Clustering of childhood asthma hospital admissions in New Zealand, 1999-2004

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ABSTRACT

The context for this study is public concern about aerial spraying of biological insecticides over Auckland, New Zealand between January 2002 and May 2004. We analysed childhood asthma hospital admissions for the whole of New Zealand, July 1999 – December 2004 using a spatial scan statistic. We found spatial clustering of asthma admissions in many New Zealand cities, and spatiotemporal clustering in a few cities. We hypothesize that many of the purely spatial clusters might be explained by characteristics of the local population or health services.

This explanation is less plausible in the case of the observed space-time clusters of asthma admissions, which we consider more likely to be related to local exposures. In spatiotemporal models, there were significant clusters in Auckland, Palmerston North, Lower Hutt, Christchurch and Invercargill. Two of the four Auckland clusters overlap biological insecticide spray zones, and two do not; the majority of the observed spatiotemporal clusters are unrelated to aerial spraying of biological insecticides in space and time. While the present results do not allow us to identify which local exposures are most relevant in explaining the observed spatiotemporal clusters, we hypothesize that air pollution, including fine particles of biological and non-biological origin, might play a role.

***Keywords and phrases*:** asthma, bioaerosol, spatiotemporal clustering, Satscan, *Bacillus thuringiensis*

1.0 INTRODUCTION

The detection and investigation of disease clusters has a long and controversial history in the field of spatial epidemiology. The basic interest in analyzing disease patterns is in determining whether the observed events exhibit any systematic pattern as opposed to being distributed at random over the study region. Disease clustering investigations may also help to reassure a community in which an apparent local excess of disease has been identified. Wartenberg (2001) suggests that public concern (often based on personal tragedy, perhaps with a specific point-source environmental contaminant in mind, but involving perhaps only a few cases), cannot and should not simply be dismissed as lying within acceptable statistical limits or explained away with demographic, statistical, or sampling error fluctuations. Reassurance is often required, which can be established via a carefully designed investigation.

Rothman (1990) has suggested that the payoff from clustering research comes from specific hypotheses that emerge to explain the observed pattern of excess occurrence. Whether infectious agents, genetic susceptibilities, or environmental pollutants, determining mechanisms is the goal, but only rarely have etiological insights resulted from cluster investigations. Disease clustering investigations may also be useful in actively identifying outbreaks, particularly for infectious diseases. There have been attempts to establish national active-cluster surveillance programs, which might regularly scan register-based data for evidence of elevated risk. However, investigating incidence data over a relatively large population systematically is costly and so most investigations are more passive and are often the result of an initial request from a member of the public. For example, in the United Kingdom, the Rapid Inquiry Facility developed by the Small Area Health Statistics Unit (Aylin et al., 1999) aims to produce a report within three days of a request by routinely collecting morbidity, mortality, and population data at a local spatial scale in anticipation of requests for an investigation.

The context for the present study is public concerns about aerial spraying of *Bacillus thuringiensis kurstaki (Btk)* over Auckland, New Zealand between January 2002 and May 2004. Biological insecticides have been widely used to control insect pests such as the Gypsy Moth. In New Zealand, *Bacillus thuringiensis* products *(Bt)* have been used to control several introduced species of insect in the past decade. Several studies of communities exposed to aerial spraying of *Bt* products have been carried out in New Zealand and North America and these have not shown any association between *Bt* exposure and health effects. However, all of these studies had limitations, including small sample sizes, potentially biased assessment of health effects and exposure, and limited duration of follow up (Hales, 2004).

We analysed childhood asthma hospital admissions for the whole of New Zealand, July 1999 – December 2004 using a spatial scan statistic (Kulldorff, 1998; 2002). This method has been used to test for disease clustering in a number of recent studies (Sabel et al., 2003; Sabel and Löytönen, 2004) and is a spatial, temporal, or spatiotemporal, local cluster detection method for aggregated data which can be applied to both focused and non-focused investigations and, importantly, can be adjusted for confounders, including adjusting for a heterogeneous background population density.

2.0 METHOD

Asthma admissions by date of admission and census area unit of residence for all of New Zealand (July 1999 – December 2004) were provided by NZ Health Information Service. There were 24380 admissions classified as asthma (ICD10 codes J450 – J460) in children aged 15 years and under. Of these, 6404 were discharged on the day of admission, 721 were not coded to a valid 2001 Census Area Units (CAUs), 408 were arranged admissions, and were excluded. This left 16847 cases (69%) which were included in subsequent analyses.

Cases discharged on the day of admission were excluded because of evidence that there was a change in reporting of day cases between 1996 and 2001 (NZHIS, personal communication). The latitude and longitude of the centroids of 1741 unique, populated 2001 New Zealand CAUs were calculated in a Geographic Information System. There were 1,775 CAUs and 36,808 Meshblocks in the 1996 census; 1,860 CAUs and 38,366 Meshblocks in the 2001 census, though not all of these had a resident population. There were subtle boundary changes between CAUs with the same name and code in 1996 and 2001 censuses, because meshblocks (from which CAUs are built) are developed for each census. This is to ensure that the population thresholds are maintained, and also to ensure that the workloads for the census enumerators is manageable. Boundary changes between 1996 and 2001 censuses meant that geocoding of admissions may not be exactly comparable between 1999-2001 and 2002-2004.

Exploratory Spatial Data Analysis (ESDA) can be broadly defined as the collection of techniques to describe and visualise spatial distributions, identify atypical locations, or spatial outliers, and discover patterns of spatial association, clusters or hotspots and suggest spatial regimes or other forms of spatial heterogeneity (Anselin, 1999). The main contribution of ESDA to GIS lies in visualising local patterns of spatial association. ESDA techniques are characterised by making few *a priori* assumptions about the data. Many such methods emphasise graphical views of the data, and hence there is a natural close affinity with geographic visualisation. Importance is attached to 'staying close to the original data' in the sense of using simple, intuitive methods. Exploratory approaches are used to *suggest* hypotheses about the data and generate theory, and thus cannot be used to test hypotheses.

Retrospective spatial and space-time analyses of childhood asthma (under age 15 years) were conducted in SaTScan (Kulldorff, 1998). The method imposes a circular scanning window on the map and lets the centre of the circle move over the study area so that at each position the window includes different sets of neighbouring administrative areas. For each circle centroid, the radius varies continuously from zero to a user-defined maximum. Although the choice of maximum cluster circle size is somewhat arbitrary, and there are no clear guidelines for its choice, it is important to make the choice of maximum cluster size *a priori* to avoid the problems of multiple hypothesis testing. The test statistic adopted is the likelihood ratio, which is maximized over all the windows to identify the most likely disease clusters. Unlike some other techniques such as Openshaw's GAM (Openshaw et al, 1987), the test statistic does take into account the problem of multiple hypothesis testing and reports the significance of each reported cluster. For the purely spatial analyses, we used a spatial window of 5km radius, and the 2001 usually resident census populations aged 0-14 as the denominator. We conducted separate analyses using data for July 1999 – December 2001 (pre spray) and January 2002 – December 2004 (post spray). For the spatiotemporal analyses, we used the whole dataset (July 1999 – December 2004) a spatial window of 5km radius and a time window of 3 months (decided *a priori*).

3.0 RESULTS

In spatial analyses using data for July 1999 – December 2001 (pre spray) 36 statistically significant space-time clusters were returned (Table 1), including many in urban areas (Figure 1) and 5 in the Auckland region (Figure 2).

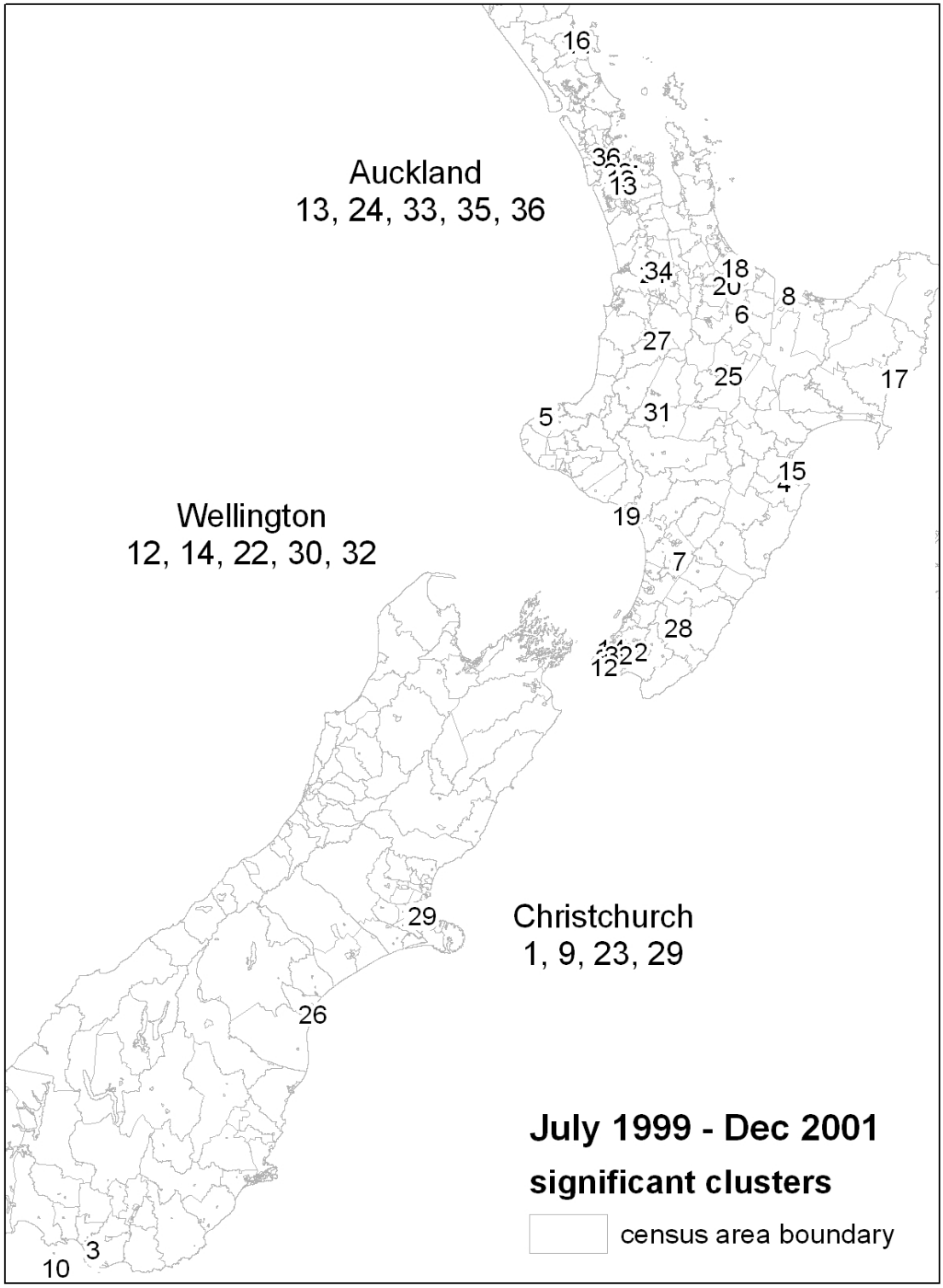

Figure 1

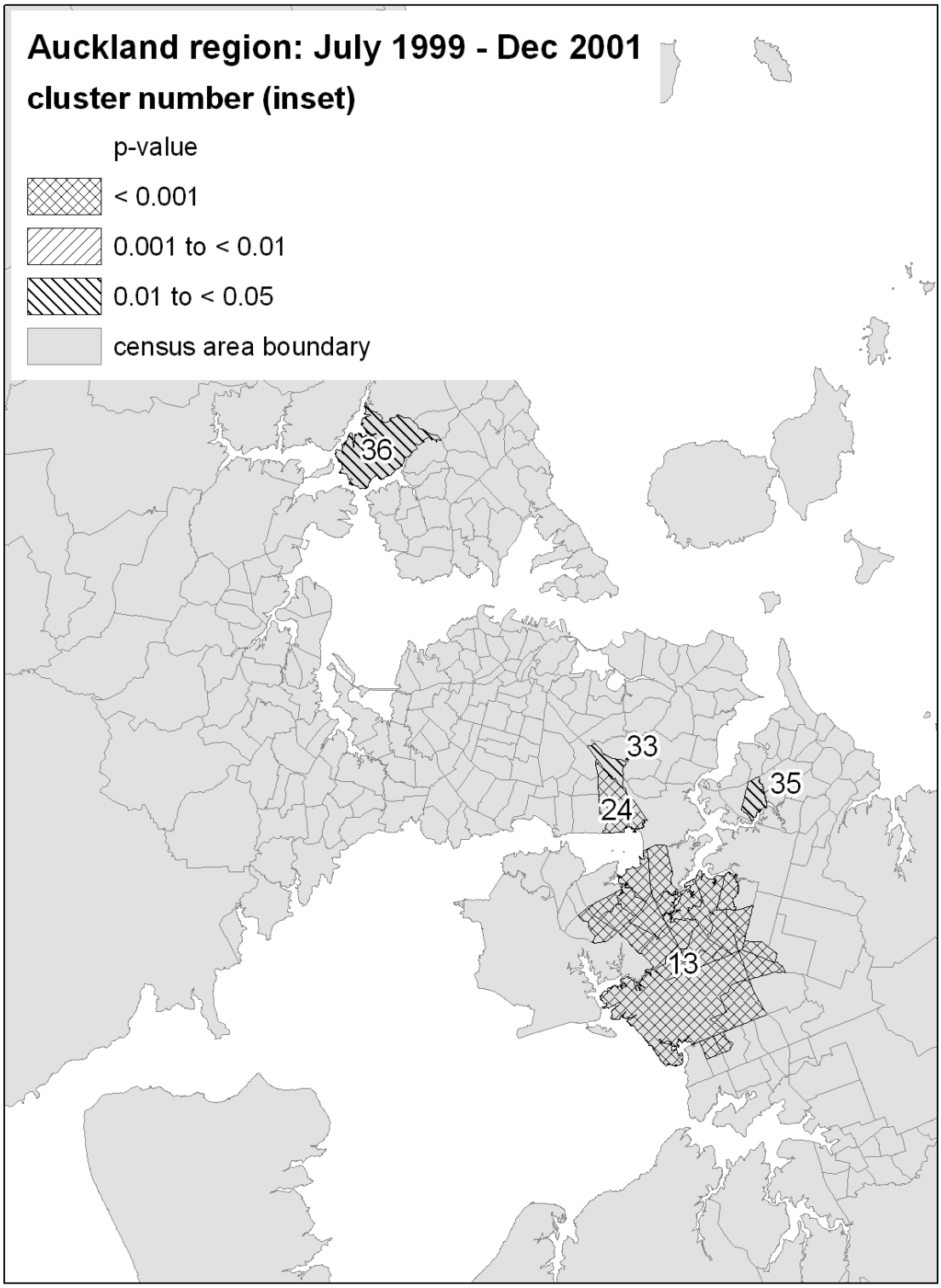
Auckland region: July 1999 - Dec 2001
cluster number (inset)
p-value
< 0.001
0.001 to < 0.01
0.01 to < 0.05
census area boundary
36
33
24
35
13

Figure 2

In spatial analyses using data for January 2002 – December 2004 (post spray) 28 statistically significant space-time clusters were returned (Table 2), including many in urban areas (Figure 3) and 6 in the Auckland region (Figure 4). One of these is a significant cluster near the spray zone that was not seen in the July 1999 – December 2001 data. One cluster (number 36) that was present in July 1999 – December 2001 has disappeared.

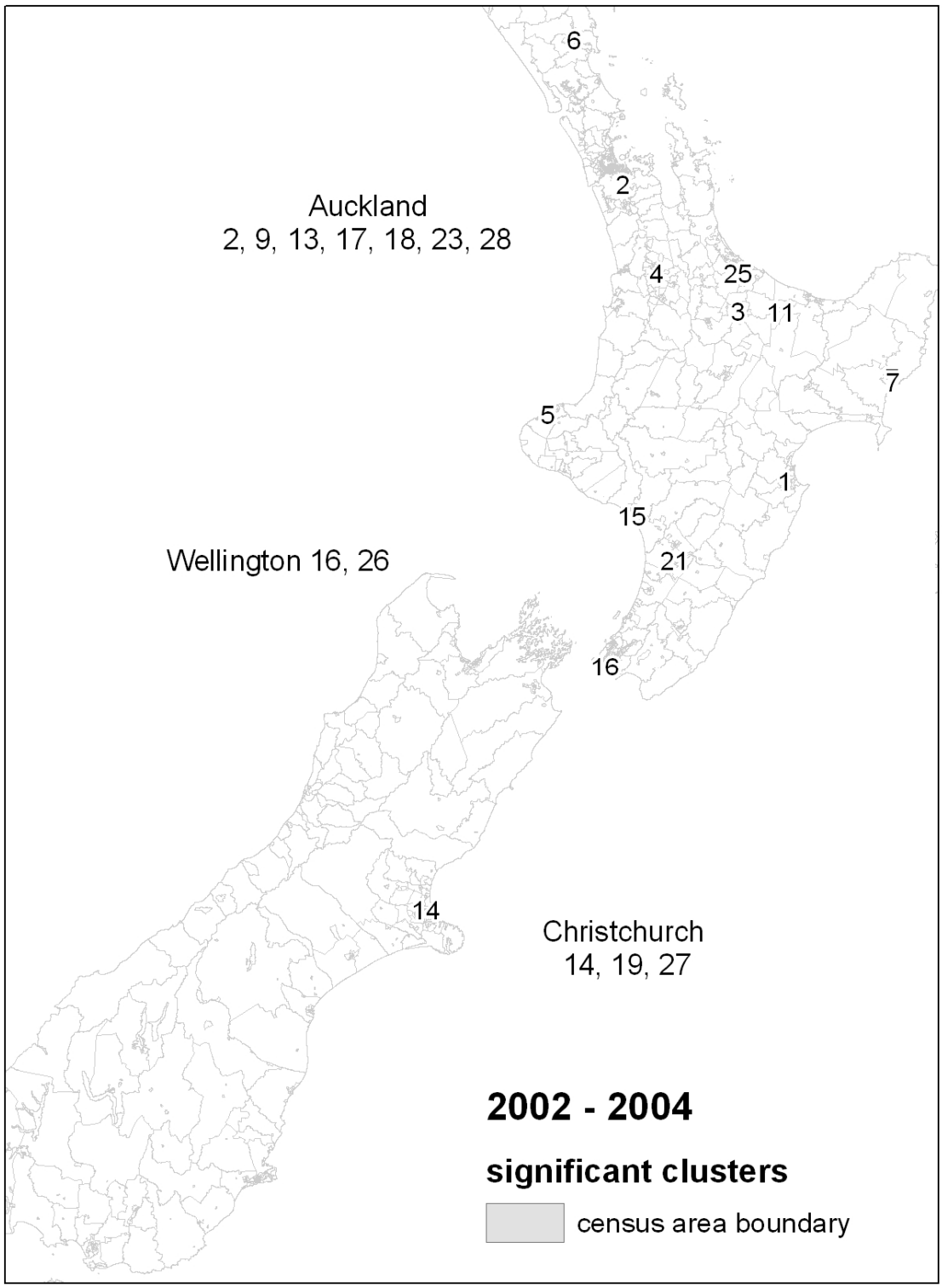

Figure 3

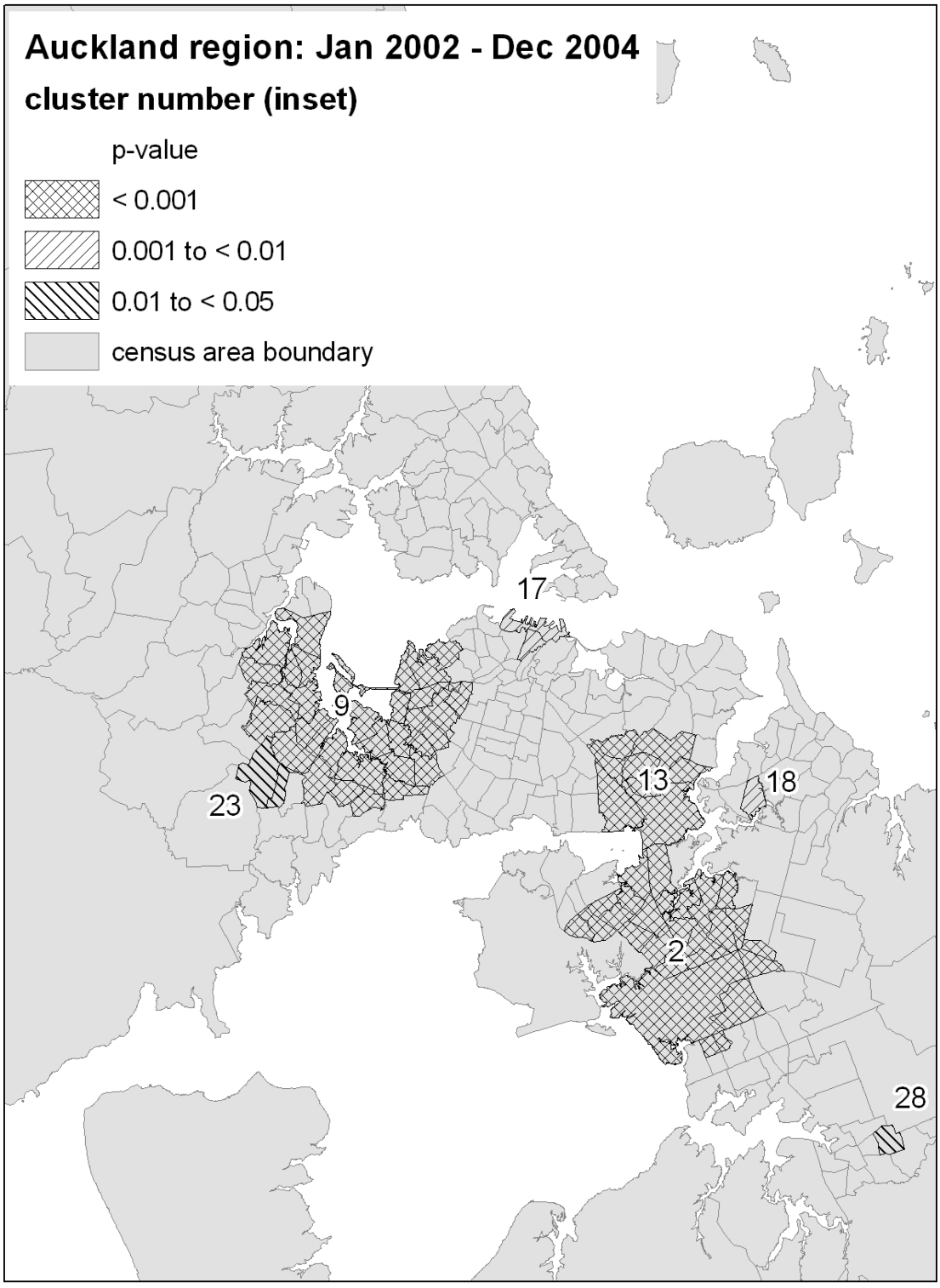
Auckland region: Jan 2002 - Dec 2004
cluster number (inset)
p-value
< 0.001
0.001 to < 0.01
0.01 to < 0.05
census area boundary
17
9
23
13
18
2
28

Figure 4

In spatiotemporal models there were significant clusters in Auckland (Figure 5), Palmerston North, Lower Hutt, Christchurch and Invercargill (not shown). Two of the four Auckland clusters overlap the spray zones, and two do not. All Auckland spatiotemporal clusters occur in later years (Table 3).

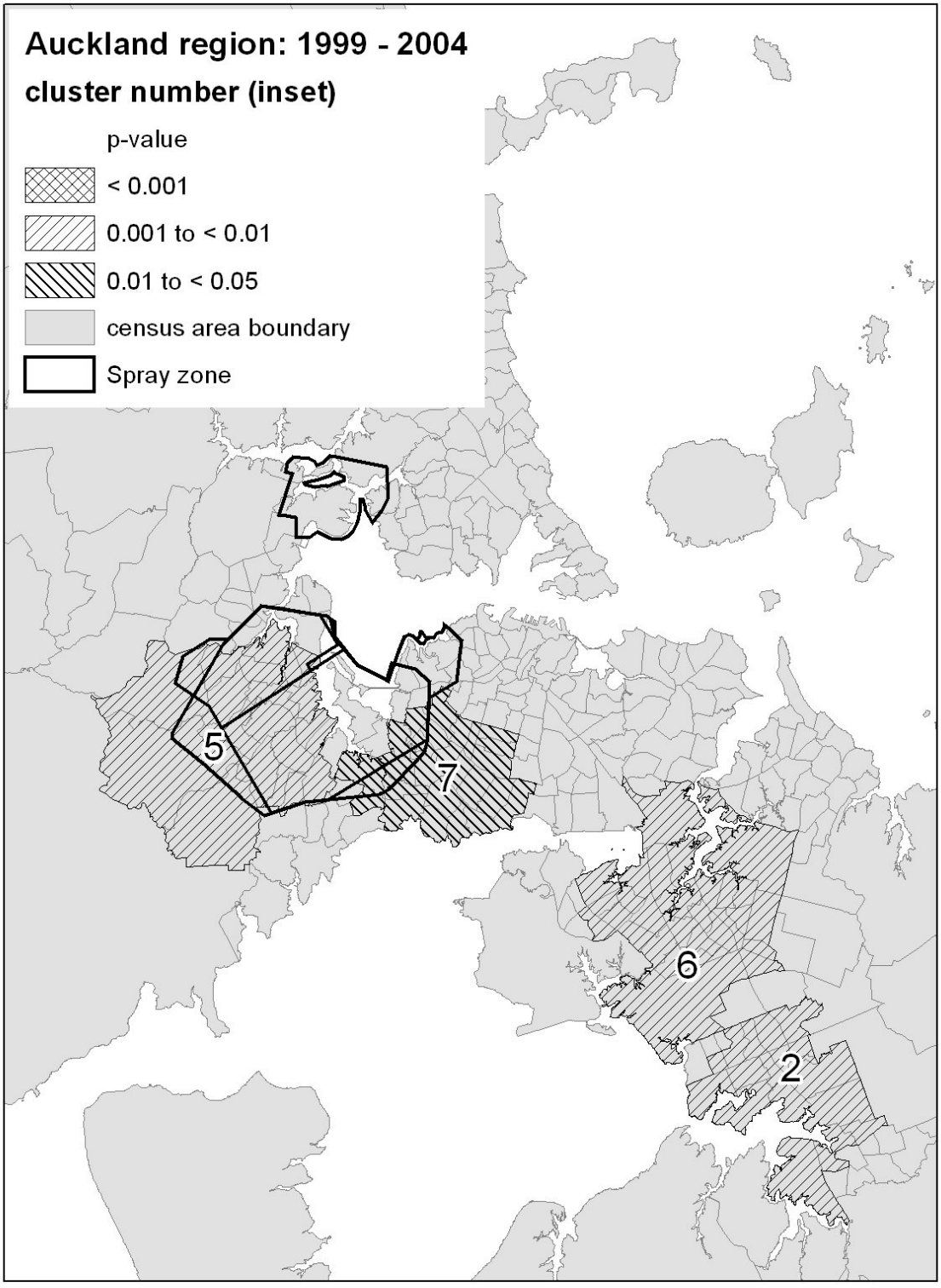

Figure 5

4.0 DISCUSSION

Several purely spatial clusters of asthma admissions persist between 1999 – 2001 and 2002 –2004. These might be related to characteristics of health services. For example, children living in cities may be more likely to go directly to a hospital with acute asthma, rather than to a primary care service. People attending a hospital rather than a primary care service may then be more likely to be admitted. Alternatively, characteristics of the local population, including social, cultural and genetic factors may affect the prevalence of asthma symptoms. Many of these potential confounders could be controlled for using individual or area level covariates such as smoking prevalence, social status, distance to the nearest general practitioner and distance to the nearest hospital.

Because they generally do not change over time scales of months to years, the above factors cannot so readily explain changes in the observed spatial clusters between the two time periods studied, or the observed spatiotemporal clusters. Access to health services might change, for example if a new hospital opened or a primary care service closed. However, such events are unlikely to explain many of the spatiotemporal trends reported here. These seem more likely to be related to environmental exposures, though which exposures may be most relevant is not clear. There are a range of environmental exposures which probably interact to affect asthma severity, including respiratory viruses, aero allergens and air pollutants.

The observed spatiotemporal clusters include several urban areas with low to moderate levels of ambient air pollution (Auckland, Palmerston North, Lower Hutt, Christchurch and Invercargill; see Figure 5 and Table 3). Short term effects of urban air pollution on asthma severity have long been suspected and are now widely considered to be causal (WHO, 2005). Many time series studies of air pollution and asthma admissions or emergency room visits have been published, but the results are inconsistent with respect to the pollutants involved and the size of the effects (Burney, 1999; WHO, 2005). Exposure to inhaled biological material may also play an important role in asthma epidemiology, as suggested by toxicological evidence (eg, Becker et al, 2005) and a large number of studies conducted in the occupational and residential indoor environment (Douwes et al, 2003). However, there is little epidemiological evidence that biological agents in the outdoor environment might play such a role.

The majority of the observed spatiotemporal clusters are unrelated to aerial spraying of biological insecticides in space and time. Nevertheless, the hypothesis that chronic exposure to biological insecticides may lead to asthma exacerbations deserves further study. Controlled studies of populations exposed to aerial spraying of *Bt* have not demonstrated short term health effects (WHO, 1999; Hales, 2004). However, these studies were not designed to detect potential long term effects of chronic exposure. A small proportion of *Btk* is present as respirable particles for a few days following aerial spraying (Teschke et al; 2001); *Bt* spores are of respirable size; may persist in the environment and subsequently be released or resuspended. These issues could be investigated further by environmental sampling for *Bt*; analyses of PM_{10} from urban monitors before, during and after aerial spraying operations; and toxicological testing of historical PM_{10} samples.

Many studies examining associations between geographical patterns of disease and causal factors assume that current residence in an area can be equated with exposure to conditions that currently (and historically) pertain there. Yet, people move and hence previous exposure to causative agents will not be taken into account. The problems will be greater for diseases that have a long lag or latency period. By adopting only the current residential address, the daily activity spaces of the children is ignored, despite the fact that exposure at school or while travelling might be important. These issues could be addressed by following up children involved in the clusters of asthma admissions reported here, and using questionnaires or GIS-based models to assess potential exposures and confounding factors.

5.0 CONCLUSIONS

We report clustering of asthma admissions in New Zealand cities. Many of the purely spatial clusters might be explained by characteristics of the local population or health services. This explanation is less plausible in the case of the observed space-time clusters, which are more likely to be related to local exposures. The present results do not allow us to identify which local exposures are most relevant. We hypothesize that the purely spatial clusters might be explained by characteristics of the local population or health services, and that the spatiotemporal clusters might be explained by air pollution exposures, including fine particles of biological and non-biological origin.

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Cluster	Latitude	Longitude	Radius	Observed	Expected	ODE	LLR	p-value	Start Date	End Date
1	-43.5283	172.4625	0.00	51	4.12	12.36	81.522896	0.00010	1999/7/1	2001/12/31
2	-37.8052	175.3249	0.00	46	4.82	9.55	62.723629	0.00010	1999/7/1	2001/12/31
3	-46.4206	168.3923	3.51	162	60.03	2.70	59.523117	0.00010	1999/7/1	2001/12/31
4	-39.6274	176.8208	3.87	204	91.04	2.24	52.456490	0.00010	1999/7/1	2001/12/31
5	-39.0753	174.0481	3.64	157	62.74	2.50	50.318439	0.00010	1999/7/1	2001/12/31
6	-38.1227	176.2382	2.76	129	48.32	2.67	46.411400	0.00010	1999/7/1	2001/12/31
7	-40.3229	175.6117	4.65	164	78.31	2.09	36.015522	0.00010	1999/7/1	2001/12/31
8	-37.9547	176.7635	0.00	42	9.18	4.57	31.106966	0.00010	1999/7/1	2001/12/31
9	-43.5281	172.6722	3.70	256	151.47	1.69	30.533157	0.00010	1999/7/1	2001/12/31
10	-46.9855	167.8586	0.00	11	0.33	33.22	27.871584	0.00010	1999/7/1	2001/12/31
11	-35.7664	174.3632	0.00	27	4.40	6.14	26.439777	0.00010	1999/7/1	2001/12/31
12	-41.3226	174.7721	1.69	72	28.81	2.50	22.874680	0.00010	1999/7/1	2001/12/31
13	-36.9867	174.8473	4.47	375	261.63	1.43	22.482533	0.00010	1999/7/1	2001/12/31
14	-41.1424	174.8540	0.00	27	5.54	4.87	21.334721	0.00010	1999/7/1	2001/12/31
15	-39.5131	176.8933	2.30	112	57.80	1.94	20.074102	0.00010	1999/7/1	2001/12/31
16	-35.7287	174.3264	0.00	7	0.15	46.50	20.029926	0.00010	1999/7/1	2001/12/31
17	-38.6640	177.9878	4.61	108	55.61	1.94	19.476803	0.00010	1999/7/1	2001/12/31
18	-37.7184	176.1790	3.83	112	58.74	1.91	19.207069	0.00010	1999/7/1	2001/12/31
19	-39.9380	175.0654	0.00	13	1.20	10.80	19.141226	0.00010	1999/7/1	2001/12/31
20	-37.8736	176.0695	0.00	38	12.34	3.08	17.114592	0.00010	1999/7/1	2001/12/31
21	-37.8007	175.2437	3.43	147	88.45	1.66	16.345064	0.00010	1999/7/1	2001/12/31
22	-41.1576	175.1175	0.00	19	3.61	5.26	16.167156	0.00010	1999/7/1	2001/12/31
23	-43.5528	172.5528	1.94	36	12.28	2.93	15.028983	0.00010	1999/7/1	2001/12/31
24	-36.9188	174.8155	0.00	10	0.96	10.38	14.367246	0.00020	1999/7/1	2001/12/31
25	-38.6884	176.1190	0.00	3	0.01	298.94	14.111320	0.00030	1999/7/1	2001/12/31
26	-44.3951	171.2503	1.03	28	8.64	3.24	13.585127	0.00050	1999/7/1	2001/12/31
27	-38.3911	175.2953	0.00	14	2.44	5.74	12.913864	0.00090	1999/7/1	2001/12/31
28	-40.9424	175.6765	3.38	60	29.17	2.06	12.499754	0.00180	1999/7/1	2001/12/31
29	-43.5441	172.5927	1.51	48	21.23	2.26	12.439808	0.00190	1999/7/1	2001/12/31
30	-41.2241	174.8374	0.00	9	0.96	9.34	12.078049	0.00240	1999/7/1	2001/12/31
31	-38.8935	175.3217	0.00	11	1.57	7.03	12.017535	0.00280	1999/7/1	2001/12/31
32	-41.2043	174.9684	2.20	50	24.33	2.06	10.392016	0.01440	1999/7/1	2001/12/31
33	-36.9017	174.8114	0.00	13	2.65	4.91	10.334351	0.01530	1999/7/1	2001/12/31
34	-37.7575	175.2885	0.73	28	10.42	2.69	10.122437	0.01790	1999/7/1	2001/12/31
35	-36.9150	174.8882	0.00	27	10.27	2.63	9.392354	0.03650	1999/7/1	2001/12/31
36	-36.7690	174.6860	0.00	28	10.93	2.56	9.290121	0.04070	1999/7/1	2001/12/31

Table 1 Spatial analysis, 1999 - 2001, 5 km window[1].

Cluster	Latitude	Longitude	Radius	Observed	Expected	ODE	LLR	p-value	Start Date	End Date
1	-39.6274	176.8208	4.53	296	115.12	2.57	100.506516	0.00010	2002/1/1	2004/12/31
2	-36.9867	174.8473	4.72	554	311.72	1.78	79.707184	0.00010	2002/1/1	2004/12/31
3	-38.1088	176.2369	4.54	186	76.25	2.44	56.785938	0.00010	2002/1/1	2004/12/31
4	-37.7840	175.2766	0.00	28	2.28	12.26	44.491535	0.00010	2002/1/1	2004/12/31
5	-39.0569	174.0752	0.61	32	3.29	9.73	44.146606	0.00010	2002/1/1	2004/12/31
6	-35.7287	174.3264	0.00	11	0.17	63.56	34.851479	0.00010	2002/1/1	2004/12/31
7	-38.6640	177.9878	4.61	137	63.93	2.14	31.649931	0.00010	2002/1/1	2004/12/31
8	-37.8052	175.3249	0.00	33	5.54	5.96	31.480598	0.00010	2002/1/1	2004/12/31
9	-36.8792	174.6757	4.99	444	300.16	1.48	31.183571	0.00010	2002/1/1	2004/12/31
10	-35.6950	174.3325	2.14	80	37.04	2.16	18.750387	0.00010	2002/1/1	2004/12/31
11	-38.0911	176.6875	0.00	59	23.88	2.47	18.309908	0.00010	2002/1/1	2004/12/31
12	-39.5062	176.9156	0.00	25	5.57	4.49	18.118763	0.00010	2002/1/1	2004/12/31
13	-36.9111	174.8313	2.34	119	65.45	1.82	17.751051	0.00010	2002/1/1	2004/12/31
14	-43.5185	172.6898	4.27	287	200.48	1.43	16.874004	0.00010	2002/1/1	2004/12/31
15	-39.9380	175.0654	1.77	30	8.48	3.54	16.410063	0.00010	2002/1/1	2004/12/31
16	-41.2935	174.7786	1.15	33	11.04	2.99	14.198342	0.00040	2002/1/1	2004/12/31
17	-36.8440	174.7701	0.78	13	2.01	6.48	13.298776	0.00120	2002/1/1	2004/12/31
18	-36.9150	174.8882	0.00	32	11.80	2.71	11.742025	0.00460	2002/1/1	2004/12/31
19	-43.6354	171.6486	0.00	13	2.39	5.44	11.421433	0.00650	2002/1/1	2004/12/31
20	-35.7664	174.3632	0.00	19	5.05	3.76	11.227228	0.00780	2002/1/1	2004/12/31
21	-40.3374	175.5680	4.04	99	60.05	1.65	10.627172	0.01220	2002/1/1	2004/12/31
22	-37.9529	176.9925	1.19	26	9.07	2.87	10.469910	0.01450	2002/1/1	2004/12/31
23	-36.9090	174.6264	1.12	50	24.85	2.01	9.841539	0.02650	2002/1/1	2004/12/31
24	-37.6745	175.1543	0.00	37	16.34	2.26	9.607464	0.03190	2002/1/1	2004/12/31
25	-37.7435	176.1600	3.77	99	61.96	1.60	9.433177	0.03780	2002/1/1	2004/12/31
26	-41.1424	174.8540	0.00	20	6.37	3.14	9.265679	0.04440	2002/1/1	2004/12/31
27	-43.5348	172.6372	0.00	6	0.52	11.56	9.204320	0.04640	2002/1/1	2004/12/31
28	-37.0598	174.9643	0.00	36	16.03	2.25	9.183624	0.04690	2002/1/1	2004/12/31

Table 2 Spatial analysis, 2002-2004, 5 km window[1].

Cluster	Latitude	Longitude	Radius	Observed	Expected	ODE	Test Stat	p-value	Start Date	End Date
1	-40.3229	175.6117	4.81	101	55.43	1.82	15.094843	0.00200	1999/6/1	2000/6/30
2	-37.0441	174.9105	3.92	184	119.63	1.54	14.970526	0.00200	2003/4/1	2004/12/31
3	-41.1912	174.9932	4.10	65	30.57	2.13	14.642400	0.00200	1999/7/1	2000/6/30
4	-46.4299	168.3675	3.31	123	73.29	1.68	14.051414	0.00400	1999/7/1	2001/3/31
5	-36.8918	174.6115	4.71	207	140.08	1.48	14.049068	0.00400	2003/1/1	2004/9/30
6	-36.9596	174.8540	4.88	408	313.80	1.30	13.174249	0.00600	2002/10/1	2004/6/30
7	-36.9177	174.7236	4.04	48	21.39	2.24	12.208545	0.01400	2004/10/1	2004/12/31
8	-43.4945	172.5615	3.25	24	7.36	3.26	11.726136	0.02400	2003/10/1	2003/12/31

Table 3 Spatiotemporal analysis, 1999-2004, 5km spatial window; 3 month time window[1]

[1] ODE: observed/expected; Test Stat: test statistic; LLR: log likelihood ratio