February 25, 1999

Dear Concerned Citizen:

The enclosed document from the Washington State Department of Health and the Seattle-King County Department of Public Health contains preliminary reports from our investigation into community health concerns around SeaTac International Airport.

Based on the August 1998 work plan outlined in cooperation with community representatives, these reports address some of the preliminary questions and are, therefore, not conclusive. They do not represent the final results of our investigation. The document enclosed with this letter includes the following five parts:

1. **Progress Report**. This addresses items in the August 1998 work plan.
2. **Cancer Incidence Report**. This is a report analyzing cancer incidence data (work plan questions 1 and 2).
3. **Literature Review**. This covers the risk factors for glioblastoma (work plan question 7).
4. **Jet Engine Emissions Review**. This is a review of the constituents of jet engine emissions (work plan question 8).
5. **Community Health Assessment**. This is an overview of health status that was requested by SeaTac-area representatives (work plan question 10, part 1).

State health department researchers looked at the occurrence of more than 25 categories of cancer between 1992 and 1996 in areas within one mile, three miles and five miles of SeaTac Airport. We found that the 10 most prevalent cancers around the airport were consistent with the 10 most prevalent cancers in both King County and in Washington State as a whole. Important findings regarding cancers of specific interest to some SeaTac-area residents were:

- Glioblastoma showed a statistically significant elevation in only one of the zones around the airport and in only one year – 1992 – for the study period 1992 through 1996. The numbers of people diagnosed with glioblastoma from 1993 through 1996 were within expected range. Based on these data, the elevation in glioblastoma appears to have been a past event that has since resolved, but we need to look at some additional years before drawing final conclusions.
- All leukemia, including the specific category of acute myeloid leukemia (AML), was not elevated around the airport.
- Breast cancer was not elevated in any of the three areas around the airport.

Addressing Community Health Concerns around SeaTac Airport
Progress Report on the Work Plan Proposed in August 1998

February 25, 1999

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Background

In response to community concerns about health around SeaTac International Airport, Senator Julia Patterson arranged meetings with community residents, the Washington State Department of Health (DOH), the Seattle King County Department of Public Health (SKCDPH) and other interested parties. As a result of these meetings and preliminary DOH findings related to glioblastoma rates in an area approximately 3 miles around the airport, Senator Patterson requested that DOH work with the SKCDPH and the community to develop a work plan addressing the community's concerns.

Community representatives presented a list of 18 questions they wanted addressed in the work plan. The work plan was divided into two phases. Phase 1 activities addressed 10 questions. Answers to the questions in Phase 1 will be necessary in determining the value and feasibility of proceeding to the remaining questions included in Phase 2.

DOH and SKCDPH issued a brief progress letter regarding the work plan on November 20, 1998. This progress report is being issued in conjunction with reports on questions 1, 2, 7, 8 and 10 (part 1). It addresses progress on the remaining questions in Phase 1 of the original work plan. Since Phase 2 requires completion of Phase 1, this progress report will not address Phase 2 activities.

Status, Progress and Revised Timeline for Phase One Questions

Questions One - Six: Descriptive Epidemiology

1. What types of cancer are the most prevalent in the proximity of the airport, and what are their risk factors?
2. Are rates of breast cancer elevated in the proximity of the airport?

Status of investigation for questions 1 and 2: completed. (Please see written report issued February 25, 1999.)

3. Do we know of all cases of glioblastoma in the proximity of the airport?
4. Can we confirm that all of the suspected cases of glioblastoma have been properly diagnosed as such?

Status of investigation for questions 3 and 4: ongoing.

Progress to date: As was recommended in the original work plan, we are combining information from available registries and community reports to identify glioblastoma among people living or working in the area around SeaTac Airport. At the meeting on August 10, 1998, we agreed to include people who were diagnosed with glioblastoma in 1985 or later and who lived within 3 miles of the airport at the time of diagnosis. We also agreed to include people with glioblastoma who lived in the 1990-1993 buyout area whether or not they were living there at the time of diagnosis. We agreed to try to identify cases among people who worked at SeaTac Airport.

Community Reports

We have received 34 reports of suspected cases of glioblastoma and other brain tumors from the community, including individuals who called the Washington State Department of Health and lists compiled by KIRO TV and by community members. The Table 1 shows the follow up status of those reports.

Table 1. Community Reports of Brain Tumors

Number (%)	Status	Comments
2 (6%)	Confirmed in Washington State Cancer Registry as brain tumors	Diagnosed 1992 – 1996; included in analyses.
3 (9%)	Confirmed in Washington State Cancer Registry as another type of cancer	Diagnosed 1992 – 1996; other cancers may spread to the brain, but they are not then counted as brain tumors.
10 (29%)	Need to confirm through cancer registry at Fred Hutchinson Cancer Research Center	Diagnosed before 1992.
8 (24%)	Will confirm through Washington State Cancer Registry	Diagnosed after 1996.
7 (21%)	Attempting to obtain sufficient information for follow-up	
4 (12%)	Unable to follow-up	Cannot obtain names.
TOTAL: 34		

Buyout Area

We have tried to find people with cancer among those who left the SeaTac area between 1990 and 1993 during the buyout by the Port of Seattle. Based on address information provided by community members, we sent packets to people who had lived in the buyout area in September 1998. The packets included a letter explaining why we were contacting them, forms requesting information on the number of people who had lived in the house and whether any of those people had cancer, and a postage-paid return envelope. In November 1998, we sent another letter requesting the same information to those who had not responded to our first mailing. The Table 2 shows the results of those mailings.

Table 2. Properties in Buyout Area

Number (%)	Status	Comments
36 (39%)	Names returned to community for additional address information	Insufficient initial contact information (20); Packets returned as undeliverable (16)
22 (24%)	Non-response after two mailings	Follow-up phone calls needed
25 (27%)	Responded, but did not report cancer	
9 (10%)	Responded and reported cancer	Follow-up of cases before 1992 or after 1996 needed
TOTAL: 92		

The nine households reported 12 people with cancer. Two of those people were diagnosed from 1992 through 1996. Of these people, one was in the Washington State Cancer Registry with the appropriate buyout area address and was already included our analyses. One individual was diagnosed after being bought out, so we changed our database to use this person's pre-buyout address. We will follow the same procedures for confirmation of the remaining 10 people diagnosed with cancer before 1992 or after 1996 as described in the previous table for community case reports.

Employment at SeaTac Airport

Finally, a letter was sent to 111 businesses located around SeaTac Airport in November 1998 asking for information regarding cancer among any of their employees from 1985 until the present. To date, we have received responses from 14 (13%) businesses. Of those, 3 (21%) reported known cases of cancer during the time period of interest.

Given the poor response rate, we recommend a different approach to this aspect of the project. Any businesses or employees with specific concerns may contact the Washington State Department of Labor and Industries at 360-902-5800 to file a complaint or request consultation. They may also contact the National Institute for Occupational Safety and Health (NIOSH) at 800-356-4674 for information on the Health Hazard Evaluation Program or to speak to a technical representative

<u>Anticipated Timeline</u>

Continue effort to verify information and confirm diagnosis: March 1999 - May 1999
Medical records review (if necessary): June 1999 - July 1999
Prepare report: August 1999

Report due: September 1999

5. Are incidence rates of glioblastoma elevated in the area west of the airport?

Status of investigation: ongoing.

Progress to date: People identified with glioblastoma through the Washington State Cancer Registry were mapped according to their residence when diagnosed for the years 1992 through 1996. One additional person was added based on responses from people who had lived in the buyout area. These cases appear to be randomly distributed with no evidence of clustering in the area west of the airport (Appendix A, Map 1). Please note that the diamonds on the map indicating people with glioblastoma have been randomized to within 1/8 mile to protect confidentiality. While the identity of a person might be obvious to those who know the person, it is not possible to learn the identity of a person from this map. A map of population densities in the same area (Appendix A, Map 2) indicates that the population density west of the airport may be somewhat less than immediately east of the airport. Formal geospatial analysis is required to determine whether the rates east and west of the airport are the same. We have postponed this analysis until we finish case identification as outlined above for questions 3 and 4.

Anticipated Timeline
Collect additional data for analysis: March 1999 - May 1999
Provide completed list to geospatial analysis experts: June 1999
Conduct geo-spatial analyses of rates of glioblastoma: July 1999 - August 1999
Prepare report: September 1999

6. Is this elevation in glioblastoma incidence rates continuing presently, or did it only occur in the past?

Status of investigation: ongoing.

Progress to date: For the period 1992 through 1996, analyses for questions 1 and 2 indicate that elevated rates of glioblastoma occurred within a 3-mile radius of the airport in 1992. (Please see Appendix B.) Cases of glioblastoma subsequently declined and were within expected ranges for the years 1993 through 1996. We have requested and are awaiting data for years before 1992 from the Fred Hutchinson Cancer Research Center; data for 1997 should soon be available from the Washington State Cancer Registry. We can look at the number of cases per year, but developing rates necessary to account for changes in the number of people living in the area may not be feasible for all years. (We have thus far been unable to obtain population data before 1990 by zipcode or census block.) After we have gathered the additional data and looked at the number of cases, we will determine whether to proceed with analyses of glioblastoma rates over time.

Anticipated Timeline
Receive data from Fred Hutchinson Cancer Research Center: March 1999
Receive data from Washington State Cancer Registry: April 1999
Develop data from cancer registries: March 1999 - May 1999
Develop appropriate census data for years of interest: May 1999 - June 1999
Conduct analyses: July 1999 - August 1999
Prepare report: September 1999

Questions Seven and Eight: Literature Review

7. What are the risk factors for glioblastoma?

Status of question 7: completed. (Please see written report issued February 25, 1999.)

8. What are the chemicals in jet engine exhaust emissions, and what happens to them after they are emitted?

Status of question 8: completed. (Please see written report issued February 25, 1999.)

Questions Nine and Ten: Potential Field Studies

9. Is it possible to monitor jet engine exhaust emissions, or to model their path using data on prevailing winds and takeoff patterns?

Status of investigation: ongoing.

The DOH is in the process of collecting and reviewing studies relevant to health and air quality issues at or near SeaTac International Airport. The focus of this search is twofold. The first is to locate information on past or current monitoring efforts of air quality in the SeaTac area. Once this is collected, the data will be reviewed to determine health implications. Collection and review of these data are likely to be completed by September 1999. The second effort will be to review models used in predicting air quality, specifically for toxic air pollutants. If it is decided that air modeling is to occur, these efforts may require up to 18 months. It should be noted that similar efforts are underway at Boeing Field/Georgetown.

A list of studies to be reviewed by DOH is provided in Appendix C. DOH requests any additional information or studies related to air quality around the SeaTac Airport.

<u>Anticipated Timeline</u>
Complete collection and review of data specific to air monitoring around SeaTac Airport: September 1999
Decision on usefulness of modeling flight path emissions: December 1999

10. Are there other important health problems, such as respiratory disease, in this community, particularly in schools located under the flight path?

<u>Part 1. Previously collected data</u>

<u>Status of investigation for part 1 of question 10</u>: completed. (Please see written report issued February 25, 1999.)

<u>Part 2. Field study</u>

<u>Status of investigation</u>: ongoing.

SKCDPH analyzed previously collected mortality and hospitalization data in response to Part 1 of this question. As noted in the original work plan, the usefulness of collecting new health data depended, in part, on information developed in answering question 9. Since question 9 has not been completed, the plan for answering this question remains unchanged. The original work plan included three steps for this question:

1. Assess the usefulness of conducting a field study based on information developed in question 9 regarding the feasibility of monitoring and modeling jet engine exhaust emissions.
2. If useful, narrow the focus of the study, assess feasibility and cost, and determine roles for agencies and the community.
3. If feasible and affordable, conduct a study.

<u>Anticipated Timeline</u>
Decision on usefulness of field study: December 1999

APPENDIX A

Map 1: Glioblastoma Cases around SeaTac International Airport, 1992-1996

Map 2: Population Density around SeaTac International Airport, 1990

APPENDIX B

Graph of Glioblastoma cases by year, 1992-1996

APPENDIX C

Table of References: Monitoring & Modeling of Air Quality

The Seattle-King County health department and the state health department also did an extensive literature review of the risk factors for glioblastoma. Although several risk factors have been explored in the literature, at this time researchers have not identified any clear causes of the disease in people.

A review of the risk factors for glioblastoma. Although several risk factors have been explored in the literature, at this time researchers have not identified any clear causes of the disease in people.

A review of the constituents of jet engine emissions by the state health department found that jet exhaust emissions can be significant sources of ground-level volatile organic compounds (primarily benzene, 1,3-butadiene, and formaldehyde), carbon monoxide, and nitrogen oxides. To date, no pollutant unique to aircraft emissions has been identified. In fact, cars and trucks also release the same pollutants as airplanes.

At the community's request, Seattle-King County health department conducted a general community health assessment. A broad range of health outcomes, including deaths, hospitalizations, physician reports and behavioral risks were examined. This assessment showed:

- Death rates for lung cancer and chronic obstructive pulmonary disease were higher in the SeaTac Airport community compared to King County as a whole. Cigarette smoking likely causes the majority of deaths from these causes.
- Late entry into prenatal care as well as rates of smoking was more common among mothers giving birth in the SeaTac Airport community.
- Hospital admissions for respiratory disease, including asthma, in children under 18 years old were elevated, although they were significantly lower than the highest asthma hospitalization rates in the county, which are found in Central and Southeast Seattle.
- Meanwhile, AIDS incidence and mortality rates are decreasing and are lower than the rates for the county as a whole.

As you review the enclosed material, please keep several things in mind:

- It is difficult to detect small differences in rates of cancer when looking at relatively small populations (such as the population in the area around SeaTac Airport).
- When doing a large number of analyses, we expect to find some statistically significant differences by chance alone.
- The state health department's analyses for questions 1 and 2 of the work plan looked at ***new cases of cancer (incidence)*** around the airport. In contrast, the health assessment by Seattle-King County health department for question 10 looked at ***death due to cancer (mortality)***. An ***incidence rate*** reflects the occurrence of the disease being studied. A ***mortality rate*** reflects deaths due to the disease. The term cancer includes a variety of diseases characterized by uncontrolled growth and spread of abnormal cells. In general, the most common types of cancer are not as fatal as less common types. While the state health department found that the occurrence of all

cancers in the area within 5 miles of the airport was less than expected in comparison to King County, the Seattle-King County health assessment found an increase in cancer deaths around SeaTac Airport. We offer some possible explanations for this pattern below. We realize these results may be an area of confusion, and we are available to discuss them further.

- Although all types of cancer occurred slightly less often in the SeaTac area, those that occurred were more likely to result in death. Factors that can affect whether a cancer leads to death include the type of cancer, the person's access to and use of health care, and other health conditions. Of particular interest regarding the results from these two evaluations, lung cancer occurred more often and was also the leading cause of cancer deaths in the area around the airport.
- The increase in cancer mortality may reflect an earlier increase in cancer incidence. There is generally some period of time between diagnosis and death, so increased cancer deaths for 1993-1997, as found in the Seattle-King County health assessment, may indicate an increase in the occurrence of cancer before 1993. We are obtaining earlier and later years of data to further examine these questions.
- When all cancers for the five-year period from 1992 through 1996 are combined, the numbers become quite large (i.e., more than 5,300 cancer cases and more than 1,000 cancer deaths were analyzed). Just as small numbers make finding small differences problematic in statistical analyses, analysis of large numbers often allows small differences to be statistically significant. A statistically significant difference is not always meaningful from a clinical or public health perspective. For instance, the rate of cancer *cases* was only 4% less than expected using King County as the comparison group; the rate of cancer *deaths* was only 9% higher than the county.

- The investigation of health concerns around SeaTac Airport is ongoing. In particular, we still are awaiting requested data for years before 1992, and data for 1997 should be available in the next few months. Although the elevation in glioblastoma appears to have been a past event that has since resolved, we need to look at some additional years before drawing final conclusions.

We hope that the enclosed reports are helpful in providing background information. If you have questions about any of these reports, please call the point of contact listed on the cover page of the individual report. Otherwise, please contact Filiz E. Satir (360.236.4077) of the state health department Communications Office if you have questions regarding this investigation.

There will also be an opportunity to ask questions at a community forum on March 4. Senator Julia Patterson has scheduled a 7 p.m. meeting, Thursday, March 4. The meeting will take place at the Highline Community College, Building 2 (maps enclosed). Representatives from the state health department and Seattle-King County health department will present a brief summary of the major findings reported in the enclosed document and answer questions from community members. We hope you will take advantage of this opportunity to join us at the community meeting.

Respectfully yours,

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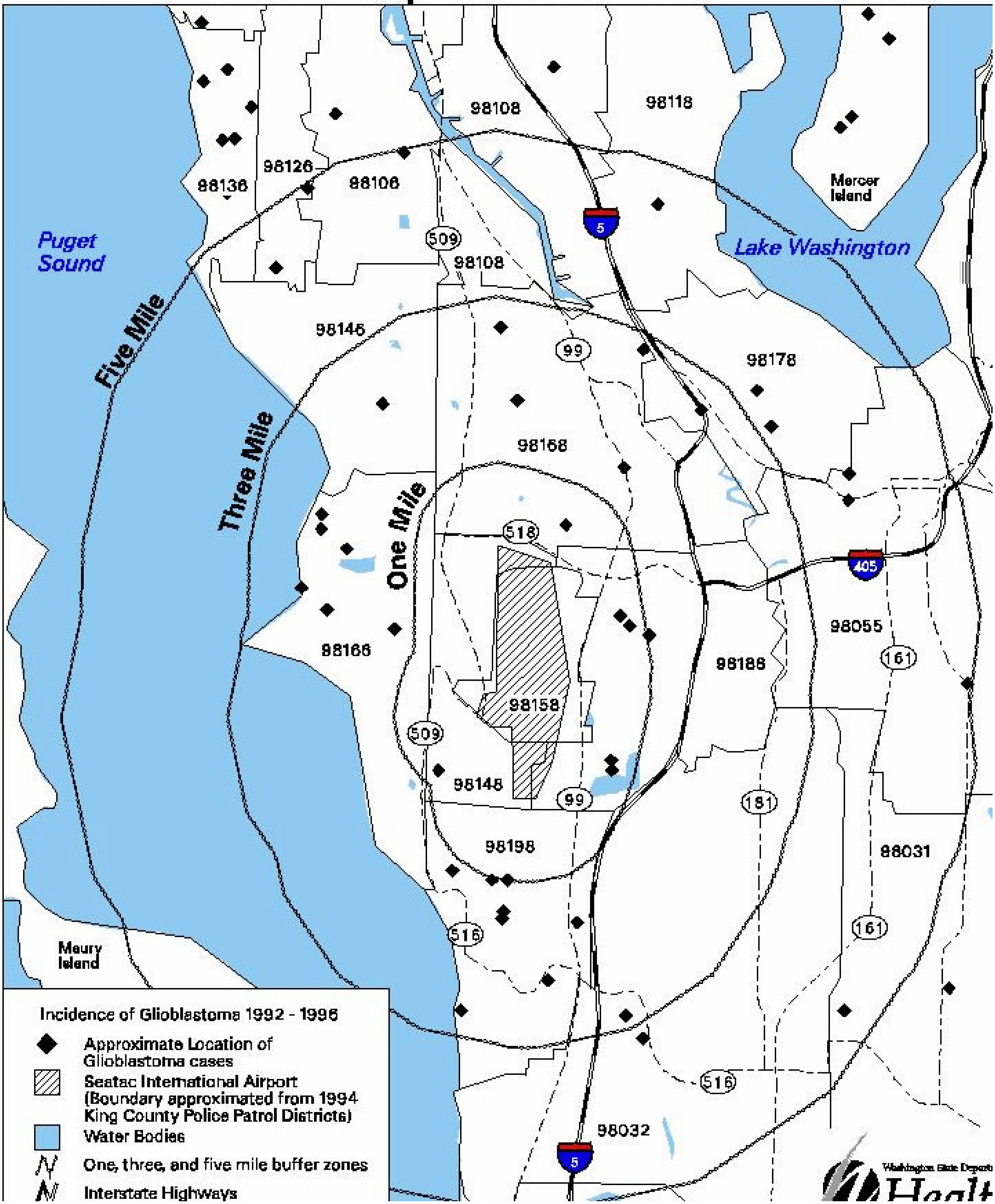
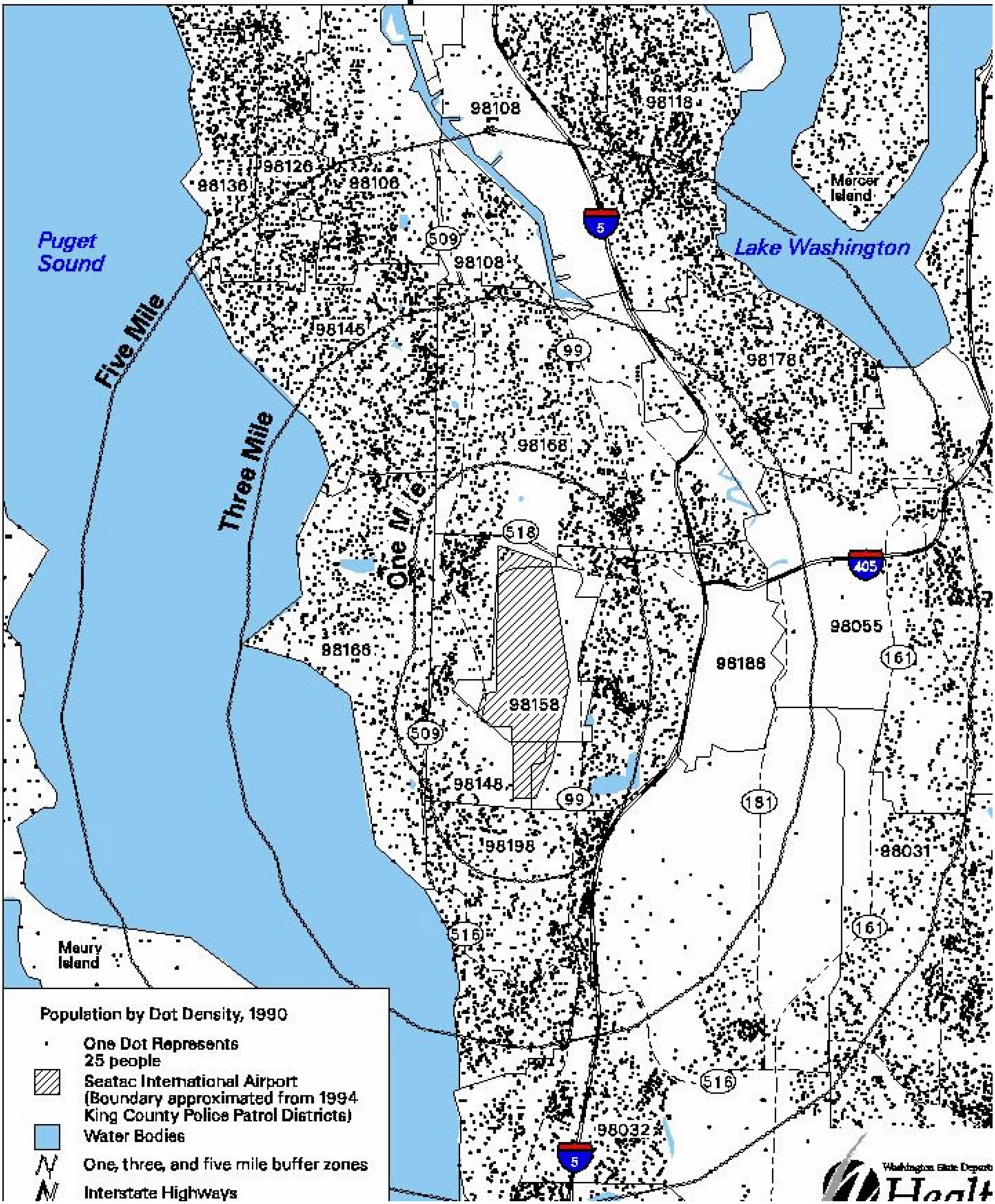
Enclosures

Seatac International Airport and Associated Buffer Zones



Seatac International Airport and Associated Buffer Zones



Cancer Rates in the Proximity of SeaTac International Airport
(Questions 1 and 2 of the August 1998 Work Plan)

Prepared by
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February 25, 1999

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EXECUTIVE SUMMARY

This report is being issued in conjunction with several other documents as part of our investigation outlined in the work plan developed by SeaTac community representatives in cooperation with the Washington Department of Health and the Seattle-King County Department of Public Health in August 1998. Questions 1 and 2 from that work plan are the primary focus of this report:

1. What types of cancer are the most prevalent in the proximity of the airport, and what are their risk factors?
2. Are the rates of breast cancer elevated in the proximity of the airport?

The proximity of SeaTac International Airport was divided into three areas for analysis: Area 1 is equivalent to 1 mile around the airport; Area 2 is within 3 miles of the airport; Area 3 is within 5 miles of the airport. More than 25 categories of cancer for the period from 1992 through 1996 were analyzed to determine statistically significant differences between observed cases in the area of interest and expected cases based on two comparison groups, King County and Washington State as a whole.

Results of numerous analyses found that the ten most prevalent cancers around SeaTac Airport were consistent with the ten most prevalent cancers in both King County and Washington State. However, some cancers, depending upon the comparison group, were found to be higher than expected in one or more of the areas around SeaTac Airport, and other cancers were found to be less than expected.

In Area 1, endometrial and lung cancers were higher than expected when compared to both King County and Washington State; cancers of the oral cavity or pharynx were higher than expected only in comparison to Washington State. No cancers were statistically less than expected in Area 1.

In Area 2, glioblastoma was higher than expected when compared to both King County and Washington State; laryngeal cancer was higher than expected when compared to King County; liver cancer was higher than expected when compared to Washington State. Prostate cancer was lower than expected in Area 2 when compared to Washington State.

In Area 3, laryngeal cancer was higher than expected when compared to both King County and Washington State; kidney/renal cancer was higher than expected when compared to King County; liver cancer was elevated when compared to Washington State. Melanoma and prostate cancer were less than expected when compared to both King County and Washington State; all cancers, breast cancer and thyroid cancer were less than expected when compared to King County.

Although substantially more types of cancer than originally specified by the work plan were analyzed for this report, a few categories were of special interest to SeaTac-area residents. Brain cancers (particularly glioblastoma), breast cancer, and leukemia (particularly acute myeloid leukemia) were of specific interest. Of these, only glioblastoma was higher than expected for the period from 1992 through 1996, and this elevation was restricted to within 3-miles of the airport. Observed cases of breast cancer were consistently within expected range regardless of comparison group except that they were even less than expected for Area 3 when compared to King County. All types of leukemia, including acute myeloid leukemia, were consistently within the expected range.

Of the cancers for which observed cases were found to be higher than expected, review of the literature did not reveal any definitive causes of the increased numbers that can be specifically attributed to proximity to the airport. Despite an extensive review of the literature by the Seattle-King County Department of Health and the Washington State Department of Health, no proven risk factors for glioblastoma in people were identified.

Tobacco exposure is the best-established risk factor for cancers of the larynx, lung and oral/pharynx. Alcohol abuse, particularly in combination with tobacco use, tends to increase the risk of laryngeal, oral and pharyngeal cancers, and air pollution has also been proposed as a possible risk factor for cancers of the lung and larynx. Asbestos exposure is another known risk factor for lung cancer. Liver cancer has been associated with numerous risk factors such as alcohol abuse, viral diseases, dietary intake, hereditary factors, and chemical exposures. Kidney/renal cancers have been best associated with obesity, radiation exposure, tobacco use, chemotherapy for other cancers, and family history; less clear associations include alcohol use, dietary factors, exposure to heavy metals, and occupational exposure to asbestos and a variety of volatile chemicals. Hormonal and family history factors are known to influence endometrial cancer, but nutritional factors have also been implicated as possible causes.

Considering that our investigation of community concerns about health around SeaTac Airport is ongoing, final conclusions would be premature. Investigation of historical data before 1992 requires continued efforts, and follow-up of community case reports is still in progress. Following completion of tasks outlined in Phase 1 of the work plan, the feasibility and desirability of further investigation will be more appropriately evaluated.

BACKGROUND

In response to community concerns about health around SeaTac International Airport, Senator Julia Patterson arranged two meetings in 1998 with community residents, the Washington State Department of Health (DOH), Seattle-King County Department of Public Health (SKCDPH) and other interested parties. Those meetings and preliminary DOH findings of elevated glioblastoma for 1992 through 1995 in an area roughly 3 miles around the airport led to Senator Patterson's request that DOH work with SKCDPH and the community to develop a work plan to address the community's concerns.

Community representatives presented a list of 18 questions they wanted addressed in the work plan. The August 1998 work plan was divided into two phases. Answers to questions from Phase 1 activities were necessary to determine the value and feasibility of Phase 2 activities. Phase 1 activities included 10 questions. This report focuses on the first two questions in the work plan.

Question 1 of Phase 1 addressed concerns about which cancers are most prevalent around SeaTac Airport and known risk factors that may contribute to their being elevated. Question 2 asked whether rates of breast cancer are elevated in the proximity of the airport.

METHODS

We looked at data from the Washington State Cancer Registry to assess whether rates of cancer around SeaTac International Airport were higher than expected. In order to assure analytic precision, we first defined the SeaTac area using geospatial coding of both cancer and census data. After geocoding available data, we designated three areas around the airport for analysis: Area 1 is within 1 mile of the airport, Area 2 is within 3 miles, and Area 3 is within 5 miles.

We compared the number of cases for more than 25 cancer categories diagnosed from 1992 through 1996 in the areas around SeaTac Airport to the number of expected cases if the rates around the airport were the same as rates from two comparison groups. We used Washington State as one comparison group in calculating expected cases within the three areas around the airport. However, the population used for comparison in an epidemiologic study should be similar to the population under investigation, and using Washington State for comparison combines various types of populations. Since the area around SeaTac Airport is urban, we also used King County as a more comparable, predominantly urban comparison group.

The expected number of cases is the number of cases expected in the SeaTac Airport area if the rate around the airport is the same as the rate in King County or Washington State. To calculate the expected number of cases, we multiplied the population in a specific age range and sex category in the SeaTac Airport area by the rate of glioblastoma for the same age range and sex category in King County or Washington State. (Since the area around the airport is part of King County and comprises more than 10% of its population, we subtracted the SeaTac area of interest from both numerator and denominator in calculations of expected cases when using King County as the comparison group.) We then added the results for all the age and sex categories together and rounded to the nearest whole number to get a total number of expected cases.

Confidence intervals are used to assess variation in a rate related to random factors. We calculated 95%

Poisson confidence intervals around the observed number of cases to assess random variation. If the confidence interval did not include the expected number of cases, we concluded that the observed number was statistically different from the expected. If the expected number of cases was less than the lower limit of the interval, we concluded that the number of observed cases was higher than expected. If the expected number of cases was greater than the upper limit of the interval, we concluded that the number of observed cases was lower than expected.

Although inclusion of case reports from concerned citizens and buyout area residents were part of questions 3 and 4 of the work plan, we have made some progress on these questions. As a result, one case of glioblastoma diagnosed in 1993 after the person moved from the buyout area was included in our analyses as a resident of Area 1. Since this individual had originally been included in records for King County outside any of the three SeaTac zones, we also subtracted this person from the King County comparison group. Many other reports received for 1992 through 1996 were already appropriately included in our existing databases.

RESULTS

The results of our analyses are presented in Table 1 on page 7 of this report. The ten most common types of cancer around SeaTac Airport were breast, lung, prostate, colorectal, melanoma, non-Hodgkin's lymphoma, bladder, endometrium, oral/pharynx, and kidney/renal; these are also the ten most common types of cancer in both King County and Washington State as a whole.

Of the cancer categories assessed, results varied according to the designated distance from SeaTac Airport. For Area 1, we found that the number of observed cases was higher than expected for cancers of the endometrium and of the lung when compared to both King County and Washington State; oral/pharyngeal cancer was higher than expected only in comparison to Washington State. For Area 2, glioblastoma was higher than expected when compared to both King County and Washington State, laryngeal cancer was higher than expected when compared to King County, and liver cancer was elevated when compared to Washington State. Prostate cancer was lower than expected in Area 2 when compared to Washington State. For Area 3, laryngeal cancer was elevated when compared to both King County and Washington State; kidney/renal cancer was elevated when compared to King County; liver cancer was elevated when compared to Washington State. Also for Area 3, melanoma and prostate cancer were less than expected when compared to both King County and Washington State; all cancers, breast cancer and thyroid cancer were less than expected when compared to King County.

Brain cancers, particularly glioblastoma, were of specific interest to SeaTac-area residents. After including one glioblastoma case of a buyout area residence in our analyses, we found that observed cases of glioblastoma were slightly higher than expected in Area 2 when data from 1992 through 1996 were combined. Of the 28 people with glioblastoma in Area 2 during this period, 10 (36%) were diagnosed in 1992. The other years had between 3 and 6 people diagnosed each year, all of which were within expected range for the year (Page 8, Figure 1).

All leukemia and the subcategory of acute myeloid leukemia were also mentioned as specific concerns to SeaTac community representatives, but we found no elevation in either of these categories. Question 2 of the work plan specified interest in evaluating rates of breast cancer around SeaTac International Airport, but we found that observed cases of breast cancer were consistently within or less than the expected range.

DISCUSSION

Perhaps the most important consideration in assessing the results of these analyses involves the issue of random chance when doing statistical tests and the problem of increasing chance results when doing multiple comparisons. We used the usual scientific standard in which there is a 5% probability that a statistically significant result is by chance alone. In other words, there is a 5% chance that a statistically significant result does not represent a true difference from the expected result for each individual analysis. The probability of statistically significant results being due to chance alone increases proportionately with the number of individual tests of significance. By conducting our analyses using more than 25 cancer categories, we expect random variation of statistically significant results, some of which will be greater than expected and some of which will be lower than expected. A mixture of statistically significant results in both directions is demonstrated in Table 1. Some of these may represent true variations, but some are probably due to chance. Since we cannot determine from statistical tests alone which results are true and which are due to chance, other considerations must also influence our conclusions. In particular, consistency with prior studies and biologic plausibility are primary factors in interpreting results.

In May 1998, DOH did a preliminary analysis of State cancer data for the years 1992 through 1995 using zipcodes to roughly estimate the population around SeaTac Airport. That analysis focused on all cancer, all leukemia, and brain tumors (including gliomas and glioblastomas). The results suggested that the number of cases of all cancer and of glioblastoma were higher than expected, particularly in the area approximately 3 miles around the airport. Using more precise methods to define the areas around SeaTac Airport and adding another year of data to the analysis, we did not find an elevation of all cancer in any of the three areas evaluated. Although results of our latest analyses were not identical to the preliminary analyses, we did find elevated glioblastoma in the area within 3 miles of the airport. Our results were also consistent with the preliminary analyses in that the numbers of people diagnosed with glioblastoma were only elevated in 1992 and the numbers for the years 1993 through 1996 were not elevated.

The term cancer is nonspecific and refers to a variety of different diseases, most of which involve more than one risk factor. To identify risk factors associated with the types of cancer that were elevated in areas around SeaTac airport, we used the textbook edited by Schottenfeld and Fraumeni[1], a comprehensive review of the scientific literature related to causes of cancer through about 1995. No definitive causes of the increased numbers that could be specifically attributed to proximity to the airport were found. In responding to Question 7 of the August 1998 work plan, SKCDPH and DOH did an extensive review of the literature regarding environmental causes of glioblastoma. The consensus among researchers at this time is that causal factors for glioblastoma in people have not yet been identified. (Please refer to the summary of the literature review being issued in conjunction with this report and also dated February 25, 1999.)

Tobacco exposure is a well-established risk factor for cancers of the larynx, lung and oral/pharynx. Asbestos exposure is another known risk factor for lung cancer, and the combination of tobacco use with asbestos exposure substantially increases the risk of lung cancer. Alcohol abuse, particularly in combination with tobacco use, tends to increase the risk of laryngeal, oral and pharyngeal cancers. Air pollution has been proposed as a possible risk factor for cancers of the lung and larynx, but analytic studies to date have been inconclusive regarding the strength of this association.

[1] *Cancer Epidemiology and Prevention*, Schottenfeld D and Fraumeni JF (eds.), Oxford University Press, New York, 1996.

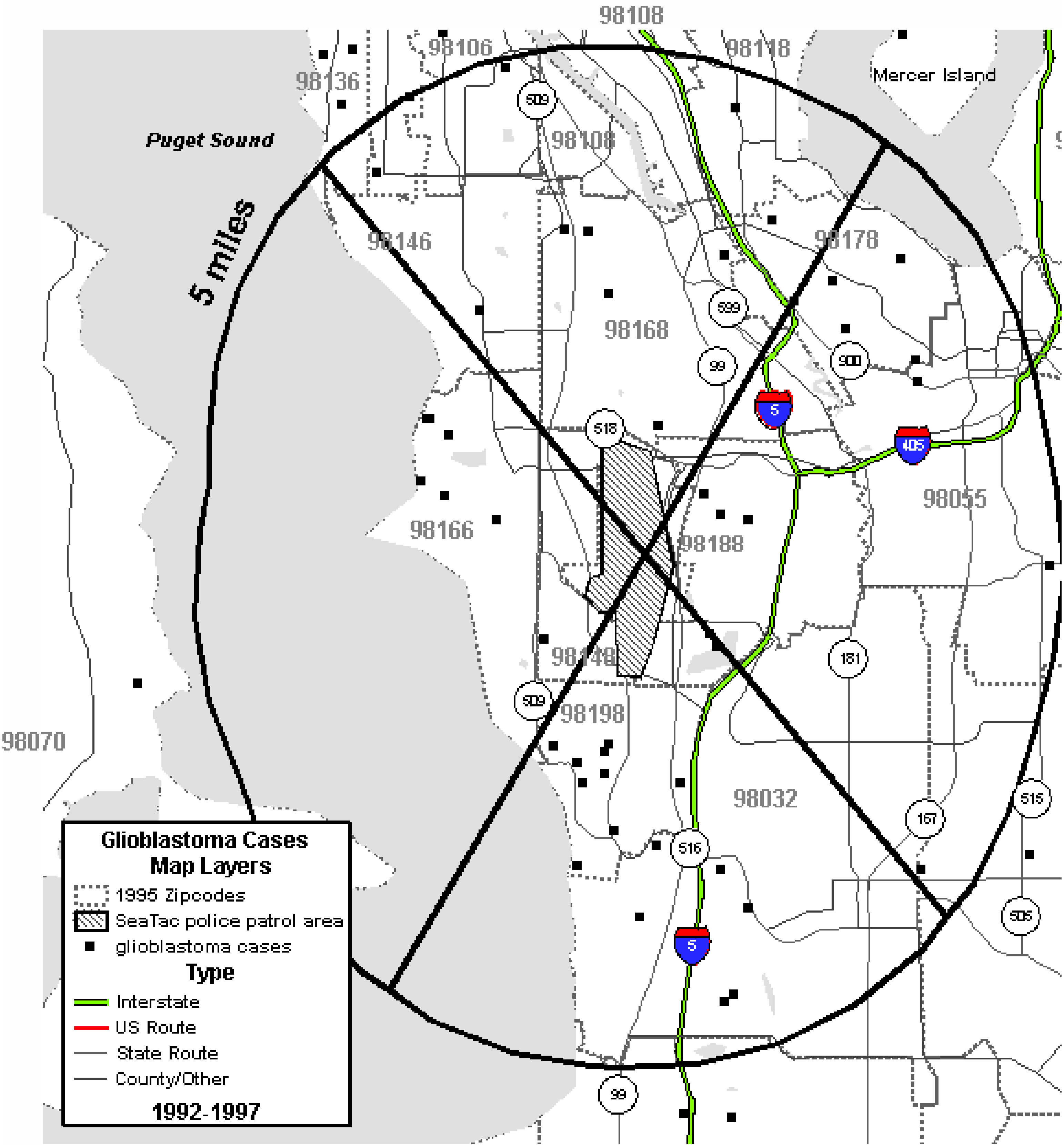
Liver cancer has been associated with numerous risk factors. Among the most prominent risk factors for liver cancer are alcohol abuse, viral diseases, dietary intake, hereditary factors, and chemical exposures.

Kidney/renal cancers have been best associated with obesity, radiation exposure, tobacco use, chemotherapy for other cancers, and family history; less clear associations include alcohol use, dietary factors, exposure to heavy metals, and occupational exposure to asbestos as well as a variety of volatile chemicals. Hormonal and family history factors are known to influence endometrial cancer, but nutritional factors have also been implicated as possible causes.

The DOH analyses for questions 1 and 2 of the work plan looked at ***new cases of cancer (incidence)*** around the airport. In contrast, the health assessment by SKCDPH for question 10 looked at ***death due to cancer (mortality)***. An ***incidence rate*** reflects the occurrence of the disease being studied. A ***mortality rate*** reflects deaths due to the disease. The term cancer includes a variety of diseases characterized by uncontrolled growth and spread of abnormal cells. In general, the most common types of cancer are not as fatal as less common types. While DOH found that the occurrence of all cancers in the area within 5 miles of the airport was less than expected in comparison to King County, the SKCDPH health assessment found an increase in cancer deaths around SeaTac Airport. We offer some possible explanations for this pattern:

- Although all types of cancer occurred slightly less often in the SeaTac area, those that occurred were more likely to result in death. Factors that can affect whether a cancer leads to death include the type of cancer, the person’s access to and use of health care, and other health conditions. Of particular interest regarding the results from these two evaluations, lung cancer occurred more often and was also the leading cause of cancer deaths in the area around the airport.
- The increase in cancer mortality may reflect an earlier increase in cancer incidence. There is generally some period of time between diagnosis and death, so increased cancer deaths for 1993 through 1997, as found in the Seattle-King County health assessment, may indicate an increase in the occurrence of cancer before 1993. We are obtaining earlier and later years of data to further examine these questions.
- When all cancers for the five-year period from 1992 through 1996 are combined, the numbers become quite large (i.e., more than 5,300 cancer cases and over 1,000 cancer deaths were analyzed). Just as small numbers make finding small differences problematic in statistical analyses, analysis of large numbers often allows small differences to be statistically significant. A statistically significant difference is not always meaningful from a clinical or public health perspective. For instance, the rate of cancer *cases* was only 4% less than expected using King County as the comparison group; the rate of cancer *deaths* was only 9% higher than the County.

Considering that the investigation of community concerns about health in the proximity of SeaTac International Airport is ongoing, final conclusions would be premature. (Please refer to the work plan progress report being issued in conjunction with this report and also dated February 25, 1999.) Investigation of historical data before 1992 requires continued efforts, and follow-up of community case reports is still in progress. Answers to some of the remaining questions may help us interpret these findings better.


98108
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5 miles
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98166
98188
98148
98198
98070
98032
Glioblastoma Cases
Map Layers
1995 Zipcodes
SeaTac police patrol area
glioblastoma cases
Type
Interstate
US Route
State Route
County/Other
1992-1997

Table 1. Cancer in the Proximity of SeaTac International Airport, 1992-1996

	Area 1 = Within 1 Miles of Airport			Area 2 = Within 3 Miles of Airport			Area 3 = Within 5 Miles of Airport		
	Observed	Expected (County Rate)	Expected (State Rate)	Observed	Expected (County Rate)	Expected (State Rate)	Observed	Expected (County Rate)	Expected (State Rate)
All Cancer[1]	797	783	771	2,794	2,874	2,827	† 5,334	5,575	5,455
Bladder	32	32	33	117	123	127	217	238	244
Brain, All Types	15	11	11	48	39	37	73	79	73
Brain, All Gliomas	15	11	10	48	38	36	73	77	72
Brain, Astrocytomas	1	2	3	7	8	9	13	17	18
Brain, Glioblastomas	7	5	5	*** 28	19	18	37	38	34
Breast	134	142	134	490	510	480	† 889	1,005	930
Cervix	5	7	8	22	23	26	45	46	52
Colorectal	86	80	79	284	306	301	564	580	573
Endometrium	*** 39	22	23	93	82	83	167	158	160
Esophagus	9	8	8	34	28	28	62	53	54
Kidney / Renal	20	17	17	73	60	63	* 138	114	122
Larynx	9	6	7	* 34	23	25	*** 67	42	49
Leukemia, All Types	16	19	18	65	69	67	137	133	131
Leukemia, Acute Myeloid (AML)	3	5	5	19	19	17	40	36	33
Liver	6	6	5	** 27	20	17	** 50	38	33
Lung	*** 132	104	108	406	386	399	779	738	767
Lymphoma, Hodgkin's	6	6	5	16	20	17	30	42	35
Lymphoma, non-Hodgkin's	28	33	30	106	120	109	222	233	212
Melanoma	36	46	42	141	163	146	††† 242	330	286
Multiple Myeloma	5	8	8	24	31	31	57	58	59
Oral / Pharynx	** 29	20	19	82	74	68	140	144	131
Ovary	16	17	16	60	62	58	117	121	113
Pancreas	18	16	16	58	63	59	122	118	112
Prostate	99	113	119	†† 393	429	448	††† 762	827	860
Stomach	6	11	11	35	42	40	82	78	77
Testis	6	6	6	16	20	20	42	39	41
Thyroid	9	11	10	32	37	35	† 59	76	70
All Other Cancer Categories	60	66	63	238	240	230	451	469	445

* Higher than expected using King County rate
** Higher than expected using State rate
*** Higher than expected using both King County and State rates
† Lower than expected using King County rate
†† Lower than expected using State Rate
††† Lower than expected using both King County and State rates

[1] The totals of cancers broken into categories are greater than "All Cancer" because some individuals have multiple cancers. The "All Cancer" category counts only the total number of individuals with any type of cancer during the study period.

LITERATURE REVIEW ON RISK FACTORS FOR GLIOBLASTOMA MULTIFORME

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LITERATURE REVIEW ON RISK FACTORS FOR GLIOBLASTOMA MULTIFORME

TABLE OF CONTENTS

REPORT SUMMARY

An extensive review of the scientific literature related to environmental causes of glioblastoma multiforme (GBM) has found that no proven risk factors for this disease in people have yet been identified. Studies looking at risk factors for GBM alone are relatively few in number, but some have evaluated environmental and occupational risk factors. Those factors (discussed in more detail in the report below) include the employment in the petrochemical industry, agriculture and at airfields. Most of these studies were designed to generate questions for further research. At this time, however, there is a consensus among researchers that causal factors for GBM in people have not yet been found.

Below is the full text of the review.

INTRODUCTION

Organization of the report

The report has four parts: *Introduction, Summary of Findings, Description of Studies and Appendices.* The Summary of Findings includes a review of studies of GBM, astrocytomas, gliomas and all brain cancer in people, followed by a review of animal studies, and studies of viral causes. A Description of Studies includes details of the studies involving humans. In the appendices, we included a discussion of relevant epidemiological concepts that are intended to aid the reader in interpreting the literature (see Appendix A). Appendix B discusses the classification of human brain cancers. Appendix C discusses the relevance of the p53 suppressor gene for determining the cause of brain cancer.

In the Description of Studies, articles related to human environmental and occupational exposures of people are grouped according to the exposure examined or occupational category. Although these groups are not necessarily mutually exclusive, we grouped studies based on the primary exposure being evaluated by the study. For each article the main findings related to GBM or astrocytic tumors are described.

We have attempted to write clearly and avoid overly technical terms in this report, but we recognize that this is a highly complex field. We are available to discuss this review with anyone who would like clarification or further information.

Methods

This literature review was prepared by the Seattle-King County Department of Public Health and the Washington State Department of Health as part of an investigation of elevated incidence of glioblastoma multiforme (GBM) in the vicinity of SeaTac International Airport. We conducted a search of medical and toxicological scientific publications through the National Library of Medicine's MEDLINE, TOXLINE and TOXNET databases. We searched for articles published in past 20 years on GBM, brain tumors, brain cancer, astrocytoma, glioma, and environmental causes of cancer. We reviewed the references in the articles located through our computer search to identify additional articles.

In evaluating risk factors for brain cancer, researchers often aggregate across brain cancer subsets. For example, many studies identify risk factors for brain cancer in general, others for gliomas and others for astrocytic brain cancer. Fewer focus solely on GBM. For studies involving people, we initially reviewed studies that focused specifically on GBM or calculated risk estimates for this subset. However, to increase the thoroughness of the search, we expanded our initial effort to include tumors classified as astrocytic, if these studies examined occupational or residential risk factors related to airports, or other potentially related occupational risk factors. Finally, when we found studies of broader groups of brain cancer (such as glioma or brain cancer as a whole), we included them here if they examined risk factors that could conceivably be related to airports. For instance, some of these studies looked at brain cancer and aircraft or airfield-related occupations or employment in the petrochemical industry (the latter included because of community concerns about possible fuel dumping). There is, however, no consensus among brain cancer researchers that different types of brain cancer are related, and GBM made up either no part, a relatively small part of an unknown portion of the brain cancers reported in these studies.

Scope of Review

This is primarily a review of the literature of environmental and occupational risk factors for GBM in people. For each environmental and occupational study, we present the country, condition, time period and population studied, the type of study, the factors examined and findings. The GBM studies are included in Table 1 (attached). We have also provided a summary of information obtained from animal studies and studies looking at the relationship of brain cancer to infectious disease. Other suspected risk factors for brain cancer, such as head injury and exposure to ionizing radiation are not reviewed in depth. They are mentioned in this review to give a feel for the scope of factors that have been investigated. Our complete collection of literature is open to review on request.

Because of our focus on GBM, this is not a comprehensive review of the literature on brain cancer in general. There are some recent reviews in the literature that cover this issue (Preston-Martin, 1996; Ashan, 1995).

Background on GBM

GBM is an aggressive, rapidly fatal type of brain cancer. The five-year survival rate is 6% (Axtell, 1976). GBM has been reported to make up anywhere from 26% to slightly more than 40% of the total of diagnosed primary brain cancers (Preston-Martin, 1989; Ashan, 1995). GBM makes up a subset of astrocytomas, which in turn are part of the glioma category (See Appendix B for a discussion of brain cancer classification).

There is controversy in the scientific community over whether new cases of GBM (along with brain cancer overall) are increasing over time (Schottenfeld, 1996). Investigators have found that new cases of GBM reported to regional cancer registries in the Surveillance, Epidemiology and End Results (SEER) program among those age 75 and older increased in the U.S. by about 30% between 1980 and 1990. The increase, however, was not statistically significant and was not seen in all age groups combined. It is not known how much of the increase may be related to the increasing availability of more sensitive diagnostic techniques (specifically Magnetic Resonance Imaging or MRI) or greater exposure to environmental or other factors (Ashan, 1995). GBM is more common in men (Schottenfeld, 1996), especially among people age 75 and older, where the incidence in men is about double that seen in women (Ashan, 1995).

GBM is uncommon. According to data from the Washington State Cancer Registry, from 1992 through 1996 GBM accounted for 0.6% of all cancers in King County and affected approximately 3/100,000 people. There were approximately 47 new cases each year in the County between 1992 and 1996.

SUMMARY OF FINDINGS

There is a consensus in the literature that the causes of GBM in people have not been identified and risk factors are poorly understood. In fact, rather little is known about the etiology (causal factors) of brain cancer in general.

There have been, however, several studies that have generated research questions that have not yet been explored further by medical researchers. The majority of these studies have examined workers and occupational exposures.

Environmental and Occupational Studies of People

Glioblastoma Multiforme

Studies that have looked at GBM and environmental or occupational factors have identified a correlation of GBM risk with a variety of factors, such as working at an airfield (Olin, 1987) or petrochemical plant (Alexander, 1980); living near a petrochemical plant, pharmaceuticals plant or saw mill/lumber yard (Olin, 1987); living in a county where rice/cotton are cultivated or that has wood industries or living near a farm (Smith-Rooker, 1992); exposure to insecticides or herbicides, increasing fuel expenditures on farms, increasing number of dairy cattle on farms (Morrison, 1992); and others. The findings of several studies have not been followed up or consistently demonstrated; for instance, a Swedish study that found increased risk of astrocytoma (mostly GBM) among those who reported working at an airfield has not, to our knowledge, been reproduced in other analytic studies. While it is certainly conceivable that some of these factors may have some exposures in common, such as exposure to solvents from jet fuel at airfields or petrochemical plants, or exposure to pesticides in farms, and that these exposures cause disease, evidence of a link between a specific exposure and GBM (e.g., studies linking specific chemical exposures with specific outcomes) among different studies is lacking.

All Astrocytomas

While no causal factors have been established, elevated risk of astrocytic tumors has been noted in several occupational settings including polyvinyl chloride production, petrochemical production, and solvent exposed workers.

Glioma, All Brain Cancer, and Cancer of the Central Nervous System

Some studies of glioma, brain cancer as a whole or cancer of the central nervous system (most of which are brain cancers) have looked at aircraft-related employment as a risk factor. Some of these studies examined risk to individuals in this occupation, while some have looked at the brain cancer risk to children of those so employed. The studies have found contradictory results, with some showing excess risk associated with employment in the aircraft industry or with aviation (Peters, 1981; Preston-Martin, 1989) or the aerospace industry (Olshan, 1986), while others have shown no excess risk for aircraft-related occupations (Spirtas, 1991; Nasca, 1988; Preston-Martin, 1989).

Other studies of all brain cancer have examined risk associated with employment in oil refineries (as reviewed by Schottenfeld, 1996, and Thomas, 1986). Although specific exposures have not been well characterized, several studies have shown excess risk of brain cancer in this group of workers. Studies that identify exposures in this industry that may be responsible have not yet been published.

Some of the difficulties in assessing this literature on GBM and astrocytic tumors include the lack of good exposure data. Many studies look at risks to occupational groups in order to understand potential harmful exposures, but specific exposures have not been thoroughly investigated. A further complication of evaluating older studies is lack of consistency in the criteria for classifying different kinds of brain tumors.

A more detailed description of the environmental and occupational studies of people begins on page 9.

Possible Clues from Animal Studies

Experimental studies have shown that nitrosamides are by far the most potent chemicals that cause brain cancer in rodents and primates. (Kleihues et al 1976, Magee et al 1976, National Academy Press 1981, Bogovski and Bogovski 1981), Nitrosamides are members of N-nitroso compounds (NOCs) that include compounds such as nitrosoureas, N-nitrosoguanidines and N-nitrosocarbamates. The most common types of brain cancer caused by nitrosourea are gliomas and schwannomas (Koestner 1990). Different alkylnitrosoureas vary in their ability to cause brain cancer in rodents depending on their chemical structure.

Other experimental evidence has shown that nitroamines, a related class of NOCs, also cause brain cancer in animals. (Kleihues 1976). In general, the biological activity of NOCs is thought to be related to alkylation of DNA, a process that changes the chemical structure of the DNA. This type of DNA damage is repaired less efficiently in the brain than other tissues (Goth and Rajewsky 1974). There is evidence that animals and people with lower levels of the enzyme, alkyltransferase, are more susceptible to cancer from alkylating agents, such as NOCs, compared to animals and people with higher levels of the enzyme. This suggests a possible source of individual variation in susceptibility (Pegg 1990).

NOCs originate from both the external environment and as part of human biological processes. External environmental sources have declined over the past decade. NOCs are found in food both naturally (for example in beets and turnips) and artificially (for example, in nitrate-cured meats) and in beverages (e.g. malt beverages), cosmetics, occupational exposures, and rubber products (Hotchkiss 1989). Other environmental sources include well water that has been contaminated with nitrate containing fertilizers or though leaky septic tanks. NOCs that are formed through human biological processes in the stomach and mouth may actually constitute the greatest exposure (Casarett and Doull 1996).

Other chemicals that have been shown to cause brain tumors in rodents include hydrazines (used in rocket fuel), dimethyl sulfate (used in chemical manufacturing), and acrylonitrile (used in the manufacturing of acrylic fibers and pesticides), as well as other chemicals whose use we have not been able to determine. These chemicals include azoxyalkanes , aryldialkyltriazenes, methylmethanesulfonate, propane sultone, and propylene imine (Ulland et al. 1971, Maltoni et al. 1982, Ward and Rice 1982, and Bigner et al. 1986). Like NOCs, the toxic effect of these chemicals appears to be related to their ability to act as alkylating agents (Kleihues et al. 1976).

There are other chemicals that cause brain tumors in experimental animals, but the ability of these chemicals to cause cancer is not related to their ability to alkylate DNA. These include lead subacetate (used in chemical analysis), vinyl chloride (used in manufacturing plastics), urethane (used in solvents, pesticides, and resins), propylene oxide (used in manufacturing polyurethanes, and as a surfactant and fumigant), ethylene oxide (used as a fumigant and fungicide, for sterilizing surgical instruments, to make ethylene glycol), and elaiomycin (antibiotic substance), as well as chemicals whose use we have not been able to determine, including 2-acetylaminofluorene and glycidol (Kleihues et al. 1976, Maltoni et al. 1982, Schoental 1969, Oyasu et al. 1970, Garman 1985).

Some of these chemicals belong to classes of compounds that have been identified as either a component of jet fuel or a byproduct, which is released after burning. However, none of the specific chemicals noted above is known to be a component of jet fuel or its byproducts. (See report for question 8: What are the chemicals in jet engine exhaust emissions, and what happens to them after they are emitted?) We are investigating whether chemical reactions of several byproducts of jet fuel could form NOCs.

Infectious Agents in Animals and People

Several types of viruses cause brain tumors in rodents if they are implanted in the animals' brains. (Ward and Rice 1982, Bigner 1978, Walsh et al. 1982, Solleveld et al. 1986). Viruses tend to cause specific types of brain tumors. Retroviruses, DNA papovaviruses, and adenoviruses have been shown to cause specific types of gliomas (Wrensch et al. 1993), including glioblastomas and astrocytomas.

In Connecticut in the late 1950s to the early 1960s, the polio vaccination was contaminated with simian virus 40 (SV 40); SV 40 may have been related to medulloblastoma in children of mothers who were vaccinated during pregnancy (Farwell 1984). A similar incident occurred in Europe at approximately the same time period, and was associated with increased numbers of glioblastoma, oligodenderogliomas, spongioblastomas, and medulloblastomas in the exposed group. However, the increases were not statistically significant (Wrensch et al. 1993). A Finnish study raised concerns over chicken pox with regard to brain tumors. In this study, children who developed medulloblastoma were more likely (relative risk 10) to have been born to mothers who contracted chicken pox during pregnancy (Bithell et al 1973).

Other infectious diseases may also play a role in the development of brain tumors. For example, a 1989 study of Seventh-Day Adventists showed that individuals who reported a positive tuberculosis skin test were more than twice as likely to develop gliomas compared to those who tested negative (Mills et al 1989).

Despite the possibility that viral and bacterial pathogens may be involved in human brain cancers, few studies have been conducted to investigate such a link.

<u>DESCRIPTION OF ENVIRONMENTAL AND OCCUPATIONAL STUDIES OF PEOPLE</u>

Glioblastoma Multiforme

Agricultural

A descriptive study with no comparison group looked at herbicide exposure among patients diagnosed with GBM and found that a significant proportion resided in agricultural and wood products counties (Smith-Rooker, 1992).

A retrospective cohort study of brain cancer mortality examined agricultural practices and risk of dying from brain cancer (Morrison et al 1992). The authors report a statistically significant association between the risk of dying of GBM and increasing fuel/oil expenditures.

Petrochemical

Alexander et al (1980) reported on a case series of 18 brain cancer deaths among men who worked at a Texas petrochemical plant. Fifteen of the eighteen tumors were GBM, which is considered an unusual distribution. The authors review other epidemiological literature that examines occupational exposures among petrochemical workers or those in related occupations. The authors conclude that based on the published literature, vinyl chloride exposure will be the preliminary focus of their investigation.

General occupational

A Swedish case-control study (Olin, et al 1987) that examined occupational risk factors for high-grade astrocytomas, the majority of which appear to be GBM, reported increased risk among those who had worked at an airfield or lived near a petroleum plant or a sewage treatment plant. The same study reported slightly increased risk among those who lived near a saw mill/lumber yard. The authors attribute the finding of increased risk among airfield workers potentially to exposure to polycyclic hydrocarbons[1]

A descriptive study (Gold and Kathren 1998) of causes of death in 260 plutonium workers identified 6 deaths due to GBM. All 6 individuals worked at the Rocky Flats Nuclear Facility. Based on an assessment of the literature and the fact that cases were not found at other nuclear weapons sites, the authors conclude that radiation exposure is not likely to be the cause of these GBM deaths. They suggest that a factor specific to the Rocky Flats site or surrounding area is likely to be responsible, however other potential risk or causal factors are not considered.

Thomas reviewed the literature on brain tumors and occupational risk factors (1986). He reports on various occupational groups found to be at higher risk of brain cancer. Many of the reports concern brain cancer in general and do not specify the proportion of cases that are GBM. However, some clustering in occupational settings is reported in which the majority of the tumors are identified as GBM. These occupational settings include polyvinyl chloride production, aviation electronics, and cosmetology.

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[1] Polycyclic hydrocarbons or polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. (Excerpted from ATSDR ToxFAQs-Polycyclic Aromatic Hydrocarbons; http://atsdr1.atsdr.cdc.gov:8080/tfacts69.html).

General non-occupational

Moranz et al (1985) report descriptive findings of a brain cancer cluster in Western Missouri. Seven cases were identified, during a period of 10 years, of which 6 were GBM. Age-adjusted mortality from brain cancer was over 4 times greater than expected. The authors report on residential and occupational exposures that might be associated. Six of the seven lived within 200 yards of a chicken hatchery; 4 were employed at the local shoe factory; 4 fished in abandoned coal mining strip pits and 5 regularly ate fish caught in strip mining pits.

Hochberg, et al (1984), using a case-control design found an association between prior head injury and GBM among those who had sustained the injury after age 15. A later study by the same principal author (1990) used a case-control design to assess association between GBM and a number of non-occupational risk factors. A slight, non-significant increased risk was detected for radiation to the head for treatment of acne or tinea capitis. Additionally, a slight, non-significant increase in risk associated with decreasing maternal age at birth was reported.

Helseth et al (1989) looked at the relationship between pre-morbid height and weight as risk factors for CNS neoplasms using a case-control design. They report a statistically significant association between height and GBM in males. Another case-control study of non-occupational risk factors (Ahlbom, 1986) for high grade astrocytoma (the majority of cases appear to be GBM) found a slightly elevated risk for eating smoked foods and having lived in the vicinity of a farm.

A case-control study (Cantor, 1993) examined the relationship between reproductive factors and risk of cancers of non-reproductive origin. The authors report that ever-parous women had a lower risk of glioma; this lower risk was observed for both astrocytoma and GBM. The authors suggest a potential role for hormonal factors in brain cancer etiology.

Therapeutic Radiation

Hodges et al (1992) reports on a descriptive study of GBM among patients who had had prior therapeutic radiation to the head. Seventeen out of 100 patients with GBM had received previous therapeutic radiation and 4 fit the criteria for radiation induced tumor.

Astrocytic Brain Cancer

Solvents

In a case-control study, Thomas (1987b) examined the risk of brain tumor mortality among men with electrical and electronics jobs. Cases and controls were identified from death certificates. The authors report a significantly increased risk of astrocytic tumors in men who worked in electronics manufacturing and repair. This increased risk did not appear to be due solely to exposure to microwave and radiofrequency (MW/RF) electromagnetic radiation since some working in manufacturing are not exposed to MW/RF radiation and some job classes that are did not show increased risk. The authors note that workers in electronics manufacturing and repair are exposed to lead, solder fluxes, solvents and other chemicals and that these exposures should be considered in future studies.

Heineman et al (1994), following up on earlier studies that found associations between solvent exposure and brain cancer, used a case-control design to analyze occupational exposure to specific solvents. The authors report an association between likely exposure to methylene chloride and astrocytic brain cancer. Smaller increased risks were detected for carbon tetrachloride, tetrachloroethylene and tricloroethylene. Risk of astrocytic brain tumor increased with length of employment in methylene chloride exposed jobs and with increased likelihood of exposure.

A retrospective cohort study of anatomists (Stroup, 1986) (an occupational class that is exposed to a variety of solvents, alcohols, and stains as well as viruses and other biologic agents examined causes of death in this group between 1888 and 1969. During this time period the authors report that 10 died of brain cancer. All of the cancers were classified as astrocytoma or GBM. Brain cancer was identified as the only cause of death for which mortality was significantly greater in anatomists compared to male psychologists and US males.

Petrochemical

Thomas et al (1987a) used a case-control design to examine occupational risk factors for astrocytic tumors associated with chemical exposures. They report a slightly elevated, non-significant risk of astrocytic tumors among men employed in production or maintenance activities at petroleum refineries. Slightly elevated, non-significant risks were also reported for several chemical exposures including solvents, lubricating oils and cutting fluids.

General Occupational

A case control study of brain tumor mortality in WA State (Demers et al, 1991) found cleaning service workers, petroleum refinery workers and forestry workers to be at excess risk for astrocytoma.

Brain Cancer Overall

A retrospective cohort study of mortality among workers at an aircraft maintenance facility found no excesses in CNS cancer (Spirtas, 1991). The workers in the study were civilian employees of an air force base that maintains and overhauls missiles and aircraft.

Ninety-two of approximately 117 (inferred number) childhood brain cancers reported to the LA county SEER registry between 1972 and 1977 were reported in this case-control analysis (Peters, 1981). Elevated risk of brain cancer in children was found for those whose fathers worked in the aircraft industry either before the pregnancy, during pregnancy or at the time of diagnosis. Exposure measurement was through interview with the mother (the father was not interviewed). This study included no GBM cases in the aircraft industry, although there were several astrocytomas.

A case-control study using WA state SEER data in 51 cases of childhood brain tumor diagnosed between 1978 and 1981 looked at parental exposure and childhood brain tumors (Olshan, 1986). The study includes benign tumors and those not histologically confirmed. Parental occupation in the aerospace industry was examined to confirm the Peters study (above). No excess risk was found for children diagnosed from 0-15 years old, but nonsignificantly elevated risk was found

for children under 10 (the Peters study's age group). However, a nonsignificantly decreased risk was found for 10-15-year-olds.
A case-control study utilizing childhood cancer cases from the New York State cancer registry looked at childhood nervous system cancers and parental occupational exposures (Nasca, 1988). Cases were childhood cancers (<15 years old) diagnosed between 1/1/68 and 12/31/77 (New York City, Rockland, Westchester, Nassau and Suffolk counties not included). No association between childhood CNS cancer and employment in the aircraft industry was observed, but increases were seen with parental occupational exposure to ionizing radiation and electromagnetic radiation and parental employment in hydrocarbon-related occupations and the petroleum industry.

A descriptive study looked at occupation among cases from the SEER registry in Los Angeles County for cases of brain cancer diagnosed between 1972 to 1985 (Preston-Martin, 1989). Occupation and industry coded were those reported at the time of diagnosis. Only males 25-64 years old were included in occupational analyses. Occupations with statistically elevated incidence for gliomas were aeronautical and astronautical engineers and airplane pilots; industrial coding showed those involved in the aircraft and parts industry had an elevated incidence of gliomas. An unknown proportion of these gliomas were GBM.

A case-control study collected information on job history and other experiences among cases of gliomas and meningiomas using SEER registry in LA county (see above) for the years 1980-1984 (Preston-Martin, 1989). Forty-four of the total of 202 gliomas were GBM, a somewhat surprising distribution in view of the exclusions of proxy responders to the questionnaire and the typically rapidly fatal course of the disease. A similar number of glioma cases and controls worked in the aircraft industry.

LITERATURE CITED

Ahlbom A, Navier IL, Norell S, et al, "Nonoccupational Risk Indicators for Astrocytomas in Adults," American Journal of Epidemiology, 1986, 124(2): 334-337.

Alexander V, "Brain tumors risk among United States nuclear workers," Occupational Medicine, 1991, 6: 695-714.

Alexander V, Leffingwell S, Lloyd JW, Waxweiler R, and Miller R, "Brain Cancer in Petrochemical Workers: A Case Series Report," American Journal of Industrial Medicine, 1980, 1: 115-123.

Ashan H, Neugut A, and Bruce J, "Trends in incidence of primary malignant brain tumors in USA, 1981-1990," International Journal of Epidemiology, 1995, 24(6): 1078-1085.

Axtell LM, Ardyce JA, Myers MH, "Cancer patient survival: Report no. 5. Bethesda, MD: National Cancer Institute, 1976; DHEW Publication no. (NIH) 77-992. Quoted in Hockberg F, Toniolo P, and Cole P, "Head trauma and seizures as risk factors of glioblastoma," Neurology, 1984, 34: 1511-1514.

Bartsch H and Hietanen E, "The role of individual susceptibility in cancer burden related to environmental exposure," Environmental Health Perspectives, 1996, May, 104 Suppl 3: 569-577.

Beall C, Delzell E, Cole P, and Brill I, "Brain tumors among electronics industry workers," Epidemiology, 1996, Mar, 7(2): 125-130.

Bigner D, Bigner S, and Burger D, "Primary brain tumors in Fischer 344 rats chronically exposed to acrylonitrile in their drinking-water," Food Chemistry and Toxicology, 1986, 24: 129-137.

Bithell J, Draper G, and Gorbach P, "Associations between malignant disease in children and maternal virus infections," British Medicial Journal, 1973, 706-709.

Bogler O, Huang H, Kleihues P, and Cavenee W, "The p53 gene and its role in human brain tumors," Glia, 1995, Nov, 15(3): 308-327.

Bogovski P and Bogovski S, "Animal species in which N-nitroso compounds induce cancer," International Journal of Cancer, 1981, 27: 471-474.

Brem S, Rozental J, and Moskal J, "What is the etiology of human brain tumor? A report on the first Lebow conference," Cancer, 1995, Aug 15, 76(4): 709-713.

Cantor KP, Lynch CF, Johnson D, "Reproductive factors and risk of brain, colon, and other malignancies in Iowa (United States)," Cancer Causes and Control, 1993, 4: 505-511.

Casarett L and Doull J, (eds.), Casarett and Doull's Toxicology: The Basic Science of Poisons Klaassen, McGraw-Hill, NewYork, 1996, pp 463-486.

Demers PA, Vaughan TL, Schommer RR, "Occupation, socioeconomic status, and brain tumor mortality: A death certificate-based case-control study," Journal of Occupational Medicine, 1991, 33(9): 1001-1006.

Farwell J, Dohrmann G, and Flannery J, “Medulloblastoma in childhood: An epidemiological study,” Journal of Neurosurgery, 1984, 61: 657-664.

Garman R, Snelling W, and Maronpot R, “Brain tumors in F344 rats associated with chronic inhalation exposure to ethylene oxide,” Neurotoxicology, 1985, 6: 117-137.

Gold B and Kathren RL, “Causes of Death in a Cohort of 260 Plutonium Workers,” Health Physics, 1998, 75(3): 236-240.

Goth R and Rajewsky M, “Persistence of O6-ethylguanine in rat-brain DNA: correlation with nervous system-specific carcinogenesis by ethylnitrosourea,” Protocols of the National Academy of Science, 1974, 71: 639-643.

Hardell L, Holmberg B, Malker H, and Paulson L, “What is the etiology of human brain tumors?,” Cancer, 1996, Mar 1, 77(5): 1006-1009.

Harris C, “P53 tumor suppressor gene: at the crossroads of molecular carcinogenesis, molecular epidemiology, and cancer risk assessment,” Environmental Health Perspectives, 1996, May, 104 Suppl 3: 435-439.

Heineman EF, Cocco P, Gomez MR, Dosemeci M, Stewart PA, Hayes RB, Zahm SH, Thomas TL, and Blair A, “Occupational Exposure to Chlorinated Aliphatic Hydrocarbons and Risk of Astrocytic Brain Cancer,” American Journal of Industrial Medicine, 1994, 26: 155-169.

Helseth A and Tretli S, “Pre-Morbid Height and Weight as Risk Factors for Development of Central Nervous System Neoplasms,” Neuroepidemiology, 1989, 8: 277-282.

Hochberg F, Toniolo P, and Cole P, “Head trauma and seizures as risk factors of glioblastoma,” Neurology, 1984, 34: 1511-1514.

Hochberg F, Toniolo P, and Cole P, “Nonoccupational risk indicators of glioblastoma in adults,” Journal of Neuro-Oncology, 1990, 8: 55-60.

Hodges LC, Smith JL, Garrett A, and Tate S, “Prevalence of Glioblastoma Multiforme in Subjects with Prior Therapeutic Radiation,” Journal of Neuroscience Nursing, 1992, 24(2): 79-83.

Hollstein M, Sidransky D, Vogelstein B, and Harris C, “P53 mutations in human cancers,” Science, 1991, July 5, 253(5015): 49-53.

Hotchkiss J, “Relative exposure to nitrite, nitrate, and N-nitroso compounds from endogenous and exogenous sources,” In Taylor, Scanlan (eds), Food toxicology: Aperspective on the Relative Risks, Marcel Dekker, New York, 1989, 57-100.

Inskip P, Linet M, and Heineman E, “Etiology of brain tumors in adults,” Epidemiological Review, 1995, 17(2): 382-414.

Kessler E and Brandt-Rauf P, “Occupational cancers of the nervous system,” Seminars in Occupational Medicine, 1987, 2(4): 311-314.

Kleihues P, Aguzzi A, and Ohgaki H, "Genetic and environmental factors in the etiology of human brain tumors," Toxicology Letters, 1995 Dec, 82-83: 601-605.

Kleihues P, Lantos P, and Magee P, "Chemical carcinogenesis in the nervous system," Int Review of Experimental Pathology, 1976, 15: 153-232.

Kleihues P, Lubbe J, Watanabe K, Von Ammon K, and Ohgaki H, "Genetic alterations associated with glioma progression," Verh Dtsch Ges Pathol, 1994, 78: 43-47.

Koestner A, "Characterization on N-nitrosourea-induced tumors of the nervous system; their prospective value for studies of neurocarcinogenesis and brain tumor therapy," Toxicology and Pathology, 1990, 18: 186-192.

Kraut A, Chan E, Lioy P, Cohen F, Goldstein B, and Landrigan P, "Epidemiologic investigation of a cancer cluster in professional football players," Environmental Research, 1991, 56(2): 131-143.

Li F, Dreyfus M, Russell T, Verselis S, Hutchinson R, and Fraumeni J, "Molecular epidemiology study of a suspected community cluster of childhood cancers," Medical and Pediatric Oncology, 1997, Apr, 28(4): 243-247.

Li Y, Millikan R, Carozza S, Newman B, Liu E, Davis R, Mike R, and Wrensch M, "P53 mutations in malignant gliomas," Cancer Epidemiolical Biomarkers Prev 1998 Apr; 7(4): 303-308.

Magee P, Montesano R, and Prussmann R, "N-nitroso compounds and related carcinogens," Searle CE (ed), Chemical Carcinogens, American Chemical Society, Washington DC, 1976, 491-625.

Maltoni C, Ciliberti A, and Carretti A, "Experimental contributions in identifying brain potential carcinogens in the petrochemical industry," Annals of New York Academy of Science, 1982, 381: 216-249.

Mausner JS and Kramer S, Epidemiology: An Introductory Text, W.B. Saunders Company, Philadelphia, 1985.

McMahon G, "The genetics of human cancer: implications for ecotoxicology," Environmental Health Perspectives, 1994, Dec, 102 Suppl 12: 75-80

Mills P, Preston-Martin S, Annegers J, Beeson W, Philips R, and Fraser G, "Risk factors for tumors of the brain and cranial meninges in Seventh-Day Adventists," Neuroepidemiology, 1989, 8: 266-275.

Moranz RA, Neuberger JS, Baker LH, et al., "Epidemiological findings in a brain-tumor cluster in Western Missouri," Journal of Neurosurgery, 1985, 62: 856-860.

Morrison HI, Semenciw RW, Morison D, et al., "Brain Cancer and Farming in Western Canada," Neuroepidemiology, 1992, 11: 267-276.

Nasca PC, Baptiste MS, MacCubbin PA, et al., "An epidemiologic case-control study of central nervous system tumors in children and paterntal occupational exposures," American Journal of Epidemiology, 1988, 128(6): 1256-1265.

National Academy Press, "The health effects of nitrate, nitrite, and N-nitroso compounds," Part I, 1981.

Olin RG, Ahlbom A, Lindberg-Navier I, et al., "Occupational Factors Associated with Astrocytomas: A Case-Control Study," American Journal of Industrial Medicine, 1987, 11: 615-625.

Olshan AF, Breslow NE, Daling JR, et al., "Childhood brain tumors and paternal occupation in the aerospace industry," *JNCI* 1986, 77(1): 17-19. Peters J and Preston-Martin S, "Brain tumors in children and occupational exposure of parents," Science, 1981, 213.

Oyasu R, Battifora H, and Clasen R, "Induction of cerebral gliomas in rats with dietary lead subacetate and 2-acetylaminofluorene," Cancer Research, 1970, 30: 1248-1261.

Pegg A, "Mammalian O6-alklguanine-DNA alkyltransferase: regulation and importance in response to alkylating carcinogenic and therapeutic agents," Cancer Research, 1990, 50: 6119-6129.

Petruska D and Engelhard H, "Glioblastoma multiforme occurring in a patient following exposure to polychlorinated biphenyls," Journal of Kentucky Medical Association, 1991, 89(10): 496-499.

Prados MD, Berger MS and Wilson CB, "Primary central nervous systems tumors: Advances in knowledge and treatment," CA—A Cancer Journal for Clinicians, 1998, 48(6): 331-360.

Preston-Martin S, "Descriptive epidemiology of primary tumors of the brain, cranial nerves and cranial meninges in Los Angeles County," Neuroepidemiology, 1989, 8: 283-295.

Preston-Martin S, Mack W and Henderson B, "Risk factors for gliomas and meningiomas in males in Los Angeles County," Cancer Research, 1989, 49: 6137-6143.

Preston-Martin S, Mack W, "Neoplasms of the Nervous System," Cancer Epidemiology and Prevention, Schottenfeld D, Fraumeni JF (eds), Oxford University Press, New York, 1996, 1231-1281.

Schoental R, "Carcinogenic action of elaiomycin in rats," Nature, 1969, 221: 765-766.

Smith-Rooker JL, Garrett A, Hodges LC, and Shue V, "Prevalence of Glioblastoma Multiforme Subjects with Prior Herbicide Exposure," Journal of Neuroscience Nursing, 1992, 24(5): 260-264.

Solleveld H, Bigner D, Averill D, Bigner S, Beermanm G, Burger P, Gillespie Y, Hubbard G, Laerum O, McComb R, McGrath J, Morgan K, Peters A, Rubinstein I, Schoenberg B, Schhold S, Swenberg J, Thompson M, Vandevedlde M, and Vinores S, "Brain tumors in man and animal: Report of a workshop," Environmental Health Perspectives, 1986, 68: 155-173.

Spirtas R, Steart PA, Lee JS, et al., "Restrospective cohort mortality study of workers at an aircraft maintenance facility" and "I. Epidemiological results," British Journal of Industrial Medicine, 1991, 48: 515-530.

Stark M, Wolff J, and Korbmacher A, "Modulation of glial cell differentiation by exposure to lead and cadmium," Neurotoxicology and Teratology, 1992, 14(4): 247-252.

Steck P and Saya H, "Pathways of oncogenesis in primary brain tumors," Current Opinions in Oncology, 1991, Jun 3(3): 476-484.

Stroup NE, Blair A, and Erikson GE, "Brain Cancer and Other Causes of Death in Anatomists," Journal of the National Cancer Institute, 1986, 77(6): 1217-1224.

Thomas T, Fontham E, Norman S, Stemhagen A, and Hoover R, "Occupational risk factors for brain tumors. A case-referent death-certificate analysis," Scandinavian Journal of Work, Environment and Health, 1986, 12(2): 121-127.

Thomas TL and Waxeiler J, "Brain Tumors and Occupational Risk Factors," Scandinavian Journal of Worker and Environmental Health, 1986, 12: 1-15.

Thomas TL, Stewart PA, Stemhagen A, et al., "Risk of astrocytic bran tumors associated with occupation chemical exposures," Scandinavian Journal of Worker and Environmental Health, 1987a) 13: 417-423.

Thomas TL, Stolley PD, Stemhagen A, et al., "Brain Tumor Mortality Risk among Men with Electrical and Electronics Jobs: A Case-control Study," Journal of the National Cancer Institute, 1987b, 79(2): 233-238.

Ulland B, Finkelstein M, and Weisburger E, "Carcinogenicity of industrial chemicals propylene imine and propane sultone," Nature, 1971, 230: 460-461.

Walsh J, Zimmerman S, and Perdue M, "Role of viruses in the induction of primary intracranial tumors," Neurosurgery, 1982, 10: 643-662.

Ward J and Rice J, "Naturally occurring and chemically induced brain tumors of rats and mice in carcinogenesis bioassays," Annals of New York Science Academy, 1982, 381: 304-309.

Wrensch M, Bondy M, Wieneke J, and Yost M, "Environmental risk factors for primary malignant brain tumors: a review," Journal of Neuro-Oncology, 1993, 17: 47-64.

Appendix A. Epidemiological Concepts

Epidemiology is "the study of the distribution and determinants of diseases and injuries in human populations (Mausner & Kramer 1985)." By studying patterns of disease occurrence and risk in populations, epidemiologists help to shed light on risk factors and causes of disease that can lead to developing strategies for intervention and prevention. Epidemiologists do their work by conducting studies. There are several different types of studies that epidemiologists may conduct. Different types of studies serve different purposes. In the following paragraph we briefly describe the different types of studies that are used in the research on GBM.

Study Design

Descriptive studies are the most basic and easily conducted epidemiological study. In a descriptive study, the characteristics of people with a disease are described, such as their age, occupation and gender. Several of the studies reviewed here are descriptive; they simply describe the characteristics people with GBM or other astrocytic tumors. An example of a descriptive study is the paper that reports that patients with GBM disproportionately resided in agricultural and wood products counties. This finding alone is not powerful enough to confirm that residing in agricultural and wood products counties is a *cause* of GBM. However it could point the way to other investigators to examine in more depth potential associations between exposure to herbicides and wood products manufacturing.

Several of the studies included in this review are retrospective cohort studies. These studies compare a "cohort" or group of individuals who have had similar exposures, such as workers in petroleum refineries, to a comparable cohort without the exposure. The risk of disease in the exposed group is compared to the unexposed group. This type of study can tell us if, for example, petroleum workers have a higher risk of brain cancer than similarly aged men in a different occupation. One of the potential weaknesses of retrospective studies is that the researcher is trying to quantify exposures that may have occurred many years ago. This can be challenging when accurate records don't exist.

Many studies described here are case-control studies. This type of study is useful for examining risk factors for relatively rare diseases. In a case-control study, people with the disease (cases) are compared to people who don't have the disease (controls). A comparison of presence or absence of risk factors is made, typically through an interview with the individual or next-of-kin. An example of this is the Olin study that identified people with GBM (cases) and compared their risk factors to people without GBM.

Both retrospective cohort and case-control studies are considered analytical designs. The choice to conduct these types of more intensive, expensive studies is often based on a hypothesis that the particular risk factors under investigation are related to development of the disease. Both case-control and cohort studies can be powerful tools for identifying risk factors and assessing causality.

Each study that is reported here includes a description of the study design. This information is intended to aid the reader in understanding the potential significance of the findings.

Criteria for Causal Association

Epidemiological studies may identify many risk factors for a disease. However, it's likely that only some are actually capable of causing the disease in question. In order to evaluate the potential that a risk factor may cause a disease, epidemiologists use the following criteria:

Strength of an Association: How strong is the association between the exposure and the disease? This is usually measured by relative risk, which compares the risk of disease in the exposed population to the risk of disease in the population that is not exposed. An example of a strong association between exposure and disease is cigarette smoking and lung cancer. Numerous studies have examined rates of lung cancer in smokers (exposed) versus non-smokers (unexposed). These studies have consistently found that smokers are much more likely to die of lung cancer than non-smokers.

Dose-Response Relationship: Is there a dose-response relationship between exposure and disease? That is, as the level of exposure increases does the risk of disease also increase? For our example of cigarette smoking, we would examine whether or not the risk of lung cancer increases with number of cigarettes smoked. Research has shown that the risk of lung cancer varies according to the number of cigarettes smoked: the lowest risk is found in occasional smokers while very heavy smokers are at high risk.

Consistency. Is the association found in one study observed in others? The more often exposure is associated with disease and the more circumstances in which this observation is made, the more likely a relationship is to be causal. The results of one study are never considered strong enough to determine causality More than one study is always necessary to rule out chance or error. The smoking example also satisfies this criterion since numerous studies in diverse populations have demonstrated increased risk of lung cancer due to smoking.

Temporally correct. Does the exposure precede onset of the disease? In order to establish causality studies must demonstrate that exposure came before the disease. To continue the lung cancer example, smoking must occur before the development of lung cancer in order for smoking to be causal. This criterion is easily met since the exposure is readily observable and known. For some causes of disease this criterion can be fairly straightforward to satisfy. For example, when the time between exposure and onset is relatively short as with many food-borne illnesses it's easier to pin-point the cause of the illness. However, with many environmental factors where the time between exposure and onset of disease can be decades, this criterion can be difficult to satisfy.

Specificity. Is the exposure necessary and sufficient to cause the disease? In the case of lung cancer, is smoking cigarettes necessary for developing lung cancer and, is smoking alone capable of causing lung cancer. This is the most difficult criterion to satisfy since different etiologic agents may cause the same disease. However, over 90% of lung cancers occur in current or former smokers.

Biologic Plausibility: Is it biologically plausible that the exposure could cause the disease? Are there animal or laboratory studies that have shown that the agent is capable of causing the disease? In the case of cigarette smoke, there are many carcinogens that could potentially cause lung cancer

APPENDIX B. GRADING AND CLASSIFICATION OF BRAIN CANCER

Brain cancer reported to the Washington State Cancer Registry is classified using the International Classification of Diseases-Oncology codes (ICD-O codes). Glioblastoma is a sub-type of the broader category, glioma (see Table 1 for the categories of brain cancer reported to the registry), a cancer of the cells that support neurons, or nerve cells. (Cancers do not arise in the nerve cells themselves.) Gliomas are a common type of brain cancer, accounting for approximately 42% of cases in women and 59% in men (LA County data). The majority (83%) of gliomas are astrocytic gliomas, that is they affect the astrocyte cells of the brain.

Although currently there are different schemes for classifying astrocytomas, lower-grade (or less aggressive) astrocytic gliomas are typically classified as *astrocytomas* and higher-grade (more aggressive) astrocytic gliomas are classified as *glioblastomas.* Historically, several pathology grading systems have been in use describing astrocytic brain tumors. Although recently, the trend has been to use either of two systems (the WHO definition of the Daumas-Duport method), there is still debate among pathologists about classification lower-grade tumors (Prados, 1998), and disagreement on the classification of glioblastoma. For instance, using the Daumas-Duport method, there is some disagreement as to whether both grade III and IV astrocytic gliomas should be classified as glioblastoma (Schottenfeld, 1996) or only grade IV.[2] Astrocytomas are usually classified as grades I and II astrocytic gliomas (Schottenfeld, 1996). In addition, there appears to be some inconsistency in the categorization of astrocytic tumors in cancer data sets in different geographic areas, based on the observation that there is regional variation in the proportion of brain cancers categorized as astrocytic gliomas versus glioblastomas (Preston-Martin, 1996).

Table 1. Pathologic classification of tumors of the central nervous system*

Histologic type

Gliomas
- Astrocytoma
- Glioblastoma multiforme
- Ependymoma
- PNET
- Oligodendroglioma
- Other gliomas

Meningioma

Nerve sheath tumors

Other/Unspecified

*adapted from Schottenfeld, 1996

[2] The American Brain Tumor Association supports a Word Wide Web site that provides the following listing (from which this information is drawn): "Types of Brain and Spinal Cord Tumors and Tumor-like Conditions." The Web address is http://www.abta.org/types.htm.

APPENDIX C: P53 TUMOR SUPPRESSOR GENE: CLUES TO CANCER ETIOLOGY

Recent advances in molecular toxicology have shed some light on genetic and environmental factors in the development of brain tumors in people. Some carcinogens cause typical, or even specific, changes in certain genes and thereby, allow conclusions regarding the sources of the cancer causing substances (Kleihues et al. 1995). To understand this process, one must first understand the role of the tumor suppressor gene *p53*. The following is background information on the tumor suppressor gene *p53* and its role in human brain tumors.

Cancer can be thought of simply as uncontrolled cell growth and division. Normally cells are in a resting phase called G_0. After receiving signals to divide, cells progress through several phases before becoming two new cells. Progression through the cycle of cell division can be slowed down by activation of a gene known as the tumor suppressor gene *p53*. The activation of this gene allows time for repair of errors in the DNA molecule before the cell completes the cycle ending in cell division. This repair is important, because errors in the DNA may lead to genetic damage, resulting in diseases such as cancer.

Gene mutations are changes in the DNA sequence in a gene. Inherited genetic mutations which inactivate this suppressor gene are responsible for some kidney tumors in children (Wilms' tumor), familial polyposis which increases risk for colon cancer, and Li-Fraumeni syndrome (an inherited trait that predisposes individuals to the development of several types of cancer, including brain tumors, early in life). Mutations of the *p53* gene are among the most common lesions in a variety of human tumors, including those of the central nervous system. Loss or a mutation of the *p53* gene has been detected in many types of glioma including glioblastoma. (Bogler 1995)

Two principal kinds of gene mutations are base-pair substitutions and frame-shift mutations. Of particular importance here is the former. A base pair refers to a purine (adenine or guanine) that is paired with a pyrimidine (cytosine, thymine or uracil) in the DNA molecule. There are two types of base-pair substitutions resulting in transitional mutations and transversional mutations. In transitional mutations, one purine is replaced by the other purine or the pyrimidine is replaced by another pyrimidine. In transversional mutations, a purine is replaced by a pyrimidine or vice versa. Transitional mutations in the *p53* gene reflect causes of cancer arising from normal biological processes within an individual, whereas transversional mutations are known to be associated with external causes of cancer. Thus, if one determines whether the *p53* gene mutations are transitional or transversional, one can determine whether the origins of the mutations that gave rise to the cancer were caused by normal biological processes or external factors. (Hollstein et al. 1991, Harris 1996, Bogler et al.1995, McMahon 1994, Greenblatt et al.1994, Kleihues et al. 1995, and Li et al 1998).

In astrocytic brain tumors (including glioblastoma), *p53* mutations are mainly reported to be transitional mutations that can best be explained as being due to endogenous factors and not caused by environmental carcinogens. (Kleihues et al. 1995, Bartsch and Hietanen 1996, Steck and Saya 1991, and Greenblatt et al. 1994). However, one recent study screening more of the *p53* gene found a higher prevalence of transversional mutations, suggesting that exposure to exogenous environmental factors should be considered in the etiology of malignant gliomas (Li et al. 1998). Further scientific studies are needed to clarify this question.

Table 1. Studies of Glioblastoma Risk Factors										
Primary author, year published	***Country***	***Disease***	***Time period***	***Population Age***	***Population Gender***	***Study type***	***Factors examined***	***Number of cases***	***Findings***	***Notes***
<u>STUDIES ON OCCUPATIONAL/ENVIRONMENTAL RISK FACTORS</u>										
Ahlbom, 1986	Sweden	Astrocytoma (mostly GBM)	Deaths, 1980-1981	20-75	Both	Case-control	Environmental and behavioral	78 (72 were astrocytomas grade III or higher)	Elevated risk for: --ate smoked ham at least one a week --lived at least 5 years "in vicinity of farm" --exposure to herbicides or insecticides (not further characterized)	Findings on smoked ham consistent with N-nitroso compound causal factor. Other studies have found brain cancer elevated in farmers (see Authors do not explain how herbicide exposure measure was constructed.
Alexander, 1980	US (TX)	Brain cancer and GBM	Yr of death: 1962-1980	30-66	Males	Case series	Occupation	18 brain cancer; 15 gbm	Possible excess mortality from brain cancer No particular occupation suspected	Follow-up investigation promised but apparently not published Specific exposure data lacking
Bakshi, 1991(letter)	US	Glioblastoma	1985-1990	48, 49 & 54	Not stated	Case series	Occupation	3	3 opthamologists with GBM, 0.21 expected	
Gold, 1998	US	All cause mortality; GBM	?	Mean age at death: 64	Male (all but 5)	Biased cohort	Occupational factors	260 from all causes; 6 astrocytoma or gbm	For astrocytoma/gbm deaths: --all worked at one facility --no apparent relationship with radiation exposure	Cohort is self-selected, so not "unbiased" Only radiation monitoring was done, so other exposures in these cases are not reported and are unknown
Morantz, 1985	US (MO)	Brain cancer and GBM	Yr of dx: 1974-1982	28-68	Both	Cross-sectional; Case series	Env. & occ.	6 gbm; 1 malignant ependymoma	Community GBM mortality 2X expected History of: --Living near a chicken factory --Employment (inc. spouse) in shoe factory --Fishing in coal mining strip pits was common among cases	Possible pesticide exposure from chicken farming? Possible solvent exposure from shoe factory? (A later case control study failed to implicate these possible exposures.) Of 6 cases, 2 were siblings. Follow up investigation promised but apparently not publ
Morrison, 1992	Canada	Brain cancer and GBM	1971-1987	35+	Males	Cohort	Farming practices	210 brain cancer, 69 gbm	Increasing GBM mortality with: Increasing fuel expenditures Increasing number of dairy cattle + (ns) Increasin number of poultry + (ns)	Exposure to fuels and oils? Exposure to insecticides from dairy and poultry herds? However, measure of exposure is indirect at best

Olin, 1987	Sweden	Astrocytoma (mostly GBM)	Deaths, 1980-1981	20-75	Both	Case-control	Occupational and residental	78 (72 were astrocytomas grade III or higher)	Elevated risk for: --Worked at airfield --Lived near petrochem plant --Lived near pharm. plant --Lived near saw mill/lumber yard	Of 78 cases, 72 were astrocytomas grade III or higher (GBM) Elevated airfield risk sig in pop controls but not hospital patient controls Cases working at airfield were all military Cases working at airfield were exposed during or shortly after WWII
Smith-Rooker, 1992	US (AK)	GBM	1984-1991	21-78 (mean of 50)	Both	Case series	Residence and occupation	100	A high percentage of cases: --Lived in counties where rice/cotton are cultivated or with wood industries --Had agricultural or wood products occupations	Since the distribution of population by county or occupation is not evaluated or given, results are difficult to evaluate
STUDIES ON OTHER RISK FACTORS										
Cantor, 1993	US (IA)	Brain cancer (and other sites)	1984-1987	40-85	Female	Case-control	Reproductive history	169 brain cancer, with unknown distribution of astrocytoma and gbm.	Ever-parous women had a significantly lower risk of gbm and brain cancer overall, and a nonsignificant lower risk of astrocytoma	Occupation and education were not controlled for in the analysis, although they were collected. Education is a potential confounder that could have artificially depressed the odds ratio calculated.
Helseth, 1989	Norway	CNS cancer	?-1987	?	Both	Case-control	Pre-morbid height and weight	1538 CNS; 581 GBM	Positive relationship between GBM and increasing height in men.	Findings may be related to incrasing brain size with height, thus increasing chance for tumor to grow. Findings are limited; height is confounded with socioeconomic status.
Hochberg, 1984	US	GBM	1977-1981	15-81	Both	Case-control	Head trauma	160	Increased risk of GBM in those with a history of head trauma, esp. severe trauma. Markedly increased risk for those reporting a history of seizures.	Pathway of increased risk is speculative; unable to separate direct effect of trauma from medical treatment (e.g., effects of exposure to x-rays in severe trauma cases or medicinal treatment).
Hochberg, 1990	US	GBM	1977-1981	15-81	Both	Case-control	Range of non-occupational risk factors	160	Negative except for suggestion of decreased risk for those who never received treatment for allergies (although this finding is stated unclearly).	An apparent contradiction in presentation of the results on allergy is confusing and hard to interpret.
Hodges, 1992	US	GBM	Not stated	25-75	Female	Case series	Exposure to therapeutic radiation	100 (17 exposed to radiation)	Four cases were attributed to radiation based on authors' criteria	

Chemicals in Jet Fuel Emissions
(Question 8 of the August 1998 Work Plan)

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Background

In response to community concerns about health around SeaTac International Airport, Senator Julia Patterson arranged two meetings in 1998 with community residents, the Washington State Department of Health (DOH), Seattle-King County Department of Public Health (SKCDPH) and other interested parties. Those meetings and preliminary DOH findings of elevated glioblastoma for 1992 through 1995 in an area roughly 3 miles around the airport led to Senator Patterson's request that DOH work with SKCDPH and the community to develop a work plan to address the community's concerns.

Community representatives presented a list of 18 questions they wanted addressed in the work plan. The August 1998 work plan was divided into two phases. Answers to questions from Phase 1 activities were necessary to determine the value and feasibility of Phase 2 activities. Phase 1 activities included 10 questions. This report focuses on question 8 in the work plan. Question 8 asked, "**What are the chemicals in jet engine exhaust emissions and what happens to them after they are emitted?"**

Components of Jet Fuel

Jet fuel, like other petroleum products, is derived from crude oil. It is a complex mixture of hundreds of different types of hydrocarbon compounds. The size and arrangement of the molecules differ for different petroleum products. During the refining process, crude oil is separated into fractions that have similar boiling points, modified further through a variety of chemical processes, and then formulated into commercial products such as gasoline, jet fuel, and fuel oil. Jet fuels are kerosene-based. This means they consist of hydrocarbon compounds ranging from 9 to 16 carbons in length. Most of these hydrocarbons are considered to be either volatile organic compounds (VOCs) which can evaporate easily or semi-volatile compounds (SVOCs) which evaporate less easily. For comparison, gasoline is also derived from crude oil and consists of hydrocarbon compounds with lengths ranging from 4 to 12 carbons.

The major types of hydrocarbons in the jet fuels used in commercial aircraft are alkanes, cycloalkanes, and alkenes (ATSDR 1993, Smith et al. 1997). These give the fuel its characteristic kerosene odor. These compounds have a great deal of stored energy and burn cleaner than do aromatic hydrocarbons. Aromatic hydrocarbons (e.g., toluene, xylene, and benzene) and polycyclic aromatic hydrocarbons (e.g., benzo(a)pyrene, and chrysene) are also found in jet fuels. Aromatic hydrocarbons usually make up only a small fraction of jet fuel. Jet fuel also contains additives which are included for various reasons including metal deactivators, antioxidants, static dissipaters, octane boosters, and corrosion and ice inhibitors (ATSDR 1995, ATSDR 1998a, and ATSDR 1998b).

Pollutants Emitted or Formed from Aircraft Engines

Jet fuel emissions include the evaporation of fuel during storage and fuel handling, such as fueling of aircraft, as well as jet engine exhaust emissions. Jet fuel, which evaporates prior to being burned, contains numerous VOCs and SVOCs mentioned above (EPA 1994).

Jet engine exhaust contains the same type of air pollutants as car, truck and bus engines. Gaseous emissions are composed principally of carbon monoxide (CO) and hydrocarbons. As with cars, these emissions result from incomplete combustion. With aircraft, VOCs and CO are primarily produced during lower power settings for descent or when idling or moving on the ground. Other major gaseous pollutants are oxygenated organic compounds (such as, formaldehyde) and oxides of nitrogen (NO_x). NO_x is produced when engines are at their hottest, such as during takeoff. Carbon is an important particulate emission. Carbon is found in the form of smoke, the major particulate emission in jet engine exhaust. Engine smoke is composed for the most part of fine particles of nearly pure carbon with diameters of 0.6 microns or less (US DOC 1970).

In addition to pollutants released from either evaporation or through incomplete combustion, NO_x and VOCs can combine in the atmosphere to form ground-level ozone, the primary component of smog (NRDC 1996). Formation of ozone usually occurs during periods of strong sunlight during summer months. Interestingly, in the central Puget Sound area, the highest concentration of ozone is not usually seen where the NO_x or VOCs are emitted. This is due, in part, to the fact that ozone formation requires time. In the central Puget Sound area, generally the highest ozone measurements are seen in the foothills of the Cascades (Ecology, 1998; PSAPCA1999).

Summary

To date, no pollutant unique to aircraft emissions has been identified. All pollutants emitted by airplanes are also emitted by cars and trucks. The following are the major emission products released from the evaporation of jet fuel or emitted from jet aircraft engines. This list names general classes of compounds. Within a given class, there are numerous chemicals. The general classes and examples of specific chemicals are:

- **Inorganic gases**, including carbon monoxide (CO), carbon dioxide (CO_2) and nitrogen oxides (NO, NO_2, NO_X);
- **Volatile Organic Compounds (VOCs)**, including hydrocarbon compounds such as alkanes, cycloalkanes, alkenes and aromatic hydrocarbons, which include compounds such as pentane, butane, acetylene, naphthalene, 1,3-butadiene, benzene, toluene, and xylene;
- **Oxygenated organics**, including a variety of carbonyl compounds such as aldehydes which include compounds such as formaldehyde and acetaldehyde; and
- **Aromatic hydrocarbons**, including polycyclic aromatic hydrocarbons (PAHs), such as benzo(a)pyrene, chrysene, benzo(a)anthracene, and fluoranthene.

References

ATSDR 1993. Case Studies in Environmental Medicine: Jet Fuel Toxicity. US Department of Human Health Services Public Health Service, Agency for Toxic Substances and Disease Registry. September 1993.

ATSDR 1995. Toxicological Profile for Fuel Oils. US Department of Human Health Services Public Health Service, Agency for Toxic Substances and Disease Registry. June 1995.

ATSDR 1998a. Toxicological Profile for Jet Fuels (JP-5 and JP-8). US Department of Human Health Services Public Health Service, Agency for Toxic Substances and Disease Registry. August 1998.

ATSDR 1998b. Toxicological Profile for Total Petroleum Hydrocarbons (THP). US Department of Human Health Services Public Health Service, Agency for Toxic Substances and Disease Registry. September 1998.

Ecology 1998. Ozone. Washington State Department of Ecology Publication No. FA-93-07. May 1998. Available at http://www.wa.gov/ecology/air/focusoz.htm.

EPA 1994. Mobile Source Hazardous Air Pollutant Emissions in the Seattle-Tacoma Urban Area. US Environmental Protection Agency, Air and Energy Engineering Research Laboratory. EPA/600/A-95/024.

NRDC 1996. Flying Off Course: Environmental Impacts of America's Airports. Natural Resources Defense Council. October 1996.

PSAPCA 1998. Ozone. Puget Sound Air Pollution Control Authority.Updated June 1998. Available at http://www.psapca.org/compol.htm.

Smith LB, Bhattacharya A, Lemasters G, Succop P, Puhala E, Medvedovic M, and Joyce J. Effect of Chronic Low-level Exposure to Jet Fuel on Postural Balance of US Air Force Personnel. Journal of Occupational and Environmental Medicine. July 1997;39(7):623-632.

US DOC 1970. Air Pollution by Jet Aircraft at Seattle-Tacoma Airport. US Department of Commerce, Environmental Science Services Administration, Weather Bureau. Weather Bureau Technical Memorandum WR-58. October 1970.

Health Profile for the SeaTac Airport Community

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DATA SUMMARY
Health Profile for the SeaTac Airport Community

People of All Ages

- The percent of the population living in poverty and without a high school diploma is higher in the SeaTac Airport Community compared to King County as a whole. Although SeaTac is diverse, it is predominantly a blue-collar community: in 1990, seven of 10 SeaTac adults were in working-class occupations.
- Chronic diseases such as heart disease, stroke, cancer and chronic obstructive pulmonary disease (COPD) are the leading causes of death in SeaTac and King County.
- Cancer of the lung and chronic obstructive pulmonary disease, two diseases closely linked with cigarette smoking, were elevated in the SeaTac Airport Community compared to the County as a whole. Lung cancer is the leading cause of cancer death in SeaTac, King County and the U.S.
- There is no adult smoking data specifically for the SeaTac community. However, the percent of adults in South County as a whole who were smokers is somewhat higher compared to King County (24% and 19%, respectively).
- AIDS death rates are lower in SeaTac than in the County, and have turned downward in recent years.
- Firearm-related deaths (suicide and homicide) were 50% higher in the SeaTac Airport Community compared to King County.

Maternal and Child Health

- Women giving birth were more likely to have had late prenatal care compared to their King County counterparts.
- Women giving birth were also more likely to have smoked during pregnancy.

Children and Younger Adults

- The birth rate in adolescents age 15 to 17 was almost twice as high in SeaTac compared to King County.
- Teens and younger adults (age 15 to 24 and 25 to 44) had higher hospitalization rates for illicit drug use, while younger adults had higher hospitalization rates for alcohol abuse.
- Teens and young adults aged 15 to 24 had higher incidence rates of chlamydia and gonor-rhea.
- Children under 18 were more likely to be hospitalized for asthma and other respiratory diseases.

Older Adults

- Adults 65 and older had lower death rates than their King County counterparts for heart disease and stroke.
- Hospitalization rates for unintentional injury and stroke were also lower than for King County for those aged 65 and older.
- However, death rates for chronic obstructive pulmonary disease was higher among older SeaTac residents compared to King County.

Health Profile for the SeaTac Airport Community

Prepared by
Seattle-King County Department of Public Health
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February 1999

Introduction

In response to a request for an overall community health assessment from residents living near SeaTac Airport, the Seattle-King County Department of Public Health has analyzed a range of data in order to create a profile of the health of this community. The result is the following report which identifies the major causes of death and illness, as well as other health indicators, in the SeaTac Airport Community, and compares them to King County as a whole. For the sake of brevity, the community will sometimes be referred to as "SeaTac", although it should not be confused with the City of SeaTac. A precise description of the geographical boundaries of the community is provided in Technical Appendix A.

The selection of health indicators, or events, examined in this report is limited to data currently available to the Seattle-King County Department of Public Health. For example, while data about the number of deaths related to diabetes are readily available from death certificates, there is currently no way to count the number of living people who have at some point been diagnosed with diabetes.

The results of the primary data analysis can be found in Technical Appendix A. The body of this report contains a summary of the most notable findings, although it is by no means comprehensive. Indicators were chosen for special consideration when statistical tests showed that there was either a significant difference between the occurrence of that indicator in the SeaTac Airport Community and the county as a whole, or there has been a significant change over time in its occurrence. The term "significant" as used throughout the report refers to this statistical definition and is not meant as a judgement about the severity of a problem.

Further information about statistical methods, rate calculation, and data sources is provided in Appendices B and C.

For additional information, or assistance in interpreting the data included, please contact the Seattle-King County Department of Public Health's Epidemiology, Planning and Evaluation Unit at 296-6817.

Health Profile Summary

Population Estimates
(Appendix A – Table 1)

- In 1997, the total population of the SeaTac Airport Community was 98,608 out of a total King County population of 1,652,775.
- The population distribution of SeaTac closely reflected that of King County as a whole in terms of the age, gender, and race/ethnicity of its residents.

Figure 1: Population Distribution of SeaTac Airport Community By Age and Gender, 1997

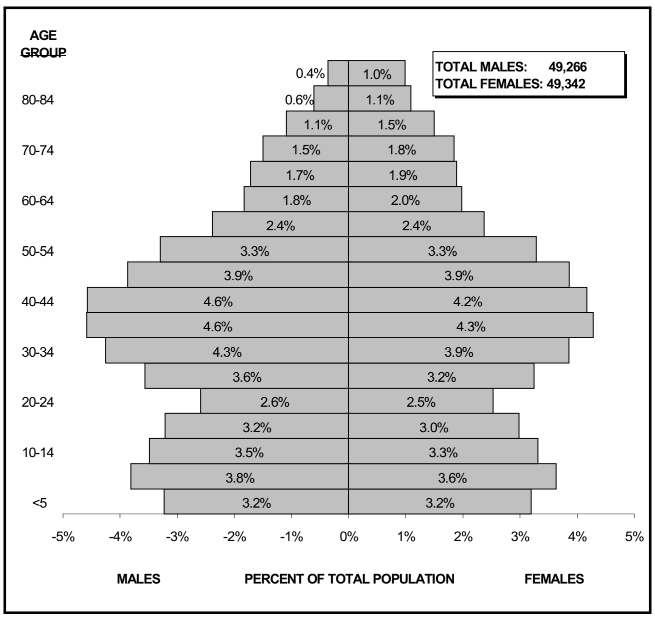
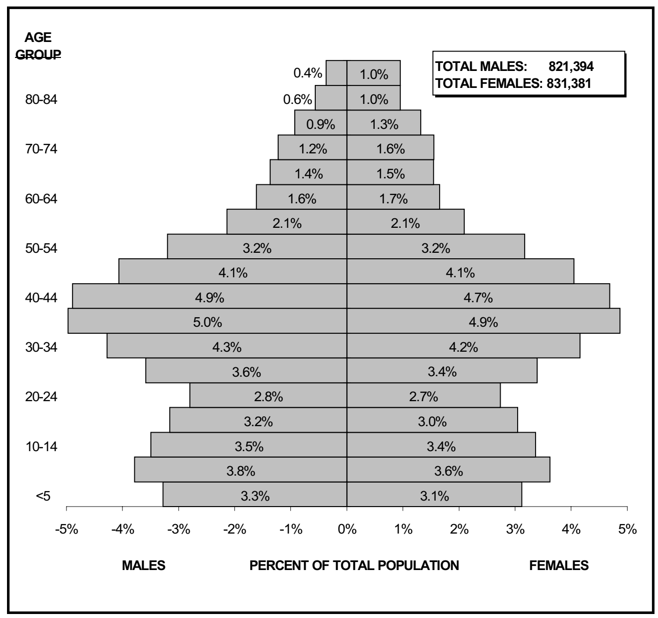


Figure 2: Population Distribution of King County By Age and Gender, 1997

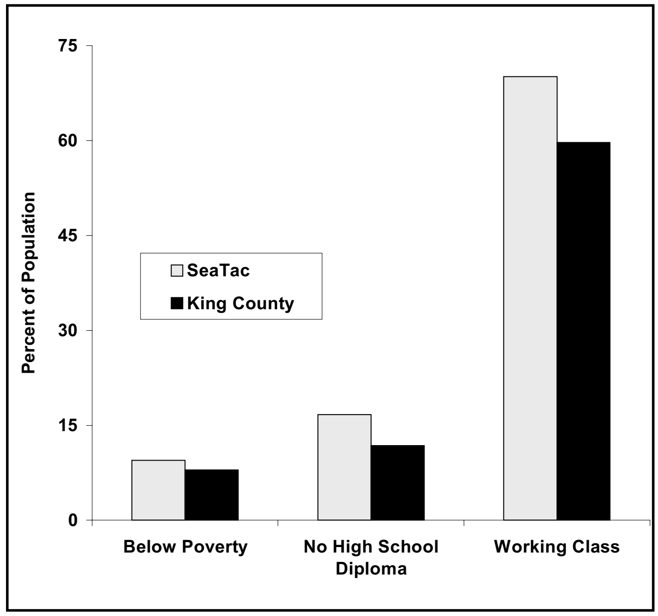


Socioeconomic Status
(Appendix A – Table 1)

- Data from the 1990 U.S. Census indicates that there was a higher percentage of persons below the federal poverty level in SeaTac (9.5%) than in the whole county (8%), particularly among the Asian/Pacific Islander and African American populations.

- As of 1990, the percent of persons age 25 and over without a high school diploma was higher in the SeaTac Airport Community (16.7%) than in King County (11.8%).
- The percentage of the SeaTac population who were working class in 1990 (70.1%) also exceeded the county percentage (59.7%).

Figure 3: Socioeconomic Measures
SeaTac Airport Community and King County
1990

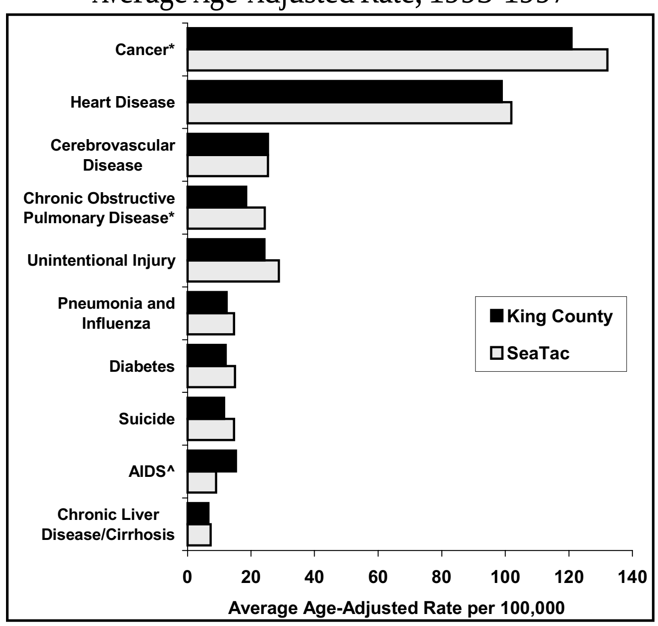


Leading Causes of Death

(Appendix A – Tables 2, 3, 6, 8 & 10)

- Between 1993 and 1997, six chronic illnesses were among the top ten leading causes of death for all age groups in SeaTac. They were cancer, heart disease, cerebrovascular disease (stroke), chronic obstructive pulmonary disease, diabetes, and chronic liver disease.
- Slightly more than one half of all deaths between 1993 and 1997 in SeaTac (52%) were from cancer and heart disease combined (51% in King County).
- Other leading causes of death among all ages in SeaTac included unintentional injury, pneumonia/ influenza, suicide, and AIDS.
- While the leading causes of death were the same in SeaTac and King County between 1993 and 1997, there were some significant differences in the death rates between the two areas. Death rates from cancer, and chronic obstructive pulmonary disease were significantly higher in SeaTac than in the county as a whole. The overall death rate was also higher in SeaTac.
- The elevation in the overall cancer death rate in SeaTac over King County is mainly due to respiratory cancer. There was no significant difference in the death rates from any other major cancer type.

Figure 4: Leading Causes of Death
SeaTac Airport Community and King County
Average Age-Adjusted Rate, 1993-1997



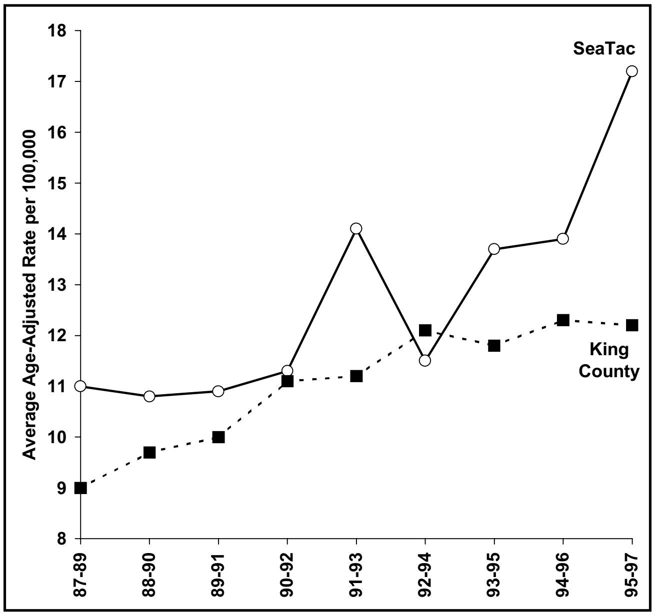
* Statistically significantly higher in SeaTac Airport Community than in King County.
^ Statistically significantly lower in SeaTac Airport Community than in King County.
"Diabetes" includes only deaths for which diabetes was the underlying (primary) cause.

- Respiratory cancer is the leading cause of cancer death in the SeaTac community, King County, Washington State and the U.S.
- In SeaTac, respiratory cancer was followed by breast cancer in women, colorectal cancer among men and women, and prostate cancer in men as the leading causes of cancer death.
- Among children (age 0-17), the three leading causes of death in SeaTac and the county between 1993 and 1997 were unintentional injuries (primarily motor vehicle accidents), homicide, and cancer.

Trends in Selected Causes of Death

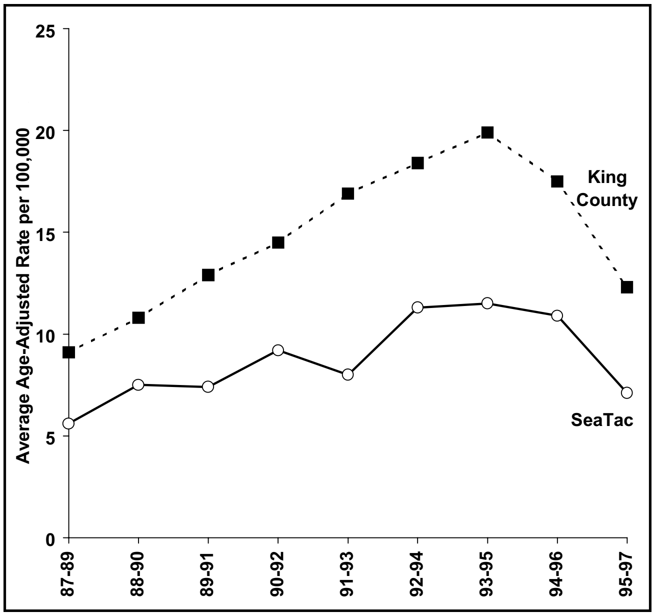
- Most of the leading causes of death in the SeaTac Airport Community have not significantly increased or decreased over the last decade. The exceptions are heart disease, diabetes, and AIDS. Heart disease deaths have declined in both SeaTac and King County since 1987.
- Diabetes deaths increased significantly in both SeaTac and King County from 1987 to 1997.
- "Diabetes deaths" includes only those deaths for which diabetes was determined to be the primary underlying cause. These numbers underestimate the total impact of diabetes on mortality because they exclude deaths from other primary causes, such as stroke, to which diabetes was a contributing factor.
- Furthermore, it is estimated that diabetes is cited as either an underlying or contributing cause on the death certificates of only 50% of all people who had diabetes.

Figure 5: Diabetes Deaths
SeaTac Airport Community and King County
3 Year Rolling Averages, 1987-1997



"Diabetes" includes only deaths for which diabetes was the underlying (primary) cause.

Figure 6: AIDS Deaths
SeaTac Airport Community and King County
3 Year Rolling Averages, 1987-1997



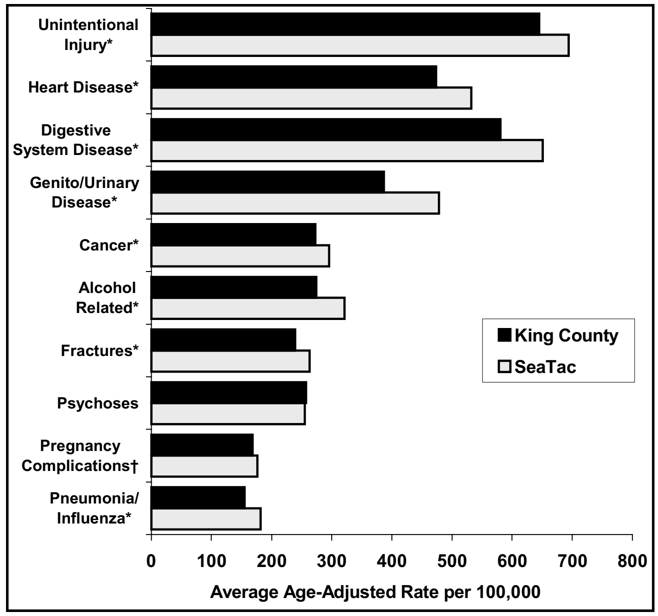
- Between 1993 and 1997, the rate of death from AIDS was significantly lower in SeaTac than in the county. AIDS death rates have been decreasing in SeaTac since 1994 (and in King County since 1995).

Leading Causes of Hospitalization

(Appendix A – Tables 4, 5, 6, 7, 8, 10 & 11)

- From 1992 to 1996, the top ten causes of hospitalization in SeaTac and the county as a whole were unintentional injury, heart disease, digestive system disease, genito/urinary disease, cancer, alcohol-related, fractures, psychoses, pregnancy complications, and pneumonia/influenza.
- Genito/urinary disease hospitalizations in SeaTac are comprised primarily of disorders of female genital organs such as endometriosis (49%) and disorders of the kidneys and urinary tract (37%).
- Hospitalization rates for all of these causes were significantly higher in SeaTac than in King County, except for psychoses and pregnancy complications (no statistical difference). The overall hospitalization rate was also higher in SeaTac.

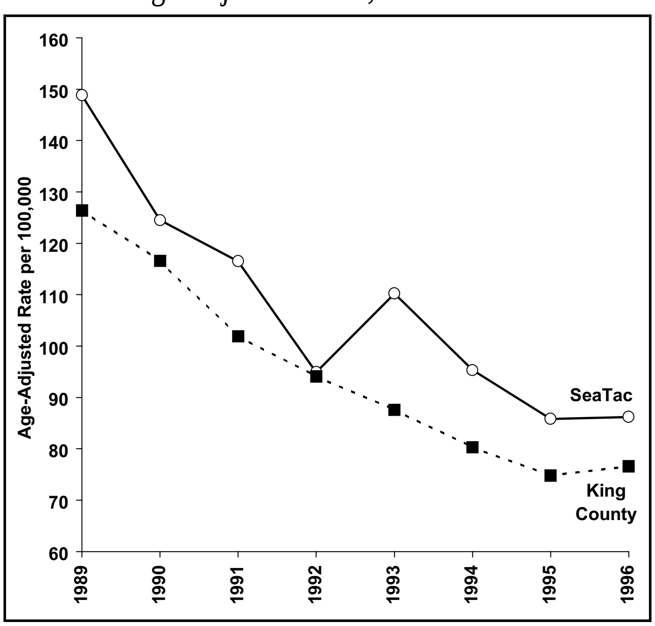
Figure 7: Leading Causes of Hospitalization
SeaTac Airport Community and King County
Average Age-Adjusted Rates,1992-1996



* Statistically significantly higher in SeaTac Airport Community than in King County.
† Rate is per 1,000 live births, not per 100,000 total population.

Trends in Selected Causes of Hospitalization

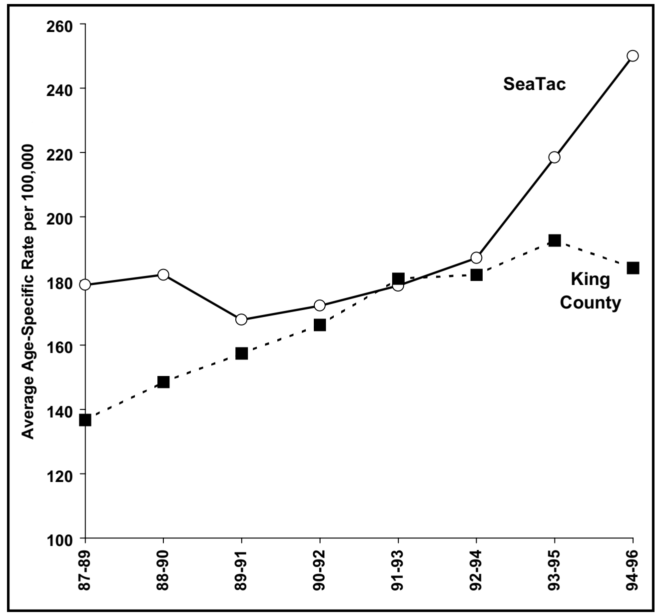
Figure 8: Hospitalizations for Motor Vehicle Accident Injuries
SeaTac Airport Community and King County
Age-Adjusted Rates, 1989-1996



- The two major types of unintentional injury hospitalizations for all ages are falls, accounting for most hospitalizations, and motor vehicle crashes. In the 15-24 age group, however, motor vehicle crashes precede falls as the greatest cause of unintentional injury.
- Between 1992 and 1996, the rate of hospitalization for motor vehicle crashes was significantly higher in SeaTac than in King County. Motor vehicle crashes are reported here by the residence of the person injured, rather than by the location of the crash.
- Rates of hospitalization in SeaTac and King County for unintentional injuries, and motor vehicle accidents specifically, declined significantly between 1989 and 1996.

- Hospitalization rates for pneumonia/influenza and asthma (constituting 36% and 19% of respiratory disease hospitalizations for all ages, respectively) were also significantly higher in SeaTac than in the county among the 0-17 age group.
- From 1987 to 1996 asthma hospitalization rates among 0-17 year olds increased in both SeaTac and King County.
- In addition to asthma, the other four leading causes of hospitalization in SeaTac for children age 0-17 were unintentional injuries, digestive system disease, infections, and perinatal conditions (ie. related to birth).
- These were the same five leading causes of hospitalization among children in the entire county.

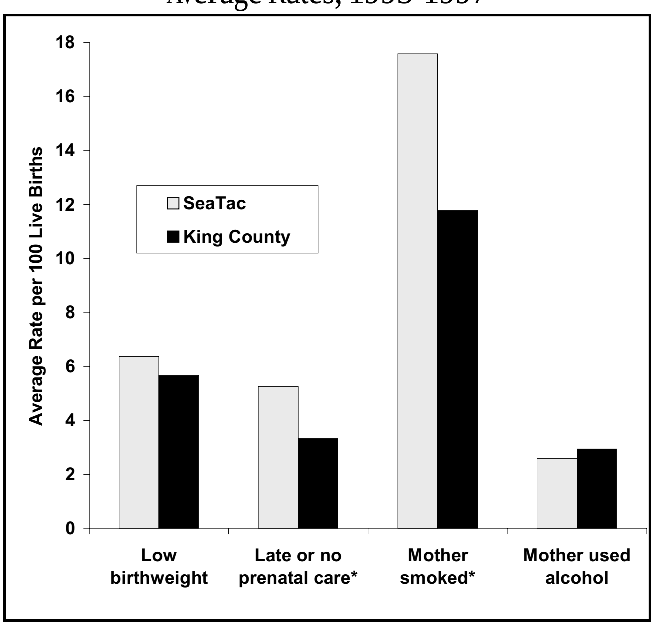
Figure 9: Hospitalization for Asthma Among 0-17 Year Olds
SeaTac Airport Community and King County
3 Year Rolling Averages, 1987-1996



Maternal and Child Health Indicators

(Appendix A – Table 9)

Figure 10: Maternal and Child Health
SeaTac Airport Community and King County
Average Rates, 1993-1997



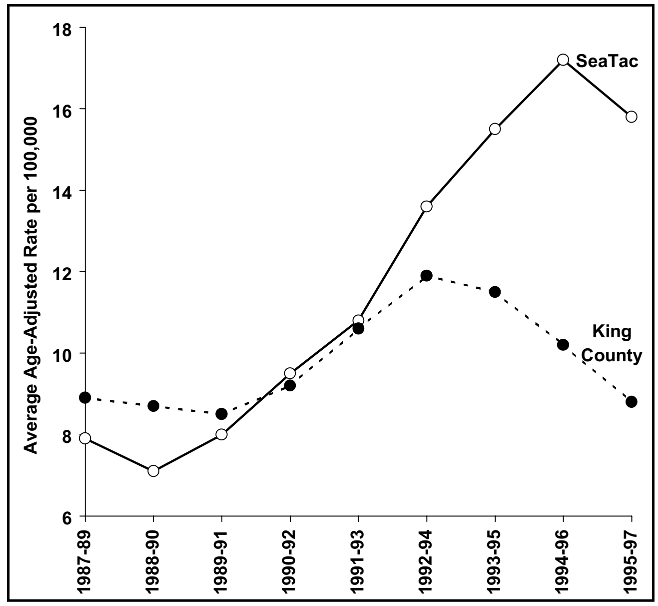
* Statistically significantly higher in SeaTac Airport Community than in King County.

- The average overall birth rate from 1993-1997 was 22% higher in SeaTac than in the entire county.
- The birth rate among teens age 15-17 in the same period was also higher in SeaTac by 94%.
- There were no significant differences between SeaTac and King County in terms of maternal use of alcohol while pregnant and low birth weight births. But the rate of maternal smoking during pregnancy and the percentage of births for which the mother received late or no prenatal care was significantly higher in SeaTac.

Violence

(Appendix A – Table 10)

Figure 11: All Firearm Deaths
SeaTac Airport Community and King County
3 Year Rolling Averages, 1987-1997

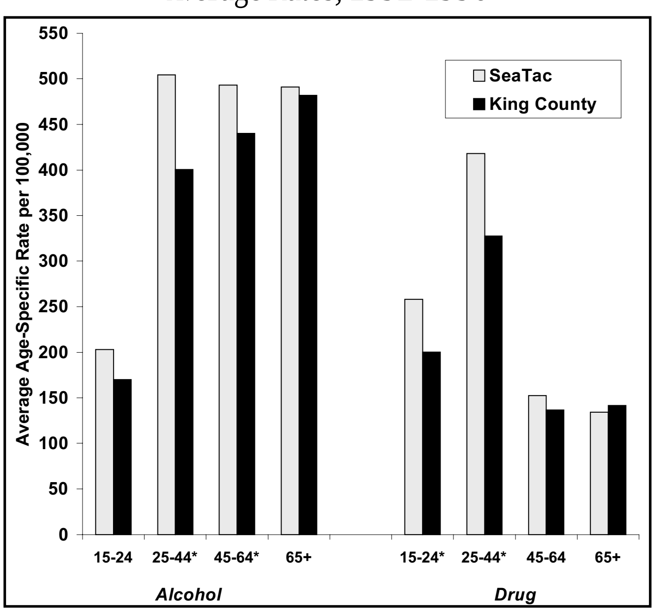


- The average hospitalization rate for assault between 1992 and 1996 was higher in SeaTac, although the homicide rate from 1993-1997 was not significantly different.
- Firearm-related deaths were 50% higher in SeaTac than in King County from 1993-1997. This rate includes accidental shootings, suicide, and homicide by firearm. Suicide accounts for the majority of these deaths in SeaTac (69%), followed by homicide (31%).
- The firearm death rate increased significantly in SeaTac and King County from 1989 to 1994. Since then, there has been a significant decline in the King County rate.

Mental Health and Substance Misuse

(Appendix A – Tables 7 & 11)

Figure 12: Alcohol and Drug Hospitalizations
in Teens and Adults
SeaTac Airport Community and King County
Average Rates, 1992-1996



* Statistically significantly higher in SeaTac Airport Community than in King County.

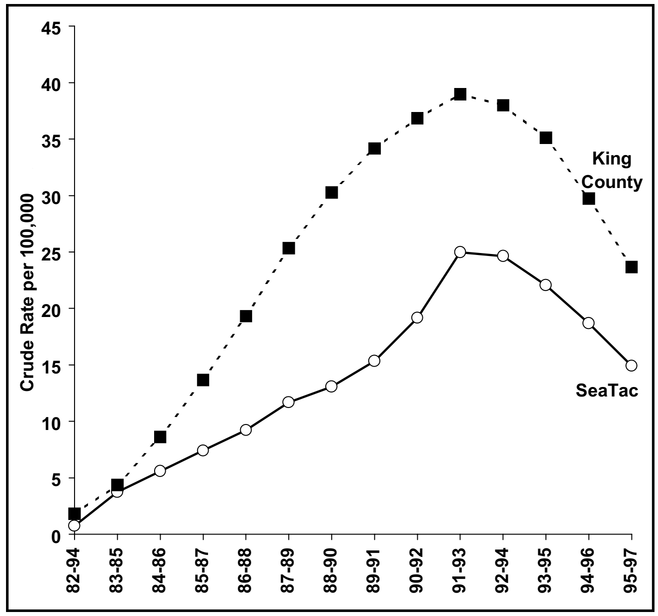
- Between 1992 and 1996, the average rates of alcohol and illicit drug related hospitalizations were higher in SeaTac than in King County for all age groups. Illicit drug related hospitalizations were also significantly higher among 15-24 year olds in SeaTac.
- The average rates of suicide (1993-1997), attempted suicide hospitalizations (1992-1996), and hospitalizations for depression (1992-1996) were not significantly different overall or among 15-24 year olds in SeaTac and King County.

Communicable Diseases

(Appendix A – Tables 7 & 12)

- The average rate of new AIDS cases from 1995-1997 was lower in SeaTac than in King County.
- Between 1993 and 1997, the average rates of chlamydia and gonorrhea were significantly higher in SeaTac among 15-24 year olds and all ages.
- There were no significantly differences in rates of TB and vaccine preventable diseases.
- From 1993-1997, the average food and waterborne disease rates were significantly lower in SeaTac than in King County.

Figure 13: New AIDS Cases
SeaTac Airport Community and King County
3 Year Rolling Averages, 1982-1997



Behavioral Risk Factors and Access to Health Care

(Appendix A – Tables 13, 14 & 15)

- There is no data available on the prevalence of behavioral risk factors or measures of access to health care specifically among the SeaTac Airport Community. However, a telephone survey conducted throughout King County provides some of this information for the South King County region to which SeaTac belongs.
- Data from the Behavioral Risk Factor Survey indicate that 17% of women in South County had not had a Pap Smear recently, versus 13% in the county as a whole.
- Among all adults surveyed, a greater percentage in South County were smokers (24%) or were over-weight (28%) than in King County (19% and 22%, respectively).
- The number of South County households with a loaded gun in the home was 16.5%, versus 12.5% for the county.
- It should be noted that although the survey data does indicate some differences between South County and the entire county in the prevalence of certain risk factors, none of those differences were statistically significant.

Summary and Discussion of Findings

Chronic disease was the major cause of death in the SeaTac Airport Community, as well as King County. Chronic diseases are those that are slow to develop and last for an extended period of time. Cancer and heart disease combined accounted for about half of all deaths. Residents of the SeaTac Airport Community were at a higher risk for death from cancer and chronic obstructive pulmonary disease (primarily emphysema) compared to the King County population as a whole. Respiratory cancer, accounting for 30% of cancer deaths, was also higher in SeaTac. The excess in chronic disease deaths as compared to the entire county appears to occur in people younger than 65. For the oldest age group (65+), the SeaTac rates were not statistically different, or were actually lower.

Chronic disease was also a major cause of disability (measured by hospitalization rates) among the SeaTac population. The hospitalization rates for heart disease and cancer were higher in SeaTac than in King County, as was the total hospitalization rate. Hospitalization can reflect factors other than incidence or prevalence of a condition, such as access to timely and appropriate primary care.

Besides chronic obstructive pulmonary disease and respiratory cancer, two other types of respiratory illness have a substantial impact on the health of SeaTac residents. Pneumonia/ Influenza - diseases caused by infectious agents - were the sixth leading cause of death among all ages. Although asthma is not usually a cause of death, it can have a debilitating impact on health. Hospitalization rates for both pneumonia/influenza and asthma were significantly higher among people younger than 65 in SeaTac than they were in the whole county. Furthermore, since 1987 those rates have been increasing among children age 0-17 both in SeaTac and in the entire county.

Unintentional injuries were the greatest cause of death among children age 0-17, and the second leading cause of hospitalization for people over 65. Falls and motor vehicle crashes account for most unintentional injuries and deaths, although their relative impact varies by age group. While motor vehicle crashes claimed the greatest number of lives among all ages, the greatest number of hospitalizations were related to falls. For older residents of SeaTac, falls accounted for the largest number of unintentional injury deaths.

Fortunately, rates of hospitalization for unintentional injuries, and particularly motor vehicle crashes, have steadily declined in SeaTac. This trend only refers to the injury rate from collisions, and doesn't necessarily indicate that traffic collisions are also declining.

While the overall birth rate in the SeaTac Airport Community was higher than in the county by 22%, the rate among teens age 15-17 was 94% higher. Some of the risk factors for poor birth outcomes were also significantly higher in SeaTac. More mothers smoked during pregnancy, and fewer received prenatal care within the first trimester of pregnancy.

The rate of death from firearms, including homicide, accidental shootings, and suicide, was higher by 50% in SeaTac than in the county as a whole. Furthermore, the rate increased significantly from 1989 to 1994 in both the SeaTac Airport Community and King County. The overall homicide rate (all weapons combined) was not significantly different in the two regions.

Violence was also a major cause of death and injury specifically among children age 0-17. Homicide was the second leading cause of death among children age 0-17.

Hospitalizations related to alcohol and illicit drug misuse were higher in SeaTac than in the county, although this difference may be partially an artifact of the way in which hospitalization data is reported. The data do not include people hospitalized in free standing substance abuse clinics or federal institutions such as the Veteran's Administration Hospital.

Reported cases of the sexually transmitted diseases chlamydia and gonorrhea were significantly higher in SeaTac than in the county by 51% and 22%, respectively.

The rate of death from AIDS was significantly lower in SeaTac and has been declining there since 1994. The rate of diagnosis of new AIDS cases also seems to be following a declining trend in SeaTac, as it is in the entire county.

Prevention of Disease, Injury and Death

Although many of the health problems in a community like SeaTac do not have a single, easily identifiable cause, there are a number of controllable factors that are known to contribute to or exacerbate the development of disease or the occurrence of injury. Because these risk factors are relatively well understood and are preventable, they represent one way to reduce the incidence of disease, injury and death. Although many of the risk factors can often be reduced by modification in individual lifestyles, the ability to make choices about healthy lifestyle behaviors is influenced and limited by norms of society, available resources, and other socioeconomic factors.

In addition to measures to reduce life-style risk factors, early detection and treatment can mitigate the impact of chronic disease. Access to and utilization of health care services to screen for high cholesterol, high blood pressure, as well as breast, colorectal, and cervical cancer is important in the prevention of unnecessary death and disability.

The most important risk factors for chronic diseases include cigarette smoking, alcohol misuse, high blood pressure, obesity, physical inactivity, high blood cholesterol, and high fat/low fiber diet. All of these factors are associated with the leading causes of death in the SeaTac Airport Community including cancer, heart disease, diabetes, cerebrovascular disease, chronic obstructive pulmonary disease, and cirrhosis.

Cigarette smoking is a major risk factor for heart disease, lung cancer, and many chronic and acute respiratory conditions. Alcohol misuse increases the risk of heart disease, high blood pressure, chronic liver disease, sexually transmitted disease, motor vehicle crashes, and other unintentional injuries including falls.

In addition to lifestyle risk factors, exposure to environmental hazards such as air pollution, toxic chemicals, and radiation can also affect health status. Poor air quality, both indoor and outdoor, contributes substantially to illness and death from respiratory diseases including cancer, chronic obstructive pulmonary disease, and asthma.

Timely access to and use of prenatal care in the first trimester of pregnancy may reduce the risk of infant death and other infant health problems. Smoking during pregnancy is also associated with an increase in poor birth outcomes. Infants born to mothers under age 18 have increased risk of mortality and low birthweight. Both the mother and the child tend to have subsequent educational, economic, and social problems.

Prevention and control of chronic disease risk factors, such as high blood pressure, can also reduce the occurrence of pregnancy complications that result in hospitalization among women, and improve health outcomes for their children.

Wearing a seat belt in a motor vehicle or wearing a helmet while riding a bicycle or motorcycle can prevent injury in an accident or mitigate injury severity. Firearms contribute to deaths and injuries in suicide, homicide, assault, and accident. Handguns are the most frequently used firearms in these incidents.

Based on availabe data, it is not possible to determine the extent to which these risk factors have contributed to the current health status of the SeaTac Airport Community, and specifically to the excess in death and disability in that community as compared to the county as a whole. However, national studies indicate that at least 50% of all deaths are associated with preventable factors. The reduction or elimination of these risk factors is, therefore, a key strategy for the prevention of disease and promotion of good health.

Opportunities for Prevention: The Impact of Risk Factors for Poor Health

Key areas for prevention	% of all deaths*	Impact on leading causes of death and other major health problems
Smoking	**19**	heart disease, stroke, lung cancer, cervical cancer, COPD, asthma, infant health
Diet/Physical Activity	**14**	heart disease, stroke, cancer, diabetes, falls and hip fracture
Alcohol Use	**5**	chronic liver disease/cirrhosis, motor vehicle crashes, falls and hip fracture, violent crimes, fetal alcohol syndrome
Exposure to Microbial agents & Immunizations/Vaccinations	**4**	AIDS, STDs, TB, Enteric diseases, hepatitis, Vaccine-preventable diseases among children, pneumonia and influenza among older adults
Toxic agents in the environment	**3**	heart disease, cancer, COPD, asthma
Firearms in the home	**2**	suicide, homicide, firearm injuries, violent crimes
Motor vehicle safety & seat belt use	**1**	motor vehicle crashes, injuries from motorcycle /bicycle accidents
Sexual behavior	**1**	HIV/AIDS, STDs, unintended pregnancy
Illicit use of drugs	**1**	drug overdose, AIDS, STDs, hepatitis B, violent crimes
Cancer screening	**NA**	breast, cervical, and colorectal cancer
Prenatal care	**NA**	infant mortality, low birth weight
Hypertension	**NA**	heart disease, stroke, and kidney failure
Mental health	**NA**	depression, suicide
Lack of Access to health care	**NA**	all, preventable hospitalization, dental health

* Percentage of total deaths caused by this factor, based on national studies. (From: McGinnis, JM and Foege, WH. Actual Causes of Death in the United States. JAMA. 270 (18): 2207-2212. 1993).

Technical Appendix A

Summary Data Tables

List of Summary Data Tables

All summary data tables include statistics for the SeaTac Airport Community (or the closest geographic approximation possible) and comparison statistics for King County overall. The following tables are included in this Technical Appendix:

SeaTac Airport Community Boundaries

The geographic boundaries of the SeaTac Airport Community were determined through consultation with a community member. Because different types of data are reported by different geographic units, three community boundary definitions were necessary in order to produce the information in this report. The community boundary used for most of the data analysis includes census tracts 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02. For data available only by zip code, the zip codes 98146, 98148, 98158, 98166, 98168, and 98188 define the boundary because they most closely match the census tracts above. The maps in Technical Appendix D visually demonstrate the differences between these two community boundary definitions. The zip code boundary for King County is also not an exact match of the county census tract boundary. Behavioral Risk Factor Survey data is only available for the entire South King County survey region which contains the SeaTac Airport Community.

Time Period Analyzed

The years reported are always the most recent years of data available. Since different data sources have different lag times for availability, the most recent year varies from 1990 for U.S. Census data to 1997 for births, deaths and some communicable diseases.

Technical Appendix B

Data Analysis Techniques and Definitions

Rates

Almost all health data are presented in the form of rates. A rate is the number of occurrences of an event divided by the size of the population that could experience that event over a specified time period. Thus, a birth rate for 15 - 17 year olds is the number of births in this age group during a given year, divided by the total number of girls age 15-17 during that year. Rates are usually multiplied by a constant, the "per" number (a percent is per 100; rates are usually per 1,000 or per 100,000), in order to make them whole numbers, which are easier to interpret. For example, the birth rate for girls age 15-17 in King County is expressed as 19.4 births per 1,000 girls that age, instead of .0194 births per girl aged 15-17.

Confidence Intervals

Some of the year to year fluctuation in the occurrence of events (such as births) in a population is due to random factors that cannot be measured. Statisticians normally report confidence intervals, or "margins of error" to show the range in which we think the true rate falls, given that there will be some random variation. The standard confidence interval is calculated so that there is a 95% probability that the true rate falls within its range. The true rate is the rate that would occur if there were no random factors (see Chiang, Chin Long, "Standard Error of the Age-Adjusted Death Rate," *Vital Statistics Special Reports,* 1961, 47(9):275-285).

Confidence intervals are also useful to determine whether rates in two areas are significantly different from one another. If the confidence intervals of the two rates overlap, we cannot say that they are statistically different from one another; the true rates of each may fall within the overlapping range. Therefore, it is only when the confidence intervals of two rates do not overlap that we conclude that the rates are "statistically significantly" different from one another. This method was used to determine whether community rates differed from King County rates.

Rolling Averages

The larger the population you're examining, the more stable or reliable you can expect rates to be. That is, there's less purely random variation in the numbers. Sometimes, in order to observe an overall time trend in rates, it helps to look at more than one year of data at a time. In this case, the rates are grouped into "rolled" averages across the total observed period. For example, to look at heart disease deaths from 1980-1997, we may use 5-year intervals. This means we would calculate the average rates from 1980-84, 1981-85, and so on. Each five year average successively advances by one year, includes a higher number of cases than a single year, and thus smoothes out random year-to-year fluctuations. This method of presenting trends can be seen in some of the graphs included in this report. Note, however, that all statistical tests to determine the presence of a statistically significant time trend were calculated using single-year data. See "Statistical Trend" below.

Data Analysis Techniques and Definitions

Age Adjusting

There are some health events (e.g., heart disease) that people are more likely to get when they get older. Others (like homicide) are more likely to affect younger people. This means that if you examined a community with a lot of older people, you would see a higher proportion of the whole population with heart disease than you would in a younger community. That doesn't necessarily mean that the first community had more of a heart disease problem, just that there were more older people living there. We often want to compare disease rates in two areas with different age structures, so we need to control for the age structure. This is called age-adjusting. Age adjusted rates tell you if one area is more likely to have a disease, leaving aside the fact that it has older (or younger) residents than the area it's being compared to.

Statistical Trend

We can do a statistical test on successive discrete years of data to give us an idea of whether there is a true overall increase or decrease in rates, or just random variation. From any given year to the next, a rate may go up or down randomly, but a rate that keeps going up or down may indicate that a real change is occurring in the population. This is called a trend. The chi square test for trend (see Armitage and Berry, Statistical Methods in Medical Research, Second Edition, 1987, pp. 372-373) gives us an idea of whether the change we see is significant. It looks at the size of the population, the amount of change in the rate, and the number of years that change occurs to tell whether the trend seems to be significant. A large population, a big chance in the rate, and a long period of time over which the rate continued to change would all give more confidence that a true statistical trend is occurring.

Technical Appendix C

Sources of Health Data

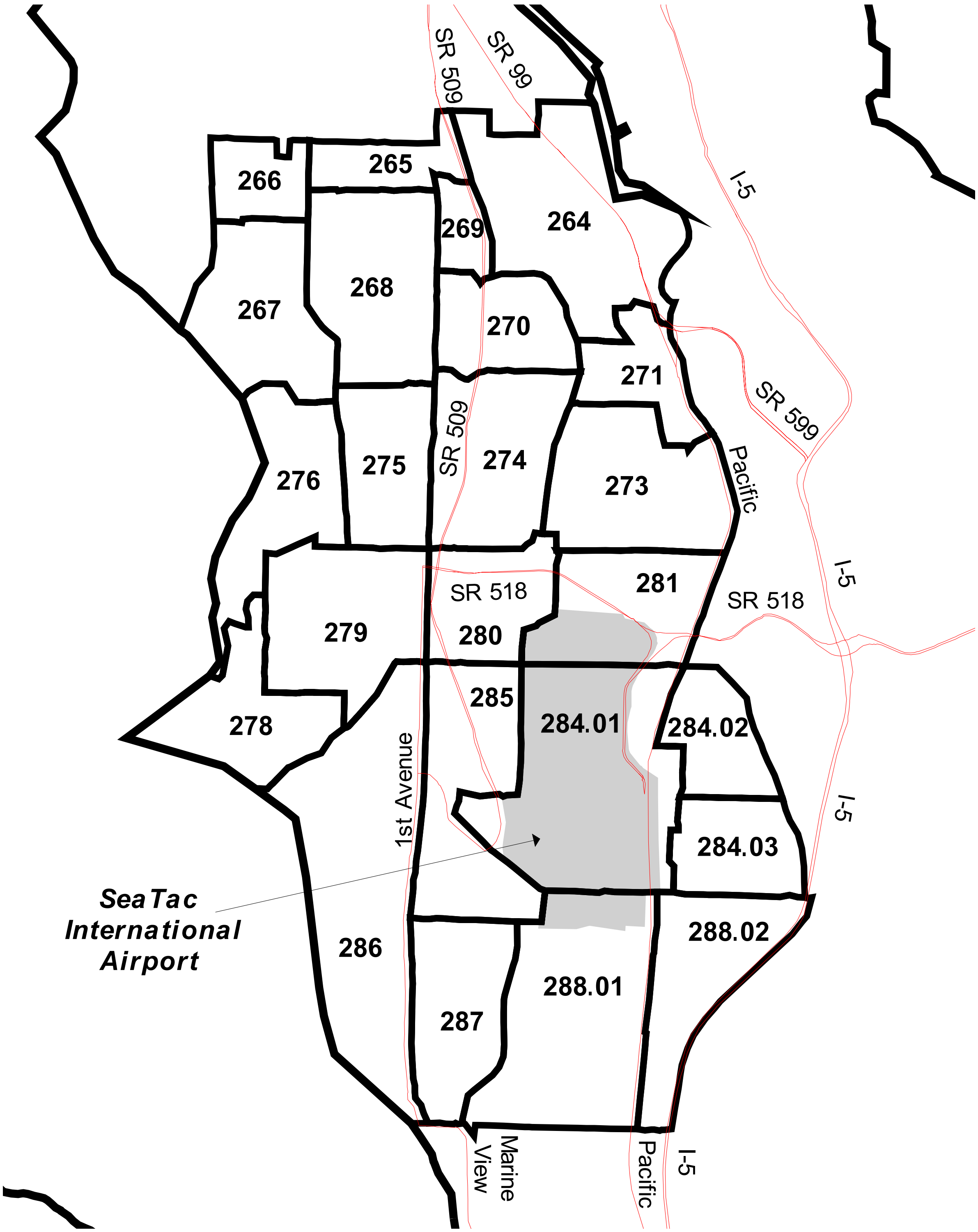
Data Type	Years Available	Data Source
Birth Certificates	1980-97	Washington State Department of Health, Center for Health Statistics
Death Certificates	1980-97	Washington State Department of Health, Center for Health Statistics
Population Demographics	1980, 1990	U.S. Census Bureau
Adjusted Population Estimates	intercensal estimates for all years thru 1997	Washington State Department of Social and Health Services, Office of Research and Data Analysis
Hospital Discharge Data	1980-96	Washington State Department of Health, Office of Hospital and Patient Data Systems, Comprehensive Hospital Abstract Reporting System (CHARS)
Reportable Disease Records	1988-97	Washington State Department of Health, STD/TB Services and Communicable Disease Epidemiology Seattle-King County Department of Public Health, HIV/AIDS Epidemiology
Accepted Child Abuse Referrals	1992-93	Washington State Department of Social and Health Services, Child Protective Services
Behavioral Risk Factor Surveillance Data	1994-1995	Washington State Behavioral Risk Factor Surveillance System Seattle-King County Department of Public Health Small Area Behavioral Risk Factor Survey

Technical Appendix D

Maps of the SeaTac Airport Community

Map 1: King County

Map 2: SeaTac Airport Community
By Census Tract



Map 3: SeaTac Airport Community By ZIP Code

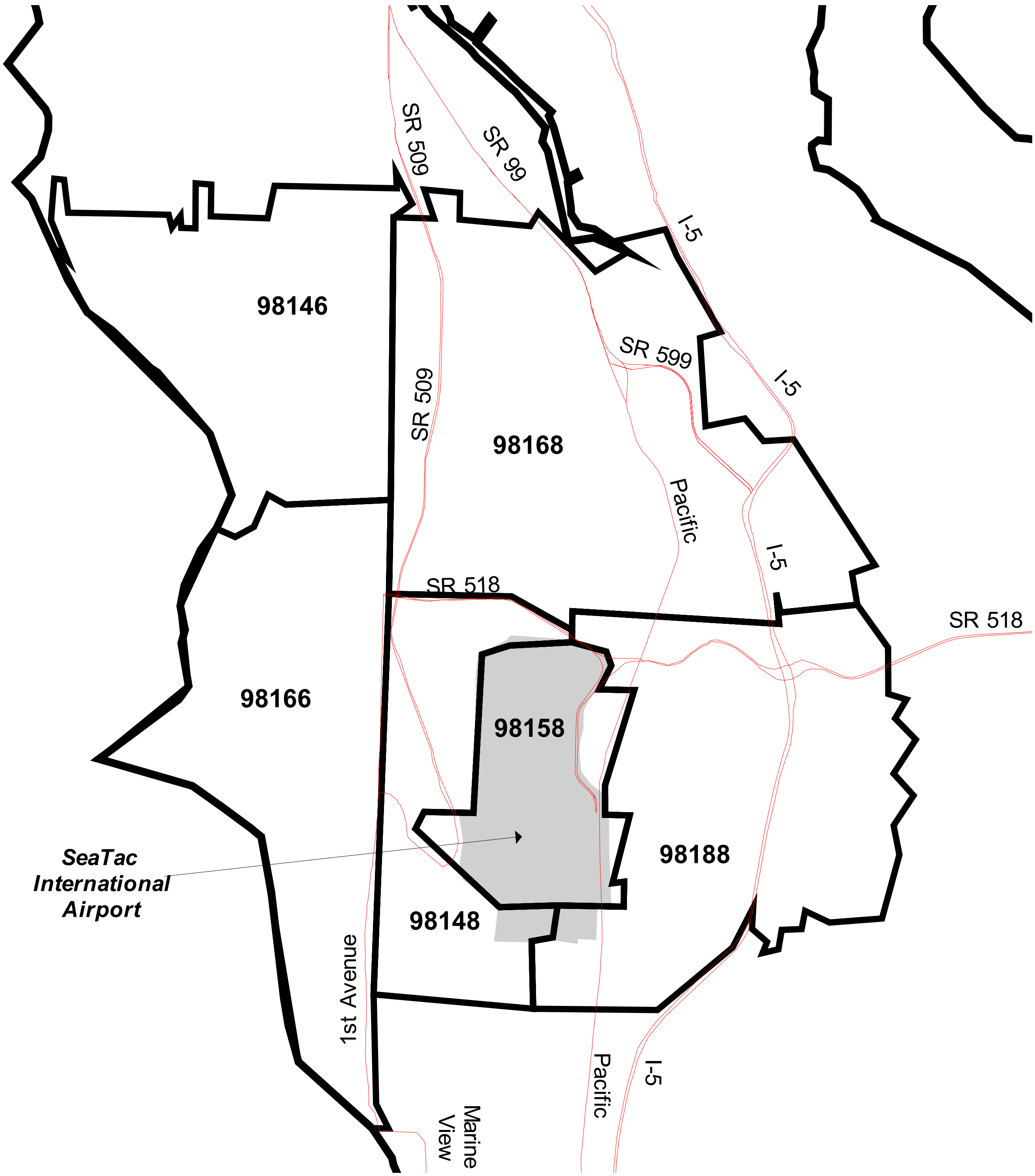


Table 1. Estimated Population and Indicators of Socioeconomic Status by Race/Ethnicity in SeaTac Airport Community† and King County

	SeaTac Airport Community					King County				
	1997 Estimate		1990 Census			1997 Estimate		1990 Census		
Race/Ethnicity	1997 Estimated Population	Percent of Total Population	% Living Below Poverty (Based on 1989 Income)	% of Employed Persons (Age 16+) in Working Class Position	% of Adults (Age 25+) Without a High School Diploma/ GED	1997 Estimated Population	Percent of Total Population	% Living Below Poverty (Based on 1989 Income)	% of Employed Persons (Age 16+) in Working Class Position	% of Adults (Age 25+) Without a High School Diploma/ GED
African American	4,112	4.2%	31.3%	NA	17.9%	93,701	5.7%	22.3%	NA	21.0%
Asian/Pacific Islander	10,301	10.4%	21.8%	NA	30.0%	177,515	10.7%	15.2%	NA	21.3%
Hispanic Ethnicity**	5,448	5.5%	13.8%	NA	27.7%	63,925	3.9%	14.9%	NA	21.6%
Native American	2,000	2.0%	26.7%	NA	21.9%	20,577	1.2%	25.7%	NA	23.9%
White	82,195	83.4%	7.3%	NA	15.5%	1,360,982	82.3%	6.1%	NA	10.3%
Total Population	98,608	100.0%	9.5%	70.1%	16.7%	1,652,775	100.0%	8.0%	59.7%	11.8%

†SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
** Hispanic ethnicity may include persons of any race.
NA = not available by race/ethnicity.
Source: Washington State DSHS Office of Research and Data Analysis, Adjusted Population Estimates, June 1997.
1990 U.S. Census

Table 2. Leading Causes of Death in SeaTac Airport Community† and King County, 1993-97

	SeaTac Airport Community					King County					Comparison to King County Rates	
				95% Confidence Interval					95% Confidence Interval			
Condition	Rank	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Rank	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Cancer	1	201	132.1	123.7	140.6	2	2,831	120.8	118.7	122.9	Higher	9
Heart Disease	2	196	101.9	95.2	108.6	1	3,023	98.9	97.2	100.6	NS	3
Cerebrovascular Disease	3	55	25.3	22.1	28.5	3	901	25.3	24.5	26.1	NS	0
Chronic Obstructive Pulmonary Disease	4	43	24.3	20.9	27.7	4	510	18.5	17.7	19.3	Higher	31
Unintentional Injury	5	33	28.8	24.1	33.6	6	463	24.2	23.1	25.2	NS	19
Pneumonia and Influenza	6	31	14.6	12.1	17.1	5	478	12.3	11.7	12.8	NS	19
Diabetes (Underlying Cause)	7	24	15.0	12.2	17.9	7	293	12.0	11.4	12.7	NS	25
Suicide	8	15	14.7	11.2	18.3	9	201	11.5	10.7	12.2	NS	28
AIDS	9	10	9.0	6.4	11.5	8	291	15.2	14.4	16.0	Lower	-41
Chronic Liver Disease/Cirrhosis	10	9	7.3	5.1	9.4	10	130	6.6	6.1	7.1	NS	11
All Causes of Death		770	464.6	449.6	479.6		11,425	427.9	424.2	431.5	Higher	9
				95% Confidence Interval					95% Confidence Interval			
			Years	Lower Bound	Upper Bound			Years	Lower Bound	Upper Bound		
Life Expectancy at Birth			77.0	76.6	77.5			78.0	77.9	78.1	Lower	-1

†SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
*Rates are age-adjusted to the 1940 U.S. Population.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Sources: Washington State DOH Center for Health Statistics and Washington State DSHS for adjusted population estimates (6/97).

Produced by: Seattle-King County Department of Public Health, EPE, 2/26/99

Table 3. Chronic Disease Mortality in SeaTac Airport Community† and King County, 1993-97

	SeaTac Airport Community				King County				Comparison to King County Rates	
			95% Confidence Interval				95% Confidence Interval			
Condition	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
All Cancer	201	132.1	123.7	140.6	2,831	120.8	118.7	122.9	Higher	9
..Respiratory Cancer	60	41.1	36.3	46.0	774	34.6	33.5	35.8	Higher	19
..Colorectal Cancer	19	11.0	8.6	13.4	282	11.2	10.5	11.8	NS	-2
..Breast Cancer in Women (F)	20	27.3	21.7	33.0	245	20.6	19.3	21.8	NS	33
..Prostate Cancer (M)	11	13.9	10.3	17.5	179	14.5	13.6	15.5	NS	-4
Heart Disease	196	101.9	95.2	108.6	3,023	98.9	97.2	100.6	NS	3
Cerebrovascular Disease	55	25.3	22.1	28.5	901	25.3	24.5	26.1	NS	0
Chronic Obstructive Pulmonary Disease	43	24.3	20.9	27.7	510	18.5	17.7	19.3	Higher	31
Diabetes (Underlying Cause)	24	15.0	12.2	17.9	293	12.0	11.4	12.7	NS	25
Chronic Liver Disease/Cirrhosis	9	7.3	5.1	9.4	130	6.6	6.1	7.1	NS	11

†SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
*Rates are age-adjusted to the 1940 U.S. Population.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Source: Washington State DOH Center for Health Statistics and Washington State DSHS for adjusted population estimates (6/97).

Table 4. Leading Causes of Hospitalization in SeaTac Airport Community† and King County, 1992-96

	SeaTac Airport Community					King County					Comparison to King County Rates	
				95% Confidence Interval					95% Confidence Interval			
Condition	Rank	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Rank	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Unintentional Injury	1	880	693.9	672.7	715.1	1	12,742	644.9	639.8	650.1	Higher	8
Heart Disease	2	809	532.3	516.7	548.0	3	10,452	473.4	469.5	477.4	Higher	12
Digestive System Disease	3	804	651.1	630.6	671.5	2	10,978	580.3	575.4	585.3	Higher	12
Genito/Urinary Disease	4	568	478.2	460.3	496.1	4	7,182	386.9	382.8	391.0	Higher	24
Cancer	5	423	295.8	283.2	308.3	5	5,518	272.5	269.2	275.7	Higher	9
Alcohol Related	6	370	321.4	306.6	336.2	6	4,931	274.3	270.9	277.8	Higher	17
Fractures	7	339	263.4	249.9	276.9	7	4,891	239.3	236.1	242.6	Higher	10
Psychoses	8	289	255.3	241.5	269.0	8	4,583	257.3	253.8	260.7	NS	-1
Pregnancy Complications (F)^	9	264	176.5	168.0	185.4	9	3,583	168.1	165.9	170.4	NS	5
Pneumonia/Influenza	10	254	181.8	171.2	192.4	10	3,412	155.0	152.5	157.5	Higher	17
All Hospitalizations (excluding childbirth)		6,020	4896.0	4851.3	4940.7		82,128	4349.2	4338.5	4360.0	Higher	13

†SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
*Rates are age-adjusted to the 1940 U.S. Population.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
^Rate=number of hospitalizations for pregnancy complications per 1,000 live births (not per total population).
Source: Washington State DOH Office of Hospital and Patient Data Systems and Washington State DSHS for adjusted population estimates (6/97).

Table 5. Hospitalization for Pneumonia/Influenza and Asthma Among 0-17, 18-64, & 65+ year olds in SeaTac Airport Community† and King County, 1992-96

Condition	SeaTac Airport Community				King County				Comparison to King County Rates	
			95% Confidence Interval				95% Confidence Interval			
	Average Annual Count	Rate per 100,000	Lower Bound	Upper Bound	Average Annual Count	Rate per 100,000	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Age 0-17:										
Pneumonia/Influenza	35	146.7	125.9	170.0	375	97.0	92.6	101.4	Higher	51
Asthma	55	227.1	201.1	255.7	726	187.8	181.7	194.0	Higher	21
Age 18-64:										
Pneumonia/Influenza	81	124.9	113.1	137.7	1,059	101.8	99.1	104.6	Higher	23
Asthma	45	69.7	60.9	79.4	540	51.9	50.0	53.9	Higher	34
Age 65+:										
Pneumonia/Influenza	138	1089.6	1009.9	1174.1	1,978	1133.7	1111.4	1156.2	NS	-4
Asthma	24	187.9	155.7	224.8	236	135.5	127.9	143.5	Higher	39

†SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Source: Washington State DOH Office of Hospital and Patient Data Systems and Washington State DSHS for adjusted population estimates (6/97).

Table 6. Leading Causes of Death and Hospitalization for Children Age 0-17 in SeaTac Airport Community†‡ and King County

	SeaTac Airport Community					King County					Comparison to King County Rates	
				95% Confidence Interval					95% Confidence Interval			
Condition	Rank	Average Annual Count	Rate per 100,000	Lower Bound	Upper Bound	Rank	Average Annual Count	Rate per 100,000	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
A. Leading Causes of Death, 1993-97†												
Unintentional Injury	1	2	9.3	4.6	16.6	1	27	6.9	5.8	8.1	NS	35
Homicide	2	1	5.1	1.9	10.9	2	13	3.4	2.6	4.3	NS	50
Cancer	3	1	3.4	0.9	8.5	3	13	3.4	2.6	4.3	NS	0
All Causes of Death		19	79.5	64.2	97.2		208	52.8	49.7	56.1	Higher	51
B. Leading Causes of Hospitalization, 1992-96‡												
Unintentional Injuries	1	75	309.2	278.7	342.2	1	1,118	289.0	281.5	296.7	NS	7
Digestive System Disease	2	64	266.1	237.8	296.9	2	911	235.6	228.8	242.6	NS	13
Asthma	3	55	227.1	201.1	255.7	3	726	187.8	181.7	194.0	Higher	21
Infections	4	47	196.5	172.3	223.1	5	575	148.6	143.2	154.1	Higher	32
Perinatal Conditions	5	42	173.3	150.6	198.4	4	626	161.9	156.3	167.7	NS	7
All Causes of Hospitalization (not childbirth)		2,059	8532.6	8368.6	8699.1		28,653	7407.7	7369.4	7446.2	Higher	15

†SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
‡SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Sources: Washington State DOH Center for Health Statistics, and Office of Hospital and Patient Data Systems.
Washington State DSHS for adjusted population estimates (6/97).

Table 7. Health of Young Persons Age 15 to 24 in SeaTac Airport Community† and King County

			SeaTac Airport Community					King County					Comparison to King County Rates	
						95% Confidence Interval					95% Confidence Interval			
Indicator	Source of Data	Time Period Analyzed	Average Annual Count	Rate	Per	Lower Bound	Upper Bound	Average Annual Count	Rate	Per	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
All Causes of Death:	Death Certificates†	93-97	9	84.2	100,000 Age 15-24	61.7	112.4	144	75.3	100,000 Age 15-24	69.9	81.1	NS	12
Unintentional Injury:	Hospitalizations‡													
All Unintentional Injury		92-96	61	524.4	"	467.4	586.5	975	508.4	"	494.2	522.9	NS	3
..Motor Vehicle Accident Injury		92-96	19	160.6	"	129.8	196.4	293	152.7	"	145.0	160.7	NS	5
..Falls		92-96	11	92.2	"	69.3	120.3	182	94.7	"	88.6	101.0	NS	-3
Birth to teenage mother:	Births Certificates†	93-97	67	37.7	1,000 15-17 Year Old Females	33.8	42.0	551	19.4	1,000 15-17 Year Old Females	18.7	20.1	Higher	94
Sexually Transmitted Disease:	Disease Reports†													
Chlamydia		93-97	207	1890.9	100,000 Age 15-24	1777.5	2009.9	2,328	1219.6	100,000 Age 15-24	1197.6	1242.0	Higher	55
Gonorrhea		93-97	48	439.3	"	385.6	498.6	575	301.2	"	290.3	312.5	Higher	46
Alcohol and Substance Abuse	Hospitalizations‡													
Alcohol-Related		92-96	24	201.6	"	166.9	241.3	326	169.9	"	161.8	178.4	NS	19
Illicit Drug-Related		92-96	30	254.5	"	215.3	298.8	384	200.0	"	191.2	209.2	Higher	27
Mental Health:	Hospitalizations‡													
Suicide Attempt		92-96	12	105.9	"	81.2	135.7	193	100.8	"	94.6	107.4	NS	5
Depression		92-96	26	220.3	"	184.0	261.8	388	202.0	"	193.1	211.2	NS	9
Injury Due to Violence:	Hospitalizations‡													
Assault		92-96	14	123.0	"	96.3	154.8	183	95.5	"	89.4	183.2	NS	29

†Mortality, birth and disease report data for SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
‡Hospitalization data for SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Sources: Mortality/Birth Data: Washington State DOH Center for Health Statistics; Hospitalizations: Wash State DOH, Office of Hospital and Patient Data Systems;
Hospitalizations: Washington State DOH, Office of Hospital and Patient Data Systems;
Disease Reports: Washington State DOH, STD/TB Services
Washington State DSHS for adjusted population estimates (6/97) used in rate calculations.

Produced by: Seattle-King County Department of Public Health, EPE, 2/26/99

Table 8. Leading Causes of Death and Hospitalization for Adults Age 65 and Older in SeaTac Airport Community†‡ and King County

	SeaTac Airport Community					King County					Comparison to King County Rates	
				95% Confidence Interval					95% Confidence Interval			
Condition	Rank	Average Annual Count	Rate per 100,000	Lower Bound	Upper Bound	Rank	Average Annual Count	Rate per 100,000	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
A. Leading Causes of Death, 1993-97†												
Heart Disease	1	163	1331.7	1241.9	1426.3	1	2,569	1461.4	1436.2	1486.9	Lower	-9
Cancer	2	139	1137.5	1054.7	1225.2	2	2,006	1141.2	1119.0	1163.8	NS	0
Cerebrovascular Disease	3	48	390.0	342.2	442.8	3	819	465.9	451.7	480.4	Lower	-16
Chronic Obstructive Pulmonary Disease	4	39	315.0	272.2	362.7	4	457	259.8	249.2	270.7	Higher	21
Pneumonia and Influenza	5	27	221.9	186.3	262.5	5	441	250.8	240.4	261.5	NS	-12
All Causes of Death		556	4533.6	4366.6	4705.4		8,439	4801.5	4755.8	4847.5	Lower	-6
B. Leading Causes of Hospitalization, 1992-96‡												
Heart Disease	1	507	4006.3	3851.9	4165.3	1	6,650	3811.5	3770.7	3852.7	NS	5
Unintentional Injury	2	356	2810.9	2681.8	2944.6	2	5,337	3058.9	3022.3	3095.8	Lower	-8
Digestive System Disease	3	282	2225.0	2110.4	2344.3	3	3,945	2261.1	2229.7	2292.9	NS	-2
Cancer	4	239	1885.5	1780.1	1995.6	4	2,950	1691.2	1664.0	1718.7	Higher	11
Cerebrovascular Disease	5	151	1193.8	1110.3	1282.1	5	2,407	1379.8	1355.3	1404.7	Lower	-13
All Causes of Hospitalization		2,171	17141.5	16820.6	17467.1		29,878	17125.8	17039.0	17212.9	NS	0

†SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
‡SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Sources: Washington State DOH Center for Health Statistics, and Office of Hospital and Patient Data Systems.
Washington State DSHS for adjusted population estimates (6/97).

Table 9. Maternal and Child Health in SeaTac Airport Community and King County

		SeaTac Airport Community					King County					Comparison to King County Rates	
					95% Confidence Interval					95% Confidence Interval			
Indicator	Time Period Analyzed	Average Annual Count	Rate	Per	Lower Bound	Upper Bound	Average Annual Count	Rate	Per	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Outcome Measures:													
Infant mortality†	93-97	11	7.7	1,000 Births	5.8	10.0	122	5.6	1,000 Births	5.2	6.1	NS	37
Hospitalizations for Pregnancy Complications (F)‡	92-96	264	176.5	1,000 Births	168.0	185.4	3,583	168.1	1,000 Births	165.9	170.4	NS	5
Risk Factors for Poor Maternal or Child Health:													
Low birthweight†	93-97	93	6.4	100 Births	5.8	7.0	1,235	5.7	100 Births	5.5	5.8	NS	12
Late or No 1st trimester prenatal care†	93-97	63	5.3	100 Births	4.7	5.9	628	3.3	100 Births	3.2	3.5	Higher	57
Mother smoked when pregnant†	93-97	246	17.6	100 Births	16.6	18.6	2,451	11.8	100 Births	11.6	12.0	Higher	49
Mother used alcohol when pregnant†	93-97	31	2.6	100 Births	2.2	3.0	512	3.0	100 Births	2.8	3.1	NS	-12
Birth to teenage mother†	93-97	67	37.7	1,000 15-17 Year Old Females	33.8	42.0	551	19.4	1,000 15-17 Year Old Females	18.7	20.1	Higher	94
Annual Births:													
Total births†	93-97	1457	69.9	1,000 15-44 Year Old Females	68.3	71.5	21,867	57.5	1,000 15-44 Year Old Females	57.2	57.8	Higher	22

†SeaTac Airport Community for this indicator includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
‡SeaTac Airport Community for this indicator includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Source: Washington State DOH Center for Health Statistics.
Washington State DSHS for adjusted population estimates (6/97).

Table 10. Injury and Violence in SeaTac Airport Community† and King County

Indicator	Type of Data	Time Period Analyzed	SeaTac Airport Community: Average Annual Count	SeaTac: Rate per 100,000	SeaTac 95% Confidence Interval: Lower Bound	SeaTac 95% CI: Upper Bound	King County: Average Annual Count	King County: Rate per 100,000	King County 95% Confidence Interval: Lower Bound	King County 95% CI: Upper Bound	Comparison to King County Rates: Significant Difference**	Comparison: Percent Difference
Unintentional Injury:												
Motor Vehicle Accident-Related Injury	Hospitalizations*	92-96	97	94.6	85.8	103.5	1,301	82.6	80.5	84.7	Higher	15
Falls	Hospitalizations*	92-96	319	213.6	202.4	224.8	4,882	208.4	205.6	211.3	NS	2
Fractures	Hospitalizations*	92-96	339	263.4	249.9	276.9	4,891	239.3	236.1	242.6	Higher	10
..Hip Fracture	Hospitalizations*	92-96	95	47.3	42.9	51.8	1,425	43.4	42.3	44.5	NS	9
All Unintentional Injury	Hospitalizations*	92-96	880	693.9	672.7	715.1	12,742	644.9	639.8	650.1	Higher	8
All Unintentional Injury	Deaths*	93-97	33	28.8	24.1	33.6	463	24.2	23.1	25.2	NS	19
Intentional Injury and Violence:												
Child Abuse (Ages 0-17)	Reports to CPS***	92-93	1,187	5002.5	4803.4	5208.0	9,704	2585.3	2549.0	2621.9	Higher	93
Assault	Hospitalizations*	92-96	57	60.2	52.9	67.4	696	45.3	43.8	46.9	Higher	33
Homicide	Deaths*	93-97	6	7.3	4.7	9.9	88	6.0	5.4	6.6	NS	22
All Firearms‡	Deaths*	93-97	15	15.7	12.0	19.5	163	10.3	9.5	11.0	Higher	52

†Hospitalization and CPS data for SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
Mortality and crime data for SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
*Rates are age-adjusted to the 1940 U.S. Population.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
***Rates on child abuse are not age-adjusted.
‡All Firearms includes Suicide, Homicide and Accidental Shootings.
Sources:
Mortality data: Washington State DOH, Center for Health Statistics
Hospitalizations data: Washington State DOH, Office of Hospital and Patient Data Systems
Child Abuse data: Washington State DSHS
Washington State DSHS for adjusted population estimates (6/97).

Table 11. Mental Health and Substance Abuse in SeaTac Airport Community† and King County

			SeaTac Airport Community				King County				Comparison to King County Rates	
					95% Confidence Interval				95% Confidence Interval			
Indicator	Type of Data	Time Period Analyzed	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Depression	Hospitalizations	92-96	145	137.2	126.7	147.7	2,231	134.5	131.9	137.1	NS	2
Suicide	Deaths	93-97	15	14.7	11.2	18.3	201	11.5	10.7	12.2	NS	28
Suicide Attempt	Hospitalizations	92-96	57	57.7	50.7	64.6	870	54.8	53.1	56.5	NS	5
Alcohol-Related	Hospitalizations	92-96	370	321.4	306.6	336.2	4,931	274.3	270.9	277.8	Higher	17
Chronic Liver Disease/Cirrhosis	Deaths	93-97	9	7.3	5.1	9.4	130	6.6	6.1	7.1	NS	11
Illicit Drug-Related	Hospitalizations	92-96	226	212.9	200.2	225.5	2,998	172.4	169.6	175.3	Higher	23

†Hospitalization data for SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
Mortality data for SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
*Rates are age-adjusted to the 1940 U.S. Population.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Sources:
Mortality data: Washington State DOH, Center for Health Statistics
Hospitalizations data: Washington State DOH, Office of Hospital and Patient Data Systems
Washington State DSHS for adjusted population estimates (6/97).

Table 12. Communicable Disease in SeaTac Airport Community† and King County

		SeaTac Airport Community				King County				Comparison to King County Rates	
				95% Confidence Interval				95% Confidence Interval			
Disease	Time Period Analyzed	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Average Annual Count	Rate per 100,000*	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Chlamydia	93-97	297	437.7	416.7	458.6	3,457	289.0	284.8	293.1	Higher	51
Gonorrhea	93-97	80	111.8	100.8	122.9	1,218	91.4	89.0	93.7	Higher	22
Food and Waterborne Disease^	93-97	53	58.0	50.9	65.2	1,144	73.7	71.7	75.6	Lower	-21
New AIDS Cases	95-97	15	14.9	10.9	19.9	387	23.7	22.3	25.1	Lower	-37
New TB Cases	90-94	9	7.6	5.2	9.9	114	6.6	6.1	7.2	NS	15
Vaccine Preventable Disease‡	91-95	4	4.5	2.4	6.6	39	2.7	2.3	3.1	NS	67

†Chlamydia, gonorrhea, food/waterborne disease and vaccine preventable disease data for SeaTac Airport Community includes census tracts: 264-271, 273-276, 278-281, 284.01, 284.02, 284.03, 285-287, 288.01, and 288.02.
AIDS and TB data for SeaTac Airport Community includes zip codes: 98146, 98148, 98158, 98166, 98168, and 98188.
^Food & Waterborne Disease includes Campylobacteriosis, Salmonellosis, Shigellosis, Giardiasis, E. coli 0157:H7, and other enteric communicable diseases.
‡Vaccine preventable disease includes measles, mumps, rubella, pertussis, diphtheria, tetanus, and invasive *Haemophilus influenzae* serotype B.
*Rates are age-adjusted to the 1940 U.S. Population.
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Sources: Washington State DOH STD/TB Services and Communicable Disease Epidemiology.
Seattle-King County DPH, HIV/AIDS Epidemiology.
Washington State DSHS for adjusted population estimates (6/97).

Table 13. Access to and Utilization of Health Care in South King County* and King County, 1994-95

Indicator	South King County				King County				Comparison to King County Rates	
			95% Confidence Interval				95% Confidence Interval			
	N^	Weighted Percent	Lower Bound	Upper Bound	N^	Weighted Percent	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Does not Have Health Insurance (age 18-64):	284	12.1	8.0	16.2	2,268	11.4	9.4	13.4	NS	6.1
Needed to see a doctor in last year but didn't because of cost (age 18 and older):	405	13.5	9.0	18.1	2,268	11.0	9.0	13.0	NS	22.7
Women over 50 not having mammogram in last 2 years:	124	22.5	14.2	30.7	740	21.4	17.7	25.1	NS	5.1
Women not having Pap Smear in last 3 years (age 18 and older):	155	16.9	9.3	24.5	622	12.9	9.4	16.3	NS	31.0

*South King County includes the following zip codes: 98001-98003, 98010, 98022, 98023, 98031, 98032, 98038, 98042, 98047, 98051, 98055, 98056, 98058, 98059, 98070, 98092, 98146, 98148, 98158, 98166, 98168, 98178, 98188, and 98198.

^N=Number of valid responses to question (excluding "Do Not Know" and no answer).

**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.

Source: Washington State Behavioral Risk Factor Surveillance System (1994) and Seattle-King County DPH Small Area Behavioral Risk Factor Survey (1995)

Table 14. Behavioral Risk Factors for Disease and Injury in South King County* and King County, 1994-95

Risk Factor	South King County				King County				Comparison to King County Rates	
			95% Confidence Interval				95% Confidence Interval			
	N^	Weighted Percent	Lower Bound	Upper Bound	N^	Weighted Percent	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Smoker:	405	23.8	18.4	29.1	2,276	18.7	16.4	21.1	NS	27
Does not eat 5 or more fruits/vegetables per day:	381	89.4	85.9	92.8	2,136	88.3	86.5	90.0	NS	1
Overweight:	399	28.3	22.6	34.0	2,244	21.8	19.4	24.2	NS	30
No physical activity in past month:	405	16.2	11.9	20.6	2,275	14.8	12.8	16.8	NS	9
Youngest child (age 5-17) does not wear helmet all or most of the time when riding bicycle:	75	33.6	22.0	45.3	336	29.8	23.6	36.0	NS	13

*South King County includes the following zip codes: 98001-98003, 98010, 98022, 98023, 98031, 98032, 98038, 98042, 98047, 98051, 98055, 98056, 98058, 98059, 98070, 98092, 98146, 98148, 98158, 98166, 98168, 98178, 98188, and 98198.
^N=Number of valid responses to question (excluding "Do Not Know" and no answer).
**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.
Source: Washington State Behavioral Risk Factor Surveillance System (1994) and Seattle-King County DPH Small Area Behavioral Risk Factor Survey (1995)

Table 15. Firearm Risk Factors in South King County* and King County, 1994-95

Risk Factor	South King County				King County				Comparison to King County Rates	
			95% Confidence Interval				95% Confidence Interval			
	N^	Weighted Percent	Lower Bound	Upper Bound	N^	Weighted Percent	Lower Bound	Upper Bound	Significant Difference**	Percent Difference
Have loaded gun in home:	399	16.5	11.9	21.2	2,239	12.5	10.5	14.5	NS	32
Have loaded handgun in home:	399	15.3	10.7	19.8	2,235	10.9	9.0	12.8	NS	40

*South King County includes the following zip codes: 98001-98003, 98010, 98022, 98023, 98031, 98032, 98038, 98042, 98047, 98051, 98055, 98056, 98058, 98059, 98070, 98092, 98146, 98148, 98158, 98166, 98168, 98178, 98188, and 98198.

^N=Number of valid responses to question (excluding "Do Not Know" and no answer).

**Lower=lower than King County rate; higher=higher than King County rate; NS=not statistically significant.

Source: Washington State Behavioral Risk Factor Surveillance System (1994) and Seattle-King County DPH Small Area Behavioral Risk Factor Survey (1995)