Infant and Perinatal Mortality and Stillbirths near Hinkley Point Nuclear Power Station in Somerset,1993-2005.

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Hinkley Point showing outfall pipe discharging waste to the sea

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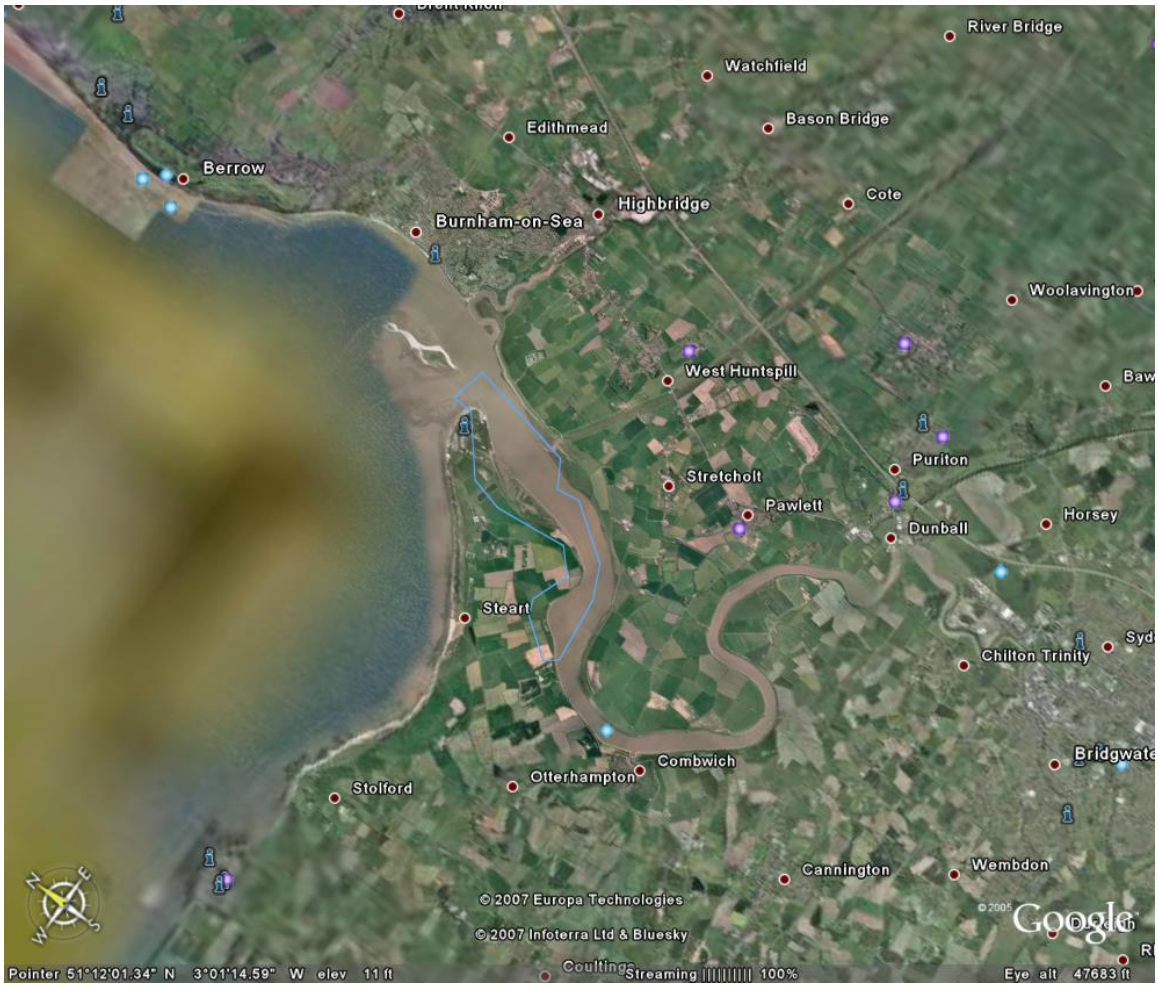
Summary

We examined infant mortality, perinatal mortality and stillbirths in a region of North Somerset surrounding and inland from the Hinkley Point Nuclear Power Station using data for the 13 year period 1993-2005 purchased from the Office for National Statistics. The purpose was to see if the sea coast effect on cancer which had been found in earlier were mirrored by birth outcomes. Because of ward changes we looked at two periods 1993-98 and 1999-2005. In 1993-1998 there was a trend in infant mortality, perinatal mortality and stillbirths by distance from the radioactively contaminated Steart Flats. For this period, Relative Risks based on the whole study area for 33 Infant Deaths in the 29 wards with centroids inside three concentric 6km annular rings centered on the offshore mud bank were 1.9, 1.54, 0.81 compared with 0.9 in the rest of 103 wards in the study area. The excess risk in the inner 6km ring was statistically significant $p = 0.03$ but the overall trend in the 18km ring was not since the numbers were low. We examined the overall trend in this period over all the wards using Poisson Regression and also introduced the Index of Multiple Deprivation as a covariate. The result was that the trend with distance from the putative point source was significant at the $p = 0.015$ level but interestingly, deprivation was not a significant factor. We also examined perinatal mortality (0-28 day + stillbirths) by distance bands. The result on 63 stillbirths in the 18km ring was a falling off of risk from the mud bank in the order 1.24, 1.35, 1.1 in the 6km bands with 0.9 in the rest of the study area.

In the second period, 1999-2005, the Poisson Regression Result was not significant for distance for the whole area and there was also a flat response to infant mortality risk by distance band in the 18km ring with relative Risks of 1.01, 0.86, 1.1 compared with 0.9 in the rest of the area. When we shifted the putative point source to Hinkley Point itself the result was that the effects disappeared. Since our earlier studies had focused on cancer in the ward of Burnham on Sea, downwind of the Steart Flats, we examined rates in this ward. Infant Mortality risk in 1993-1998 was high 4.3 ($p = 0.01$) and neonatal mortality also; RR was 6.7 ($p = 0.003$) based on 4 deaths. Sex ratios in Burnham on Sea were anomalous with a mean of 1175 over the whole period, an excess of boys born relative to the expected mean of 1050; this high level is the same as that found in those exposed to internal radionuclides after Hiroshima. To examine the source of the excess infant mortality we focused on the estuary wards. In the whole period 1993-2005 there was an excess of infant deaths in the 11 estuary wards (24 deaths in 3866 livebirths) compared with the 138 deaths in the 34005 livebirths in the rest of the area ($OR = 1.53$ ($0.99<OR<2.35$) $p = 0.053$). This was driven by a sudden peak in infant mortality which occurred in 1996 when there were 5 deaths in the 295 livebirths in the estuary wards compared with 9 deaths in the 2800 births inland ($OR = 5.27$ (1.53, 17.31; $p = 0.0009$). The trend with time shows an increase in the estuary area which persists until 2001 giving for the period 1996-2001 an Odds Ratio of 2.74 ($1.61<OR<4.65$) $p = 0.0001$) in the test of estuary vs. inland. We conclude that this event suggests the explanation for the increases in cancer in the estuary area and in Burnham on Sea which we found in the earlier studies, namely that there was a release of radioactivity from the Hinkley Point plant around 1995 and that this caused firstly an increase in infant mortality and subsequently the increases in cancer which we found. We discuss the sources of the exposures and highlight an accidental release of radioactivity in October 1994 for which the operators were fined £22,000 by the pollution inspectorate.



The Estuary of the River Parratt and the Steart Flats. Hinkley Point is on the coast in the bottom left had corner of the Google Earth satellite image. At low tide, the mud is exposed for several miles offshore.

1. Introduction

Between 2000 and 2004 Green Audit carried out a number of epidemiological studies of cancer (mortality and incidence) near the Hinkley Point nuclear site in Somerset (Busby et al 2000, Busby and Rowe 2002). These studies have been part of a general interest in the health effects of chronic low dose internal exposure to radioactive fission products and to uranium (Busby 1996, 2002, 2007). Two main conclusions have resulted from all these studies. The first is that internal exposures to certain radionuclides carry significantly enhanced risk of genetic damage relative to the risks modeled on the basis of the mean absorbed doses. The second is that those who live in regions where there is an elevated atmospheric concentration of such radioactive particles suffer an increased risk of cancer (ECRR2003, CERRIE 2004b). The matter has been brought to the attention of various risk agencies and government committees, but there has been little sign of any official change in thinking in this area (CERRIE2004a) despite what seems to be an

acceptance by some that there is a problem with applying data from acute external exposures at Hiroshima to chronic internal exposures to substances which have specific biochemical affinities for DNA (IRSN2006) and in the case of Uranium, may even amplify and focus natural background gamma radiation doses in the DNA (Busby and Fucic 2006, Busby 2005).

A large study of cancer in populations living near the Irish Sea conducted from 1998-2001 suggested the hypothesis that populations living near radioactively contaminated intertidal sediment would be exposed, by sea to land transfer, to respirable radioactive particles and would show increased cancer risk (see Busby 2007 for a review of these studies). The earlier investigation of cancer near Hinkley Point nuclear site supported this idea and showed that there was increased risk of all cancers combined, prostate cancer, lung cancer and breast cancer by distance from the main source of respirable radioactive particles, the large offshore mud bank known as the Steart Flats, west of the coastal town of Burnham on Sea and east of the Hinkley Point site and its liquid radioactive waste outfall pipe. A satellite photo of the plant showing discharges from the plant is reproduced on the cover. The mud flats at the mouth of the River Parratt become the cumulative depository of all the liquid, and most of the airborne releases (due to washout from the atmosphere) from the power station. Our hypothesis was that radioactive particles move up the tidal river Parratt and are resuspended, with the result that local proximal estuary wards would carry excess cancer risk following inhalation.

The original ward level mortality studies (Busby et al 2000) indicated significant excess breast cancer mortality risk in the town of Burnham on Sea, specifically the North Ward. These studies received considerable media attention and were attacked by the nuclear industry, the local health authorities and the government watchdog Committee on Medical Aspects of Radiation in the Environment (see the COMARE website). Since incidence data at small area level were (and are) not made available for independent research, a local community group, Parents Concerned about Hinkley (PCAH) conducted a door to door survey of cancer in Burnham North ward. The questionnaire method, developed by Green Audit, and already piloted in Carlingford, County Louth, Ireland uses the population defined by the questionnaire (i.e. those living in the town at the time) as a population at risk to which national age specific rates can be applied. Results for Burnham North showed the existence of a 2-fold excess breast cancer risk and also revealed 2.7- fold increased leukemia risks in the six years 1996-2001 (Busby et al 2002). The main results are given in Table 1.

The study was criticized by various official groups including COMARE (see www.comare.org) but a subsequent follow up study by the South West Cancer Intelligence Agency confirmed the high breast cancer and leukemia risk in the ward (SWCIS 2003). This SWCIS study argued that although there was a higher than normal risk in Burnham, it was merely the result of the natural play of chance and had no causal relationship with releases from the nuclear site. The study and its findings, particularly the questionable mathematical treatment of trend in cancer by distance from the mud banks were reviewed in Busby 2007.

Table 1 Relative Risk (see text) for cancer in Burnham North from 1996-2001 indicated by PCAH survey (based on England and Wales rates for 1997) (Busby *et al* 2002)

Cancer	**Observed**	**Expected**	**RR**	**Poisson p-value**
All malignancy	64	66	0.97	NS
Female breast	16	8.09	1.98	0.01
Kidney	5	1.26	3.96	0.01
Leukemia	4	1.46	2.73	0.05
Cervix uteri	3	0.54	5.55	0.01
Colon	6	7.8	0.77	NS
Prostate	7	7.2	0.97	NS
Lung	4			Low

By 2007, we were able to look at breast cancer in Burnham North in the whole period. Between 1995 and 2005 there were 41 deaths from breast cancer in Burnham North whilst 24 were predicted on the basis of the age breakdown of the population, the ward level socioeconomic status and national rates. This represents a 70% excess risk with a cumulative Poisson p-value of 0.001: that is one chance in 1000 that this was a chance effect.

Since cancer is a genetic disease expressed at the cellular level, areas which have a true causal increase in cancer risk due to the presence of some mutagenic agent, might also be expected to show a high risk of other health conditions associated with mutagenic stress. We were therefore interested in examining the levels of infant and perinatal mortality and stillbirth in Burnham North to see if these end points are also raised, and at the same time to examine the same area of Somerset in which we had previously looked at cancer risk. The hypothesis we are testing is that there is an increased risk of infant mortality in estuary wards near the Steart Flats which we proposed as the source of exposures which caused the excess cancer we found in earlier studies.

2. Method

The area we examined was the same area of Somerset we used to examine the cancer mortality trends. The data were split into 2 periods since after 1999 there were ward boundary and ward name changes. We examined the post-1999 set of wards separately, in addition to combining them with the pre-1999 wards for an overall examination of trend. Annual numbers of total births by census ward were purchased from the Office for National Statistics together with tables of infant and neonatal (0-28day) mortality by year and ward for the appropriate periods. Stillbirth data were also obtained from ONS up to 2001, after which the data were no longer released owing to a new decision about confidentiality. We have made a formal request to ONS under the Freedom of Information Act for these stillbirth data. In order to examine the relation between

disadvantagement and infant mortality in the area, deprivation quotients were obtained from ONS for the pre-1999 wards.

First, ward level Relative Risk was examined on the basis of the rate for the appropriate period for the whole of the study area, some 100 wards. This defined an expected number of infant deaths, neonatal, stillbirths etc in any ward or aggregate of wards, and comparison of the observed number with this expected number gave a Relative Risk, RR = O/E. We examined the effect of the contaminated mud bank and the estuary of the Parratt in a number of different ways. First we determined the distance in km of the centroid of the wards from (a) Hinkley Point and (b) the centre of the Steart Flats. Then:

1. We aggregated wards into roughly equal numbers at annular bands with distances from the two possible point sources (a) and (b) of 0-6km, 6-12km, 12-18km, all the rest. We then compared rates for the various end points in these distance bands for the two separate periods 1993-98 and (without the stillbirths) 1999-2005 and (without the stillbirths) for the whole period.
2. We examined the rates and Relative Risks in Burnham on Sea.
3. We constructed coloured risk maps for the two periods and two sets of wards.
4. We looked at wards which were proximal to the estuary or the sea and compared these with those which were not.
5. We examined the trend for infant mortality from the two putative point sources using a General Linear Poisson Regression model of Numbers of cases versus both Distance and Deprivation for the two periods and for the whole period:

   $$\text{Log}(N/B) = \alpha \log(\text{Distance}) + \beta \log(\text{Deprivation}) + \text{Intercept}$$

6. We compared the trend in infant mortality risk over the whole period by estuary and inland wards.

3 Results

3.1 The whole study area

The whole area was the same as that used in the earlier cancer mortality study and consisted of the wards listed in Appendix A. Details of the total numbers of births and the various rates for infant mortality, neonatal mortality and stillbirths are given in Table 2.

Rates for all three end points in the overall study area were slightly lower than those of England and Wales. Risk maps for infant mortality are given in Fig 1 and Fig 2. Perinatal mortality risk (Neonatal and Stillbirths) is mapped for 1993-98 in Fig 3.

Fig 1. Infant mortality risk in area near Hinkley Point (red dot) 1993-1998.

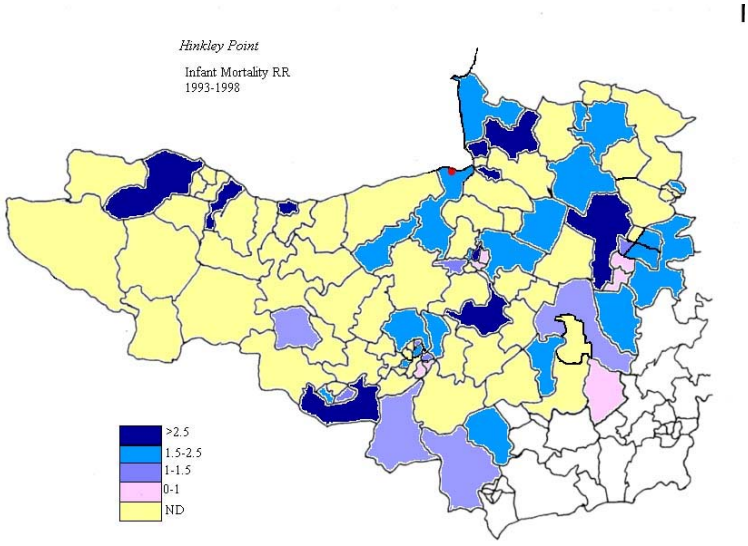


Fig 2. Infant mortality Risk near Hinkley Point (red dot) 1999-2005 . Note that the wards are slightly different after 1999

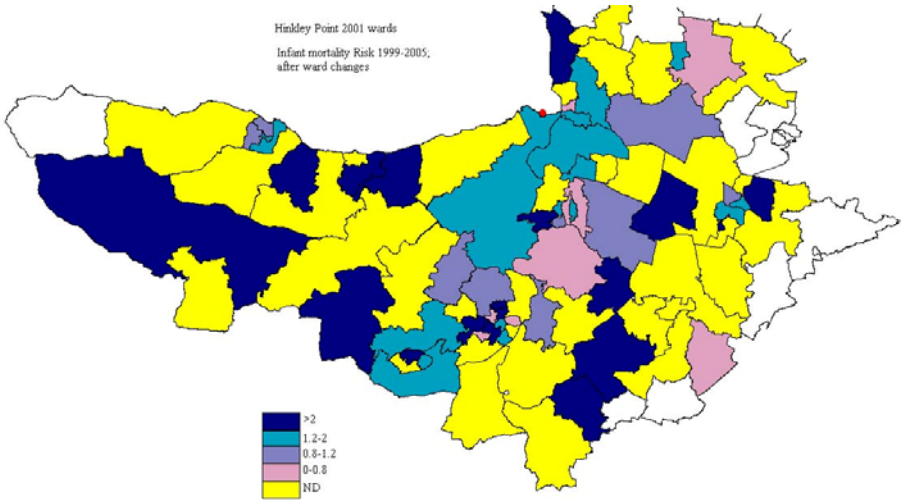


Fig 3 Perinatal (0-28day + stillbirth) mortality risk in study area 1993-98

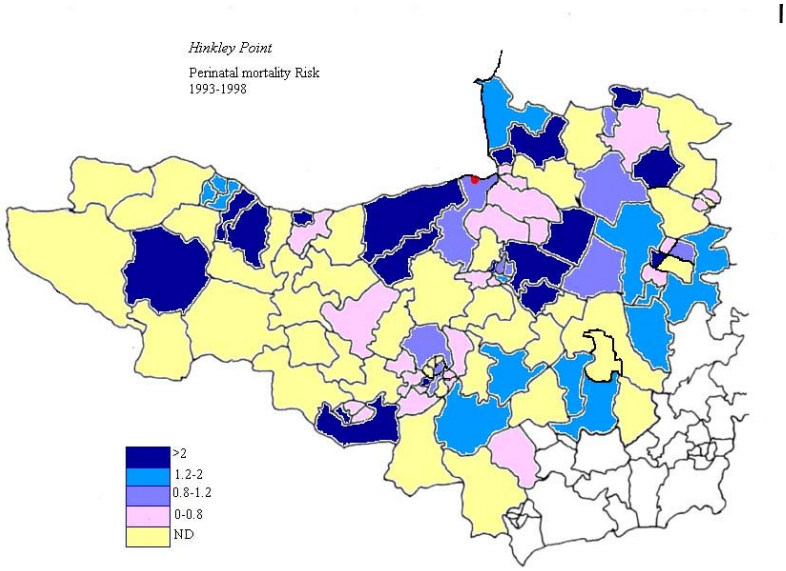


Table 2. Total births in the study area for the two periods 1993-98 and 1999-2005 with overall rates of infant and neonatal mortality and still births compared with England and Wales rates 1997.

	1993-1998 (6y)	**Rate 93-98 Per 1000**	**1999-05 (7y)**	**Rate 99-05 Per 1000**	**E & W 1995 Rate/1000**	**E & W 2002 Rate/1000**
Live Births	19097		21687			
Infant	79	4.13	96	4.42	6.1	5.3
Neonatal	51	2.67	71	3.27	4.1	NA
*Stillbirth	101	5.29	NA	NA	5.52	NA

**Stillbirth data not available after 2001. In 1995, the Infant Mortality Rate for all Somerset was 3.2/1000; in 2002 it was 4.2/1000*

3.1 Burnham on Sea North

Overall results for the two periods are given in Tables 3 and 4 below. There was a significantly high level of neonatal (0-28days) mortality in the first period but no infant mortality in the second period

Table 3 Total births, infant and neonatal mortality and stillbirths in Burnham on Sea North 1993-1998. Also given is the sex ratio, the number of boys born per 1000 girls.

	Number Persons	*Sex Ratio M/F	Rate/1000 livebirths	Relative Risk O/E	P-value (Poisson)
Live births	225	1163	-	-	-
Infant deaths	4	-	17.7	4.3	0.01
neonatal	4	-	17.7	6.7	0.003
stillbirths	0	-	0	-	-

*Sex ratio in England and Wales was 1055 in 1996 and is generally stated to be 1055 males per 1000 females with a Standard Deviation of 2.6

Table 4 Total births, infant and neonatal mortality in Burnham on Sea North 1999-2005. Also given is the sex ratio, the number of boys born per 1000 girls.

	Number Persons	*Sex Ratio M/F	Rate/1000 livebirths	Relative Risk O/E	P-value (Poisson)
Live births	304	1187	-	-	-
Infant deaths	0	-	0	0	0
neonatal	0	-	0	0	0

*Sex ratio in England and Wales was 1055 in 1996 and is generally stated to be 1055 males per 1000 females with a Standard Deviation of 2.6.

3.2 Comparison by distance bands from Steart Flats.

The distances of the centroids of the wards from the centre of the Steart Flats were determined using the GIS program ARCVIEW. Then wards within 18km were aggregated into 3 groups according to these distances 0-6, 6-12 and 12-18km. Relative Risks were determined for each aggregated group. Results are shown in Table 5 for the period 1993-1998 and a plot with an exponential fit of the Relative Risks (based on the whole area rates for the period) in Fig 4.

The numbers of cases in the 18km study radius circle were too low for statistical significance of the trend line either using Chi-square for linear trend or Poisson regression ($p = 0.09$) but a Poisson regression analysis for the whole area for the period 1993-98 was carried out and showed a significant trend (see below).

In the period 1999-2005, the trend in infant mortality was flat (see Table 6) but stillbirths were not available after 2001.

Table 5. Births, Infant and Perinatal Mortality and Relative Risks in wards with centroids in radial band areas by distance from the centre of the Steart Flats near Hinkley Point Nuclear site 1993-1998.

Distance (N) wards	Obs: Inf (births)	Expect: Infant	RR inf. (p-value)	Obs: Perinatal	Exp: Perinatal	RR peri. (p-value)
0-6 (5)	11 (1415)	5.8	1.9 (0.03)	14	11.3	1.24 (NS)
6.1-12 (9)	10 (1580)	6.5	1.54 (NS)	17	12.6	1.35 (NS)
12.1-18 (15)	12 (3602)	14.9	0.81 (NS)	32	29	1.1 (NS)
18.1+ (74)	46 (12500)	51	0.9 (NS)	89	99	0.9 (NS)

Table 6. Births, and Infant Mortality and Relative Risk in wards with centroids in radial band areas by distance from the centre of the Steart Flats near Hinkley Point Nuclear site 1999-2005.

Distance (N) wards	Obs: Inf (births)	Expect: Infant	RR inf. (p-value)
0-6 (5)	7 (1555)	6.9	1.01 (NS)
6.1-12 (12)	14 (3695)	16.3	0.86 (NS)
12.1-18 (9)	11 (2237)	10	1.1 (NS)
18.1+ (67)	64 (13960)	62	1.03 (NS)

3.3 Coastal and estuary wards.

We aggregated wards on the basis of their proximity both to the tidal River Parratt the estuary and to the sea coast and compared these wards with the inland ones. For the initial analysis we set up a coastal/ tidal category which had all the wards which were either adjacent with the sea or along the tidal section of the River Parratt as far as Bridgwater. Infant mortality results are shown in Table 7. Interestingly, the clear effect present in the first period was missing in the second period. Table 8 gives these results. In order to further investigate these results and to examine what was driving the effect, we localized the examination to the outer estuary wards and compared the infant mortality trend in these wards with the trend in the inland wards. The trend in infant mortality in these two areas, represented as Relative Risk, based on Somerset rates, are shown in Fig 5 where we have also shown LOESS regression fits of the two datasets. The wards are given in Table 9, the numbers in Table 10 and the statistics in Table 11.

Fig 4. Exponential fitted line and Relative Risk of Infant Mortality by ward and distance from Steart Flats 1993-1998 in the restricted 18km radius area.

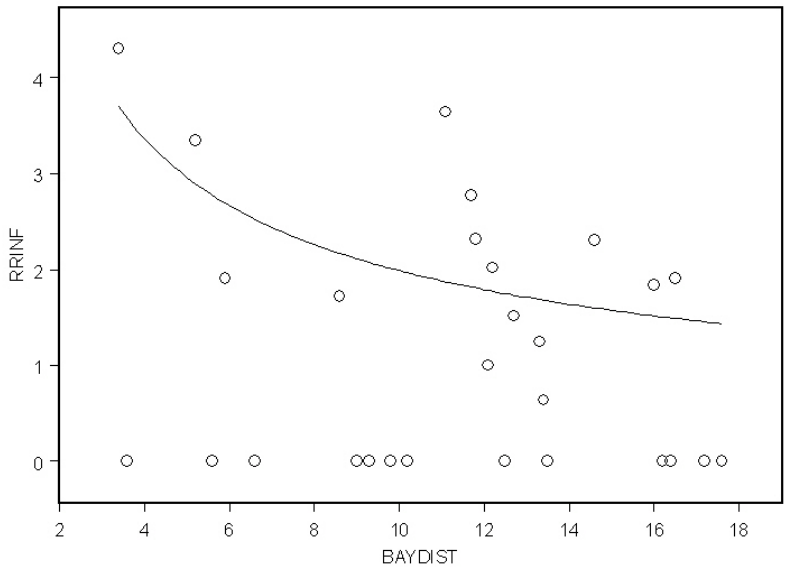


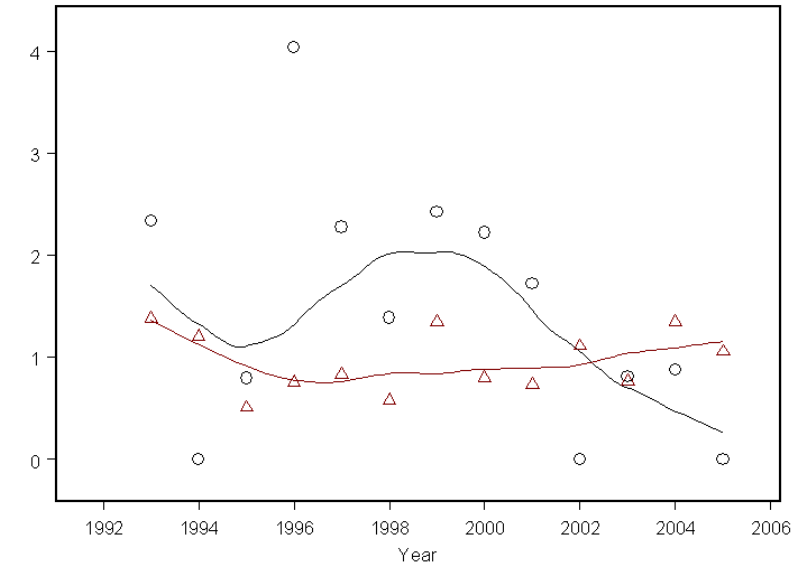
Table 7. Infant mortality in coastal/ estuary and inland wards 1993-1998

	N wards	**Births**	**Infant deaths**	**Expected**	**RR (p-) 95%CI**
Coast	26	4859	33	21.5	1.53 (0.01)
Inland	77	14238	46	62.9	0.73
Test of Coast vs. Inland	OR = 2.1 P = 0.001; 95% C.I. is 1.34<RR<3.27 (Mantel Haenszel)				

Table 8. Infant mortality in coastal/ estuary and inland wards 1999-2005

	N wards	**Births**	**Infant deaths**	**Expected**	**RR (p-)**
Coast	24	5162	25	22.8	1.1 (NS)
Inland	69	16285	71	71.9	0.98
Test of coast vs. inland	Not significantly different				

Fig 5 Trend in infant mortality risk by years 1993-2005 comparing the estuary (blkue) wards near Hinkley Point (listed in Table 9) with all the inland (red) wards. Upper: LOESS fitted line. Note high value in 1996. (Estuary: circles and black line; inland: triangles and red line.)



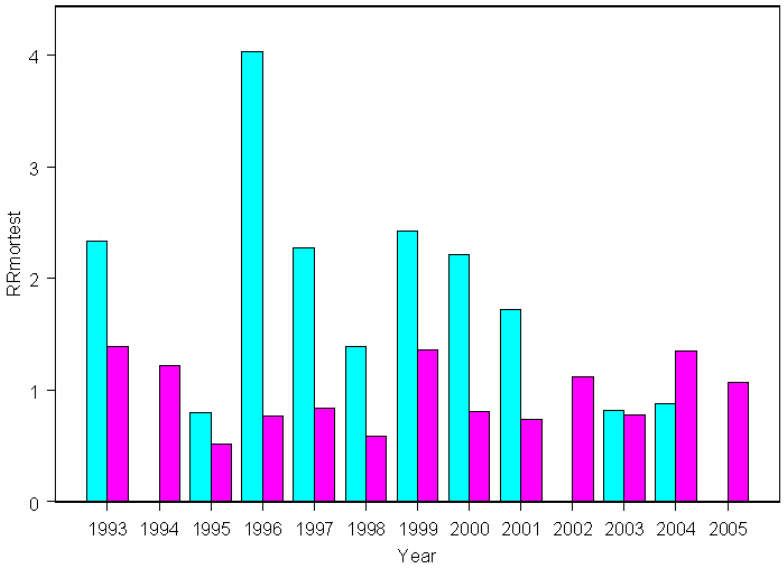


Table 9 Estuary wards. In period 2 (1999-2005) there were boundary changes and new wards were defined. We have employed these where this has occurred.

Ward	Period (1993-8 = 1)
Cannington and Combwich	1
Cannington and Quantocks	2
Huntspill	1
Huntspill and Pawlett	2
Highbridge	1, 2
Burnham North	1,2
Burnham South	1,2
Berrow	1,2
Brent	1
Brent North	2

Table 10 Infant mortality trend in estuary wards (Table 9) and inland wards by year. We should expect about 1.25 deaths per year in the estuary wards (on 300 births) and11.8 deaths inland on 2800 births.

Year	**Estuary**		**Inland**	
	Births	Infant deaths	Births	Infant deaths
93	306	3	3081	18
94	310	0	2932	15
95	300	1	2755	6
96	295	5	2800	9
97	314	3	2829	10
98	343	2	2832	7
99	295	3	2450	14
00	322	3	2363	8
01	277	2	2250	7
02	256	0	2337	11
03	292	1	2454	8
04	272	1	2463	14
05	284	0	2459	11

Table 11 Statistical test of Estuary vs Inland (Mantel Haenszel, Chi-square, 95% Confidence intervals and p-values).

Period	**Odds Ratios, (95%CI), p-value**
93-05	OR = 1.53 (0.99<RR<2.35) p = 0.053
96-01	OR = 2.74 (1.61<RR<4.65) p = 0.0001

3.4 Poisson regression and the effect of distance and also deprivation

Coefficients of overall deprivation are available from various sources. In the past we have employed Social Class by census ward as a weighting when examining cancer mortality risk (Busby et al 2000). In this study we use the more recent Index of Multiple Deprivation, which is available from ONS for each pre- 1999 census ward. Since it is generally found that infant mortality and deprivation are correlated, we ran Poisson Regression modeling infant mortality (DEATHS) on Deprivation (DEPP) and also on distance from the Steart Flats (FROMBAY) for the period 1993-98. Results, which showed a significant effect for distance from the Steart Flats (p= 0.015), but no effect for Deprivation are given in Table 12.

The regression equation was of the usual form:

$$\text{Log (DEATHS/BIRTHS)} = \alpha \log (\text{FROMBAY}) + \beta \log (\text{DEPP}) + \text{Intercept}$$

Table 12 Poisson Regression results for analysis of infant mortality by distance from Steart Flats (BAYDIST) and Deprivation (DEPP) for all wards in the 1993-1998 period.

Coefficients:	**Value:**	**Std Error**	**t-value**	**ANOVA Pr (Chi)**
(Intercept)	-5.22615206	0.43581618	-11.9916431	-
FROMBAY (α)	-0.02630337	0.01252011	-2.1008888	0.0156347
DEPP (β)	0.01204164	0.01222829	0.9847358	0.3290212

4. Discussion.

4.1 Radiation and infant mortality

The association between increased infant mortality and exposure to internal fission product radionuclides at the time of atmospheric testing 1955-1965 was first pointed out by Sternglass (1971) but has been disputed in that context by others (Lindop and Rotblat 1971, Nishiwaki et al 1972). In 1992 the issue was reopened in a paper in the British Medical Journal by Robin Whyte who argued that there was a real environmental effect in the period 1959-63 in the USA and also in England and Wales (Whyte 1992). In rodents, exposure to Strontium-90, caused foetal death (Luning and Froelen 1963, Smirnova and Lyaginskaya 1969) and the effect (due largely to development effects including to congenital heart defects) was also present in England and Wales populations at the time of the weapons fallout where it correlated with the Sr-90 levels (Busby 1995). More recently, infant mortality effects have been found in Germany and in the former

Soviet Union following the radiation exposures of the Chernobyl accident (Koerblein and Kuchenoff 1997, Petrova et al 1997 Scherb et al 2000,Koerblein 2004, Koerblein 2006,). The risk agencies (UNSCEAR 2000) do not consider infant mortality as a radiation effect either from exposure to weapons fallout nor the Chernobyl exposures. The ECRR (ECRR2003) on the other hand have provisionally allowed for the effect of radiation on infant mortality and stillbirths and based its risk factor on a 14% increase per mSv (as calculated by the conventional ICRP risk model).

Infant mortality as an end point for mutagenic effect is of course epidemiogically difficult (Doll 1973). The reason is simple. The foetus can only sustain a certain level of damage, and high levels of damage cause miscarriages. Therefore the dose response has to be biphasic: increasing the dose beyond a certain point can cause a decrease in infant mortality. Of course this may also be accompanied by a decrease in birth rate, and this has been reported in the period 9 months following the Chernobyl accident (see e.g. Bentham and Haynes 1991 and Busby 1995 for a discussion). With regard to nuclear sites, Mangano et al have shown effects in downwinders and significantly also that these effects cease after the plants are shut (Mangano et al 2002). Mangano et al's study is relevant to the present one. Subsequent to 1987, 8 U.S. nuclear plants located at least 113 km from other reactors ceased operations. Strontium-90 levels in local milk declined sharply after closings. But so did deaths among infants who had lived downwind and within 64 km of each plant. These reductions occurred during the first 2 yr that followed closing of the plants, were sustained for at least 6 yr, and were especially pronounced also for birth defects. Trends in infant deaths in proximate areas not downwind, and more than 64 km from the closed plants, were not different from the national patterns.

4.2 Hinkley Point and Cancer effects

As outlined in the introduction, we set out to examine infant and perinatal mortality near the Hinkley Point nuclear site because we had previously examined cancer rates in the same area of north Somerset and found a significant excess risk, particularly of breast cancer, in those living near the contaminated offshore intertidal sediment (Busby et al 2000). There was a decreasing trend in risk by distance from the mud bank known as the Steart Flats between 1995-1998 for 'All malignancy', Prostate, Lung and Breast cancer and the highest risk, for breast cancer, was found to be in the ward of Burnham North, downwind from the mud bank. Incidence statistics at the ward level are not made available for research and so a small area questionnaire study was carried out with a local group, Parents Concerned About Hinkley, PCAH. Results confirmed the breast cancer excess and showed also an excess in leukemia, cervical and kidney cancer also. (Busby and Rowe 2002). The Somerset Coast Primary Care Trust commissioned the South West Cancer Intelligence Agency to follow this with their own examination of the cancer incidence in the area (SWICS 2003). Their findings showed that the PCAH survey of Burnham North had accurately determined the excess breast cancer and also leukemia risk, but the author of the SWCIS study, Dr Julia Verne, argued that the effect was one of chance and was not related to exposures from the plant or contaminated mud.

Nevertheless, there was an increased risk of cancer in those who live near the contaminated estuary and offshore mudflats and the effect was greatest in Burnham on Sea North. And it was also true that earlier studies of child leukemia in the area made by

Somerset health Authority had found that this was significantly high nearer the Hinkley point plant. And also a study published by Alexander et al in 1990 had associated increased child leukemia with living near estuaries (Alexander et al 1990). Since the radiation exposures that cause cancer (by damaging genetic material) also cause infant deaths (by damaging genetic material) it is not unreasonable to want to see if there might be increased infant mortality in the same area where previously we (and Verne) found increased cancer risk.

4.3 Infant mortality

We turn to the results. The sequence of investigation began with the largest area and then attempted to move forward a step at a time to see what was driving the effects we found. So having noted a significant effect by distance from the mud bank, we then looked to see if it was a general coastal effect or an estuary effect. After refining the study to look at the estuary we looked to see which wards carried the highest risk, and which years carried the highest risk. This sequence is summarized with the results in Table 13.

First, we were unable to examine stillbirths throughout the whole period since these data were no longer made available after 2001. We will examine each result of our study in turn. The study area consisted of 103 wards with 19097 births between 1993 and 1998 and 21697 births in 93 wards between 1999 and 2002. There were 79 infant deaths including 51 neonatal (0-28 day) deaths in 1993-98 and 96 infant including 71 neonatal deaths in 1999-2005. These define rates of 4.13 and 4.42 infant deaths per 1000 live births, slightly under the England and Wales mean for the period of 5.2 infant deaths per 1000 live births. The rate for stillbirths in the first period was 5.29 per 1000 live and dead births.

The infant and perinatal mortality results for the period 1993-1998, that is in the pre 1999 wards, were mapped by risk level (Figs 1 and 2). These maps suggest a clustering of risk near the coast, and this is confirmed in the case of infant mortality in this period by the aggregation of wards into radial distance bands of 6km depth. Table 5 shows that in the first period, based upon the rates for the whole area, there was a falling off of risk as we move away from the putative point source with the highest risk for infant mortality of 1.9 in the 5 wards which are within the 6km centroid band (a statistically significant effect with Poisson $p = 0.03$). The trend line is fitted to the individual ward risk points using an exponential regression in Fig 4. The overall trend was not significant however, probably because of the low numbers of deaths. Perinatal mortality risk was also higher in the 0-6km strip and lowest in the area outside the 18km radius chosen for the examination. In both cases the linear trend in proportions was not statistically significant.

We then examined Infant Mortality in the first period 1993-1998 by distance from the putative point source, the Steart Flats, using Poisson Regression. In this approach we also included deprivation as a covariate and used the whole of the area, rather than the restricted are near the point source in order to bring in more cases. For this we employed the ONS Index of Multiple Deprivation (see www.ons.gov.uk). The result was that there was a significant correlation between distance from the point source ($p = 0.016$, Table 12) but interestingly no effect in relation to deprivation. It is quite interesting that in this location, infant mortality is not driven in any way by deprivation, or at least we can say that the main source of infant death is not deprivation.

Table 13 Identifying the main driver for infant mortality risk in North Somerset wards 1993-2005 by distance from Steart Flats mud bank.

Area and period tested	**Test and Result**	**Conclusion**
1. All pre 1999 wards 93-99	Radial 6km rings show continuous reduction in risk; inner risk p = 0.03	1. Effect near the mud bank before 1999
2. All post 1999 wards: 99-05	Radial 6km rings do not show continuous reduction in risk	2 Effect not there after 1999
3. All pre 1999 wards 93-99 Poisson Regression on distance	Significant Regression line on distance p = 0.015; no effect for deprivation	3. Too few cases for significance in 18km ring but significant with all the wards using Poisson Regression
4. All post 1999 wards: 99-05	Regression not statistically significant; deprivation not available for wards	4. No effect in second period after 1999
5. Coastal and estuary wards vs. the rest pre 1999 wards	Statistically greater risk; OR = 2.1; (1.34, 3.27)	5. Effect increases if we look at coastal and estuary wards before 1999
6. Estuary wards only vs. the rest pre-1999	Statistically greater risk than for the coastal and estuary wards	6. Effect increases if we look at the estuary wards
7. Trend by year in estuary wards	Pinpoints increase in risk beginning in 1996 and ending in 2001	7. Effect is due to an increase in 1996 which falls off by 2001
8. Estuary wards only vs. the rest between 1996 and 2001	Effect is driven by the increase in the estuary wards between 1996 and 2001; OR = 2.74 (1.61, 4.65) p = 0.0001	
9. Burnham on Sea	Burnham on Sea shows the highest Risk in the period of high risk above; RR = 4.3 (vs whole of study area; p = 0.01)	8. Whatever is causing the effect is close to or upwind of Burnham on Sea and began in 1994/1995

The trends seen in the first period were not apparent in the second period after 1999 as we show in Table 6; Poisson regression for the second period also showed no significant effects on distance. A Poisson regression using Hinkley Point Nuclear Power Station as the centre of risk gave no significant result either by the concentric ring aggregation approach or by Poisson Regression. We have not recorded these negative results here but it should be borne in mind that we would not predict an effect centered in the nuclear power plant since the radioisotopes accumulate in the mud following their dispersion from the plant and in our hypothesis, it is the mud that is the source of risk.

To try and tease out the driver of these interesting results we looked at the wards which were proximal to the sea combined with those which were estuary wards. We

included all the seaside wards and also those which bordered on the tidal section of the River Parratt as far inland as Bridgwater. Results showed a highly significant effect when these were tested against the inland wards for the first period 1993-1998 (Odds Ratio = 2.1 CI (1.34<OR<3.27) p = 0.001) Table 7. Again, there was no significant effect in the period 1999-2005.

The effect was clear in Burnham on Sea North, as we show in Table 3. The Relative Risk for Infant Mortality was 4.3 (p = 0.01 in the period 1993-98 and for neonatal mortality was 6.7 (p = 0.003). Again this effect did not persist into the later period 1999-2003 (Table 4). In order to follow up on whether there was a genetic effect on the birth outcomes we calculated the Sex Ratio (SR, the number of males born per 1000 females). The sex ratio SR is considered to be a good indicator of genetic damage from radiation. A discussion of its use in the Hiroshima survivors is given in Busby 2007. It should not differ significantly from 1055. In Burnham it is as high as 1163 and this persist to the second period. More boys are being born: this is exactly what happened after Hiroshima in the city entrants (those who entered after the bomb) and it follows irradiation of the mothers by internal radioactivity. However, owing to the small number of births, we cannot say that this is not due to chance in this case.

To further examine the reason why there was a clear effect in the first but not second period we next focused on the estuary wards only. These 11 wards are given in Table 9. In Table 10 and 11 the infant mortality trend analysis suggests the explanation. As Fig 5 shows, the trend in infant mortality shows that levels increased sharply around 1996 and persisted until 2001. In 1996 there were 5 infant deaths in the 295 births in the estuary wards compared with 9 deaths in the 2800 births in the rest of the area (OR = 5.3 (1.75<OR< 15.4) p = 0.0009). Taking the whole period 1996-2001 there were much higher risks in the estuary wards than the inland wards OR = 2.74 (1.61, 4.65) p = 0.0001. Thus it is these estuary wards rather than the coastal wards that drive the effect and Burnham on Sea where the effect is greatest. From the map it is clear that Burnham onb Sea, and particularly the north ward, which has the largest population close to the mud flats and also directly downwind of the plant. The Hook of the south bank of the estuary funnels the wind directly to the Burnham North ward. We feel that this is the result which explains the other findings and possibly also the later increases in cancer which we discovered in the same ward areas in earlier studies. These results suggest that some radiation release from the nuclear plant occurred around 1995 which resulted in exposures to people living near the estuary wards and mainly those in Burnham North.

Table 14 Total reported and Relative Risks for some cancers in Burnham North 1997-2001, with Standardised Incidence Ratio SIR based on England and Wales

Site	Reported 1997-2001	Expected 1997-2001	SIR	p-value
Breast	14	5.85	2.4	0.003
Leukemia	4	1.2	3.3	0.04
Kidney	4	1.05	3.8	0.02

For this reason we return to re-examine the incidence risks for cancer which we found in the PCAH questionnaire Table 1. We focus now on the period 1997-2001. Since the

increase in infant mortality occurred in 1996, we assume that the exposure that caused this was in 1995. We should therefore look for increases in cancer starting in about 1997. This is the response trend that occurred after Chernobyl and is due to induction of genomic instability and the progression of pre-cancerous genomes into clinical expression of the disease. Table 14 lists the main cancers found in the PCAH survey with their expectations and risks in this period. There were 5 breast cancers reported diagnosed in Burnham North in the single year 1997, 3.7 times the expected number of cases, and this occurred one year after a similar sharp increase in infant mortality which was some four times the level expected.

4.4 Conclusion: sources of radiation from the plant activities

Both infant mortality in the estuary wards and cancer rates in Burnham North remained high for some years. Again, it is hard to avoid the most likely explanation for all of these findings. That is that there was some exposure to a harmful mutagenic agent released in 1995 to the estuary of the River Parratt which affected individuals who lived in these wards. Since the only source of such an agent in the area known to us is the nuclear power station at Hinkley Point we tentatively conclude that there was a release of radioactivity in 1995, and that this is the cause both of the infant mortality and the subsequent cancer. Traces of such a release may yet remain in the environment e.g. in tree rings, mud cores, house dust etc.

There are three interesting possibilities in connection with Hinkley Point as a putative source.

(1) The period before 1999 when the main infant mortality effects were found also corresponds with the operation of the Hinkley Point A Magnox nuclear power station which reduced and finally halted operation in May 2000. Magnox stations have graphite cores cooled by Carbon Dioxide gas and release large amounts of radioactive noble gases, Krypton-85 and Argon-41. In fact the Hinkley A station released more radioactive noble gases in the period 1990-1997 than any other UK nuclear power station, roughly 3,000,000 GBq per annum (compare Hinkley B that releases about 40,000GBq/a) (UNSCEAR 2000 Table 31 p 246). This is a large amount of radioactivity. These dense radioactive noble gases are released upwind of Burnham on Sea and roll along the mud flats toward the town, dissolving in the muddy silt and in any non aqueous material to build up a reservoir. They will be inhaled as gases or adsorbed on particles. It may be significant that after the closure of Hinkley A the infant mortality rate downwind fell sharply and in Burnham on sea there were no infant deaths after Hinkley A closed and there were no more exposures to the high local levels of radioactive noble gases.

(2) Prior to 1995 there were three nuclear waste incinerators operating on the Hinkley site; they were PUP incinerators which had no filtration and were shut down in 1995 when activists were attempting to prevent a fourth large (filtered output) incinerator being built on the site to burn waste from Hinkley and also from the Trawsfynydd reactor in Wales which had just been closed down. At the time, (1995) one of the workers who was retiring contacted Jim Duffy of Stop Hinkley and told him that when waste was sent for

burning at the PUP incinerators only the outer drums were monitored. The inner drums contained intermediate level waste which was burned with the low level waste in the outer drums. This operative refused to allow his name to be used as he was concerned about his pension and would not go public for the same reason: however he was concerned about what was being done.

(3) There was a release of 2 tons of radioactive carbon dioxide and radioiodine vented to the atmosphere in two separate accidents in October 1994 (Burnham and Highbridge Weekly News 20 Oct 1995). Nuclear Electric, the operators, were fined £220,000 in a case brought by Her Majesty's Inspectorate of Pollution. Exposures would thus have been to parents in October 1994, effects would have been seen in those born 9-12 months later (due to the delay in effects on sperm development). These children, born in Oct 1995 would have been aged 0-1 in 1996 when the peaks in infant mortality occurred in the estuary wards.

So any of these circumstances may be relevant, and the release in late 1994 seems to line up with the increases in infant mortality in the area. We feel that it is not likely that both the infant mortality increases and cancer trends also both with time and with specific location, near a nuclear site and polluted environment are the result of chance, although this is conceivable.

Alternatively, it is fair to say that if there were evidence of some chemical exposure in 1995 which was severe enough to mainly affect Burnham on Sea but also cause problems in the other estuary wards, then this would have to be a possible explanation.

As we suggest in the title of this report, infant mortality is a valuable indicator of environmental pollution, and these results imply that it may be used to signal possible unreported historical pollution events which could then also be investigated in other ways. Access to ward level cancer incidence data, and also stillbirth data, both of which are presently restricted, would make the matter of ascribing causes to such ill health more easy and would therefore make it more difficult for industry to discharge harmful substances into the environment. The Policy Information Network on Child Health and Environment PINCHE (www.pinche.org) concluded that a routine computer-based real time monitoring of all ward level health data would be straightforward and would enable rapid determination of risk from pollution sources (van Den Hazel et al, 2006). Infant mortality would seem to be a valuable component of such a scheme.

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APPENDIX A
Wards used in the analysis

1993-1998	1999-2005
Cannington and Combwich	2Cannington and Quantocks
Central	2East Poldens
Dowsborough	2Bridgwater Eastover
East Poldens	2Bridgwater Hamp
Eastern Quantocks	2Huntspill and Pawlett
Eastover	2North Petherton
Hamp	2Puriton
Huntspill	2Bridgwater Quantock

Newton Green	2Sandford
North Petherton	2Bridgwater Sydenham
Parchey	2Bridgwater Victoria
Pawlett and Puriton	2West Poldens
Quantock	2Woolavington
Sandford	2Alcombe East
Sowey	2Alcombe West
Sydenham	2Aville Vale
Victoria	2Carhampton and Withycombe
West Poldens	2Crowcombe and Stogumber
Westonzoyland	2Dulverton and Brushford
Woolavington	2Minehead North
Alcombe	2Minehead South
Aville Vale	2Old Cleeve
Carhampton and Withycombe	2Porlock and District
Crowcombe and Stogumber	2Quantock Vale
Dulverton and Brushford	2Watchet
Dunster	2West Quantock
Holnicote	2Williton
Minehead North	2Burrow Hill
Minehead South	2Curry Rivel
Old Cleeve	2Islemoor
Porlock And Oare	2Langport and Huish
Quantock Vale	2Martock
Watchet	2Turn Hill
West Quantock	2Wessex
Williton	2Blackdown
Burrow Hill	2Neroche
Curry Rivel	2Dulverton and Brushford
Islemoor	2Quarme
Langport and Huish	2Bishop's Hull
Martock	2Bishop's Lydeard
Turn Hill	2Bradford-on-Tone
Wessex	2Comeytrowe
Blackdown	2Milverton and North Deane
Neroche	2Monument
Dulverton and Brushford	2Neroche
Exmoor	2Blackdown
Haddon	2North Curry
Quarme	2Norton Fitzwarren
Bishop's Hull	2Ruishton and Creech
Bishop's Lydeard	2Staplegrove
Bradford-on-Tone	2Stoke St. Gregory
Comeytrowe	2Taunton Fairwater
Milverton	2Taunton Halcon
Monument	2Taunton Blackbrook and Holway
Neroche	2Taunton Lyngford
Blackdown	2Taunton Manor and Wilton
North Curry	2Taunton Pyrland and Rowbarton
North Deane	2Trull
Norton Fitzwarren	2Wellington North

Ruishton and Creech	2Wellington Rockwell Green and
Staplegrove	2Wiveliscombe and West Deane
Stoke St. Gregory	2West Monkton
Taunton Fairwater	2Axbridge
Taunton Halcon	2Axe Vale
Taunton Holway	2Berrow
Taunton Lyngford	2Brent North
Taunton Manor	2Burnham North
Taunton Priory and Wilton	2Burnham South
Taunton Pyrland	2Cheddar and Shipham
Taunton Rowbarton	2Highbridge
Taunton Trinity	2Wedmore and Mark
Trull	2Avalon
Wellington North	2Glastonbury St Benedict's
Wellington Rockwell Green	2Glastonbury St Edmund's
Wellington South	2Glastonbury St John's
West Deane	2Glastonbury St Mary's
West Monkton	2Moor
Wiveliscombe	2Rodney and Priddy
Axbridge	2Street North
Axe Vale	2Street South
Berrow	2Wells Central
Brent	2Wells St Cuthbert's
Burnham North	2Wells St Thomas'
Burnham South	2Bridgewater bower
Cheddar	2Knoll
Highbridge	2Taunton killams and mountfield
Mark	2Wellington east
Shipham	2Illminster
Wedmore	2Knowle
Avalon	2Kings isle
Ebbor	2Street west
Glastonbury St. Benedict's	2St Cuthberts out N and West
Glastonbury St. Edmund's	2Hutton and Locking
Glastonbury St. John's	
Glastonbury St. Mary's	
Moor	
Rodney	
Sheppey	
Street North	
Street South	
Wells Central	
Wells St. Cuthbert's	
Wells St. Thomas	