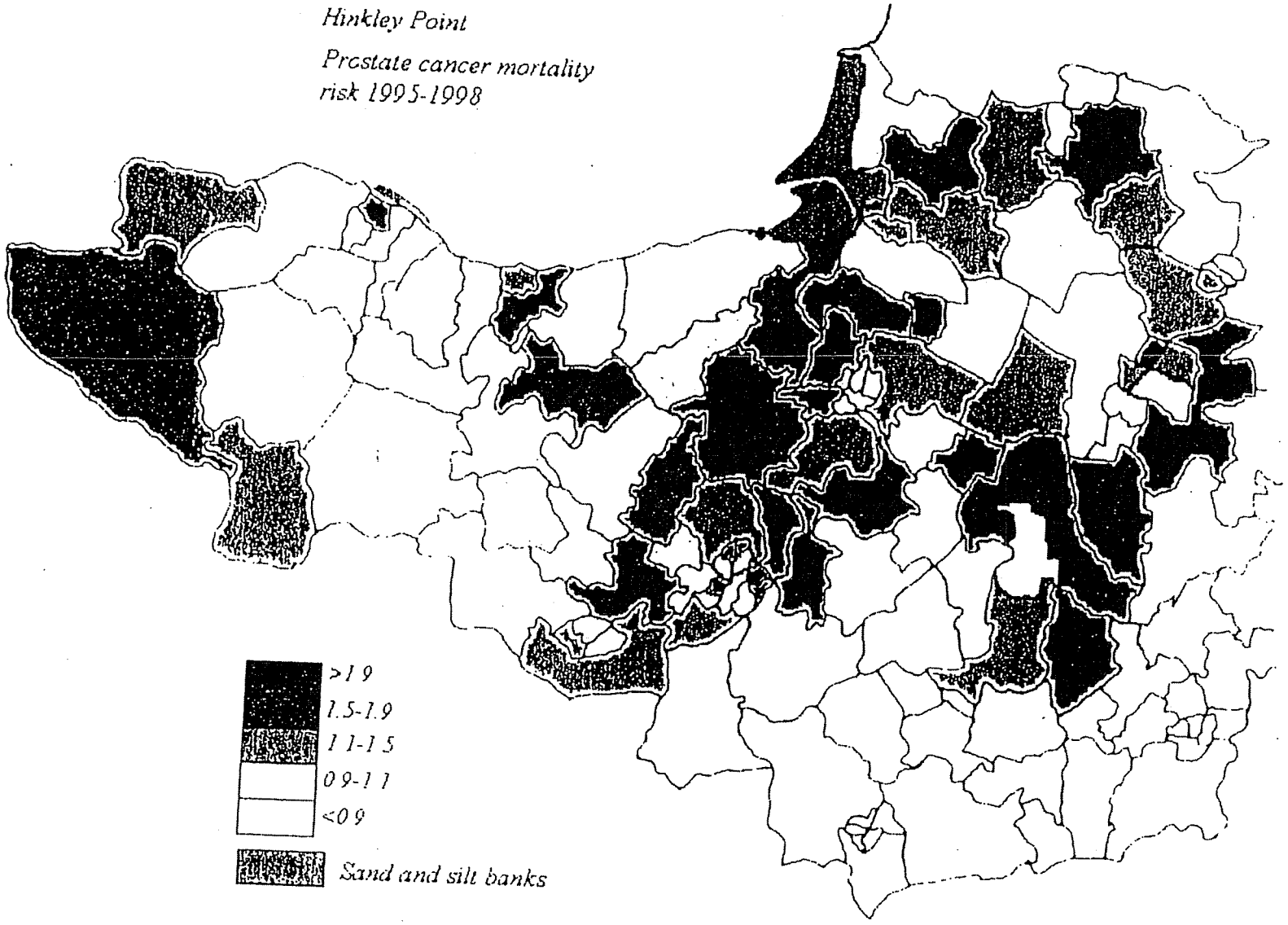


Fig 2. Relationship between Relative Risk of mortality from prostate cancer 1995-1998 and distance (km) of cumulated population from the point source of risk in the offshore mud banks


Hinkley Point
Prostate cancer mortality risk 1995-1998
>1 9
1.5-1.9
1 1-1 5
0 9-1 1
<0 9
Sand and silt banks

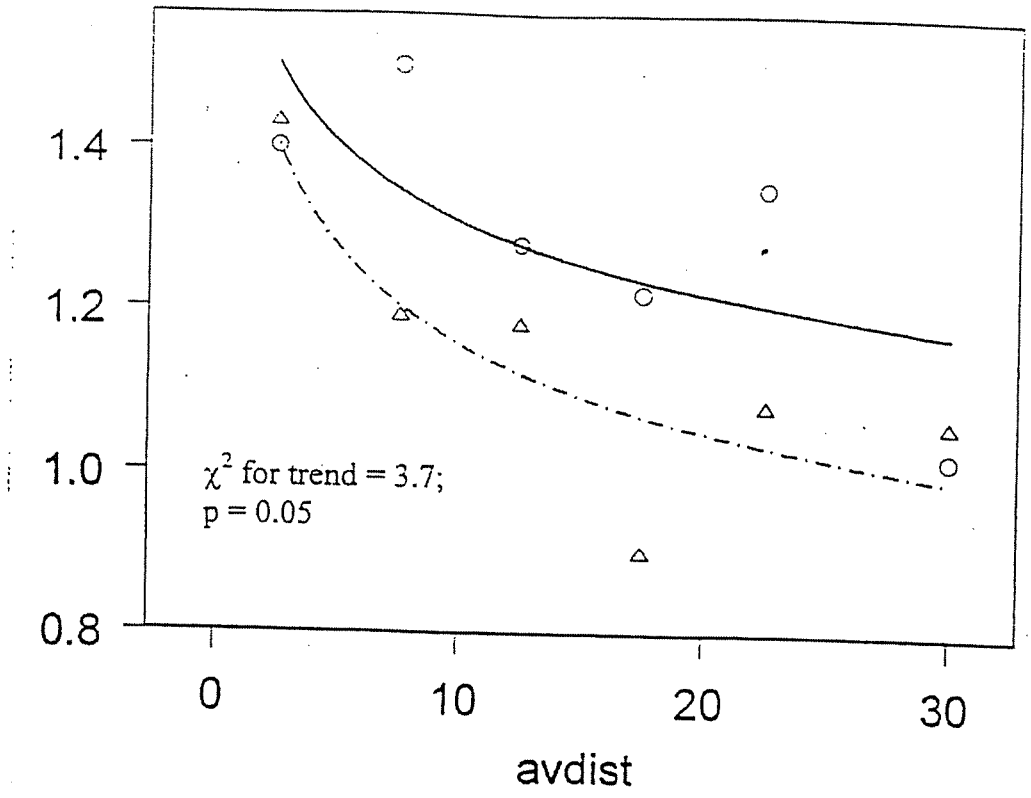


Fig 4. Relationship between Relative Risk of mortality from prostate cancer (open circles) and breast cancer (triangles) 1995-1998 and mean distance (km) of populations in 5km annular rings with increasing radial distance from the point source of risk in the offshore mud banks. Data points also included from inner 2.5km ring and all wards >25km in the study area.

	Observed (deaths)	Expected (deaths)
Low land	229	182
High land	51	53
Both	280	235

Relative Risk 1.25
Chi square = 12.1
p-value <0.01

Table 3. Risk of prostate cancer mortality in wards below and above the 200m contour in the whole study area (uncorrected for social class).

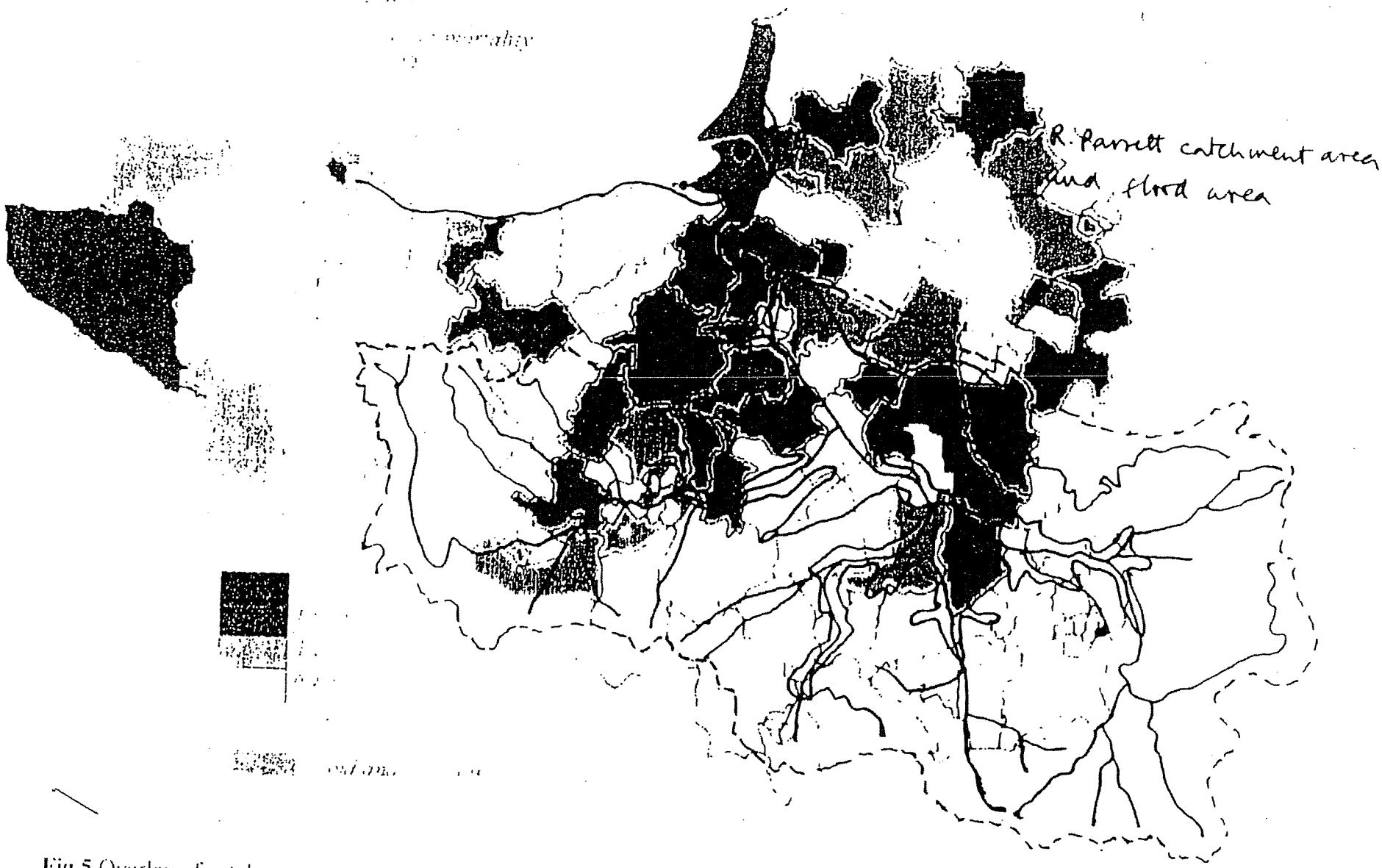


Fig.5 Overlay of catchment area and flood plain of river Parrett with prostate cancer mortality results.

Location	Gamma dose rate at height of 1m (nGy/hr)
Hinkley Point Outfall beach dose	98
East of outfall beach dose	68
West of outfall beach dose	111
Burnham on Sea beach dose	60
Combwich	78
Average dose Somerset (NRPB)[a]	34

[a] No grid point in Somerset reported by NRPB exceeds 40nGy/hr

Table 4. Gamma background levels near Hinkley Point compared with average levels in Somerset 1988. *(Source: Nuclear Electric Annual Report 1994 and NRPB, 1988)*

5
Discussion

The hypothesis that risk is highest inside the 5km ring and falls off significantly with distance is supported by the results. As in the case of breast cancer mortality, the proximal effect is driven by high Relative Risk in Burnham on Sea. For breast cancer the highest risk was in Burnham North but for prostate cancer excess is found in both wards. For the combined population of Burnham 21 prostate cancer deaths were observed in the four year period where 14 were expected were expected (RR = 1.5; p = 0.05). In addition, the risk map Fig.2 appears to show a distinct annular region of higher risk to the east of the offshore mud banks but also following the low-lying land and the river which drain it. The pattern of risk for prostate cancer mortality over the period is similar to that of breast cancer, however, the overall risk in the study area is greater for prostate cancer. This finding is in agreement with levels of prostate cancer in Somerset found by Swerdlow and Santos Silva in 1988.

Shifting the origin of the circles of risk to Hinkley Point itself markedly reduced the Relative Risk in the 0-5km circle but merely moved the highest risk to the 10km circle, falling thereafter in the same pattern. This procedure produced a pattern of risk similar to that found by Bowie and Ewings for leukemia in 1989

In addition to the above, examination of the trend with distance in consecutive annular rings shows a pattern which supports the exponential fall off of concentration shown in the levels of plutonium in air shown in Figure 1. .

The hypothesis was based on theoretical considerations of our findings on the north Wales coast. The towns of Bangor and Conwy are both on estuaries (if we consider the Menai Strait to be an estuary), close to an extensive mud bank, the Lavan Sands, which contains considerable quantities of radioactive silt. In our unpublished study we found that in the six years 1984-89, relative risk of incidence of prostate cancer in all ages was higher in the coastal towns relative to a control group of inland towns. The effect was just statistically significant but much less pronounced than the breast cancer coast effect. In addition, the trend in risk with distance from the sea in Wales did not fall off so abruptly as it did with breast cancer, an effect which we see also here in Somerset. In Wales, concentration of Caesium-137 in the intertidal sediment varied with where the sample was taken but close to the respective towns values were about 300Bq/kg dry in 1989. (Garland *et al.* 1989). This level of Caesium-137 was associated with plutonium- 239+240 levels of about 60Bq/kg . The origin of this material was mainly Sellafield but in the river material from Chernobyl

contributed about half the radioactivity [illegible] These two towns are on the north coast and are sheltered from all winds west of north, and so sea to land transfer of radioactivity from the silt (which has been measured) is less than it might be were the towns facing the prevailing weather. The drying area of the offshore banks near Conwy and Bangor is about 50km^2.

The offshore drying banks near Hinkley Point are roughly the same size forming a triangular area of 58km^2 stretching from the power station in the south to Brean Down in the north. The main population centre close to the banks is the town of Burnham on Sea but unlike the towns of Bangor and Conwy, Burnham faces the prevailing wind and is directly downwind of the power station. The banks themselves seem to have lower levels of man-made radioactivity in them. MAFF has measured levels of Caesium-137 in the sediment a kilometre east and west of the outfall pipe from the station. Representative values are given in Table 5 below. In Table 4 is given the measured levels of gamma background taken by NRPB surveys and Nuclear Electric in the early 1990s. These show that there are components of the mud and shoreline sediment which increase the background significantly over the values obtained inland. These components probably include radioactive isotopes from Hinkley Point, weapons fallout and some natural radioactivity. Note the higher value for Burnham on Sea.

Year	[A]Cs-137 Hinkley mud. (Bq/kg)	Cs-137 Cemlyn Bay mud (Bq/kg)	Pu-239 Cemlyn Bay Mud (Bq/kg)
1970	375	207	Not listed
1975	250	1150	Not listed
1980	42	1000	56
1985	16	700	58
1990	44	320	48
1995	35	180	23

[a] corrected for measurements made on wet mud

Table 5 Values of Caesium-137 land Plutonium-239 activity in intertidal mud from north Wales and Hinkley Point

It is clear that levels on the Somerset offshore banks are between a quarter and a tenth of those in north Wales, where the effects of Sellafield are predominant. Nevertheless, this lower level of radioactive contamination is more than compensated by the openness of the coast and hinterland to the prevailing wind. The sea is always brown in onshore winds, and seaspray, carrying its radioactive burden, will penetrate far inland. It will be deposited inside the hollow area defined by the Quantock and Mendip Hills and be washed on to the Somerset Levels, where its drainage through the rivers and dykes back to the sea will result in transfer back to the depot of the offshore banks. This cyclical process will retain the radioactive particles in the area and increase the chance of inhalation and ingestion. Examination of the topological and river schematic shown in Figure 5 reveals that those areas to the east of the rivers are the areas that carry the highest risk. Comparison of two groups of wards categories by whether they are above or below the 200m contour shows that the aggregate of wards on the low ground carries 1. 25 times the risk of the high-ground wards ($\chi^2 = 12.1$; $p < 0.05$).

The results of this examination of prostate cancer mortality risk demonstrate that proximity to the offshore drying banks close to Hinkley point power station may be seen to

be the main source of risk. It is not being maintained that discharges from the nuclear power station are the only source of radioactivity in the mud. There will be a proportion of material from weapons fallout and also from man-made isotopes in the Uranium and Thorium series. The relative cancer hazard from specific internal isotopes has not been established, and no attempt will be made in this report to review the evidence that man-made isotopes are more hazardous that natural isotopes (see e.g. Busby, 1995).

The results of an analysis mortality from of all malignancies, and other specific cancer sites in the area near Hinkley Point will be reported separately.

6
Conclusions

This second report of the results of a study of mortality in wards near Hinkley Point between 1995 and 1998 reveals a pattern of risk in prostate cancer that is similar to that found for breast cancer mortality. It also resembles the risk of leukemia in young people living in the same area in 1989 (Bowie and Ewings, 1989). Results also support earlier findings in of our group in Welsh data of an association of elevated prostate cancer risk with proximity to offshore, drying mud-banks containing radioactive contamination originating with discharges from nuclear plant and other sources. In the Somerset study area Relative Risk was highest in the ward adjacent to the mud-banks and downwind from Hinkley point, namely Burnham on Sea (RR = 1.5) 21 observed, 14 expected). Risk within the five kilometre and 10km radii of the centroid of the mud flats was 1.4 and thereafter risks fell continuously as larger radius rings of population were analysed. The mean trend with distance from the flats in five consecutive rings showed a similar exponential decay pattern to that shown by the penetration of radioactivity inland following sea-to-land transfer via seaspray. In the whole of the study area, risk higher risk was associated with living on low lying land near rivers compared with higher land above 200m (RR=1.25 $p < .01$). None of the results could be explained by socioeconomic variables which contributed only about 3 percent of the risk.

It is concluded that the most likely explanation for the finding is that the cancer risk follows exposure to inhaled radioactive particles, originating mainly from the nuclear site, resuspended by wave action and driven ashore by the prevailing wind. In the case of the most local downwind wards, there may also be a contribution from exposure to aerial discharges from the power station. In addition, there will be a component from weapons fallout. Although the prostate cancer pattern of risk is broadly similar to that found for breast cancer, there are some differences. In particular, the trend with distance from the coastal mud bank is not so abrupt and in addition, there is an underlying higher risk of prostate cancer in the whole study area.

This study is an ecological study and can only show association, not causality. Although we believe that the results support the hypothesis that the radioactive discharges from Hinkley Point may have contributed to the risk of both prostate and breast cancer, it is possible that some other explanation which we are unaware of may be found for the results we report. Futher work is in progress to investigate the hypothesis .

Recommendations

This research was made possible by the recent release of small area cancer mortality data by the Office of National Statistics (ONS). Before this it was impossible to obtain cancer data for small areas either from ONS or from the Regional Cancer Registries. The argument given by these registries continues to be that the data are confidential, because their release might make those who provided them feel that they were bing in some way identified. We do not accept this argument and have found that those who suffer cancer, and their friends and family, are most interested any information that might throw light on the cause of their condition. We believe that the true origin of the secrecy over small area health data is political. In a world that is increasingly filling up with toxic chemicals and radioactive isotopes released from point sources, and one in which the illness rates are increasing, particularly from cancer, the only sensible course is to allow access to incidence data so that risk can be mapped and causes examined.

Accordingly, we recommend the release of small area cancer incidence and mortality data for all years since 1974.

Second, since the results of the present study support many other studies which show that the cancer hazards of internal man-made radioactivity are hundreds of times higher that the presently accepted risk models, it is recommended that no further release to the environment of such materials is permitted, pending research which must show these substances to be harmless.

Acknowledgements

This study was supported by 'Stop Hinkley' , 'West Somerset CND' and 'The Low level [illegible] Campaign'.

Summary

This study of prostate cancer mortality is the second in a short series which uses national data to examine the relative risk of dying of cancer in 103 wards within 30km of Hinkley Point nuclear power station between 1995 and 1998. The first study, which reported the results of an analysis of breast cancer mortality was published in April 2000.

It is argued that the main source of radioactive exposure of populations living in the area is radioactive material discharged from the nuclear power station and trapped in the intertidal sediment which forms the extensive offshore mud banks which characterise the coast from Hinkley Point to Brean Down. These banks, which extend to over 50 square kilometres in area have become a depot for materials discharged from the power station since it was commissioned. It is argued that particles of intertidal sediment containing man-made radioisotopes are re-suspended by wave action and transferred to land where they are inhaled by those living close to the sea. This leads to increases in cancer in such populations. The hypothesis being tested is that the centre of the offshore mud banks is a point source of risk. The mechanism underlying the hypothesis follows recent analysis of cancer incidence data for coastal and inland populations in Wales, which showed that the population of certain towns in north Wales close to offshore mud banks had elevated risk of cancer including breast and to a weaker extent prostate cancer.

Results of the present study support the hypothesis and show that there was a statistically significant excess risk of dying of prostate cancer in the aggregate wards within 5km of the centre of the offshore mud banks near Hinkley Point (RR= 1.4; p =.05). The risk falls off with increasing distance from this point source and in wards contained within 5, 5-10, 10-15, 15-20, 20-25km and above 25km, the Relative Risk (based on England and Wales national rates) were 1.4, 1.49, 1.28, 1.22 and 1.35 and 1.02 with overall RR for the study area 1.19. ($p<.05$ throughout, χ^2 for trend 3.47, p= .05). As in the case of breast cancer mortality, the wards in the closest town to the mud banks and downwind of the nuclear power station, Burnham on Sea had high risk also for prostate cancer mortality, with 21 deaths observed and 14 expected (RR=1.5; p=.05). Social class was shown to affect the overall result by less that 3 per cent. The trend with distance from the centre of the mud banks in consecutive 5km annular rings was similar to published trend data for inland penetration of plutonium and seaspray.

The inland penetration of airborne man-made radioactive particulates is discussed and it is suggested that apart from increasing risk in coastal towns near intertidal mud or silt deposits, this effect might also result in increased risk in low-lying areas and river valleys. Comparison of the populations living in wards below the 200m contour with those above gives a relative risk of 1.25 ($p <.02$) between the two groups.

This result for prostate cancer identifies a similar high risk area to that reported by Bowie and Ewings in a 1989 Somerset Health Authority study of leukemia in young people living in near Hinkley Point. It shows a similar pattern of risk to that found for breast cancer in the preceding report. It also supports earlier studies by Rooney *et al.* of excess prostate cancer risk in nuclear workers who had been monitored for internal contamination by man-made radioactive isotopes.

The present study was made possible by the release, for the first time, of limited ward level data on mortality by cause of death. It is recommended that such data is released to wards level for earlier years and also that cancer incidence data for small areas be made available so that further and more powerful statistical methods can be used to examine the effects of point source pollution from nuclear sites.

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Appendix A

prostate obs4all	prostate expyr	prostate rr4	prostate expctall	
4	0.519	1.926782	2.076	Cannington + Combwich
3	0.853	0.87925	3.412	Central
2	0.46	1.086957	1.84	Dowsborough
2	0.427	1.17096	1.708	East Poldens
3	0.268	2.798507	1.072	Eastern Quantocks
2	0.487	1.026694	1.948	Eastover
3	0.695	1.079137	2.78	Hamp
1	0.418	0.598086	1.672	Huntspill
3	0.267	2.808989	1.068	Newton Green
3	0.647	1.159196	2.588	North Petherton
1	0.222	1.126126	0.888	Parchey
5	0.47	2.659574	1.88	Pawlett + Puriton
12	1.16	2.586207	4.64	Quantock
3	0.333	2.252252	1.332	Sandford
3	0.188	3.989362	0.752	Sowey
4	0.936	1.068376	3.744	Sydenham
1	0.675	0.37037	2.7	Victoria
1	0.431	0.580046	1.724	West Poldens
1	0.253	0.988142	1.012	Westonzoyland
3	0.375	2	1.5	Woolavington
2	0.895	0.558659	3.58	Alcombe
0	0.263	0	1.052	Aville Vale
0	0.3027	0	1.2108	Carhampton + Withycombe
2	0.2934	1.704158	1.1736	Crowcombe + Stogumber
0	0.239	0	0.956	Dunster
0	0.1226	0	0.4904	East Brendon
0	0.1562	0	0.6248	Holnicote
11	1.159	2.372735	4.636	Minehead North
4	1.329	0.752445	5.316	Minehead South
2	0.534	0.93633	2.136	Old Cleeve
3	0.52	1.442308	2.08	Porlock + Oare
1	0.4137	0.604303	1.6548	Quantock Vale
3	0.5938	1.263052	2.3752	Watchet
1	0.401	0.623441	1.604	West Quantock
5	0.5907	2.116133	2.3628	Williton
2	0.4175	1.197605	1.67	Burrow Hill
2	0.69147	0.723097	2.76588	Curry Rivel
1	0.297	0.841751	1.188	Islemoor
0	0.8055	0	3.222	Langport + Huish
6	0.912	1.644737	3.648	Martock
4	0.53057	1.884765	2.12228	Turn Hill
7	1.0965	1.595987	4.386	Wessex
1	0.365	0.684932	1.46	Blackdown
1	0.684	0.365497	2.736	Neroche
3	0.531	1.412429	2.124	Dulverton + Brushford
1	0.154	1.623377	0.616	Exmoor
0	0.167	0	0.668	Haddon
0	0.214	0	0.856	Quarme
3	0.8057	0.930868	3.2228	Bishop's Hull
5	0.6741	1.854324	2.6964	Bishop's Lydeard

4	0.314	3.184713	1.256	Bradford-on-Tone
3	0.905	0.828729	3.62	Comeytrowe
0	0.379	0	1.516	Milverton
2	0.343	1.457726	1.372	Monument
3	0.684	1.096491	2.736	Neroche
0	0.365	0	1.46	Blackdown
1	0.321	0.778816	1.284	North Curry
0	0.295	0	1.18	North Deane
1	0.249	1.004016	0.996	Norton Fitzwarren
6	0.792	1.893939	3.168	Ruishton + Creech
3	0.54	1.388889	2.16	Staplegrove
1	0.25	1	1	Stock St. Gregory
5	1.04	1.201923	4.16	Taunton Fairwater
6	0.666	2.252252	2.664	Taunton Halcon
3	0.439	1.708428	1.756	Taunton Holway
1	0.925	0.27027	3.7	Taunton Lyngford
5	0.662	1.888218	2.648	Taunton Manor
3	1.18	0.635593	4.72	Taunton Priory + Wilton
4	0.568	1.760563	2.272	Taunton Pyrland
5	0.722	1.731302	2.888	Taunton Rowbarton
2	0.913	0.547645	3.652	Taunton Trinity
2	0.435	1.149425	1.74	Trull
3	0.863	0.869061	3.452	Wellington North
1	0.227	1.101322	0.908	Wellington Rockwell Green
5	1.14	1.096491	4.56	Wellington South
1	0.266	0.93985	1.064	West Deane
3	0.428	1.752336	1.712	West Monkton
1	0.555	0.45045	2.22	Wiveliscombe
1	0.261	0.957854	1.044	Axbridge
2	0.448	1.116071	1.792	Axe Vale
0	0.728	0	2.912	Berrow
5	0.475	2.631579	1.9	Brent
10	1.946	1.284687	7.784	Burnham North
11	1.614	1.703841	6.456	Burnham South
5	0.797	1.568381	3.188	Cheddar
7	1.21	1.446281	4.84	Highbridge
2	0.397	1.259446	1.588	Mark
0	0.235	0	0.94	Shipham
2	0.621	0.805153	2.484	Wedmore
4	0.436	2.293578	1.744	Avalon
0	0.224	0	0.896	Ebbor
2	0.343	1.457726	1.372	Glastonbury St. Benedict's
2	0.254	1.968504	1.016	Glastonbury St. Edmund's
3	0.366	2.04918	1.464	Glastonbury St. John's
1	0.425	0.588235	1.7	Glastonbury St. Mary's
1	0.369	0.677507	1.476	Moor
2	0.395	1.265823	1.58	Rodney
2	0.443	1.128668	1.772	Sheppey
3	0.951	0.788644	3.804	Street North
2	0.802	0.623441	3.208	Street South
1	0.512	0.488281	2.048	Wells Central
5	0.91	1.373626	3.64	Wells St. Cuthbert's
3	0.874	0.858124	3.496	Wells St. Thomas
280			234.9	total