

# **Peterborough Health Study**

**GE Canada**

**Final Report**

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## Preface

The report is organized in two sections based on the two phases of the study; Phase I being the initial study to determine whether there were any differences between the plant population for cancer mortality when compared to the Ontario population. Phase II covered the case-control study to understand whether cases with lung cancer had a different exposure profile to the control group with no lung cancer.

We chose a writing style to afford employees the ability to follow the steps taken along the way i.e. using bullet points, flow diagrams and simple tables.

The study team wish to thank the members of both joint and safety committees (CAW & CEP), the local union presidents, the Union National Offices (G. Botic and J. Carr), Dr. T. Haines from McMaster University, Dr. P. Corey from University of Toronto, Dr. E. Holowaty from Cancer Care Ontario, Dr. L. Genesove from the Ontario Ministry of Labour, Dr. G. Humphreys from the Peterborough Health Center, Ms. Claire-Marie Fortin from the WSIB, Princess Margaret Hospital, Kingston General Hospital, Peterborough Health Unit, Cancer Care Ontario and Statistics Canada.

HRH  
DG

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# Peterborough Health Study, GE Canada

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# **Peterborough Health Study, GE Canada**

## **Phase I**

### **1. Introduction**

- A concern was raised through the CAW/GE Joint Health and Safety Committee about possible excess cancer deaths among Peterborough plant employees.
- The concern was not related to any particular site, but was general in nature.
- The committee requested that the plant management respond to the CAW members' concerns.

### **2. Overview**

#### **2.1 Study Design and Data Analysis**

- On the basis of a preliminary review of the nature of the concern and available epidemiologic methodologies, it was concluded that a death based analysis, such as proportional mortality ratio analysis (PMR) would be the most appropriate.
- The Committee's Union representatives Dr. Ted Haines - professor at McMaster University, as well as Dr. Gary Humphreys - the local medical officer of health acknowledged the method, which formed the basis of this Phase I.
- A proportional mortality study includes only deaths.
- Proportional Cancer Mortality Rates (PCMR) is a screening technique. It only examines the degree of departure of a given observed value from the expected one. In other words, to what extent an observed number of deaths for a given cancer site measured up when compared to an external population. If both the observed and expected values are equal, the PCMR is said to be one. This would be interpreted as exhibiting a similar mortality experience in the plant and the comparison population. However, not all ratios will be equal to one; some will be higher and some will be lower which could be due to some chance statistical variations or due to other factors.

Advantages PCMR design	Disadvantages PCMR design
<ul style="list-style-type: none"> <li>• Most suitably used to explore for disease excesses and deficits on preliminary analysis of the available data.</li> </ul>	<ul style="list-style-type: none"> <li>• Validity depends on whether the deaths included are representative of all deaths that would be identified if complete follow-up of the full cohort.</li> </ul>
<ul style="list-style-type: none"> <li>• Good approximations to SMRs (Standard Mortality Rates) from cohort studies when all-causes combined SMR=1 (observed=expected).</li> </ul>	<ul style="list-style-type: none"> <li>• Does not directly measure the risk of dying from (e.g.) lung cancer, but the difference in mortality from other causes of death.</li> </ul>
<ul style="list-style-type: none"> <li>• Greater confidence for PCMR, since the Healthy Worker Effect less affects cancer mortality.</li> </ul>	

- The analysis was carried out using a widely used USDR program of statistical interpretations developed by Dr. Richard Monson at Harvard University.
- There also is a need to examine statistical variability. For example, if given PCMRs equal to 1.05 or 1.25, do they mean they are in excess? Or is it possible that they do not differ from one? A question can be raised: what is the variability in the PCMR observed if similar analysis is carried out many more times? This question can be answered by calculating the 95% Confidence Intervals for PCMR.
- There are two possible outcomes: 1) If the confidence intervals cover one, i.e. 0.85-1.10, then PCMR is not statistical significant. 2) If the confidence intervals do not cover one, i.e. 0.75-0.97 or 1.11-2.30, then the observed PCMR demonstrates a significant deficit or is significant higher than 1.
- The calculation of PCMR requires an external comparison population. Although the local population would be the most suitable, it generally falls short on data stability.
- The criteria for selecting the comparison group was to see that it meets the goals, readily available and large in numbers, therefore stable. The Ontario cancer data provided such an opportunity.

## 2.2 Ontario Cancer Registry Data Management

- The Ontario Cancer Treatment and Research Foundation now called Cancer Care Ontario operates the Ontario Cancer Registry (OCR). The OCR is a population-

based registry that contains information on all new cases of cancer diagnosed in Ontario since 1964. The OCR also receives death certificates for all Ontario residents from the Office of the Registrar General.

- Death registration is a legal process in Ontario. A death must be registered before the body can be interred. Registration process consists of two parts:
  - the medical certificate of death (completed by the physician in attendance of the deceased during the last illness; includes the main cause of death and the chain of events leading to death. For statistical purposes the cause selected for coding and tabulation of the official cause-of-death statistics is the "underlying cause of death" which is considered the same with the main cause of death.
  - the non-medical portion of death registration (completed by the funeral director; includes date of birth, sex and residence at time of death).
- Both parts are submitted by the funeral director to the office of the municipality, which completes the burial permit. The municipal office forwards the two forms to the Registrar General in Ontario.
- At the Office of Registrar General, both medical and non-medical data are coded by trained medical coders according to the International Classification of Diseases (ICD) in use at time of death (the 8<sup>th</sup> revision for death<1979, the 9<sup>th</sup> revision for death >1979). Autopsy findings are rarely taken into account in determining the certified cause of death, since the death must be registered immediately, before autopsy findings are available. In addition, there are no administrative procedures for routine reporting of autopsy findings to the Registrar General once the death has been registered.
- Coded data are provided every 6 months to OCR and Statistics Canada.
- The malignant neoplasm section of ICD-9 comprises codes 140 to 208. Most of the 3-digit codes in this range represent the organ of origin of the neoplasm, such as lip, stomach or prostate. The fourth digits of the ICD-9 codes permit more precise classification of the site or type of cancer. The OCR using standard conversion tables has converted all cancer data to ICD -9<sup>th</sup> codes.
- "Proof of death" form is not an official document. It is a formula used by the Insurance Company.



## **3. Methods**

### **3.1 Study population**

- The initial premium list included all GE Canada employees; those with a Peterborough address were checked against the Peterborough employee records to validate that they did work in Peterborough.
- The study population (GE cohort) consists on all GE Peterborough plant's employees (alive or dead) between Jan. 1970 and June 1998. Because of the study design and analytical tool selected, all the alive employees have been excluded from the beginning (no information was collected). (See Figure 1; Page 23).
- Before 1986, according to the legislation, only people who worked 10 or more years for GE were entitled to pension benefits.
- After 1986, the legislation was reduced to two years in order to be entitled to pension benefits.

### **3.2. Data Collection**

#### **3.2.1 Data Sources**

- Three lists represented the data-sources for this review:
  - List I with 1818 names, received in June 1997 for period 1970-1994 from GE Canada Pension Records (Initial Period of Study). (See Page 24).
  - List II from CAW, with 242 names, received in February 1998. (See Page 25).
  - List III with 368 names, from Meadowvale Pension Records for period 1995-Mid 1998, received in October 1998 (Second Period included in study). (See Page 26).
- For all the names (2428) included in the three lists we used the following inclusion/exclusion criteria.

#### **3.2.2 Inclusion Criteria**

- Name found in the GE Canada Pension List, Meadowvale (See Figure 1; Page 23).

- Date of Death: - 1970-1994 - because it was thought the quality of the data will be good as both the Company records and the Ontario Cancer Registry data were shown to be better organized from 1970. A test was done on the data from 1965 to 1969, which confirmed the lack of good records in both places. Later on as the review extended into late 1998, a joint decision was made to increase the population size by including the period 1995 to mid-1998.
- Each name must be found in OCR's mortality database.

### **3.2.3 Exclusion Criteria**

- Name not found in the GE Canada Benefits List (See Figure 1; Page 23).
- Name not found in OCR-mortality database.
- Name without employee work file or chart.
- Date of Death out of the study period (January 1970 - June 1998).

### **3.2.4 List I**

- The data collection started with List I. (see Figure 2; Page 24).
- A database was created from the annual logs from GE Canada Benefits Department for the years 1970-1994. These logs contain data of death, employee name, status (i.e. active, pension), and in most of the cases location plus an internal file number. In order to appear on this log, the employees must have been eligible for payment under the company benefits program. An attempt was made to further extend the population to 1965-1969. It was found that a significant number of cases were missing compared to other five years intervals and therefore this population dropped.
- The database was created with the following fields: Surname, First name, Second name, Third name, Sex, SIN, Employee Number, Date of Birth, Date of Death. This information was then sorted in alphabetical order by surname, then given names.
- The database was submitted to OCR (1<sup>st</sup> submission) in October 1997. For 1726 names the ICD-codes were found, 92 names were not in OCR mortality data.
- In April 1999, the 92 names were re-submitted to OCR for a record linkage and 6 more ICD-codes were found, one name was presented twice and 85 names were still not found in OCR mortality data.
- So, from the initially 1818 names, we have 1732 available for analysis, 1 presented twice, and 85 not in OCR mortality data. (See Figure 2; Page 24).

### 3.2.5 List II

- The CAW provided a list with 242 names in February 1998. (See Figure 3; Page 25).
- From this list, 131 were identified as included in the List I.
- Except 70 names (Not qualified by the inclusion/exclusion criteria established for the review) the rest of the list was submitted to OCR (1<sup>st</sup> time).
- For the initial period, only deaths occurred between 1970-1994 were included in the study.
- From OCR we received 41 ICD-codes, but only 34 names were found in M-vale Benefits List, so we excluded the 7 missing names.
- In April 1999, when the inclusion period for the study was extended to June 1998, we re-submitted the 70 names previously "not qualified" to OCR. No additional information was found.
- In the same time we cross-validated the 34 names with ICD-codes with the List I, checking for transposition of Surname/First Name, wrong SIN #. We found that 17 names were presented twice.
- So, finally, from List II we have 17 additional] names for analysis, 148 names presented twice, and 77 "not qualified" (under the review criteria). (See Figure 3; Page 25).

### 3.2.6 List III

- This list contains deaths from January 1995 to June 1998. (See Figure 4; Page 26).
- Initially the list was cross-validated with CAW-List and GE Canada Benefits List; 14 names were presented twice.
- The rest of the names were submitted to OCR (1<sup>st</sup> time) and for 257 names the ICD-codes were found, and 97 names were "not qualified".
- In April 1999, the list with "not qualified" was re-submitted to OCR and 1 more ICD-code was found.
- The 257 names with ICD-codes were cross validated against List I, checking for transposition of Surname/First Name or wrong SIN # and 6 names were presented twice.
- So, from List III, we have 252 additional names for analysis, 21 names presented twice, and 95 not qualified.

### 3.2.7 List IV

- Finally, the names available for analysis (1732 from List I+ 17 from List II+ 252 from List III= 2001) were included in the final data set (List IV).(See Figure 5; Page 27).

### 3.3. Data Handling at OCR

- A diskette was provided to OCR containing the following information: Surname, First, Second and Third Name, Sex, SIN, Date of Birth, Date of Death. AutoMatch<sup>R</sup>, a generalized probabilistic record linkage program, was used to link the GE file to the Ontario Mortality File, males and females combined, for the years 1970 to 1998 inclusive. The record linkage process involves blocking and matching on a given variable. In total, five passes were implemented for this linkage.
- Each pass used different data fields common to both files for blocking to allow records, which failed to match in a previous pass to match in a subsequent pass.
  - Pass #1: blocking variables: SIN and Sex variables
  - Pass #2: Surname initial, Given name initial, Birth year and Sex variables
  - Pass #3: Full Date of Birth, Sex variables
  - Pass #4: Surname and Sex variables
  - Pass #5: Date of Death and Sex variables
- By using this information, the Ontario Cancer Registry office identified the cause of death code (ICD-8<sup>th</sup> or ICD-9<sup>th</sup>). There were a number of cases that could be not matched. These were likely deaths that occurred outside the province of Ontario.
- All ICD-8<sup>th</sup> codes were converted into ICD-9<sup>th</sup> codes, to facilitate the data analysis

### 3.4 Macro Job Coding

#### 3.4.1 Primary job status codes

- The output file from the OCR was transformed in a list, which included only identifying information and not the ICD-codes. This list was sent to Peterborough for primary job status coding.
- For each person included in the List IV, 4 variables were created as primary job status coding:
  - First time with GE: H (hourly), S (salary)
  - Last time with GE: H (hourly), S (salary)

- Last time with GE: A (active), R (retired)
- Manufacturing: M (manufacturing- at least 1 year on the floor),
- Non-Manufacturing: nM (did not work on the floor)

### 3.4.2 Final job status codes

- The primary job status coding was converted in the final job status coding (which included 3 variables).
- First variable designated the job status the very first date the employee was hired.

There are 2 codes:

1= HA ; hourly active

3= SA ; salary active

- Second variable designated the job status at the last date of employee worked.

There are 4 codes:

1= HA ; hourly active

2= HR ; hourly retired

3= SA ; salary active

4= SR ; salary retired

Active means, job status at or prior to time of death. Those who retired with early disability were considered as active for epidemiological purpose. Retired were those with death benefits.

- Third variable indicated manufacturing exposure -there are 4 codes:

1= MA ; manufacturing active

2= MR ; manufacturing retired

3= nMA ; non-manufacturing active

4= nMR ; non-manufacturing retired

Manufacturing meant working on the floor for one year or more.

Non-manufacturing meant that the employee did not work on the floor.

### 3.4.3 The job status codes were introduced in the list created from the OCR output.

- The data was transformed into a format that was suitable for input to the Epidemiology Records and Reporting System (ERRS) Program for data analysis.

### 3.4.4 Comparison population

- OCR provided a Comparison population database.
- They extracted the number of cancer deaths (ICD-codes 140-208) by year and Sex from OCR mortality database for Ontario 1970-1998.
- The record layout contained as variables: year of death (1970,1971,1972....1998), sex, cancer site (ICD-9 Code), data by five-year age group (18 age groups: 0-4, 5-9, 10-14, .....85+).

### 3.5 The Analytical Tool

- The Monson computer program (See Page 7) was used to determine the expected number of deaths in a cohort, and to calculate Proportional Cancer Mortality Rates, utilizing cause-age-time-sex-specific mortality rates.
- Briefly, for each sex and in each 5-year age and calendar time group, the proportion of Ontario residents who died of each cause of cancer was applied to the number of cancer deaths in the study cohort and the expected values summed over all age-time subgroups.
- The steps in the computer program:
  - Step 1. The proportional cancer mortality data matrix is read.
  - Step 2. The age-year-of-death distribution of all deaths in the study population is determined in five-year groupings.
  - Step 3. The age-time distribution of the observed deaths is determined.
  - Step 4. The age-time-specific expected deaths for each cause is calculated by multiplying the proportional cancer mortality matrix times the age-time distribution of total deaths.
  - Step 5. Proportional cancer mortality ratios are calculated by dividing the observed ratios by the expected ratios.
  - Step 6. The Mantel-Haenszel Chi square is calculated.
- The PCMR analysis consisted of 4 parts:
  - Arranging the external data set (i.e. study-population) into a format readable by the ERRS program.
  - Arranging the Comparison population data set (i.e. Ontario cancer data) into a format readable by the ERRS program.
  - Using the Comparison population to generate the Rates.

- Run the analysis for each data set (i.e. All\_male, Hourly, Salary)

## **4. Data Validation**

### **4.1 Data - Source Level**

- A manual search was performed, including search for transposition of Day/Month in Date of Birth or Date of Death and Surname/First Name.
  - **List I** - Cross-validated with GE Canada Benefits list.
  - **List II** - Cross-validated with List I (in order to exclude the subjects presented twice) and with GE Canada Benefits List.
  - **List III** - Cross-validated against list I and II (in order to exclude the subjects presented twice), and GE Canada Benefits list.

### **4.2 The Office of the Registrar General**

- The underlying cause of death is coded by trained medical coders using ICD in use at time of death (the 8<sup>th</sup> revision for the years: 1970-1978; the 9<sup>th</sup> revision since 1979).
- When important information is either missing or inadequately provided on the Death certificate, the Registrar General Office routinely requests clarification from the individual responsible (i.e. the funeral director or physician).

### **4.3 OCR**

- All data has been converted by the OCR to ICD-9<sup>th</sup> using standard conversion tables.
- For the cases that could not be found by record linkage, a manual search was performed (including a search for transposition of Day/Month in Date of Birth or Date of Death and Surname/First Name)

### **4.4 For subjects with ICD-codes from OCR**

- The names with ICD codes from List I, II and III were cross-referenced against employee card files. In the majority of the cases, a match was made on the basis of the name and date of death. Additional data from the card files were used to fill in any missing information. A database was further checked taking into consideration information available from the union. With the exception of 7 cases, which were not

included in the analysis, The GE Canada Benefits list was found to be reliable. The final list (List IV) contained 2001 names.

- For the 7 missing names, the annual death logs were checked 5 times; for
- transposition of day/month in the Date of Death, or for Surname/First Name.
- A sample of 60 subjects was checked for validation of the inclusion of Date of Birth and Date of Death. Each 30<sup>th</sup> subject from the final list (2001 names) was compared with the original data-source. The data matched.
- All cancer cases were controlled for the conversion of ICD-8<sup>th</sup> into ICD-9<sup>th</sup> version. The data was sorted using Excel -program, and the columns of ICD-8 and ICD-9 were checked for each row and compared with the conversion-table received from OCR.
- Finally, a sample of 75 subjects was checked (each 25<sup>th</sup> name in the 2001 list). For cancer as cause of death, the codes were compared with OCR's conversion table.
- For other causes of death, the codes were compared with the books ICD-8<sup>th</sup> and ICD-9<sup>th</sup> revisions. The data matched.

#### **4.5 Job Status Coding (Primary) - Peterborough**

- The results for primary coding were checked for consistency (a sample of 45 subjects). (No error was found).
- We used: - list with 2001 names for analysis, (with all the information except the ICD-8 and ICD-9 codes), - the Human Resources Charts (HR charts), - a list with explanation for each code used in the HR chart (in order to verify if the person worked on the floor, or had a chance to be exposed).
- The selection was done using the 10<sup>th</sup> name from the alphabetical list (2001) for 20 subjects, the 25<sup>th</sup> name from the list for other 10 subjects, the 50<sup>th</sup> name from the list for other 10 subjects and finally 5 subjects were randomly selected from the three boxes with charts. There were no discrepancies.

#### **4.6 Job Status Coding - final coding and inclusion in the final data set level**

- In order to validate the final coding for job status, a sample of 50 subjects was checked. Each 25<sup>th</sup> name on the alphabetical list with primary job status coding for 30 subjects and each 50<sup>th</sup> name for other 20 subjects. (No error was found).
- The results were checked for consistency (a sample of 50 subjects) for inclusion



in the final data set. Each 30<sup>th</sup> name from the list was selected in the sample. (No error was found).

#### **4.7. Final pre-analysis check**

- The final pre-analysis check was conducted by an individual not previously associated with the review. An examination of a portion of the data and documentation was done to identify any systematic problems with the procedures. The examination consisted of three parts: a horizontal data evaluation, a vertical data evaluation, and a categorization comparison.

##### **4.7.1. Horizontal Data Evaluation**

- Examination of data processing results across a group of variables and individuals.
- ICD 8 to ICD 9 conversion – compared on specific categories. (No miss-matches found).
- Proof of death forms. Examined consistency of coding, inclusion, and OCR results. (No systematic miss-matches identified).
- Age at death calculation – compared death-birth year to calculated value. (No errors found).

##### **4.7.2. Vertical Data Evaluation**

- Examination of individuals from different lists to check for application of criteria and documentation.
- Examination of 5% sample from List II. The status of each member of the list was noted and, if appropriate, the reasons for exclusion.
- Other combined List IV. Tracked individuals through the process of initial identification to final analysis. ( No systematic problems identified).
- Documentation of past data validation of work histories and list creation was reviewed. The documentation allowed re-creation of the steps used for the actual processing.

## **4.8. Analytical Tool Validation**

- To check on reliability of the ERRS program categorization a parallel categorization was done using SPSS (a standard statistical package). The following steps were used:
  1. Imported the data from Excel into SPSS.
  2. Created frequency tables, ICD categorization, age groups, and decade of death variables for comparison.
  3. Compared results of each variable to ERRS subsets (by gender, pay class, and manufacturing status).
  4. Minor discrepancies were noted and resolved. (No major discrepancies were detected).
- The final results of both data sets were consistent.

## **4.9 Data validation for PCMR analysis**

### **4.9.1. Off Site Data Analysis**

- The PCMR analysis was first done in off site (May, 1999), as they had resources to handle the data. The external data set (List IV, ICD-8) was transformed into a readable data by the ERRS, using a program written in off site.
- The results obtained for the Observed cases did not match the numbers obtained by descriptive statistics.
- The Comparison population from OCR (ICD-9), with some modifications in order to convert it into ICD-8, was used to generate the Rates.
- A parallel ERRS program was installed in Canada, to cross validate with the off-site. A new program was written in Canada in order to convert the data set into a format readable by the ERRS program. The same rates generated off-site were used in Canada for PCMR analysis.
- The results obtained in Canada were different than the ones obtained off site, but they were validated by descriptive statistics.
- The "readable" data sets created in Canada (using the program written here), were sent off site, and the PCMR analysis was redone there. The results were validated with the ones obtained in Canada.

#### **4.9.2. On Site Data Analysis (Canada)**

- In Canada (June, 1999), we started the validation of the Rates-generation (ICD-8) obtained off site (4.9.1). The validation included 4 levels:
  1. The Comparison population (ICD-8) used off site, was compared with the one obtained from OCR (ICD-9) and modified into ICD-8.
  2. For the conversion of the Comparison data into a readable file by the system we could not use the program written offsite; the program was entering into an infinite loop because of mis-matching of the input data format. We did the necessary modifications to the program in order to run correctly.
  3. The On-Site "Candeath data" (an intermediate step for rates generation) obtained with the new program was the same with the one from Off-Site.
  4. From "Candeath data", the Rates for 1970-1998 were generated, and compared with the ones obtained off site. The rates were the same.
  5. At that point, the validity of the PCMR analysis results obtained in section 4.9.1 was confirmed.

#### **4.9.3. Comparison Population conversion to ICD 9**

- In this study, both the initial data set and the Comparison population contained ICD-9 codes. Because in this study we are interested in PCMR using only Ontario population as comparison, we were able to run the analysis using ICD-9 codes. The previous limitation of using just ICD-8 codes was because the Monson program was based on US population as comparison (records were kept as ICD-8 codes).
- Starting from the Comparison population, we re-generated the rates for ICD-9 codes, following which the PCMR analysis was performed. The observed numbers obtained were cross validated using descriptive statistics.
- All the numbers were correct.

## **5. Results**

### **5.1 Demographics**

- The demographic characteristics of the review subjects are provided in Table 1; Page 29.

- The Peterborough group of deceased employees included in the review was largely male (92.9%), had worked in manufacturing at some time (87.4%), and started as hourly employees (81.9%).
- The majority of the subjects died after age 70 and the decade of death was evenly distributed over the time period (1970s 31.6%, 1980s 34.1%, and 1990s 34.3%).
- Table 2 (Page 30) compares the hourly to salary demographic characteristics. The average (mean) age at death was nearly identical for hourly employees (70.8 years) and salaried employees (70.3 years). Slightly more females were in the salaried group than in the hourly group. The majority of the hourly employees were on the manufacturing floor (99.9%) compared to the salaried group (62%).

## **5.2 Cancer Comparisons**

- The demographic comparison between those who died from cancer is made with those who died from other causes in Table 3, Page 31.
- The hourly employees were less likely to die from cancer (65.8%) compared to other causes (72.1%).
- The salaried employees were more likely to have died from cancer (34.2%) compared to other causes (28%).
- The cancer deaths were more slightly younger (69.1 years) compared to other deaths (71.31 years) and there have been more cancer deaths in the 1990 decade than previous decades compared to other causes of death.

## **5.3 Male Employees**

- The results of the male PCMR analysis are presented in tables 4-10 (Pages 32-38).
- For all males presented in Table 4 there is a statistically significant excess of lung cancer based on 198 cases (PCMR 1.35, 95% CI 1.21-1.51) and for Hodgkin's disease based on 7 cases (PCMR 3.35, 95% CI 1.68-6.68).
- Table 5 presents the data for hourly male employees, which shows a similar pattern; a statistically significant excess of lung cancer based on 131 cases (PCMR 1.34, 95% CI 1.16-1.54) and for Hodgkin's disease based on 4 cases (PCMR 2.96, 95% CI 1.18 - 7.44).
- The parallel analysis for salaried employees is shown in Table 6 showing a statistically significant excess of lung cancer based on 66 cases (PCMR 1.4, 95% CI

1.15-1.70) and for Hodgkin's disease based on 3 cases (PCMR 4.13, 95% CI 1.47 – 11.56). In addition, the salaried male employees show an excess of prostate cancer (PCMR 1.5, 95% CI 1.01 – 2.22).

- When the analysis is done by manufacturing status the excess persists in those in manufacturing (Table 7) but not those in non-manufacturing jobs (Table 8) for those with lung cancer. The excess for Hodgkin's disease is significantly elevated in both groups. A combination analysis of the job and manufacturing status is shown in Tables 9 and 10. The salaried employees in the manufacturing group show the increase in lung cancer and the non-manufacturing group shows the excess for Hodgkin's disease. The parallel analysis for hourly employees was not done because virtually all were in the manufacturing group.

#### **5.4 Female Employees**

- The results of the female PCMR analysis are presented in tables 11-14 (Pages 39-42).
- The all-female analysis in Table 11 shows a similar excess of lung cancer to the males, based on 16 cases (PCMR 2.23, 95% CI 1.44 – 3.44). No cases of Hodgkin's disease were observed.
- When the analysis was restricted to hourly employees in Table 12 the lung cancer excess persisted, based on 9 cases (PCMR 2.29, 95% CI 1.29 – 4.08).
- Table 13 analyzes the females by manufacturing status, again the lung cancer excess is observed with 11 cases (PCMR 2.21, 95% CI 1.31 – 3.73).
- The salaried female employees also showed the excess of lung cancer (Table 14) with 6 observed cases compared to 2.93 expected (PCMR 2.05, 95% CI 1.01-4.18).

### **6. Discussion**

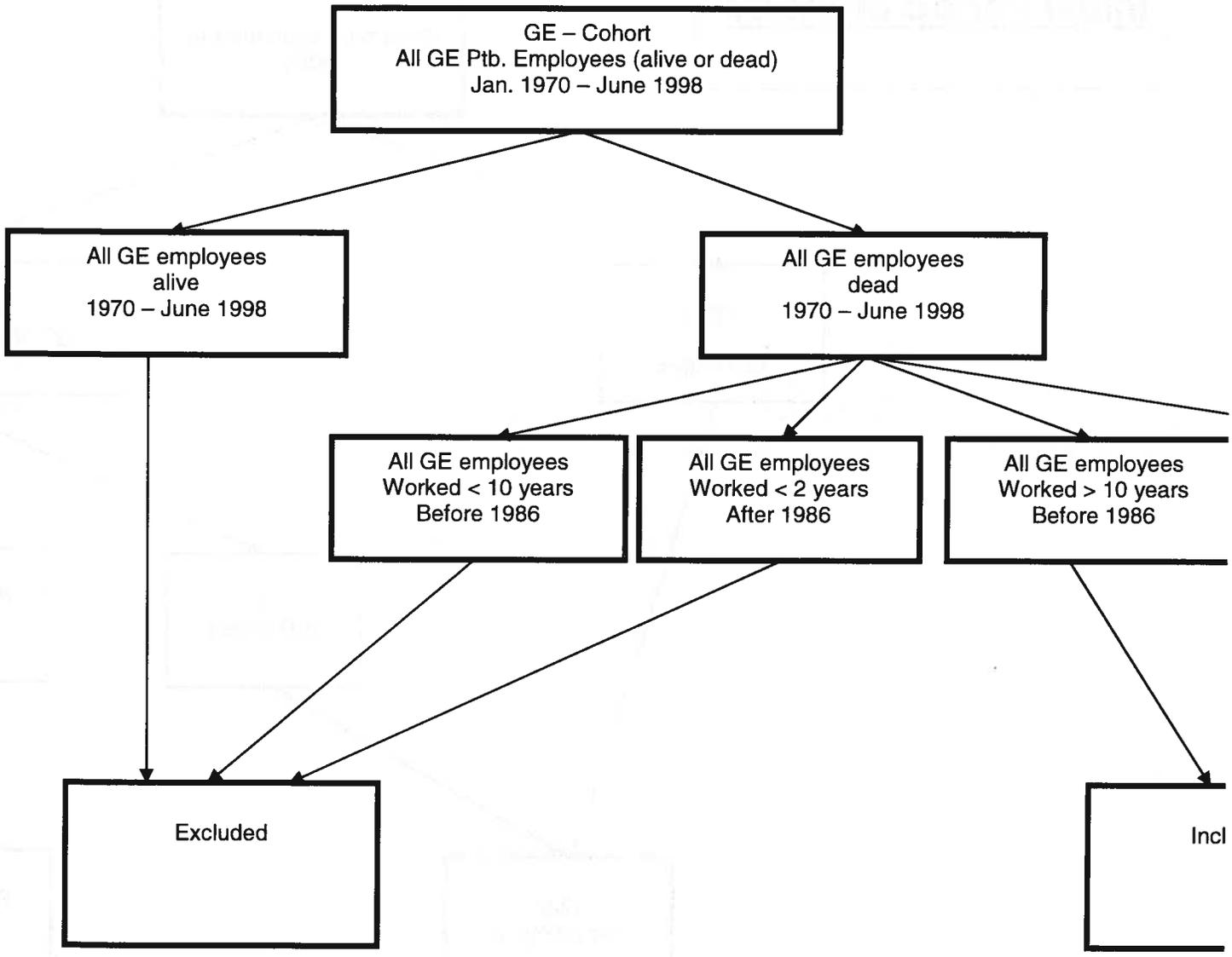
- A review of deceased employees from the Peterborough plant focused on the distribution of type of cancer identified as the cause of death and compared it to the Ontario distribution of cancer.
- Table 15 (Page 43) summarizes the statistically significant results. A statistically significant excess of lung cancer was observed in several analyses: for male employees - all males, hourly, salary, manufacturing, and salaried manufacturing;

female employees - all females, hourly, salaried, and manufacturing. Hodgkin's disease showed a similar significant excess pattern in the males but not in the manufacturing groups. No excess of Hodgkin's disease was observed in the females. An excess of prostate cancer was identified in the male salaried group.

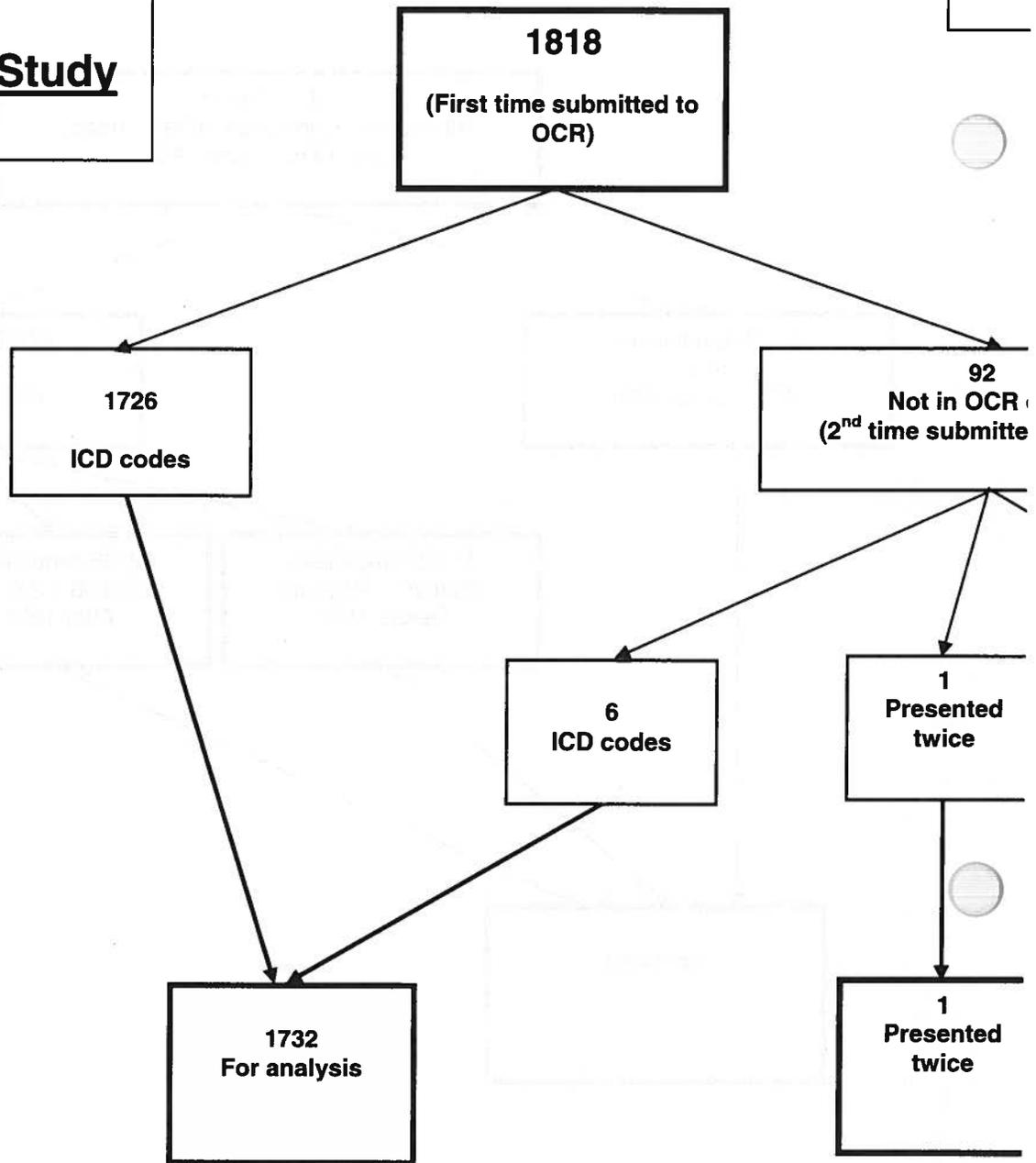
- As with any study the possibility of misclassification of the subjects is possible (i.e., to incorrectly assess their cancer status) however there is no reason to think this would bias the results in one direction or another. Misclassification is a greater problem for those whose cancer was not reported or diagnosed at the time of death. This should be identical in the GE population and the Ontario population. The primary exposure variables, manufacturing and pay class status are reliable throughout the Peterborough plant and are at minimal risk for misclassification.
- Confounders, which could be related to the observed excesses in lung cancer, include smoking and exposure to known human carcinogens. These will be examined in greater detail in a follow-up study (Phase II).
- The observed excesses between the GE group and the larger Ontario population require further investigation. The cause for the difference is not clear – it could be specific to the location of the plant, to exposures at the plant, or due to other factors.
- A follow-up plan to investigate the type of lung cancer, the smoking histories and detailed work histories was taken. This was done at Peterborough using available records from Human Resources and Industrial Hygiene data.

## **7. Conclusions**

- The PCMR study design comparing the deceased Peterborough employees and the Ontario population found several statistically significant excesses of cancer.
- The Peterborough employees were more likely to have died from cancers of the lung, Hodgkin's, and prostate (salary only). The excess was observed in several groups. No detailed work exposure or smoking history was available to assess further workplace effects.
- A plan for further data collection and analysis has been developed.
- The map-process of Phase I is presented in Figure 6 (Page 28).

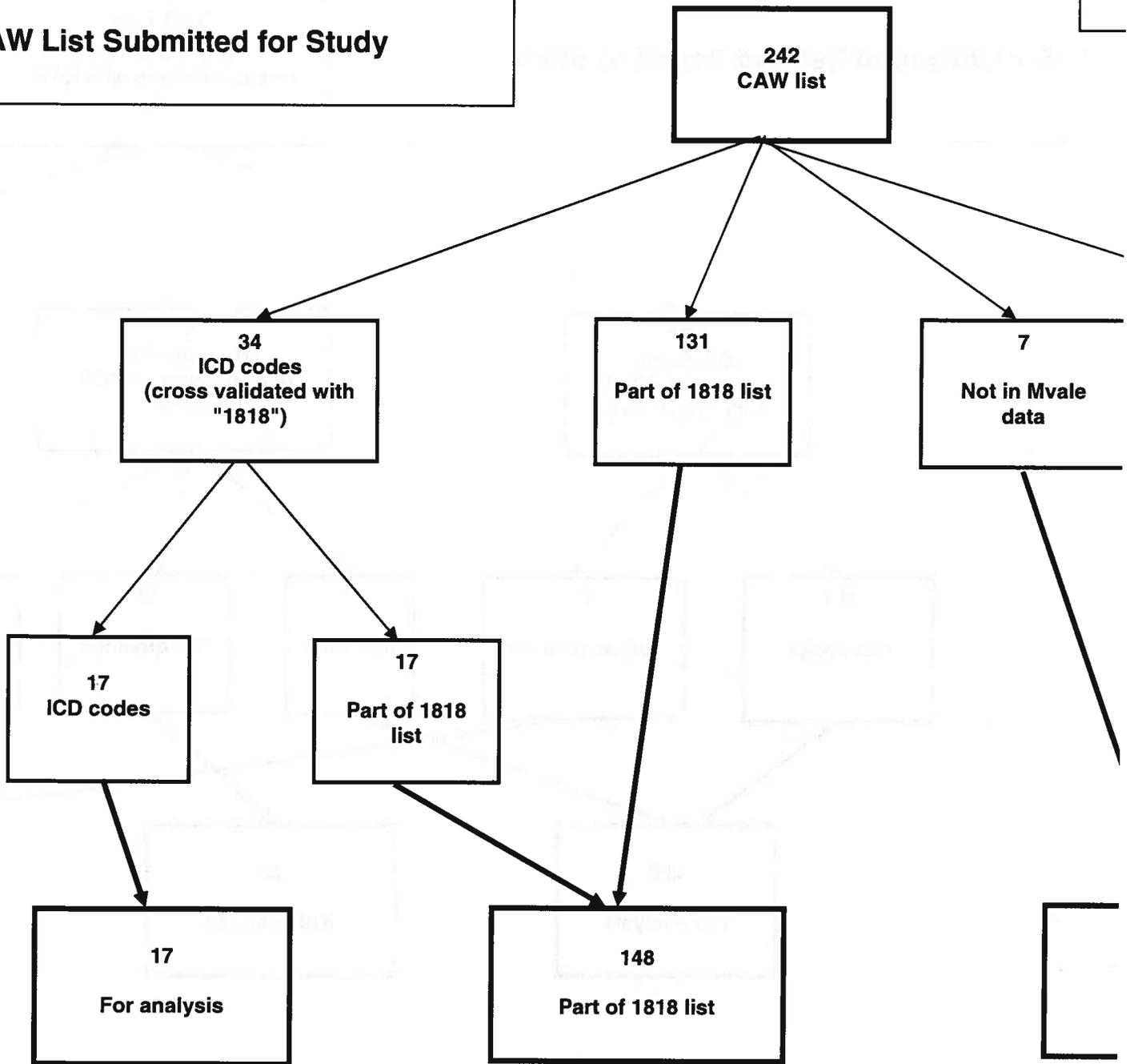


**Initial Period of Study**

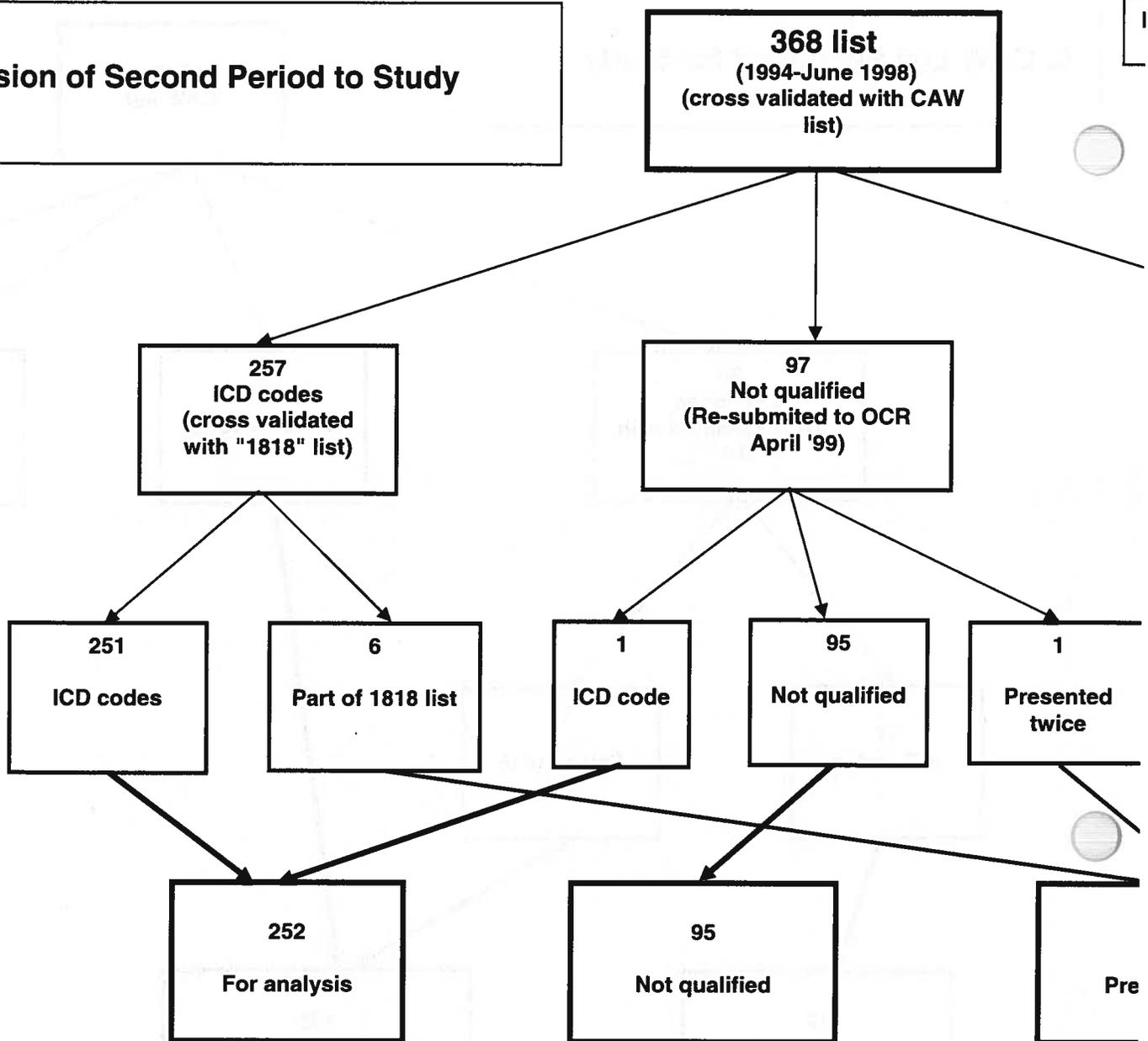




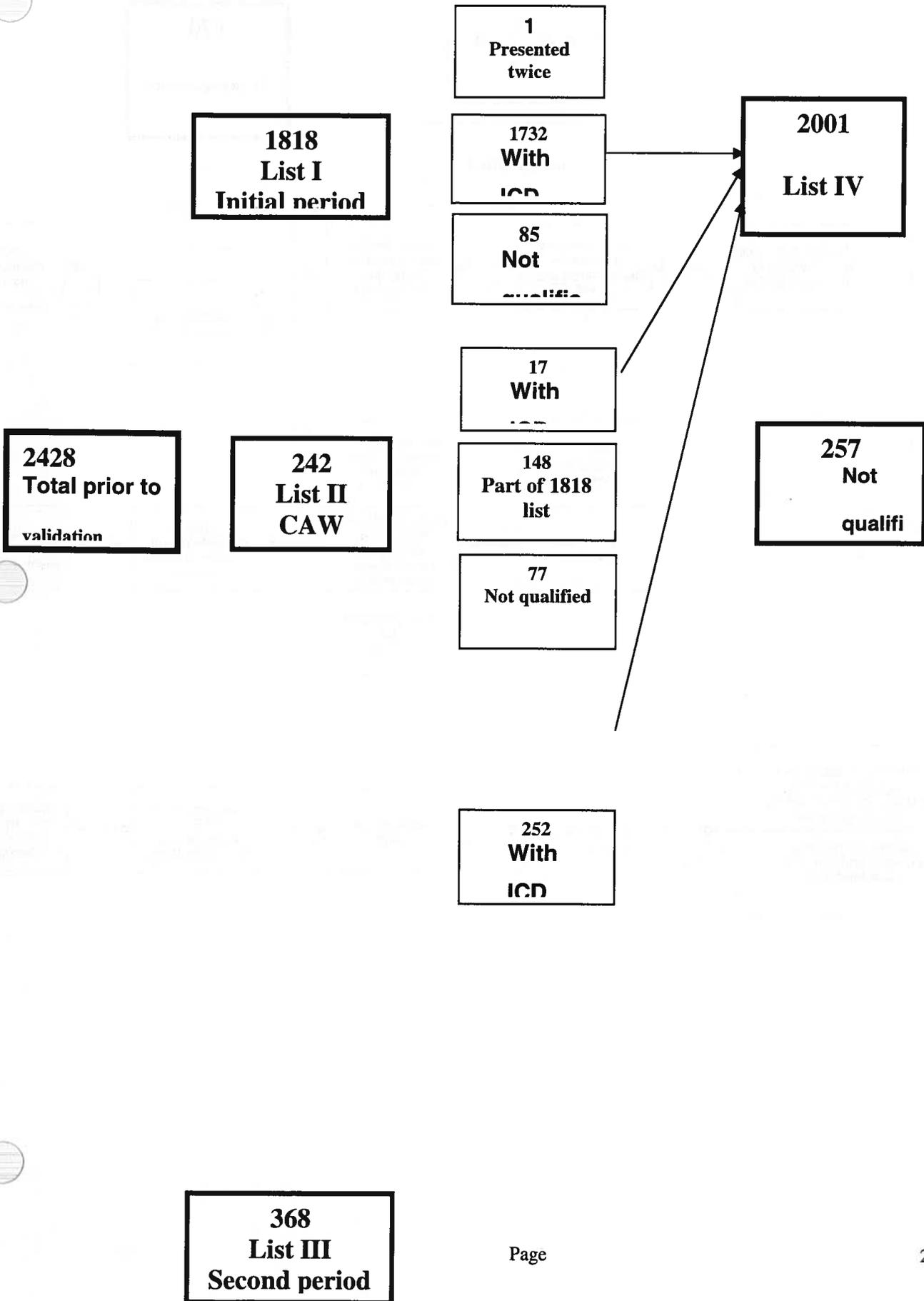
**A. CAW List Submitted for Study**



## B. Inclusion of Second Period to Study

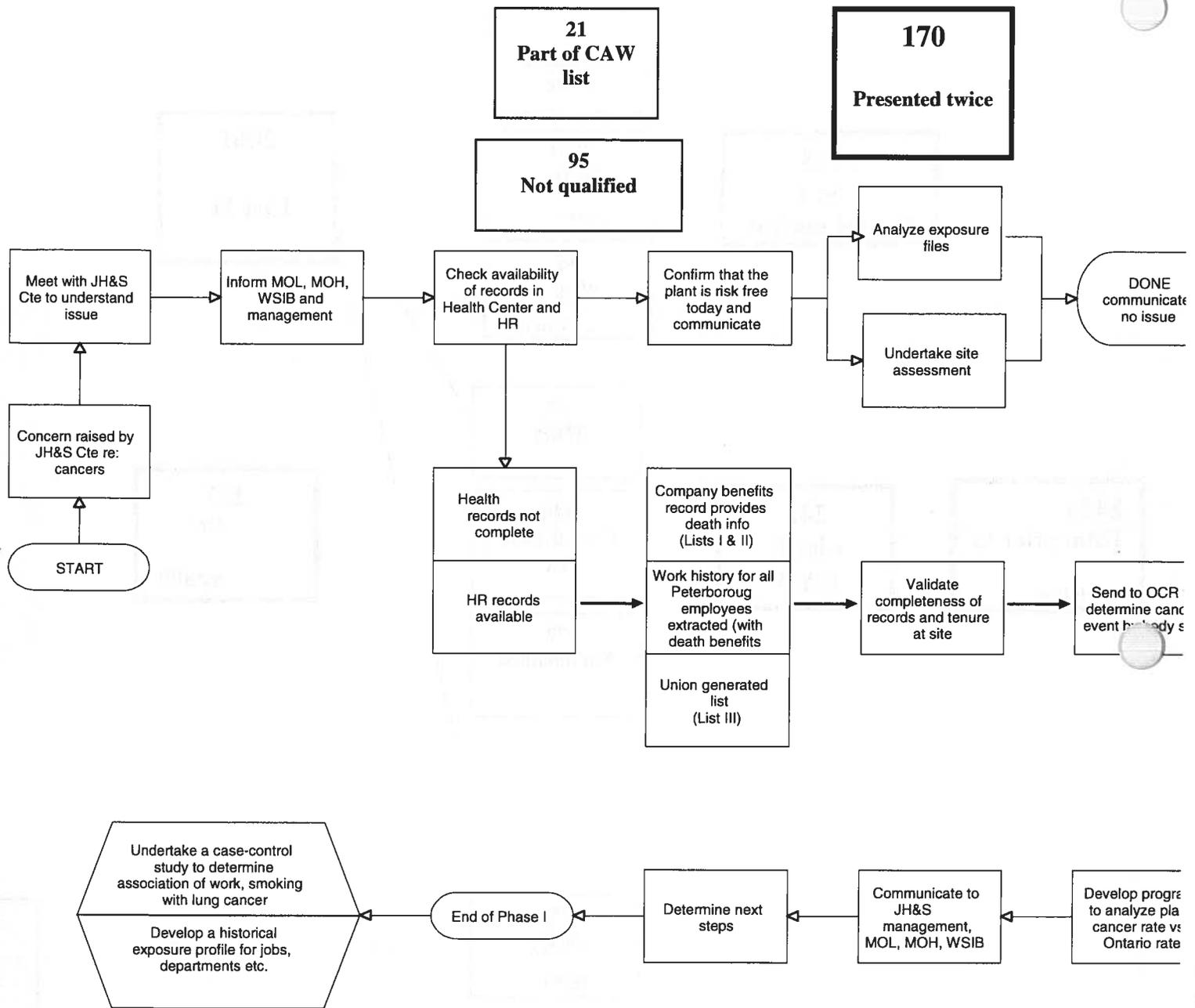


# Summary of Final List for Analysis



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# Phase I Map-Process



Number (%)

<b>VARIABLE</b>	<b>ENTIRE GROUP</b>	
<b>Gender</b>		
Male	1859	(92.9)
Female	142	(7.1)
<b>Manufacturing &gt;1 year</b>		
Mfg-Active	326	(16.3)
Mfg-Retired	1423	(71.1)
Not Mfg – Active	49	(2.4)
Not Mfg – Retired	176	(8.8)
Missing data	27	(1.3)
<b>First GE Payclass</b>		
Hourly	1639	(81.9)
Salary	335	(16.7)
Missing data	27	(1.3)
<b>Last GE Payclass</b>		
Hourly – Active	260	(13.0)
Hourly – Retired	1127	(56.3)
Salary – Active	115	(5.7)
Salary – Retired	472	(23.6)
Missing data	27	(1.3)
<b>Age group at death</b>		
19-29	17	(.8)
30-39	18	(.9)
40-49	71	(3.5)
50-59	207	(10.3)
60-69	510	(25.5)
70-79	713	(35.6)
80-89	396	(19.8)
90-99	68	(3.4)
100+	1	(.0)
<b>Decade of death</b>		
1970-79	632	(31.6)
1980-89	682	(34.1)
1990-98	687	(34.3)

Number (% of column)

\*All cells do not sum to 2001 due to missing observations

<b>VARIABLE</b>	<b>HOURLY</b>		<b>SALARY</b>	
<b>Gender</b>				
Male	1303	(93.9%)	533	(90.8%)
Female	84	(6.1)	54	(9.2)
<b>Manufacturing &gt;1 year</b>				
Mfg-Active	259	(18.7)	67	(11.4)
Mfg-Retired	1126	(81.2)	297	(50.6)
Not Mfg – Active	1	(.1)	48	(8.2)
Not Mfg – Retired	1	(.1)	175	(29.8)
<b>First GE Payclass</b>				
Hourly	1381	(99.6)	258	(44.0)
Salary	6	(.4)	329	(56.0)
<b>Last GE Payclass</b>				
Hourly – Active	260	(18.7)	0	
Hourly – Retired	1127	(81.3)	0	
Salary – Active	0		115	(19.6)
Salary – Retired	0		472	(80.4)
<b>Age group at death</b>				
19-29	12	(.9)	5	(.9)
30-39	12	(.9)	6	(1.0)
40-49	49	(3.5)	21	(3.6)
50-59	147	(10.6)	58	(9.9)
60-69	354	(25.5)	153	(26.1)
70-79	475	(34.2)	227	(38.7)
80-89	288	(20.8)	101	(17.0)
90-99	49	(3.5)	17	(2.9)
100+	1	(.1)	0	
<b>Mean age –SD</b>	70.8 – 12.1		70.2— 12.0	
<b>Decade of death</b>				
1970-79	470	(33.9)	161	(27.4)
1980-89	474	(34.2)	202	(34.4)
1990-98	443	(31.9)	224	(38.2)

Number (% of column)

\*All cells do not sum to 2001 due to missing observations

VARIABLE	CANCER DEATH		OTHER DEATH	
Gender				
Male	524	(91.0%)	1335	(93.7%)
Female	52	(9.0)	90	(6.3)
Manufacturing >1 year				
Mfg-Active	107	(19.0)	219	(15.5)
Mfg-Retired	389	(69.0)	1034	(73.3)
Not Mfg - Active	16	(2.8)	33	(2.3)
Not Mfg - Retired	52	(9.2)	124	(8.8)
First GE Payclass				
Hourly	452	(80.1)	1187	(84.2)
Salary	112	(19.9)	223	(15.8)
Last GE Payclass				
Hourly - Active	84	(14.9)	176	(12.5)
Hourly - Retired	287	(50.9)	840	(59.6)
Salary - Active	39	(6.9)	76	(5.4)
Salary - Retired	154	(27.3)	318	(22.6)
Age group at death				
19-29	4	(.7)	13	(.9)
30-39	3	(.5)	15	(1.0)
40-49	23	(4.0)	48	(3.4)
50-59	77	(13.4)	130	(9.1)
60-69	163	(28.3)	347	(24.4)
70-79	215	(37.3)	499	(35.2)
80-89	79	(13.7)	317	(22.0)
90-99	12	(2.1)	56	(3.9)
100+	0		1	(.1)
Mean age -SD	68.9 - 11.12		71.35 - 12.40	
Decade of death				
1970-79	141	(24.5)	491	(34.5)
1980-89	196	(34.0)	486	(34.1)
1990-98	239	(41.5)	448	(31.4)

Table 4

**CMR - ANALYSIS - ALL MALE**

<b>ODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-val</b>
01-998	All causes	1859				
0-209	All cancers	524				
0-149	Buccal Cavity-Pharynx	10	12.19	0.82	0.45 - 1.51	
0-159	Digestive System	153	135.45	1.13	0.99 - 1.29	
5-156	Liver/Gallbladder,etc	13	9.96	1.31	0.76 - 2.23	
7	Pancreas	18	23.31	0.77	0.49 - 1.21	
<b>2-163</b>	<b>Lung</b>	<b>198</b>	<b>146.69</b>	<b>1.35</b>	<b>1.21 - 1.51</b>	<b>p&lt;0.0</b>
0	Bone	1	0.94	1.07	0.15 - 7.52	
2-173	Skin	7	6.19	1.13	0.54 - 2.35	
0-189	Genito-Urinary Organ	72	70.44	1.02	0	
5	Prostate	49	43.83	1.12	0.86 - 1.45	
8	Bladder	12	14.97	0.8	0.46 - 1.39	
9	Kidney	10	10.15	0.98	0.53 - 1.82	
1-192	Brain-Central Nervous	8	10.98	0.73	0.37 - 1.43	
0-209	Lymphopietic Cancer	44	37.38	1.18	0.89 - 1.56	
0	Lympho-Reticulo	1	3.75	0.27	0.04 - 1.64	
<b>1</b>	<b>Hodgkin's Disease</b>	<b>7</b>	<b>2.09</b>	<b>3.35</b>	<b>1.68 - 6.68</b>	<b>p&lt;0.0</b>
4-207	Leukemia-Aleukemia	12	11.35	1.06	.61 - 1.85	
2-3,8	Lymphatic Tissue	24	20.19	1.19	.8 - 1.76	



Table 5

**CMR - ANALYSIS - HOURLY MALE**

<b>ODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
01-998	All causes	1303				
10-209	All cancers	343				
10-149	Buccal Cavity-Pharynx	9	8.2	1.1	0.58 - 2.09	
10-159	Digestive System	99	90.57	1.09	0.93 - 1.29	
15-156	Liver/Gallbladder,etc	9	6.64	1.36	0.71 - 2.58	
17	Pancreas	9	15.57	0.58	0.31 - 1.08	
<b>12-163</b>	<b>Lung</b>	<b>131</b>	<b>98.02</b>	<b>1.34</b>	<b>1.16 - 1.54</b>	<b>p&lt;</b>
20	Bone	1	0.62	1.62	0.23 - 11.19	
2-173	Skin	3	4.09	0.73	0.24 - 2.23	
20-89	Genito-Urinary Organ	42	47.2	0.89	0	
25	Prostate	26	29.36	0.89	0.62 - 1.26	
28	Bladder	10	10.04	1	0.54 - 1.83	
29	Kidney	5	6.81	0.73	0.31 - 1.74	
31-192	Brain-Central Nervous	5	7.25	0.69	0.29 - 1.62	
40-209	Lymphopietic Cancer	25	24.82	1.01	0.69 - 1.46	
40	Lympho-Reticulo	0	2.5	0	0	
<b>41</b>	<b>Hodgkin's Disease</b>	<b>4</b>	<b>1.35</b>	<b>2.96</b>	<b>1.18 - 7.44</b>	<b>p&lt;</b>
44-207	Leukemia-Aleukemia	10	7.54	1.33	0.72 - 2.44	
42-3,8	Lymphatic Tissue	11	13.44	0.82	.46 - 1.46	

Table 6

**CMR - ANALYSIS - SALARY MALE**

<b>ODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
1-998	All causes	533				
0-209	All cancers	171				
0-149	Buccal Cavity-Pharynx	1	3.87	0.26	0.04 - 1.55	
0-159	Digestive System	51	43.78	1.16	0.93 - 1.46	
5-156	Liver/Gallbladder,etc	4	3.22	1.24	0.47 - 3.26	
7	Pancreas	8	7.55	1.06	0.54 - 2.08	
<b>2-163</b>	<b>Lung</b>	<b>66</b>	<b>47.25</b>	<b>1.4</b>	<b>1.15 - 1.7</b>	<b>p&lt;0.</b>
0	Bone	0	0.31	0	0	
2-173	Skin	3	2.06	1.46	0.48 - 4.42	
0-189	Genito-Urinary Organ	28	22.61	1.24	0	
<b>5</b>	<b>Prostate</b>	<b>21</b>	<b>14.03</b>	<b>1.5</b>	<b>1.01 - 2.22</b>	<b>0.</b>
8	Bladder	2	4.82	0.41	0.11 - 1.55	
9	Kidney	5	3.26	1.54	0.65 - 3.63	
1-192	Brain-Central Nervous	3	3.65	0.82	0.27 - 2.48	
0-209	Lymphopietic Cancer	16	12.24	1.31	0.82 - 2.08	
0	Lympho-Reticulo	1	1.24	0.81	0.12 - 5.66	
<b>1</b>	<b>Hodgkin's Disease</b>	<b>3</b>	<b>0.73</b>	<b>4.13</b>	<b>1.47 - 11.56</b>	<b>p&lt;0.</b>
4-207	Leukemia-Aleukemia	2	3.72	0.54	0.14 - 2.06	
2-3,8	Lymphatic Tissue	10	6.56	1.53	0.84 - 2.78	

Table 7

**CMR - ANALYSIS - MANUFACTURING MALE**

<b>ODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
01-998	All causes	1651				
00-209	All cancers	460				
00-149	Buccal Cavity-Pharynx	10	10.85	0.92	0.50 - 1.70	
00-159	Digestive System	131	119.97	1.09	0.95 - 1.26	
05-156	Liver/Gallbladder, etc	11	8.8	1.25	0.70 - 2.24	
07	Pancreas	15	20.71	0.72	0.44 - 1.18	
<b>02-163</b>	<b>Lung</b>	<b>183</b>	<b>131.09</b>	<b>1.4</b>	<b>1.24 - 1.57</b>	<b>p&lt;0.</b>
00	Bone	1	0.79	1.27	0.18 - 8.93	
02-173	Skin	4	5.25	0.76	0.29 - 2	
00-189	Genito-Urinary Organ	61	61.57	0.99	0	
	Prostate	38	38.19	1	0.74 - 1.34	
	Bladder	12	13.16	0.91	0.52 - 1.59	
09	Kidney	10	9.03	1.11	0.60 - 2.04	
01-192	Brain-Central Nervous	6	9.56	0.63	0.29 - 1.37	
00-209	Lymphopietic Cancer	34	32.56	1.04	0.76 - 1.44	
00	Lympho-Reticulo	1	3.32	0.3	0.05 - 1.89	
<b>01</b>	<b>Hodgkin's Disease</b>	<b>5</b>	<b>1.74</b>	<b>2.88</b>	<b>1.26 - 6.59</b>	<b>p&lt;0.</b>
04-207	Leukemia-Aleukemia	11	9.8	1.12	0.63 - 2.01	
02-3,8	Lymphatic Tissue	17	17.7	0.96	0.60 - 1.53	

Table 8

**CMR - ANALYSIS - Not MANUFACTURING MALE**

<b>ODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
1-998	All causes	185				
0-209	All cancers	54				
0-149	Buccal Cavity-Pharynx	0	1.22	0	0	
0-159	Digestive System	19	14.39	1.32	0.91 - 1.92	
5-156	Liver/Gallbladder,etc	2	1.06	1.88	0.49 - 7.24	
7	Pancreas	2	2.41	0.83	0.22 - 3.2	
2-163	Lung	14	14.17	0.99	0.64 - 1.52	
0	Bone	0	0.15	0	0	
2-173	Skin	2	0.9	2.21	0.59 - 8.27	
0-189	Genito-Urinary Organ	9	8.23	1.09	0	
5	Prostate	9	5.21	1.73	0.96 - 3.12	
8	Bladder	0	1.71	0	0	
9	Kidney	0	1.04	0	0	
1-192	Brain-Central Nervous	2	1.34	1.5	0.39 - 5.68	
0-209	Lymphopietic Cancer	7	4.51	1.55	0.78 - 3.09	
0	Lympho-Reticulo	0	0.41	0	0	
<b>1</b>	<b>Hodgkin's Disease</b>	<b>2</b>	<b>0.34</b>	<b>5.86</b>	<b>1.77 - 19.33</b>	<b>p&lt;0.05</b>
4-207	Leukemia-Aleukemia	1	1.46	0.68	0.10 - 4.59	
2-3,8	Lymphatic Tissue	4	2.29	1.75	0.68 - 4.48	

Table 9

**PCMR - ANALYSIS - SALARY MANUFACTURING MALE**

<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
001-998	All causes	350				
140-209	All cancers	117				
140-149	Buccal Cavity-Pharynx	1	2.65	0.38	0.06 - 2.41	
150-159	Digestive System	32	29.39	1.09	0.82 - 1.45	
155-156	Liver/Gallbladder,etc	2	2.16	0.93	0.24 - 3.64	
157	Pancreas	6	5.15	1.17	0.54 - 2.54	
<b>162-163</b>	<b>Lung</b>	<b>52</b>	<b>33.07</b>	<b>1.57</b>	<b>1.26 - 1.96</b>	<b>p&lt;0.05</b>
170	Bone	0	0.17	0	0	
172-173	Skin	1	1.15	0.87	0.12 - 6.06	
180-189	Genito-Urinary Organ	19	14.38	1.32	0	
185	Prostate	12	8.83	1.36	0.80 - 2.31	
188	Bladder	2	3.11	0.64	0.17 - 2.48	
189	Kidney	5	2.21	2.26	0.97 - 5.24	
191-192	Brain-Central Nervous	1	2.31	0.43	0.07 - 2.81	
200-209	Lymphopietic Cancer	9	7.74	1.16	0.62 - 2.18	
200	Lympho-Reticulo	1	0.82	1.22	0.17 - 8.51	
201	Hodgkin's Disease	1	0.39	2.6	0.40 - 17.03	
204-207	Leukemia-Aleukemia	1	2.26	0.44	0.07 - 2.91	
202-3,8	Lymphatic Tissue	6	4.27	1.41	0.65 - 3.06	



Table 10

**PCMR - ANALYSIS - SALARY Not MANUFACTURING MALE**

CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	183				
140-209	All cancers	54				
140-149	Buccal Cavity-Pharynx	0	1.22	0	0	
150-159	Digestive System	19	14.39	1.32	0.91 - 1.92	
155-156	Liver/Gallbladder,etc	2	1.06	1.88	0.49 - 7.24	
157	Pancreas	2	2.41	0.83	0.22 - 3.2	
162-163	Lung	14	14.17	0.99	0.64 - 1.52	
170	Bone	0	0.15	0	0	
172-173	Skin	2	0.9	2.21	0.59 - 8.27	
180-189	Genito-Urinary Organ	9	8.23	1.09	0	
185	Prostate	9	5.21	1.73	0.96 - 3.12	
188	Bladder	0	1.71	0	0	
189	Kidney	0	1.04	0	0	
191-192	Brain-Central Nervous	2	1.34	1.5	0.39 - 5.68	
200-209	Lymphopoietic Cancer	7	4.51	1.55	0.78 - 3.09	
200	Lympho-Reticulo	0	0.41	0	0	
201	<b>Hodgkin's Disease</b>	<b>2</b>	<b>0.34</b>	<b>5.86</b>	<b>1.77 - 19.33</b>	<b>p&lt;0.05</b>
204-207	Leukemia-Aleukemia	1	1.46	0.68	0.10 - 4.59	
202-3,8	Lymphatic Tissue	4	2.29	1.75	0.68 - 4.48	

Table 11

**PCMR - ANALYSIS - ALL FEMALE**

CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	142				
140-209	All cancers	52				
140-149	Buccal Cavity-Pharynx	0	0.59	0	0	
150-159	Digestive System	10	12.66	0.79	.47-1.33	
155-156	Liver/Gallbladder,etc	0	1.13	0	0	
157	Pancreas	1	2.28	0.44	.07-2.8	
62-163	<b>Lung</b>	<b>16</b>	<b>7.18</b>	<b>2.23</b>	<b>1.44 - 3.44</b>	<b>p&lt;0.05</b>
70	Bone	0	0.12	0	0	
72-173	Skin	2	0.57	3.54	0.98 - 12.76	
	Breast	11	10	1.1	0.66 - 1.84	
80-189	Genito-Urinary Organ	4	6.79	0.59	0	
88	Bladder	0	0.6	0	0	
89	Kidney	0	0.72	0	0	
91-192	Brain-Central Nervous	2	1.22	1.64	0.43 - 6.32	
200-209	Lymphopoietic Cancer	3	3.95	0.76	0.26 - 2.2	
200	Lympho-Reticulo	0	0.39	0	0	
201	Hodgkin's Disease	0	0.3	0	0	
204-207	Leukemia-Aleukemia	1	1.11	0.9	0.13 - 6.15	
202-3,8	Lymphatic Tissue	2	2.15	0.93	0.24 - 3.6	

Table 12

**PCMR - ANALYSIS - HOURLY FEMALE**

<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
001-998	All causes	84				
140-209	All cancers	28				
140-149	Buccal Cavity-Pharynx	0	0.32	0	0	
150-159	Digestive System	5	6.6	0.76	0.36 - 1.59	
155-156	Liver/Gallbladder, etc	0	0.56	0	0	
157	Pancreas	0	1.21	0	0	
162-163	<b>Lung</b>	<b>9</b>	<b>3.93</b>	<b>2.29</b>	<b>1.29 - 4.08</b>	<b>p&lt;0.05</b>
170	Bone	0	0.9	0	0	
172-173	Skin	1	0.34	2.98	0.47 - 18.75	
174	Breast	4	5.49	0.73	0.31 - 1.7	
180-189	Genito-Urinary Organ	1	3.68	0.27	0	
188	Bladder	0	0.31	0	0	
189	Kidney	0	0.39	0	0	
191-192	Brain-Central Nervous	1	0.71	1.41	0.21 - 9.56	
200-209	Lymphopietic Cancer	3	2.25	1.33	0.47 - 3.82	
200	Lympho-Reticulo	0	0.21	0	0	
201	Hodgkin's Disease	0	0.23	0	0	
204-207	Leukemia-Aleukemia	1	0.64	1.56	0.23 - 10.43	
202-3,8	Lymphatic Tissue	2	1.17	1.71	0.45 - 6.48	



Table 13

**PCMR - ANALYSIS - MANUFACTURING FEMALE**

<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
001-998	All causes	98				
140-209	All cancers	36				
140-149	Buccal Cavity-Pharynx	0	0.43	0	0	
150-159	Digestive System	6	8.98	0.67	0.34 - 1.30	
155-156	Liver/Gallbladder,etc	0	0.78	0	0	
157	Pancreas	0	1.62	0	0	
162-163	<b>Lung</b>	<b>11</b>	<b>4.98</b>	<b>2.21</b>	<b>1.31 - 3.73</b>	<b>p&lt;0.05</b>
170	Bone	0	0.11	0	0	
172-173	Skin	1	0.42	2.39	0.37 - 15.61	
174	Breast	7	7.23	0.97	0.51 - 1.85	
180-189	Genito-Urinary Organ	2	4.97	0.4	0	
188	Bladder	0	0.43	0	0	
189	Kidney	0	0.52	0	0	
191-192	Brain-Central Nervous	2	0.91	2.21	0.59 - 8.31	
200-209	Lymphopoietic Cancer	3	2.88	1.04	0.36 - 3.01	
200	Lympho-Reticulo	0	0.29	0	0	
201	Hodgkin's Disease	0	0.25	0	0	
204-207	Leukemia-Aleukemia	1	0.82	1.22	0.18 - 8.31	
202-3,8	Lymphatic Tissue	2	1.52	1.32	0.34 - 5.06	

**Table 14 PCMR - ANALYSIS - SALARY-NON-MANUFACTURING- FEMALE**

<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>
001-998	All causes	40			
140-209	All cancers	14			
140-149	Buccal Cavity-Pharynx	0	0.14	0	0
150-159	Digestive System	4	3.25	1.23	0.55 - 2.76
155-156	Liver/Gallbladder,etc	0	0.3	0	0
157	Pancreas	1	0.58	1.72	0.26 - 11.31
162-163	Lung	4	1.87	2.14	0.90 - 5.06
170	Bone	0	0.01	0	0
172-173	Skin	0	0.11	0	0
174	Breast	4	2.22	1.8	0.76 - 4.23
180-189	Genito-Urinary Organ	2	1.54	1.29	0
188	Bladder	0	0.15	0	0
189	Kidney	0	0.18	0	0
191-192	Brain-Central Nervous	0	0.25	0	0
200-209	Lymphopietic Cancer	0	0.88	0	0
200	Lympho-Reticulo	0	0.09	0	0
201	Hodgkin's Disease	0	0.03	0	0
204-207	Leukemia-Aleukemia	0	0.23	0	0
202-3,8	Lymphatic Tissue	0	0.52	0	0

Table 15

**COMPARISON BETWEEN JOB CLASSES FOR SIGNIFICANT CASES****Observed Number of Cases****MALE**

Type of Cancer	All Male	H	S	Manuf.	NonManuf.	S-Manuf.	S-NonManuf.	H-Manuf.	H-non
Lung	*198	*131	*66	*183	14	*52	14	*131	
Melanoma	*7	*4	*3	*5	*2	1	*2	*4	
Prostate	49	26	*21	38	9	12	9	26	

**FEMALE**

Type of Cancer	All Fem.	H	S	Manuf.	NonManuf.	S-Manuf.	S-NonManuf.	H-Manuf.	H-non
Lung	*16	*9	*6	*11	4	2	4	*9	

= PCMR value significant higher than 1

Male = Hourly + Salary + No job status (131+66+1)

Female = Hourly + Salary + No job status (9+6+0)

Male (Lung): H = H -Manuf. + H-nonManuf. (131+0)

Female (Lung): Hourly = H-Manuf.(9)

## Phase II

### 1. Introduction

- In Phase I, the proportional cancer mortality rate comparison between the deceased Peterborough employees and the Ontario population found that employees had an excess of lung cancer and Hodgkin (male) and lung (female).
- In Phase II, we tried to understand whether the workplace might have accounted for the excess lung cancer in males, and whether smoking played a role.

### 2. Overview

#### 2.1. Objectives:

- Re-create an exposure profile for jobs stations where known human lung carcinogens were handled and used by considering historical data and exposure control systems.
- Using employees work histories (i.e. job and duration on each job) assign to each employee an exposure score.
- Use a case/control design and odds ratio analyses to understand the relationship between lung cancer and the exposure rating based on duration and intensity, and a combination of duration and intensity, and separately with a combination of carcinogens.

#### 2.2. Process-map

The process-map for Phase II is presented in Appendix 1, Page 80 (simplest form) and Appendix 2 , Page 81 (detailed format).

## 3. Methods

### 3.1 Study design

#### 3.1.1 Nested case-control study

- This is a (nested) case-control study developed from the initial cohort used in Phase I.
- A nested case-control study is a case-control study within either a retrospective or prospective cohort study; for phase II we used a retrospective design.
- Controls were selected from unaffected cohort members who were still alive and under surveillance at the time the cases died because of the disease.
- The controls were matched by age, gender and time of entry into the cohort.
- We chose, the optimal general strategy to adjust for differences in the distributions of suspected confounders in the analysis rather than to match.
- We found matching to be useful for a small number of variables, such as sex and date of birth, to minimize confounding from these clearly extraneous variables.

#### We chose the case-control study because:

- It can be carried out in a much shorter period of time than the cohort studies
- It did not require a large sample size
- It can examine multiple etiologic factors for a single disease.

#### Controls:

- Our controls came from the population at risk of the disease or condition being studied.
- Types of controls
  - **Dead controls:** if the cases in the study are defined as deaths from one cause (i.e. lung cancer) and the researcher wish to compare them with people who died from another cause of death
  - **Controls with similar diseases** i.e. Case-control study with cases=lung cancer, and controls=other type of cancer. Advantages: minimize recall or report bias, minimize interviewer bias.

### **3.1.2 Population**

- The original cohort was represented by all GE Peterborough plant employees who had worked in the plant for more than 10 years before 1986 and respectively more than 2 years after 1986, and who died between Jan. 1970 and June 1998 (See Appendix 3; Page 82).
- Since smoking is the major confounder for lung cancer, we attempted to collect smoking information for both cases and controls.
- In order to obtain the smoking information from hospital records (See 3.1.6. Confounders) we decided to use as the study population all subjects from List IV (2001) having as main cause of death cancer (See Appendix 4; Page 83).

### **3.1.3 Cases**

#### **3.1.3.1 Inclusion criteria:**

- Peterborough plant employee who worked in the plant >10years before 1986 and >2years after 1986
- Death in the interval Jan. 1970 – June 1998
- Cause of death: lung cancer (according to the death certificate) ICD9 classification, code: 162-163
- Included in List IV (2001) (See Appendix 3; Page 82)

#### **3.1.3.2 Exclusion criteria**

- Cause of death: other location of cancer, non-cancer
- Not included in the List IV (2001) (See Appendix 3)

### **3.1.4 Controls**

#### **3.1.4.1 Inclusion criteria:**

- Peterborough plant employee who worked in the plant >10years before 1986 and >2years after 1986
- Death in the interval Jan. 1970 – June 1998
- Cause of death: other cancer than lung cancer (according to the death certificate) ICD9 classification, code: 140-209 (except 162-163)
- Included in List IV (2001) (See Appendix 3; Page 82)

#### **3.1.4.2 Exclusion criteria:**

- Cause of death: lung cancer, non-cancer

- Not included in the List IV (2001) (See Appendix 3; Page 82)

### 3.1.5 Matching criteria

- A computer program was written in order to select the controls according to the following criteria (See Appendix 4; Page 83):
  - Gender (same for case and control)
  - DOB  $\pm$  10 years .The date of birth of control should be in the interval ( $-10$  years, DOB of case,  $+ 10$  years).
  - Point of survival. The control should be alive at the moment the matched case died.
  - First time with GE  $\pm$  11 years. The control should start to work with GE in the interval ( $-11$  years, date of 1<sup>st</sup> time with GE for case,  $+ 11$  years).

For male, 195 cases have been matched to one control.

For female, only 5/15 cases could be matched by gender and DOB criteria only. Because of the small number of females and unsatisfactory matching criteria we decided that the analysis could not be performed for a case-control study.

### 3.1.6 Power calculation

- From the table below we see that for an exposure variable with a prevalence of about 45% in this study we would have more than 80% power of detecting a relative risk of 2 or greater.
- However for an exposure variable with prevalence in the range from 5 to 10 percent we would have 80% power of detecting relative risks in the range 2.5 to 3.0.

SAMPLE SIZE DETERMINATION USING THE FORMULAE FROM STATISTICAL METHODS FOR RATES  
AND PROPORTIONS BY JOSEPH L. FLEISS SECOND EDITION JOHN WILEY AND SONS  
ADAPTED FOR CASE-CONTROL STUDIES BY SCHLESSELMAN

ALPHA = 0.05 AND POWER = 0.8 AND CONTROLS PER CASE = 1

PE	REL RISK	N
0.01	1.5	8364
0.01	2.0	2598
0.01	2.5	1380
0.01	3.0	904
0.01	3.5	661
0.01	4.0	516
0.05	1.5	1774
0.05	2.0	559
0.05	2.5	301
0.05	3.0	200
0.05	3.5	148
0.05	4.0	117
0.10	1.5	957
0.10	2.0	307
0.10	2.5	168
0.10	3.0	113
0.10	3.5	85
0.10	4.0	68
0.45	1.5	403
0.45	2.0	145
0.45	2.5	87
0.45	3.0	63
0.45	3.5	51
0.45	4.0	43



### **3.1.7 Confounders**

- The two potential confounders considered in this study are smoking status and age.
- In order to reduce the impact of age as a confounder, we matched the cases and controls according to their Date of Birth (DOB)  $\pm$  10 years and we performed a stratified analysis by different groups of age in the end.
- Smoking information is usually obtained by questionnaire or direct interview with the case/control or next of kin (when cases and controls are dead), or by checking the family's physician records.
- Since all the cases and controls died in an interval that started more than 30 years ago, we did not want to interview next of kin, in order to avoid the "re-call" potential bias. Further, we felt that such personal contact may be unnecessarily disruptive or traumatic for families. Because of the time interval, most of the family's physician records would have been difficult to locate and access not easily available.
- The hospital records represented a potentially feasible, valid choice for obtaining smoking information. Since the hospital records for people who died of cancer were easier to be identified and accessed, we decided to follow only the people involved in the study who died of cancer. Cancer patients tend to be directed to Cancer centers, where all adjacent information from other medical offices is collected. In order to validate our proposed source of data we performed a pilot study at The Princess Margaret Hospital (PMH) in Toronto (See 3.6. Health)

## **3.2. Work History**

### **3.2.1. Data Sources**

- Each employee who worked in GE Peterborough plant or has been paid by them working at other locations, has an Employee record kept in the HR office in Peterborough.
- All the Employee records for subjects included in the study and who died because of cancer have been collected and photocopied. The original cards were kept in 3 boxes in the HR office in Peterborough. The copies are kept locked in Meadowvale office. The copies kept in Toronto were important for data validation and for the final assessment of exposure for each employee.

- Each Employee record contains multiple information
  - Surname, First (Second) Name
  - Date of Birth
  - SIN #
  - Employee #
  - Address
  - Earnings record (Date and Rate): The rate information was used to distinguish Hourly jobs from Salary jobs. A “per hour” rate listed represented an Hourly job. A number of hours and amount of pay represented a Salary job.
  - Position Changes (Date, Employed As, Department, Clock No.)
    - The **Date** could be recorded in different format (i.e. January 23, 1987, 01/23/87, 1/23/1987, 23/01/87, 23/01/1987).
    - **Employed As:** in this column are mentioned the name of the job performed or sometimes the code of the job. The name of the job could have many aliases.
    - **Department** column: the name of the department or a code could be recorded. The name of the department could have many aliases. Sometimes the information was missing.
    - **Clock No.:** in most situations a number was recorded. The clock numbers are related to specific departments, buildings in the plant (See Appendix 5; Page 84). Every time a person performed a job in a different location, the clock no. had changed also. The clock no. was used when we needed to “follow” a person in all the years he/she worked in the plant. (I.e. A person worked 10 years in a non-exposed area, except 6 months when he had to perform the same job in an area with fugitive exposure from asbestos. Using the new clock no. recorded in the Employee record, we could identify the new location and assign the corresponding exposure duration and intensity).

### 3.2.2. Data Collection

#### 3.2.2.1. Primary coding

- For each subject who died of cancer, the information contained in the Employee record was transposed in electronic format. (See Appendix 6; Page 85).
- Each change of job title or department was considered a separate entry. If in one interval of time more than 1 job have been performed in a department, a separate entry was created for each job. For each entry, the job status was mentioned.
- Start Date was recorded in mm/dd/yyyy format.
- End Date: was calculated as the Start date of next entry less one day and was recorded in mm/dd/yyyy format.
- Job status: H for hourly and S for salary jobs.
- Job title: Was recorded every time it was mentioned in the employee record
- Job code: recorded when mentioned in the employee record.
- Department name: recorded when mentioned in the employee record.
- Department code: recorded when mentioned in the employee record.
- Clock #: recorded when mentioned in the employee record.

### 3.2.2.2. Advanced coding

- A salary person could work 100% of the time in an office, or spend some time on the floor, where he/she could be exposed to a carcinogen. For each Salary job a code was allocated, according to the time spent on the floor (See Appendix 7; Page 86).
  - Code 1:** < 25 % time worked on the floor
  - Code 2:** 25-50 % time worked on the floor
  - Code 3:** 50-75 % time worked on the floor
  - Code 4:** 100 % time worked on the floor

For "Hourly" everybody had code 4 (since they worked all the time on the floor).
- The HR department in Peterborough has lists of occupations for Hourly and respective Salary employees. For specific periods of time, a list included a Code # and Occupation name for each job in the plant. These lists have been used to complete all the left empty cells after the primary coding for job name and code.

- A group of former employees and HR people reassessed the departments codes and clock #, and complete the existing “blanks” in the department column.
- Using interview, a list of clock numbers related to each department was created from the information provided by HR (former and actual) employees.
- In many situations an employee performed a job in one location. For short period(s) of time the employee moved to another location without a change in the Employee record (department or job name), except for the clock #. This change was important from the exposure point of view. All these situations have been identified and a group of former/actual employee analyzed one by one each situation.

#### **3.2.2.3. Lists - creation**

- Using computer programs (See 3.8. Data validation and handling) lists of jobs, departments, clock # and combinations of them have been created. (See Appendix 8; Page 87).
- The list with job names and aliases contained 1977 names.
- The list with department names and aliases contained 447 names.
- After computer program was written to handle aliases for both Job and Department lists, a list with all combinations clock #, department, name of the subject was created. This list contained 2735 entries.

### **3.3. Exposure reconstruction**

#### **3.3.1. Updated carcinogens information**

- A list of all MSDS (Material Safety Data Sheet) used in GE Peterborough plant was created, and all MSDS's reviewed for carcinogens.
- A literature search was performed in order to update the information about confirmed human carcinogens using year 2000 as the baseline. Five major institutions were reviewed (See Appendix 9; Page 88).

- The initial agreement was that a chemical would be included in the study if 2 or more of the mentioned institutions would recognize it as a confirmed human carcinogen.
- From the created list of confirmed human carcinogens, only 8 satisfied the inclusion criteria for being analyzed (See Appendix 9; Page 88):
  - Confirmed human carcinogen (CHC) for lung
  - Recognized as CHC by 2 or more institutions
  - Present in the plant between 1940 and 1998
- For each of the 8 carcinogens we collected the TWA (Time Weighted Average exposure limit) and carcinogen status over three periods to reflect the status over time. (See Appendix 10; Page 89). In the final analysis, there was an agreement to use the TWA for year 2000.

### **3.3.2. Design**

- The design of the exposure reconstruction was created to adapt to the particular characteristics of the GE Peterborough plant. Over the time there were departments that functioned in different locations. One department could involve more than 1 building, or only half of a building. Some jobs were performed in different buildings.
- The design recreated the potential exposure to the 8 carcinogens for the interval 1940-1997, and could be used in any situations when the previous exposure of an employee should be evaluated (See Appendix 11; Page 90).
- For each carcinogen, the following steps were followed:
  - Validated the inclusion criteria
  - Updated the TWA
  - Found location(s) in the plant (departments, buildings)
  - Period of time used in the plant for each location (Start, End)
  - Identified processes, jobs related and jobs in the vicinity (fugitive)
  - Identified duration of potential exposure (% of time working using the carcinogen) for each location
  - Identified level of exposure by location using air samples data and judgments (See 3.3.4.2. Intensity).

### **3.3.3. Data sources and collection**

- The initial agreement with the JH&S Cte was that the exposure information should cover the interval 1940-1998, as this will incorporate a sufficient period to account for the latency for lung cancer.
- Various sources were used for collecting data:
  - Interviews with retired and current employees
  - Publications and historical photos
  - Industrial Hygiene notebooks
  - Company newsletters from 1945
  - Engineering Drawings
  - Employee record cards
- The flow-process of data collection is presented in Appendix 12; Page 91. The plant/department history was created by collecting information for each building in the plant (including Bldg. 9 which has been demolished and Bldg. 101 which is located in the vicinity of the plant). Each building was related to a specific department in a specific period of time. At this point we collected information on use of each of the 8 carcinogens by department/buildings/processes.
- All the validated information (See 3.8. Data Validation) made possible the creation of the plant map, with all locations of each carcinogen over time. Each location was identified with the process that involved the use of the carcinogen (See Appendix 13; Page 92).
- For each location we collected information on:
  - **Start date** of using the carcinogen
  - **End date** of using the carcinogen
  - **Frequency** (% of time) using the carcinogen during a particular process  
 The collected information was in different format: # hours/week, # months/year, # hours/year, # hours/day. All the information was converted in a percentage of full time job performed in a year. This percentage was used in calculating the duration of exposure (See 3.3.4.1)
  - **Existing control equipment** (respiratory protective equipment, local exhaust, general ventilation)
- From the interviews we learnt that the company provided respirators for most of the jobs with exposures. We noted that sometimes the workers didn't wear them

according to interview with former employees. For these situations, even though the company provided the required protection, the agreement was to consider “no personal protective equipment” in order to ensure that we reflected the reality of exposure as closely as possible

### 3.3.4. Assessment of exposure

#### 3.3.4.1. Duration

- A worker could perform a job in 3 situations (See Appendix 14; Page 93):
  - Non-exposed: in a place without exposure at any time (i.e. Salary employee who spent 100% of his/her working time in an office, without any contact with the manufacturing floor).
  - +/- exposed: in a place (on the floor) where carcinogen could be used (i.e. Salary employee who spent a part (%) of time on the floor and the remaining part (%) in an office).
  - Exposed: in a place where he/she is exposed directly or indirectly to the carcinogen
- In order to calculate the exact duration of exposure, 3 steps have were followed:
  - Duration 1 = time interval = time worked  
= “End date” – “Start date” = # years (months). This was calculated in the same manner for Hourly and Salary jobs.
  - Duration 2 = time worked with potential exposure = Duration 1 \* % of time worked on the floor  
The % of time spent on the floor was provided by “the advanced coding” (See 3.2.2.2. Advanced coding). For Hourly jobs we always used code 4 (100%) for the time spent on the floor.
  - Duration 3 = time worked exposed = Duration2 \* % time carcinogen used  
The  
The final duration of exposure was calculated in the same way for Hourly and Salary jobs.
- For particular situations we used a “score” system that is described in section 3.3.4.3.

- For examples of Duration calculation See Appendix 7; Page 86.

#### **3.3.4.2. Intensity**

- All the information collected during the exposure reconstruction confirmed that all major processes involving the eight carcinogens didn't change over time. Same machines were used from the beginning (i.e. Carding machine in Wire & cable department) until the department was closed. No major changes in the protective equipment were performed over the time.
- In order to calculate the exposure Intensity, 2 steps have been followed (See Appendix 15; Page 94).
- Intensity 1 is the initial level of intensity assigned to each job (See Appendix 16; Page 95).

In order to assign the Intensity (1) level we had to adapt the strategy according to the existence or not of industrial hygiene monitoring data.

- o For situations where we found industrial hygiene (IH) monitoring data, we decided to use 5 levels of exposure:

- Level 0 = no exposure
- Level 1 = < 25% actual TWA (year 2000)
- Level 2 = 25-75% actual TWA (year 2000)
- Level 3 = 75-100% actual TWA (year 2000)
- Level 4 = >100 % actual TWA (year 2000)

When more than 1 air sample was available for one location, an average of the values was calculated and used.

- o When we did not have IH data we use Judgments to assign the same Levels (0 to 4).

- Interview employees for opinions about jobs with similar exposure or about exposure gradient.

(i.e. We asked the employees:

- Were same type of asbestos gloves used everywhere in the plant?
- Were the gloves in relatively same condition or were locations where the gloves were deteriorated and could represent a source of higher exposure?



- Which job created more “dust”: cutting cord-sets or using asbestos tape in Armature department?)
- Assumptions (i.e. Different locations for the same job, materials, protective equipment, similar bldgs. characteristics, with IH data for only one location = all locations have the same exposure intensity).
  - A welder could perform his job in different locations in a plant. All welders would use asbestos gloves very seldom, for approximately 1% of their working time. The only direct exposure to asbestos is represented by the use of asbestos gloves, which were the same in all departments. So, we considered that all welders had the same level of direct exposure to asbestos.
  - In Building 23 brazing (which involves Cadmium use) was performed from 1966 until 1992. Very close to the brazing location a cadmium line existed from 1966 – 1983. Because of this we considered both brazing and cadmium line as one location (source) of exposure to cadmium from 1966 – 1983 (with also a higher intensity). From 1984 – 1992, brazing alone represented the source of exposure to cadmium, with a lower intensity.
- “Score system” – See 3.3.4.3.

- Intensity 2 is the final level of intensity assigned for each job (direct or fugitive exposure). After deciding the Initial four levels of intensity, each source of exposure was verified for the existence of different controls (protective equipment).

According to the source of information (air samples or judgments) different sets of criteria were used to decide the final exposure level (See Appendix 17; Page 96).

We considered that the co-existence of personal protective equipment-local exhaust-general exhaust (controls) would offer the maximum protection against exposure.

We considered that only general ventilation would not be enough to justify a reduction of the intensity level.

- Air samples: were collected while the local exhaust (if existing) and general ventilation were functioning. The value of the air sample expressed as % of TWA is the level of exposure for “fugitive” and workers without personal protective equipment.
- Judgments: The assigned Initial levels of intensity were based on jobs comparisons without taking into consideration the protective equipment.

#### **3.3.4.3. Score system (Particular situations) (See Appendix 18; Page 97)**

- After the linkage of work history with exposure reconstruction data, three situations were found:
  - A. The common one: during a particular interval of time only one job in one location was performed. There will be only one duration of exposure and only one intensity level of the source.
  - B. In an interval of time, one job was performed in different locations, with multiple sources of exposure and different intensities.
  - C. In an interval of time more than one job were performed but in only one location, with multiple sources of duration and intensity.
- For each interval of time we proposed to calculate a total score, by adding all the scores (product of duration exp. \* intensity exp.) of the jobs.
- The total score was considered equivalent to a full time job (100% time) with Intensity X. (i.e. a total score of 200 was equivalent to a full time job at level 2 intensity).

### 3.4. Linkage Work History – Exposure Reconstruction

- The lists of departments, jobs, clock # created in “Work History” (3.2.) section were linked with the information collected so far by the exposure reconstruction process.
- This linkage allowed us to create maps for sub-areas in each building (department) where carcinogens were used (See Appendix 19; Page 98).
- Each sub-area represented a main process involving the carcinogen and a list of jobs performed in the vicinity. Some jobs could involve directly the use of the carcinogen; other jobs did not use the carcinogen but could have fugitive exposure from a nearby carcinogen source.
- As an example these are the steps for the linkage information for Bldg. 22 (Wire & Cable department) (See also Appendix 19; Page 98).
- First we created a table with the following columns: Department and job (information provided by “Work History”) and Building, Sub-area, Intensity and Duration (information provided by “exposure reconstruction”).

Department	Job	Bldg.	Sub-area	Intensity	Duration	
W & C	Carder & twister operator	22	1	4	100%	
W & C	Wire cutter	22	1	4	100%	
W & C	Braider	22	2	2 - direct 1 - fugitive	30 % - direct 100% - fugitive	*
W & C	Sparkers	22	2	2 - direct 1 - fugitive	30 % - direct 100% - fugitive	
W & C	Electrode cutoff	22	3	1 -fugitive	100%	
W & C	Tuber operator	22	4	3 - fugitive 4 - no exp.	3 - 100% 4 - zero	**
W & C	Testman electrical	22	4	4 - no exp.	0	
W & C	Tractor & lift truck driver	22,24,26		?	?	***

- \* As one can see in Appendix 19, in bldg. 22 there were two sources of asbestos exposure: the carding machine which was located in sub-area 1 and braiding process located in sub-area 2. For sub-area 2, a worker could have two

sources of asbestos exposure: a direct exposure from braiding and an indirect (fugitive) exposure from sub-area 1 (carding machine). For sub-area 3, a worker could have fugitive exposure from sub-area 2. In sub-area 4 there was no asbestos-exposure.

**\*\*** In this situation a job was performed in 2 sub-areas, each with different duration and intensity of exposure.

**\*\*\*** In this situation a job was performed in 3 buildings, each with different duration and intensity of exposure.

- Second we used the score system described in 3.3.4.4. in order to calculate the duration and intensity of exposure for sub-areas with multiple sources of exposure (\* and \*\*).
- For (\*\*\*) situation we used the following assumptions:

Duration: there are 3 buildings, so we assumed that 33% of time worked was performed in each building.

Intensity: in this particular situation, 3 buildings represented W&C department: 22, 24 and 26. In bldg. 24 and 26 there was not asbestos exposure. The only possible asbestos exposure was in bldg. 22. As one can see in the bellow table, the intensity of exposure was not uniform in the building.

Sub-area	Duration*	Intensity *
1	100%	4
2	100%	2
3	100%	1
4	0%	0

\* Both Duration and Intensity are presented in the updated format (after using the score system).

Since the job was not related to a particular sub-area, we used an average of the intensities level.

- The new information was integrated in a final table used in the assessment of each work history. The jobs were grouped by building, sub-area, intensity and duration.

### 3.5. Individual assessment of exposure

- After the linkage of the work history and exposure reconstruction was finished, each work history was reviewed in order to calculate the final intensity and duration of exposure for each carcinogen. In Appendix 20; Page 99 is presented an example of how the final assessment of exposure was calculated for each subject.
- From 12/26/1955 to 12/26/1959, the subject worked as a painter (Hourly) in Industrial Motor Department. From exposure reconstruction we know that in this particular location of the paint booth, the job implied the use of Chromium VI (ingredient in the paint) for 25% of the time worked. The intensity of exposure was 1, since all protective equipment (personal, local and general) was present. The final duration (3) of exposure in this time interval was 1 (See 3.3.4.1. Duration).
- In the interval 12/27/1959 to 12/29/1960 the subject worked also as a painter, but in another location: Apparatus. For this location the Chromium VI was present for 40% of the time worked, with the same level 1 intensity.
- In the following 3 time intervals the worker performed different jobs as Hourly employee, in different sub-areas of W&C department. The intensity and % time exposed were obtained using the score system (See 3.3.4.3.)
- In the interval 2/20/1968 to 12/17/1970, the subject worked as a Dispatch-prod. Controller (Salary employee) in Armature department. In this job he spent only 25% of the time worked on the manufacturing floor (Advanced code = 1), so the Duration 2 was 25% of Duration 1 (i.e. 0.6 y). Working on the floor, the subject could be exposed to asbestos only for 50% of the time, so Duration 3 = 50% of Duration 2 (i.e. 0.3 y). The intensity of the exposure was 1.
- The final assessment of the exposure took into consideration all the carcinogens involved. For each carcinogen and each intensity level we added the Durations 3. The subject in the Appendix.. was exposed to Cr VI, level 1 for (1 + 0.3) 1.3 years. The same subject was exposed to asbestos level 1 for 1.18 years (0.12+0.13+0.3+0.63) and level 2 for 1.84 years.
- The "final" exposure was recorded in the data-format used for the analysis (See Appendix 21; Page 100). For asbestos only the Intensity and Duration components of exposure were also recorded.

## 3.6. Health (Smoking)

### 3.6.1. Pilot Study

In order to validate if hospital records could be used as source of smoking information we performed a pilot study at The Princess Margaret Hospital (PMH) in Toronto.

- **Objective:** To determine the proportion of records in lung and colon cancer patients with information on: smoking status (current/ex/never), lifetime dose, and tobacco type.
- **Rationale:** Data on smoking are important for nested case control studies of occupational respiratory diseases, especially cancer. Studies to date have surveyed subjects or proxy respondents, but there are disadvantages which include recall biases and imprecision, and the potential to elicit distress through proxy respondents. Information gathered during hospitalization may be less subject to recall errors from patients and therefore provide better quality. However, our review of the literature did not share any light on the quality or utility of hospital records for patients.
- **Methods:** From the Princess Margaret Hospital Cancer Registry database 15 patients with lung cancer and 15 with colon cancer were randomly selected from each 5 years interval from 1965-94. We examined the records for data on smoking habits; dose was determined from the data including age started/stopped, duration, and daily amount. Gender and age were also noted.
- **Results:** 180 charts were analyzed: 108 male, 72 female; mean age 62.3 years. For lung cancer, the proportion of charts with data on smoking status, dose and type were 95.5%, 91.4%, 97.5%, respectively. For colon cancer these proportions were 62.2%, 51.8%, and 53.6%. For both cancer types, completeness improved in more recent records.
- **Conclusions:** Smoking data appear to be adequately completed for lung cancer records; for one other cancer site, e.g. colon cancer, the data are less complete probably because treating physicians put a greater emphasis on getting smoking information from patients with respiratory diseases. We considered the results to be good enough to allow us to use

the hospital records as the source of smoking information for our case-control study.

### **3.6.2. Data sources and collection**

- Initially we completed the Application package for access to the Ontario Cancer Registry (OCR). Using the linkage of our existing information with their database we expected to identify for each subject in the study the hospital(s) where they have been admitted. The following steps were to contact the hospitals, identified the charts and collect the smoking information. At OCR, a couple of changes regarding the ethics approval and procedures to access their database was made. The new procedures supposed to include an interval of 6-8 months of waiting for approval at different levels. (See Appendix 22; Page 101). Because of this situation we decided to change our strategy.
- Since all the subjects in the case-control study were cancer patients, we assumed that most of them have been admitted at least once in a Peterborough hospital. All cancer patients are sent to a regional hospital where a Cancer Registry exists. The closest locations of Cancer Registries from Peterborough are Toronto (at Princess Margaret Hospital) and Kingston (at Queen's University). (See Appendix 23; Page 102).

#### **3.6.2.1. Princess Margaret Hospital (PMH)**

- PMH has a Cancer Registry data that contains updated charts for cancer patients who have been admitted to The Toronto Hospital, The Toronto Hospital Western Division and PMH.
- In order to obtain access to the medical records first we had to obtain an approval from the Ethics Review Committee of the University Health Network. With this approval we were allowed to complete the Application package for access to the PMH's medical records. The Ethics Committee at PMH discussed and approved our application and allowed our team to collect the information.
- Only Dr. Daniela Ghiculete, who signed the confidentiality of information agreement, had access to the hospital records.

- The hospital records were examined at PMH, without making copies of the records.

### **3.6.2.2. Peterborough Regional Health Care Centre**

- Peterborough Regional Health Care Centre database includes the medical records of the patients admitted in their hospital and also the records from St. James hospital.
- An Application package was completed and submitted for approval to the Ethics Committee. Initially the Ethics Committee approved the direct access of GE team to the medical records. Later, the same Committee decided that the hospital staff should collect the smoking information from the medical records. This information should be released to us without personal identifier.
- An updated list with names of subjects without smoking information was submitted to the Peterborough Regional Health Care Centre.
- We provided the hospital with a Floppy disk that included the exposure code and status (case or control) for each subject in the list and the personal identifier information (See Appendix 24; Page 103). Under the supervision of the Director of Patient Information Services, our team performed the transfer into electronic format (Floppy) of the smoking information collected by the hospital's staff. In the end the columns that contained the personal identifier (Surname, First-Second Name, SIN, DOB) have been erased from the Floppy disk. In this way we ensured that no exposure information of the subjects was retained by the hospital and that GE did not retain personal identifier information, as per our agreement.

### **3.6.2.3. Kingston Regional Cancer Center**

- An Application package was completed and submitted for approval to the Queen's University Health Sciences & Affiliated Teaching Hospitals Research Ethics Board.
- The Ethics Board approved the direct access of GE to the medical records.
- The same updated list with the names of subjects without smoking information that was sent to Peterborough was submitted to Kingston (almost



same date for submission). Only 10 subjects had medical records in Kingston Regional Cancer Center. For all of the names we found the smoking information in Peterborough charts, so no further work was performed in Kingston.

### **3.7. Analysis**

The analysis didn't take latency into account. Latency is defined as the time interval since the person started to be exposed until he/she first developed the disease. With our collected information we can find the date when the exposure started, but we do not know when the cancer was diagnosed for the first time. The date of death cannot be used in order to calculate latency. As a consequence, more cases were included in the analysis.

#### **3.7.1. Tools**

- The measure of strength of an association in a case-control study is the odds ratio estimate of the relative risk of developing the disease for those who have been exposed compared with that for those not exposed. Odds ratios can be calculated for different amounts of exposure or for subgroups stratified by other risk factors.
- Odds ratios greater than 1 indicate a greater likelihood of disease among the exposed than among the unexposed.
- The interval estimates for the odds ratio were obtained using the Woolf method or Fisher's exact test. If the 95% Confidence Interval excludes 1, one can conclude that the true odds ratio is significantly greater or lower than 1. If the 95% Confidence Interval includes 1, then the OR value is not statistically significant.
- The initial intention was to use the same tools for the analysis of exposure to each carcinogen included in the study.
- The tools used for the analysis could be classified as:

##### **3.7.1.1. Basic statistical analysis**

- In these analyses the exposure variable is considered categorical (See Appendix 25; Page 104).
- There are two designs for calculating the odds ratios:

## **A = unmatched analysis**

- **Crude analysis:** This analysis uses a dichotomous exposure classification (2 X 2 table). Cases and controls were classified as to whether they were or not exposed to the carcinogen (See Appendix 26; Page 105) OR and 95% confidence intervals were obtained.
- **Stratified analysis:** In this analysis the exposure is still dichotomous but the control of confounding involves the assembly of a separate table (2 X 2) for each level of the confounder (age and smoking status) or for each combination of levels if more than one confounder is being controlled. (See Appendix 27; Page 106). The Adjusted Mantel-Haenszel OR test was used. OR and 95% confidence intervals were obtained.
- **Multiple exposure levels analysis:** The data is considered categorical.
  - Three “aspects” of the exposure have been analyzed with this method:
    - a) **Intensity of the exposure:** the concentration of the carcinogen in the environment that potentially could enter the body and be delivered to the target organ (lung).
    - b) **Duration of exposure:** the amount of time the subject was exposed to the carcinogen
    - c) **Cumulative exposure:** the integral of exposure intensity over time
  - We split the exposure in 4 levels: No-exposure, Low, Medium and High (See 3.7.2.).
  - Inferences about the association between a disease and a factor are considerably strengthened if information is available to support a gradient between the degree of exposure to a factor and the disease in question.
  - The objective was to assess whether there is a trend in the prevalence rates of lung cancer with the exposure after controlling for age and smoking status.
  - The data was arranged in stratum (2 x K table), according to age, smoking status and exposure levels (See Appendix 28; Page

107). Odds ratios were calculated for each stratum (comparing each level of exposure with the non-exposed group. The Mantel-extension test (Chi-Square Test for Trend-Multiple Strata) was performed in order to calculate the test statistic. This test does not yield odds ratio estimates, but only provide a p-value.

### **B = matched analysis**

- Crude analysis: Matched analysis is simply a special form of stratified analysis because each matched set represents a stratum. The data was arranged in a 2 X 2 table (See. Appendix 28; Page 107). Since only one control has been matched to each case, the Mantel-Haenszel estimate is simply the ratio of the number of pairs where the case is exposed and the control is not, and the number of pairs where the control is exposed and the case is not.

#### **3.7.1.2. Advanced statistical analysis**

- In a logistic regression analysis, the exposure variable is considered continuous (See Appendix 25; Page 104). Logistic regression is a mathematical model in which the log odd is modeled as a linear combination of a set of risk factors.
- Logistic regression is a useful tool for estimating odds ratio associating a disease with one or more risk factors and potential confounding variables (i.e. in our study: lung cancer with exposure to one or more simultaneous carcinogens and age and smoking status).
- Logistic regression is the modeling analogue of the Mantel-Haenszel procedure (See 3.7.1.1.)
- Persons are classified as to whether they experienced the event of interest. Additionally, each worker is classified according to his/her final cumulative exposure, and final values of confounders.
  - Conditional logistic regression: the controls are matched for each case, based on multiple matching criteria (“genuine” pairs), and this matching is retained in the analysis.
  - Unconditional logistic regression: is used when matching has only been performed on general factors.

### 3.7.2. Cut-points

- For each component of the analysis (Intensity, Duration, Cumulative) a distribution table of the values was created for cases and controls. According to these distributions, the cut-points have been chosen in order to categorize people in: Non-exposed, Low, Medium and High groups.

➤ Intensity: Intensity is related only to the concentration (amount) of the carcinogen, without any reference to the duration of exposure.

Many employees have been exposed to different intensities of the same carcinogen over time (i.e. one worker have been exposed to asbestos level 4, level 2 and level 1).

Because of situations like the one above a decision should be taken in order to evaluate each subject in the same way. There were two options: to choose the lowest or the highest level for everybody. Because it makes more medical sense and also will err on the side of employees, the highest level of exposure was selected for each subject (only for the Intensity analysis).

In Appendix 29; Page 108, the distribution and cut points for Intensity data are presented. The cut-points used are: Low=Level 1 of Intensity, Medium=Level 2 and 3, and High=Level 4.

➤ Duration: The distribution and cut-points for Duration are presented in Appendix 30. The cut-points are: Low= <2 year exposure, Medium= 2-10 years exposure, High= >10 years exposure.

➤ Cumulative: The cumulative exposure to a specific carcinogen was calculated as the sum of all products intensity of carcinogen \* duration of exposure. This score was generated for each subject.

The distribution of the data and the cut-points are presented in Appendix 31.

The cut-points are: Low= <5, Medium= 5-10, High= >10.

### 3.7.3. Confounders

#### 3.7.3.1. Age at death

Following the data distribution we decided to use the following age groups:  
< 60 y, 60-69, 70-79, > 80 years.

#### 3.7.3.2. Smoking status

Employees were classified in 3 categories:

Smokers (In this group are included all smokers and ex-smokers.)

Non-smokers (People who never smoked).

No information (Since for almost 30% of subjects we couldn't find the smoking information, we considered them as a separate group).

### **3.8. Data Validation and Handling**

#### **3.8.1. Level: Matching controls**

A sample of 70-paired case/control have been randomly selected and verified manually to confirm that the matching criteria were correct. No error was found.

#### **3.8.2. Level: Work History**

##### ➤ Computer programs

- To validate the period start/end consistency: start after end (i.e. start: May 6, 1938 and end: June 26, 1936) or zero-length periods (i.e. same start and end date). The program identified all errors related to start and end date. For each error, we reviewed the employee record and made the correction in the electronic format of data. After the corrections were done, we ran again the program until the final result was "No error found".
- To account for overlapping jobs in the same department. I.e. 3 jobs simultaneously in the same department in the same time interval; we calculated duration of work in that department, each job contributed with 1/3 of the interval time.
- To verify the "same job-department" assumption was not violated. One job could be performed only on one department at a certain interval of time. Each entry (interval of time) should have only one job and 1 department listed.
- To identify missing data for "Job" and "Department" column.
- To determine all "Jobs" names and aliases.
- To determine all "Departments" names and aliases.
- To determine all combinations "Clock # and Department name".
- To determine all combinations "Clock #, Department, name of subjects".

- To handle aliases for both Department and Job lists.
- To find all jobs pertaining to each department.
- To calculate Duration 1 and Duration 2 (See 3.3.4.1.) in each job/department.

➤ Manually

- Every time when the clock # didn't match the regular corresponding department, two or three HR people reviewed independently the employee record and decide in what department the person worked during that time interval. The information provided has been cross-validated.

**3.8.3. Level: Exposure reconstruction**

- The interviews were conducted individually and not in groups. In this way, the cross-validation of the information collected was possible.
- When maps were created, the maps-information drawn by different employees was cross-validated.
- When MSDS of some materials used in the plant were not found, we contacted the company that made the product in order to obtain the necessary information.
- When possible, the information from interviews regarding the "Controls" or protective equipment, were cross-validated with photographs.
- Air samples results in hand written format have been compared with electronic format data. No error was found.
- Always we interviewed more than 1 person who worked in a specific area for identifying a department and processes locations.
- For detailed information (job related to a process) necessary to determine the "fugitive" exposure we also used cross-validation of information. Maps of different departments or sub-areas have been distributed to different former employees in order to record the location of the processes and jobs related. If two or more persons provided the same information for a specific area, we considered this correct.

### 3.8.4. Linkage work history – exposure reconstruction

- A sample of 80 cases (48%) and 80 controls (48%) has been verified manually with regard to the calculation of exposure duration and intensity. No error was found.

### 3.8.5. Smoking information

- When direct access to the hospital records was allowed, each page of the patient's file was checked.
- At Peterborough Health Center, under the hospital supervision, our team performed the transfer of the information collected from the hospital records into electronic format.

### 3.8.6. Analysis

- The basic statistics analysis was performed using Excel and SAS software programs. The results were the same.

## 4. Results

### 4.1. Descriptive

- The descriptive statistics for cases and controls is presented in Table 1.

Table 1 Descriptive statistics

	Cases	Controls
Gender	M	M
N	195	195
Age (mean +/- STD)	69.1 +/- 9.0	70.3 +/- 9.1

- The distribution of cases and controls by age group is presented in Table 2.

Table 2 Distribution of cases and controls by age-group

Age group	Cases	Controls	Total
< 60	30	27	57
60 - 69	65	54	119
70 - 79	76	85	161
< 80	24	29	53
TOTAL	195	195	390

- The distribution of cases and controls by smoking status is presented in Table 3.

Table 3 Distribution of cases and controls by smoking status

	Cases	Controls
Gender	M	M
Smokers ( # and % )	137 (70.26%)	88 (45.13%)
Non-smokers ( # and % )	13 (6.67 %)	52 (26.67%)
No Smoking information ( # and % )	45 (23.08%)	55 (28.21%)

- With respect to the Intensity component of exposure, the distributions of cases and controls by age group and smoking status are presented in Appendix 32.
- With respect to the Duration component of exposure, the distributions of cases and controls by age group and smoking status are presented in Appendix 33.
- With respect to the Cumulative exposure, the distributions of cases and controls by age group and smoking status are presented in Appendix 34.

#### 4.2. Crude Analysis

- Crude analysis could be performed for each carcinogen included in the study, except silica because no cases or controls have been exposed to it.
- The Odds ratios and 95% confidence intervals are presented in Table 4.



Table 4 Odds ratios and 95% Confidence intervals

<b>Carcinogen</b>	<b>OR</b>	<b>95% CI</b>
<b>Asbestos</b>	<b>1.00</b>	<b>0.59 - 1.70</b>
<b>Arsenic</b>	<b>0.66</b>	<b>0.11 - 4.03</b>
<b>Beryllium</b>	<b>1.00</b>	<b>0.24 - 4.10</b>
<b>Cadmium</b>	<b>0.42</b>	<b>0.13 - 1.42</b>
<b>Chromium VI</b>	<b>1.00</b>	<b>0.20 - 5.07</b>
<b>Ni-Cr</b>	<b>0.50</b>	<b>0.04 - 5.54</b>
<b>Uranium</b>	<b>0.76</b>	<b>0.27 - 2.13</b>

None of the odds ratio values is statistically significant different than 1.

- For asbestos the distributions of cases and controls by age and smoking status are presented in Appendix 35; Page 114.
- For smoking status, the distribution of cases and controls is presented in Table 5

Table 5 Distribution of cases and controls by smoking status

<b>Crude Analysis (Ever/Never Smoking)</b>			
	<b>Smokers</b>	<b>Non-smokers</b>	<b>Total</b>
<b>Cases</b>	137	45	182
<b>Controls</b>	88	55	143
<b>Total</b>	225	100	N=325

The Odds ratio is 5.35 and the 95% confidence interval : 2.57 – 11.13.

#### 4.3. Stratified Analysis

- This type of analysis was performed only for exposure to asbestos. Because of the small numbers of cases and controls exposed to other carcinogens than asbestos, such analysis could not be performed.

- In Table 6 the mean age was calculated for cases and controls for three situations
  - All of the data
  - Excluding the 100 persons without smoking status information
  - Including only matched pairs whose members both had smoking status information.

**TABLE 6 MEAN AGE FOR CASES AND CONTROLS**

	(1)		(2)		(3)	
	ALL DATA		EXCLUDE MISSING		COMPLETE PAIRS	
	CONTROLS	CASES	CONTROLS	CASES	CONTROLS	CASES
N	195	195	140	150	110	110
MEAN	70.3	69.1	69.1	68.6	69.5	68.3
STD	9.1	9.0	8.9	8.8	8.5	9.0
P VALUE **	0.005		-	-	0.03	
R ##	0.77				0.79	

\* The p value associated with Student's paired t test comparison of the mean age between the two groups.

## The Pearson correlation coefficient between the ages of the cases and controls.

Although the mean age is significantly lower among the cases compared to the controls the matching was, in practical terms successful, the difference being 1.2 years for all of the data and also 1.2 years for the subset of 110 pairs in which both members had smoking status information. The highly significant Pearson correlation coefficients between the ages of the cases and controls resulted in very low standard errors for the paired comparisons of the sample means.

- In Table 7 is given the 2 by 5 table that gives the association between case-control status and quintiles of age. There is no significant association between case-control status and the quintiles of age ( $p = 0.57$ ).

**TABLE 7 COMPARISON OF AGE DISTRIBUTION AMONG CASES & CONTROLS**

STATUS	QUINTILES OF AGE					Total
	1	2	3	4	5	
Frequency,						
Row Pct ,						

0	18	25	20	21	26	110
	16.36	22.73	18.18	19.09	23.64	
1	24	23	26	17	20	110
	21.82	20.91	23.64	15.45	18.18	
Total	42	48	46	38	46	220

Statistics for Table of STATUS by RAGE

Statistic	DF	Value	Prob
Chi-Square	4	2.9267	0.5702

**4.3.1. Stratified by age**

- The Exposure distribution (Exposed /Non-exposed) of cases and controls by age is presented in Table 8.

Table 8 Distribution of exposure for cases and controls by age-group

Age group	Cases		Controls		Odds Ratio
	Exp.	Non-Exp.	Exp.	Non-Exp.	
< 60	13	17	16	11	<b>0.53</b>
60 - 69	28	36	24	30	<b>0.97</b>
70 - 79	34	43	35	50	<b>1.13</b>
> 80	11	13	11	18	<b>1.38</b>

- The adjusted Mantel-Haenszel odds ratio is: 0.98 with the 95% confidence interval of 0.69,1.39.
- The Mantel-Haenszel chi-square is 1.50 with a p value  $0.5 < p < 0.75$ .

**4.3.2. Stratified by Smoking status**

- The Exposure distribution (Exposed /Non-exposed) of cases and controls by the smoking status is presented in Table 9.

Table 9 Distribution of exposure for cases and controls by smoking status

	Cases	Cases	Controls	Controls	
Smoking status	Exp.	Non-Exp.	Exp.	Non-Exp.	Odds Ratio
Smokers	66	71	41	47	1.07
Non-smokers	4	9	24	28	0.52
No Information	16	29	21	34	0.89

- The adjusted Mantel-Haenszel odds ratio is: 0.94 with the 95% confidence interval of 0.62,1.43.
- The Mantel-Haenszel chi-square is 1.03 with a p value  $0.5 < p < 0.75$ .

#### 4.4. Matched Analysis

- This type of analysis could be performed only for asbestos exposure.
- There were 43 matched sets with case exposed / control non-exposed and 43 matched sets with case non-exposed / control exposed.
- The Odds ratio is 1, with a 95% confidence interval: 0.66 to 1.52.

#### 4.5. Multiple exposure levels

##### 4.5.1. Intensity component of exposure

- The exposure distribution of cases and controls by age and smoking status is presented in Appendix 32; Page 111.
- The crude Odds ratio and the stratified by age and smoking status odds ratio are presented in Appendix 36; Page 115.
- The Mantel-extension test showed that there is not a statistically significant trend with intensity of exposure.

##### 4.5.2. Duration component of exposure

- The exposure distribution of cases and controls by age and smoking status is presented in Appendix 33; Page 112.
- The crude Odds ratio and the stratified by age and smoking status odds ratio are presented in Appendix 37; Page 116.
- The Mantel-extension test showed that there is not a statistically significant trend with duration of exposure.

#### **4.5.3. Cumulative exposure**

- The exposure distribution of cases and controls by age and smoking status is presented in Appendix 34; Page 113.
- The crude Odds ratio and the stratified by age and smoking status odds ratio are presented in Appendix 38; Page 117.
- The Mantel-extension test showed that there is not a statistically significant trend with cumulative exposure.

#### **4.6. Logistic regression**

- Using age as a continuous variable in an unconditional logistic regression analysis and using matched pairs by age in a conditional logistic regression analysis, the odds ratio of lung cancer for asbestos was never greater than 1. The results were almost identical when simultaneous exposure to different carcinogens was considered.

#### **4.7. Power of the study**

- Only for the variables asbestos, cadmium and uranium was the prevalence sufficiently high to give any reasonable statistical power for finding an association.
- Table 10 presents the power of the study to detect a certain Relative Risk due to the prevalence of different carcinogens.

Table 10.

Carcinogen	Power of the study	RR to be detected
Asbestos *	> 80 %	> 2
Cadmium *	80%	2.5 - 3
Uranium *	80%	2.5 - 3
Ni-Cr	< 80 %	4
Arsenic	< 80 %	4
Chromium VI	< 80 %	4
Beryllium	< 80 %	4
* sufficient prevalence		

## 5. Conclusions

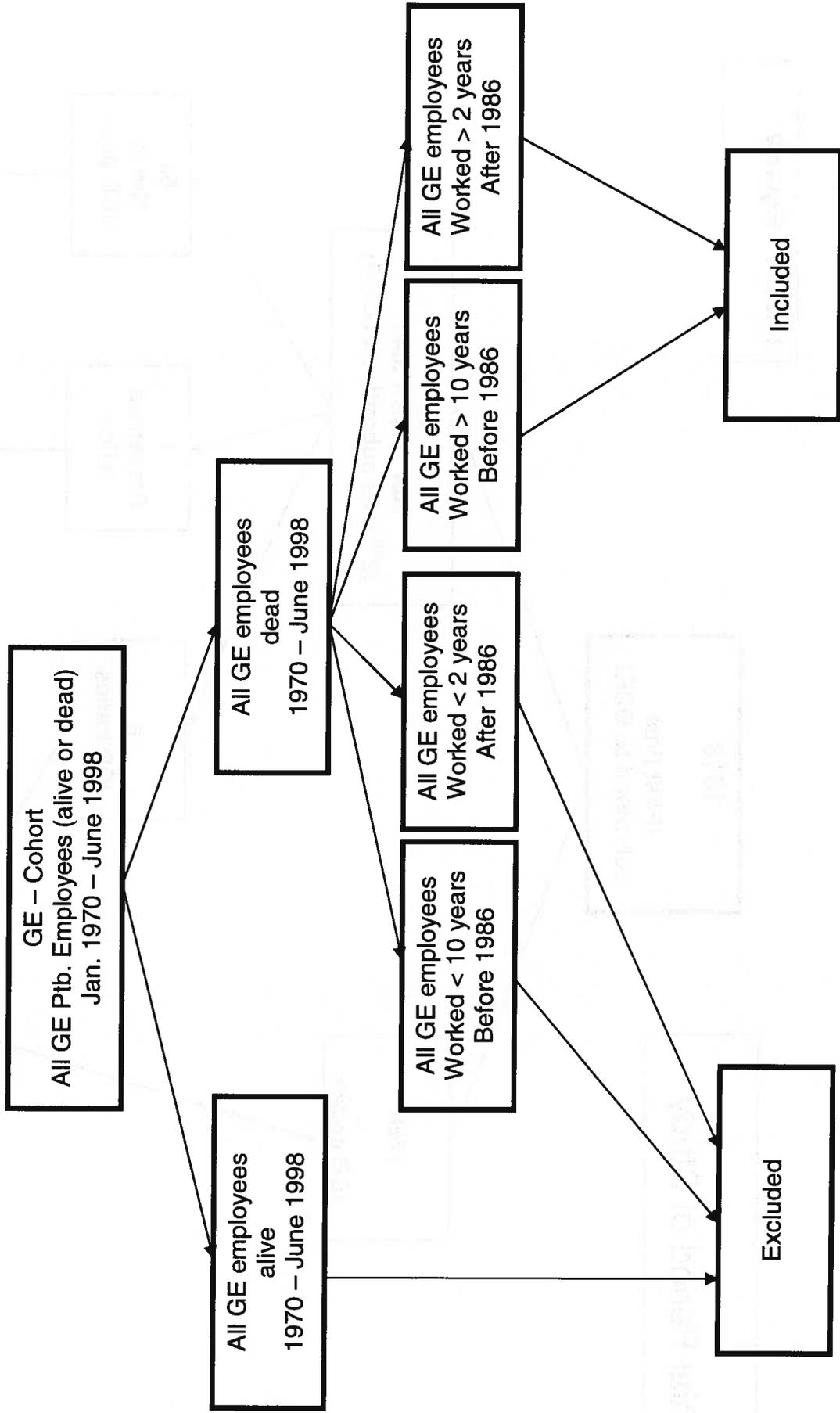
- Using an odds ratio analysis adjusted and not-adjusted for age and smoking status (Cochran-Mantel-Haenszel, unconditional and conditional logistic regression), there was no association between lung cancer deaths and any of the carcinogens.
- There was no statistically significant trend of increasing risk of death by lung cancer with cumulative exposure, exposure intensity or duration.
- There was no increase in risk of lung cancer death with multiple carcinogens exposure.
- The only significant association for lung cancer arose when smoking was considered.
- From the Industrial Hygiene data, the exposures were predominantly within the TWA limits at the time.
- From the interviews and information collection, there was generally used personal protective equipment, and local and general ventilation in areas where carcinogens were used. In our analyses, we weighted the exposure rating to consider the level of use of the controls devices.



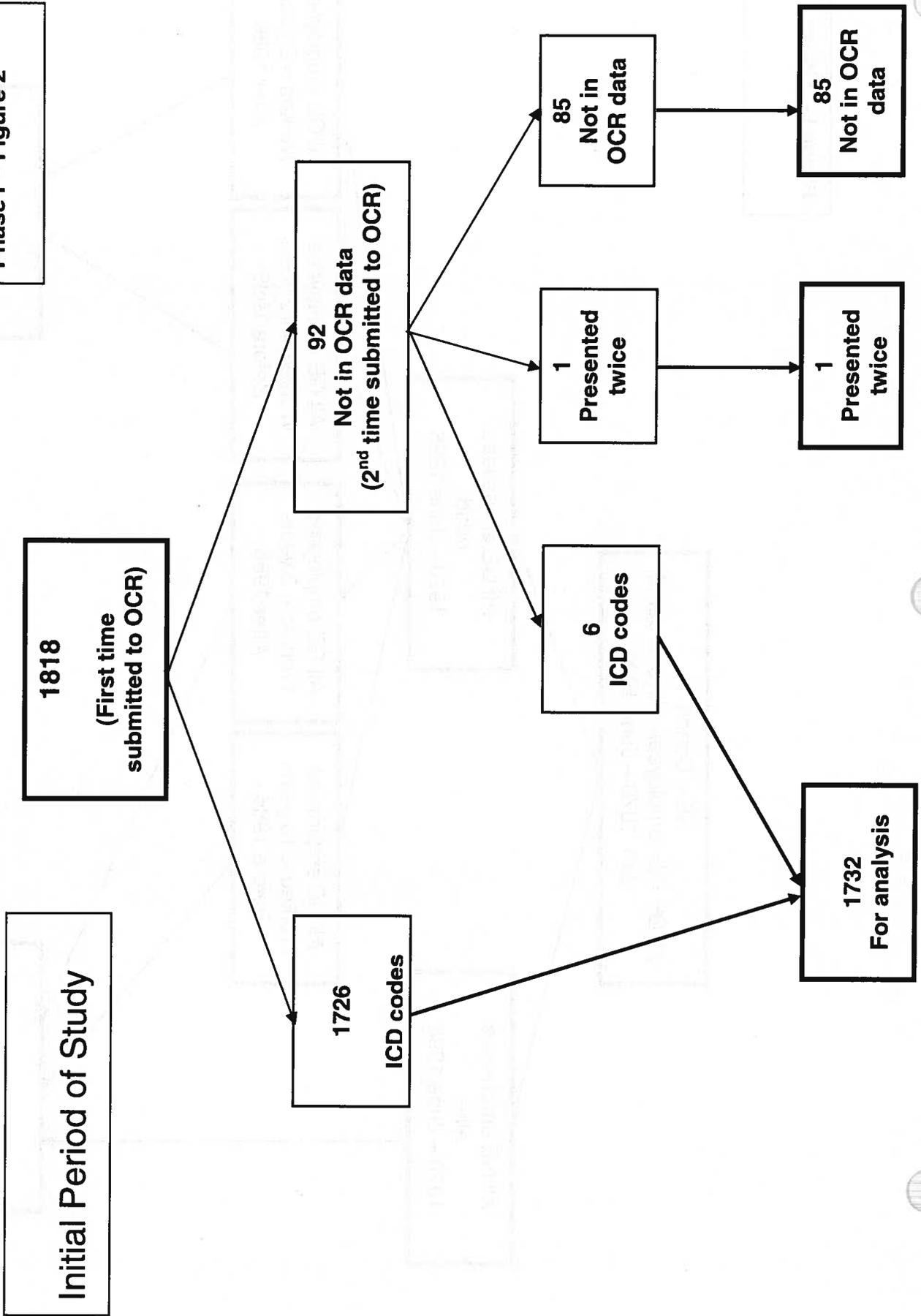




Phase I – Figure 1

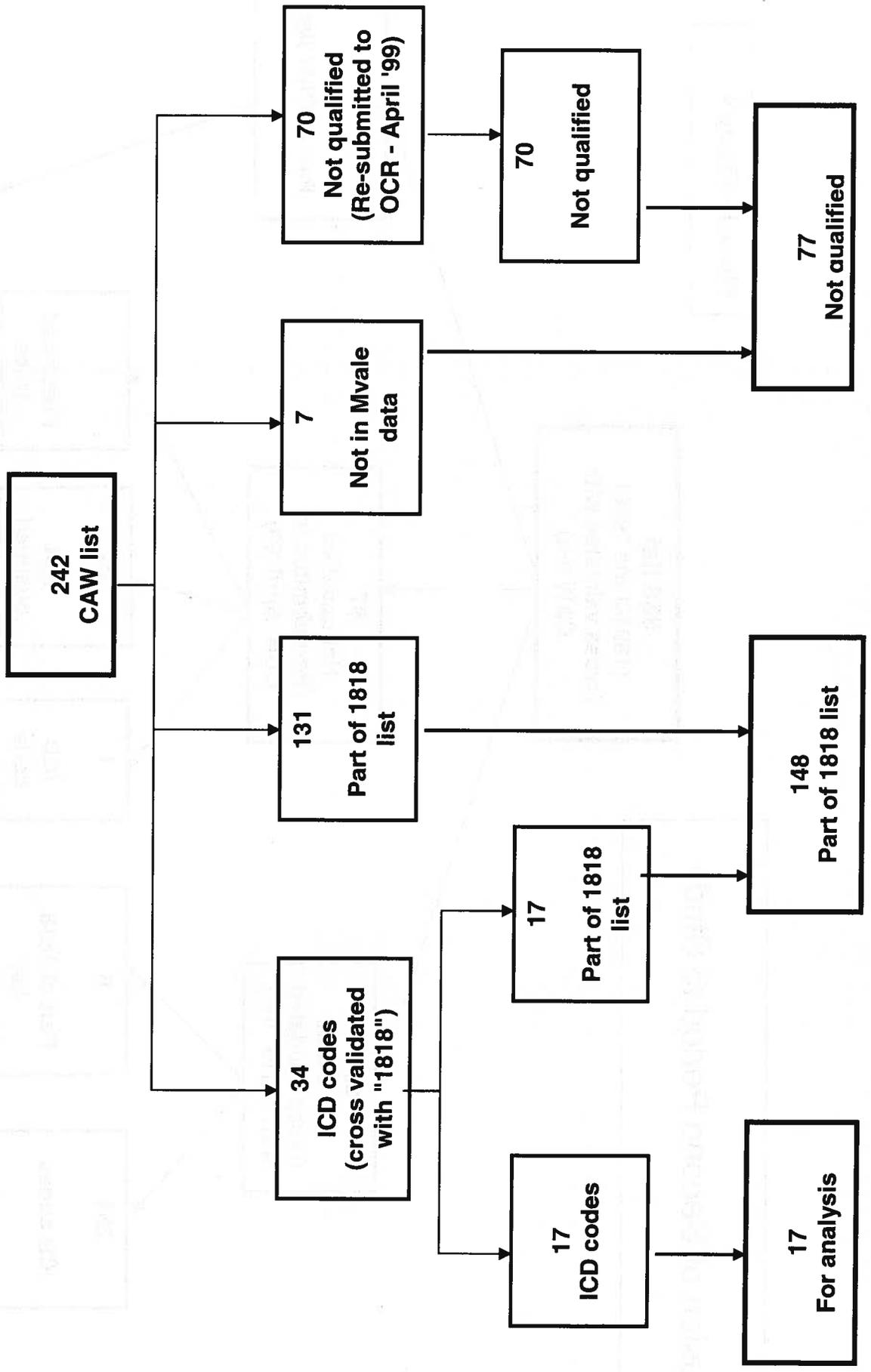


**Phase I – Figure 2**

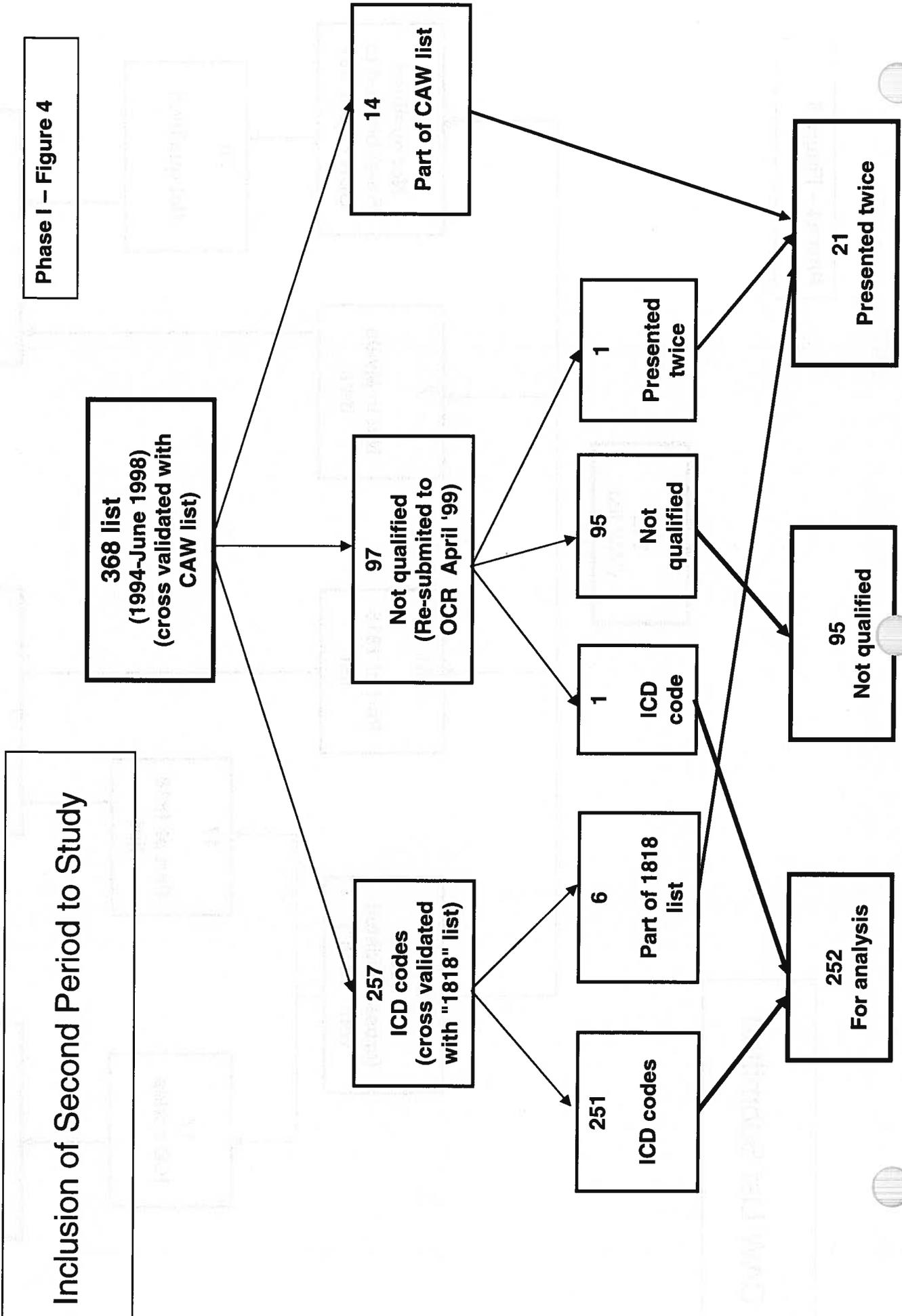


Phase I – Figure 3

CAW List Submitted



Phase I – Figure 4



Inclusion of Second Period to Study

368 list  
(1994-June 1998)  
(cross validated with  
CAW list)

257  
ICD codes  
(cross validated  
with "1818" list)

97  
Not qualified  
(Re-submitted to  
OCR April '99)

14  
Part of CAW list

251  
ICD codes

6  
Part of 1818  
list

1  
ICD  
code

95  
Not  
qualified

1  
Presented  
twice

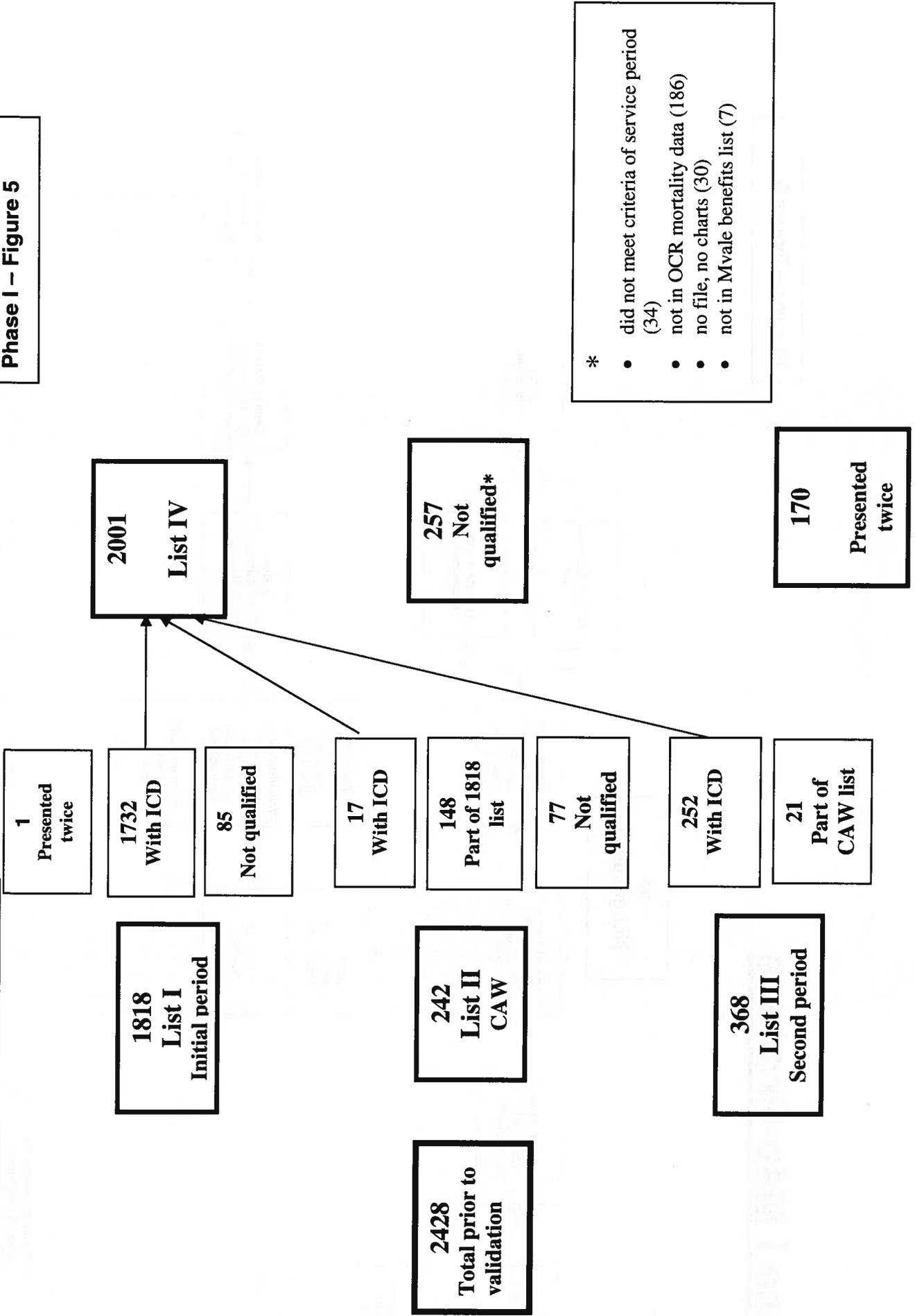
252  
For  
analysis

95  
Not  
qualified

21  
Presented  
twice

# Summary of Final List for Analysis

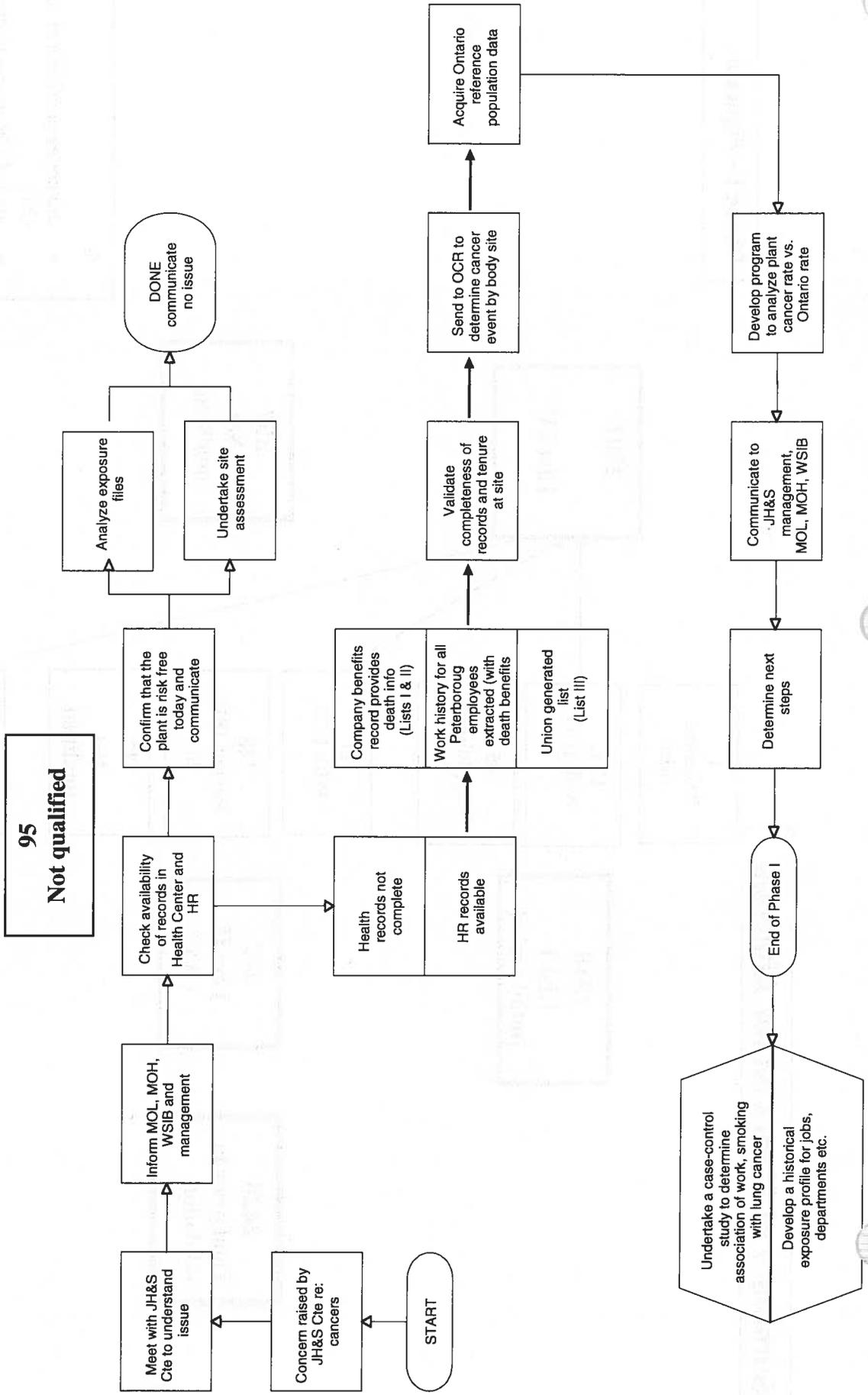
Phase I – Figure 5



\*  
 • did not meet criteria of service period (34)  
 • not in OCR mortality data (186)  
 • no file, no charts (30)  
 • not in Mvale benefits list (7)

# Phase I Map-Process

## Phase I – Figure 6





**Table 1 Demographic characteristics of entire group – all causes of death Number (%)**

**Phase I – Table 1**

VARIABLES	ENTIRE GROUP
Gender	
Male	1859 (92.9)
Female	142 (7.1)
Manufacturing > 1 year	
M fg-Active	326 (16.3)
M fg-Retired	1423 (71.1)
Not M fg-Active	49 (2.4)
Not M fg-Retired	176 (8.8)
Missing data	27 (1.3)
First GE Payclass	
Hourly	1639 (81.9)
Salary	335 (16.7)
Missing data	27 (1.3)
Last GE Payclass	
Hourly - Active	260 (13.0)
Hourly - Retired	1127 (56.3)
Salary - Active	115 (5.7)
Salary - Retired	472 (23.6)
Missing data	27 (1.3)
Age group at death	
19 – 29	17 (0.8)
30 – 39	18 (0.9)
40 – 49	71 (3.5)
50 – 59	207 (10.3)
60 – 69	510 (25.5)
70 – 79	713 (35.6)
80 – 89	396 (19.8)
90 – 99	68 (3.4)
100 +	1 (0.0)
Decade of death	
1970-79	632 (31.6)
1980-89	682 (34.1)
1990-99	687 (34.3)





Table 2: Demographic characteristics of group by last payroll class at GE  
Number (% of column)

\*All cells do not sum to 2001 due to missing observations

VARIABLE	HOURLY	SALARY
<b>Gender</b>		
Male	1303 (93.9%)	533 (90.8%)
Female	84 (6.1)	54 (9.2)
<b>Manufacturing &gt;1 year</b>		
Mfg-Active	259 (18.7)	67 (11.4)
Mfg-Retired	1126 (81.2)	297 (50.6)
Not Mfg – Active	1 (.1)	48 (8.2)
Not Mfg – Retired	1 (.1)	175 (29.8)
<b>First GE Payclass</b>		
Hourly	1381 (99.6)	258 (44.0)
Salary	6 (.4)	329 (56.0)
<b>Last GE Payclass</b>		
Hourly – Active	260 (18.7)	0
Hourly – Retired	1127 (81.3)	0
Salary – Active	0	115 (19.6)
Salary – Retired	0	472 (80.4)
<b>Age group at death</b>		
19-29	12 (.9)	5 (.9)
30-39	12 (.9)	6 (1.0)
40-49	49 (3.5)	21 (3.6)
50-59	147 (10.6)	58 (9.9)
60-69	354 (25.5)	153 (26.1)
70-79	475 (34.2)	227 (38.7)
80-89	288 (20.8)	101 (17.0)
90-99	49 (3.5)	17 (2.9)
100+	1 (.1)	0
<b>Mean age –SD</b>	70.8 – 12.1	70.2—12.0
<b>Decade of death</b>		
1970-79	470 (33.9)	161 (27.4)
1980-89	474 (34.2)	202 (34.4)
1990-98	443 (31.9)	224 (38.2)

**Phase I – Table 3**

**Table 3: Demographic characteristics of group by cancer death  
Number (% of column)**

\*All cells do not sum to 2001 due to missing observations

<b>VARIABLE</b>	<b>CANCER DEATH</b>	<b>OTHER DEATH</b>
<b>Gender</b>		
Male	524 (91.0%)	1335 (93.7%)
Female	52 (9.0)	90 (6.3)
<b>Manufacturing &gt;1 year</b>		
Mfg-Active	107 (19.0)	219 (15.5)
Mfg-Retired	389 (69.0)	1034 (73.3)
Not Mfg – Active	16 (2.8)	33 (2.3)
Not Mfg – Retired	52 (9.2)	124 (8.8)
<b>First GE Payclass</b>		
Hourly	452 (80.1)	1187 (84.2)
Salary	112 (19.9)	223 (15.8)
<b>Last GE Payclass</b>		
Hourly – Active	84 (14.9)	176 (12.5)
Hourly – Retired	287 (50.9)	840 (59.6)
Salary – Active	39 (6.9)	76 (5.4)
Salary – Retired	154 (27.3)	318 (22.6)
<b>Age group at death</b>		
19-29	4 (.7)	13 (.9)
30-39	3 (.5)	15 (1.0)
40-49	23 (4.0)	48 (3.4)
50-59	77 (13.4)	130 (9.1)
60-69	163 (28.3)	347 (24.4)
70-79	215 (37.3)	499 (35.2)
80-89	79 (13.7)	317 (22.0)
90-99	12 (2.1)	56 (3.9)
100+	0	1 (.1)
<b>Mean age –SD</b>	68.9 – 11.12	71.35 – 12.40
<b>Decade of death</b>		
1970-79	141 (24.5)	491 (34.5)
1980-89	196 (34.0)	486 (34.1)
1990-98	239 (41.5)	448 (31.4)

Phase I - Table 4

Table 4						
PCMR - ANALYSIS - ALL MALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	1859				
140-209	All cancers	524				
140-149	Buccal Cavity-Pharynx	10	12.19	0.82	0.45 - 1.51	
150-159	Digestive System	153	135.45	1.13	0.99 - 1.29	
155-156	Liver/Gallbladder,etc	13	9.96	1.31	0.76 - 2.23	
157	Pancreas	18	23.31	0.77	0.49 - 1.21	
<b>162-163</b>	<b>Lung</b>	<b>198</b>	<b>146.69</b>	<b>1.35</b>	<b>1.21 - 1.51</b>	<b>p&lt;0.05</b>
170	Bone	1	0.94	1.07	0.15 - 7.52	
172-173	Skin	7	6.19	1.13	0.54 - 2.35	
180-189	Genito-Urinary Organ	72	70.44	1.02	0	
185	Prostate	49	43.83	1.12	0.86 - 1.45	
188	Bladder	12	14.97	0.8	0.46 - 1.39	
189	Kidney	10	10.15	0.98	0.53 - 1.82	
191-192	Brain-Central Nervous	8	10.98	0.73	0.37 - 1.43	
200-209	Lymphopoietic Cancer	44	37.38	1.18	0.89 - 1.56	
200	Lympho-Reticulo	1	3.75	0.27	0.04 - 1.64	
<b>201</b>	<b>Hodgkin's Disease</b>	<b>7</b>	<b>2.09</b>	<b>3.35</b>	<b>1.68 - 6.68</b>	<b>p&lt;0.05</b>
204-207	Leukemia-Aleukemia	12	11.35	1.06	.61 - 1.85	
202-3,8	Lymphatic Tissue	24	20.19	1.19	.8 - 1.76	

PCMR - ANALYSIS - HOURLY MALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	1303				
140-209	All cancers	343				
140-149	Buccal Cavity-Pharynx	9	8.2	1.1	0.58 - 2.09	
150-159	Digestive System	99	90.57	1.09	0.93 - 1.29	
155-156	Liver/Gallbladder,etc	9	6.64	1.36	0.71 - 2.58	
157	Pancreas	9	15.57	0.58	0.31 - 1.08	
<b>162-163</b>	<b>Lung</b>	<b>131</b>	<b>98.02</b>	<b>1.34</b>	<b>1.16 - 1.54</b>	<b>p&lt;0.05</b>
170	Bone	1	0.62	1.62	0.23 - 11.19	
172-173	Skin	3	4.09	0.73	0.24 - 2.23	
180-189	Genito-Urinary Organ	42	47.2	0.89	0	
185	Prostate	26	29.36	0.89	0.62 - 1.26	
188	Bladder	10	10.04	1	0.54 - 1.83	
189	Kidney	5	6.81	0.73	0.31 - 1.74	
191-192	Brain-Central Nervous	5	7.25	0.69	0.29 - 1.62	
200-209	Lymphopoietic Cancer	25	24.82	1.01	0.69 - 1.46	
200	Lympho-Reticulo	0	2.5	0	0	
<b>201</b>	<b>Hodgkin's Disease</b>	<b>4</b>	<b>1.35</b>	<b>2.96</b>	<b>1.18 - 7.44</b>	<b>p&lt;0.05</b>
204-207	Leukemia-Aleukemia	10	7.54	1.33	0.72 - 2.44	
202-3,8	Lymphatic Tissue	11	13.44	0.82	.46 - 1.46	

Phase I – Table 6

Table 6						
PCMR - ANALYSIS - SALARY MALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	533				
140-209	All cancers	171				
140-149	Buccal Cavity-Pharynx	1	3.87	0.26	0.04 - 1.55	
150-159	Dygestive System	51	43.78	1.16	0.93 - 1.46	
155-156	Liver/Gallbladder,etc	4	3.22	1.24	0.47 - 3.26	
157	Pancreas	8	7.55	1.06	0.54 - 2.08	
<b>162-163</b>	<b>Lung</b>	<b>66</b>	<b>47.25</b>	<b>1.4</b>	<b>1.15 - 1.7</b>	<b>p&lt;0.05</b>
170	Bone	0	0.31	0	0	
172-173	Skin	3	2.06	1.46	0.48 - 4.42	
180-189	Genito-Urinary Organ	28	22.61	1.24	0	
<b>185</b>	<b>Prostate</b>	<b>21</b>	<b>14.03</b>	<b>1.5</b>	<b>1.01 - 2.22</b>	<b>p&lt;0.05</b>
188	Bladder	2	4.82	0.41	0.11 - 1.55	
189	Kidney	5	3.26	1.54	0.65 - 3.63	
191-192	Brain-Central Nervous	3	3.65	0.82	0.27 - 2.48	
200-209	Lymphopoietic Cancer	16	12.24	1.31	0.82 - 2.08	
200	Lympho-Reticulo	1	1.24	0.81	0.12 - 5.66	
<b>201</b>	<b>Hodgkin's Disease</b>	<b>3</b>	<b>0.73</b>	<b>4.13</b>	<b>1.47 - 11.56</b>	<b>p&lt;0.05</b>
204-207	Leukemia-Aleukemia	2	3.72	0.54	0.14 - 2.06	
208-3,8	Lymphatic Tissue	10	6.56	1.53	0.84 - 2.78	

Phase I – Table 7

Table 7						
PCMR - ANALYSIS - MANUFACTURING MALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	1651				
140-209	All cancers	460				
140-149	Buccal Cavity-Pharynx	10	10.85	0.92	0.50 - 1.70	
150-159	Digestive System	131	119.97	1.09	0.95 - 1.26	
155-156	Liver/Gallbladder,etc	11	8.8	1.25	0.70 - 2.24	
157	Pancreas	15	20.71	0.72	0.44 - 1.18	
<b>162-163</b>	<b>Lung</b>	<b>183</b>	<b>131.09</b>	<b>1.4</b>	<b>1.24 - 1.57</b>	<b>p&lt;0.05</b>
170	Bone	1	0.79	1.27	0.18 - 8.93	
172-173	Skin	4	5.25	0.76	0.29 - 2	
180-189	Genito-Urinary Organ	61	61.57	0.99	0	
185	Prostate	38	38.19	1	0.74 - 1.34	
188	Bladder	12	13.16	0.91	0.52 - 1.59	
189	Kidney	10	9.03	1.11	0.60 - 2.04	
191-192	Brain-Central Nervous	6	9.56	0.63	0.29 - 1.37	
200-209	Lymphopoietic Cancer	34	32.56	1.04	0.76 - 1.44	
200	Lympho-Reticulo	1	3.32	0.3	0.05 - 1.89	
<b>201</b>	<b>Hodgkin's Disease</b>	<b>5</b>	<b>1.74</b>	<b>2.88</b>	<b>1.26 - 6.59</b>	<b>p&lt;0.05</b>
204-207	Leukemia-Aleukemia	11	9.8	1.12	0.63 - 2.01	
202-3,8	Lymphatic Tissue	17	17.7	0.96	0.60 - 1.53	

Phase I - Table 8

<b>Table 8</b>						
<b>PCMR - ANALYSIS - Not MANUFACTURING MALE</b>						
<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
001-998	All causes	185				
140-209	All cancers	54				
140-149	Buccal Cavity-Pharynx	0	1.22	0	0	
150-159	Digestive System	19	14.39	1.32	0.91 - 1.92	
155-156	Liver/Gallbladder, etc	2	1.06	1.88	0.49 - 7.24	
157	Pancreas	2	2.41	0.83	0.22 - 3.2	
162-163	Lung	14	14.17	0.99	0.64 - 1.52	
170	Bone	0	0.15	0	0	
172-173	Skin	2	0.9	2.21	0.59 - 8.27	
180-189	Genito-Urinary Organ	9	8.23	1.09	0	
185	Prostate	9	5.21	1.73	0.96 - 3.12	
188	Bladder	0	1.71	0	0	
189	Kidney	0	1.04	0	0	
191-192	Brain-Central Nervous	2	1.34	1.5	0.39 - 5.68	
200-209	Lymphopoietic Cancer	7	4.51	1.55	0.78 - 3.09	
200	Lympho-Reticulo	0	0.41	0	0	
<b>201</b>	<b>Hodgkin's Disease</b>	<b>2</b>	<b>0.34</b>	<b>5.86</b>	<b>1.77 - 19.33</b>	<b>p&lt;0.05</b>
204-207	Leukemia-Aleukemia	1	1.46	0.68	0.10 - 4.59	
202-3,8	Lymphatic Tissue	4	2.29	1.75	0.68 - 4.48	



Table 9						
PCMR - ANALYSIS - SALARY MANUFACTURING MALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	350				
140-209	All cancers	117				
140-149	Buccal Cavity-Pharynx	1	2.65	0.38	0.06 - 2.41	
150-159	Digestive System	32	29.39	1.09	0.82 - 1.45	
155-156	Liver/Gallbladder,etc	2	2.16	0.93	0.24 - 3.64	
157	Pancreas	6	5.15	1.17	0.54 - 2.54	
<b>162-163</b>	<b>Lung</b>	<b>52</b>	<b>33.07</b>	<b>1.57</b>	<b>1.26 - 1.96</b>	<b>p&lt;0.05</b>
170	Bone	0	0.17	0	0	
172-173	Skin	1	1.15	0.87	0.12 - 6.06	
180-189	Genito-Urinary Organ	19	14.38	1.32	0	
185	Prostate	12	8.83	1.36	0.80 - 2.31	
188	Bladder	2	3.11	0.64	0.17 - 2.48	
189	Kidney	5	2.21	2.26	0.97 - 5.24	
191-192	Brain-Central Nervous	1	2.31	0.43	0.07 - 2.81	
200-209	Lymphopoietic Cancer	9	7.74	1.16	0.62 - 2.18	
200	Lympho-Reticulo	1	0.82	1.22	0.17 - 8.51	
201	Hodgkin's Disease	1	0.39	2.6	0.40 - 17.03	
204-207	Leukemia-Aleukemia	1	2.26	0.44	0.07 - 2.91	
202-3,8	Lymphatic Tissue	6	4.27	1.41	0.65 - 3.06	

Table 10						
PCMR - ANALYSIS - SALARY Not MANUFACTURING MALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	183				
140-209	All cancers	54				
140-149	Buccal Cavity-Pharynx	0	1.22	0	0	
150-159	Digestive System	19	14.39	1.32	0.91 - 1.92	
155-156	Liver/Gallbladder,etc	2	1.06	1.88	0.49 - 7.24	
157	Pancreas	2	2.41	0.83	0.22 - 3.2	
162-163	Lung	14	14.17	0.99	0.64 - 1.52	
170	Bone	0	0.15	0	0	
172-173	Skin	2	0.9	2.21	0.59 - 8.27	
180-189	Genito-Urinary Organ	9	8.23	1.09	0	
185	Prostate	9	5.21	1.73	0.96 - 3.12	
188	Bladder	0	1.71	0	0	
189	Kidney	0	1.04	0	0	
191-192	Brain-Central Nervous	2	1.34	1.5	0.39 - 5.68	
200-209	Lymphopoietic Cancer	7	4.51	1.55	0.78 - 3.09	
200	Lympho-Reticulo	0	0.41	0	0	
201	Hodgkin's Disease	2	0.34	5.86	1.77 - 19.33	p<0.05
204-207	Leukemia-Aleukemia	1	1.46	0.68	0.10 - 4.59	
202-3,8	Lymphatic Tissue	4	2.29	1.75	0.68 - 4.48	

Table 11						
PCMR - ANALYSIS - ALL FEMALE						
CODE - ICD-9	ORGAN	OBS	EXP	PCMR	CONF. LIMITS	p-value
001-998	All causes	142				
140-209	All cancers	52				
140-149	Buccal Cavity-Pharynx	0	0.59	0	0	
150-159	Digestive System	10	12.66	0.79	.47-1.33	
155-156	Liver/Gallbladder, etc	0	1.13	0	0	
157	Pancreas	1	2.28	0.44	.07-2.8	
162-163	<b>Lung</b>	<b>16</b>	<b>7.18</b>	<b>2.23</b>	<b>1.44 - 3.44</b>	<b>p&lt;0.05</b>
170	Bone	0	0.12	0	0	
172-173	Skin	2	0.57	3.54	0.98 - 12.76	
174	Breast	11	10	1.1	0.66 - 1.84	
180-189	Genito-Urinary Organ	4	6.79	0.59	0	
188	Bladder	0	0.6	0	0	
189	Kidney	0	0.72	0	0	
191-192	Brain-Central Nervous	2	1.22	1.64	0.43 - 6.32	
200-209	Lymphopoietic Cancer	3	3.95	0.76	0.26 - 2.2	
200	Lympho-Reticulo	0	0.39	0	0	
201	Hodgkin's Disease	0	0.3	0	0	
204-207	Leukemia-Aleukemia	1	1.11	0.9	0.13 - 6.15	
202-3,8	Lymphatic Tissue	2	2.15	0.93	0.24 - 3.6	

Table 12

**PCMR - ANALYSIS - HOURLY FEMALE**

<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
001-998	All causes	84				
140-209	All cancers	28				
140-149	Buccal Cavity-Pharynx	0	0.32	0	0	
150-159	Digestive System	5	6.6	0.76	0.36 - 1.59	
155-156	Liver/Gallbladder,etc	0	0.56	0	0	
157	Pancreas	0	1.21	0	0	
162-163	<b>Lung</b>	<b>9</b>	<b>3.93</b>	<b>2.29</b>	<b>1.29 - 4.08</b>	<b>p&lt;0.05</b>
170	Bone	0	0.9	0	0	
172-173	Skin	1	0.34	2.98	0.47 - 18.75	
174	Breast	4	5.49	0.73	0.31 - 1.7	
180-189	Genito-Urinary Organ	1	3.68	0.27	0	
188	Bladder	0	0.31	0	0	
189	Kidney	0	0.39	0	0	
191-192	Brain-Central Nervous	1	0.71	1.41	0.21 - 9.56	
200-209	Lymphopoietic Cancer	3	2.25	1.33	0.47 - 3.82	
200	Lympho-Reticulo	0	0.21	0	0	
201	Hodgkin's Disease	0	0.23	0	0	
204-207	Leukemia-Aleukemia	1	0.64	1.56	0.23 - 10.43	
202-3,8	Lymphatic Tissue	2	1.17	1.71	0.45 - 6.48	

<b>Table 13</b>						
<b>PCMR - ANALYSIS - MANUFACTURING FEMALE</b>						
<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	<b>p-value</b>
001-998	All causes	98				
140-209	All cancers	36				
140-149	Buccal Cavity-Pharynx	0	0.43	0	0	
150-159	Digestive System	6	8.98	0.67	0.34 - 1.30	
155-156	Liver/Gallbladder,etc	0	0.78	0	0	
157	Pancreas	0	1.62	0	0	
162-163	<b>Lung</b>	<b>11</b>	<b>4.98</b>	<b>2.21</b>	<b>1.31 - 3.73</b>	<b>p&lt;0.05</b>
170	Bone	0	0.11	0	0	
172-173	Skin	1	0.42	2.39	0.37 - 15.61	
174	Breast	7	7.23	0.97	0.51 - 1.85	
180-189	Genito-Urinary Organ	2	4.97	0.4	0	
188	Bladder	0	0.43	0	0	
189	Kidney	0	0.52	0	0	
191-192	Brain-Central Nervous	2	0.91	2.21	0.59 - 8.31	
200-209	Lymphopoietic Cancer	3	2.88	1.04	0.36 - 3.01	
200	Lympho-Reticulo	0	0.29	0	0	
201	Hodgkin's Disease	0	0.25	0	0	
204-207	Leukemia-Aleukemia	1	0.82	1.22	0.18 - 8.31	
202-3,8	Lymphatic Tissue	2	1.52	1.32	0.34 - 5.06	

Phase I – Table 14

<b>Table 14 PCMR - ANALYSIS - SALARY-NON-MANUFACTURING-FEMALE</b>						
<b>CODE - ICD-9</b>	<b>ORGAN</b>	<b>OBS</b>	<b>EXP</b>	<b>PCMR</b>	<b>CONF. LIMITS</b>	
001-998	All causes	40				
140-209	All cancers	14				
140-149	Buccal Cavity-Pharynx	0	0.14	0	0	
150-159	Digestive System	4	3.25	1.23	0.55 - 2.76	
155-156	Liver/Gallbladder,etc	0	0.3	0	0	
157	Pancreas	1	0.58	1.72	0.26 - 11.31	
162-163	Lung	4	1.87	2.14	0.90 - 5.06	
170	Bone	0	0.01	0	0	
172-173	Skin	0	0.11	0	0	
174	Breast	4	2.22	1.8	0.76 - 4.23	
180-189	Genito-Urinary Organ	2	1.54	1.29	0	
188	Bladder	0	0.15	0	0	
189	Kidney	0	0.18	0	0	
191-192	Brain-Central Nervous	0	0.25	0	0	
200-209	Lymphopoietic Cancer	0	0.88	0	0	
200	Lympho-Reticulo	0	0.09	0	0	
201	Hodgkin's Disease	0	0.03	0	0	
204-207	Leukemia-Aleukemia	0	0.23	0	0	
202-3,8	Lymphatic Tissue	0	0.52	0	0	

Table 15

**COMPARISON BETWEEN JOB CLASSES FOR SIGNIFICANT CASES**

**Observed Number of Cases**

**MALE**

Type of Cancer	All Male	H	S	Manuf.	NonManuf.	S-Manuf.	S-NonManuf.	H-Manuf.	H-nonManuf.
Lung	*198	*131	*66	*183	14	*52	14	*131	0
Hodgkin	*7	*4	*3	*5	*2	1	*2	*4	0
Prostate	49	26	*21	38	9	12	9	26	0

**FEMALE**

Type of Cancer	All Fem.	H	S	Manuf.	NonManuf.	S-Manuf.	S-NonManuf.	H-Manuf.	H-nonManuf.
Lung	*16	*9	*6	*11	4	2	4	*9	0

\* = PCMR value significant higher than 1

All Male = Hourly + Salary + No job status (131+66+1)

All Female = Hourly + Salary + No job status (9+6+0)

For Male (Lung): H = H -Manuf. + H-nonManuf. (131+0)

For Female (Lung): Hourly = H-Manuf.(9)

1. What is the purpose of the experiment?  
 The purpose of this experiment is to determine the effect of temperature on the rate of reaction between hydrogen peroxide and potassium iodide.

2. What is the hypothesis?  
 It is hypothesized that as the temperature increases, the rate of reaction will also increase.

Table 1: Reaction Time vs. Temperature

Temperature (°C)	Reaction Time (s)
10	120
20	60
30	30
40	15
50	8

3. What is the conclusion?  
 The experiment shows that the rate of reaction increases significantly with temperature.

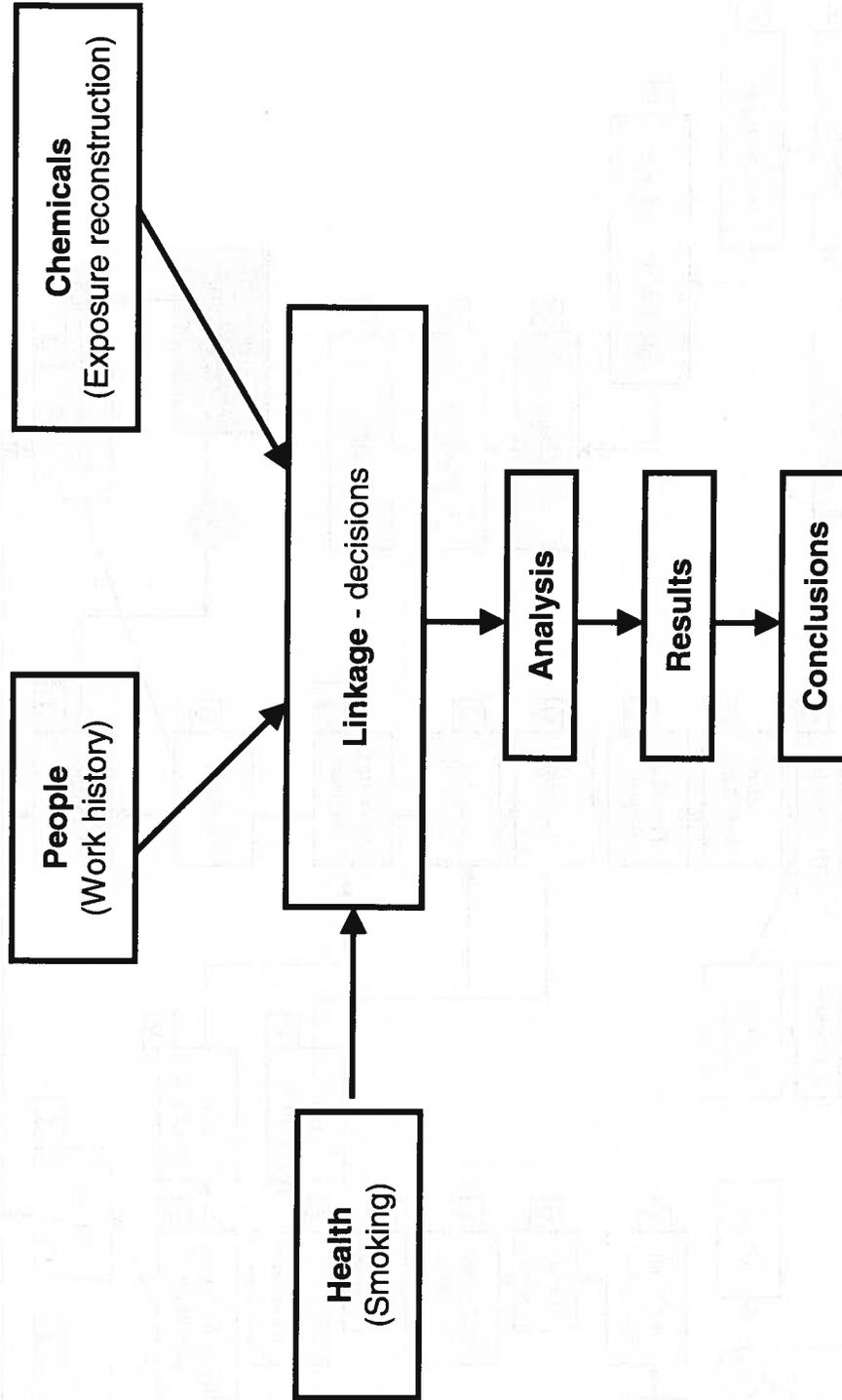
4. What are the sources of error?  
 Possible sources of error include inaccurate timing and inconsistent concentrations of reactants.

Date: \_\_\_\_\_  
 Page: \_\_\_\_\_



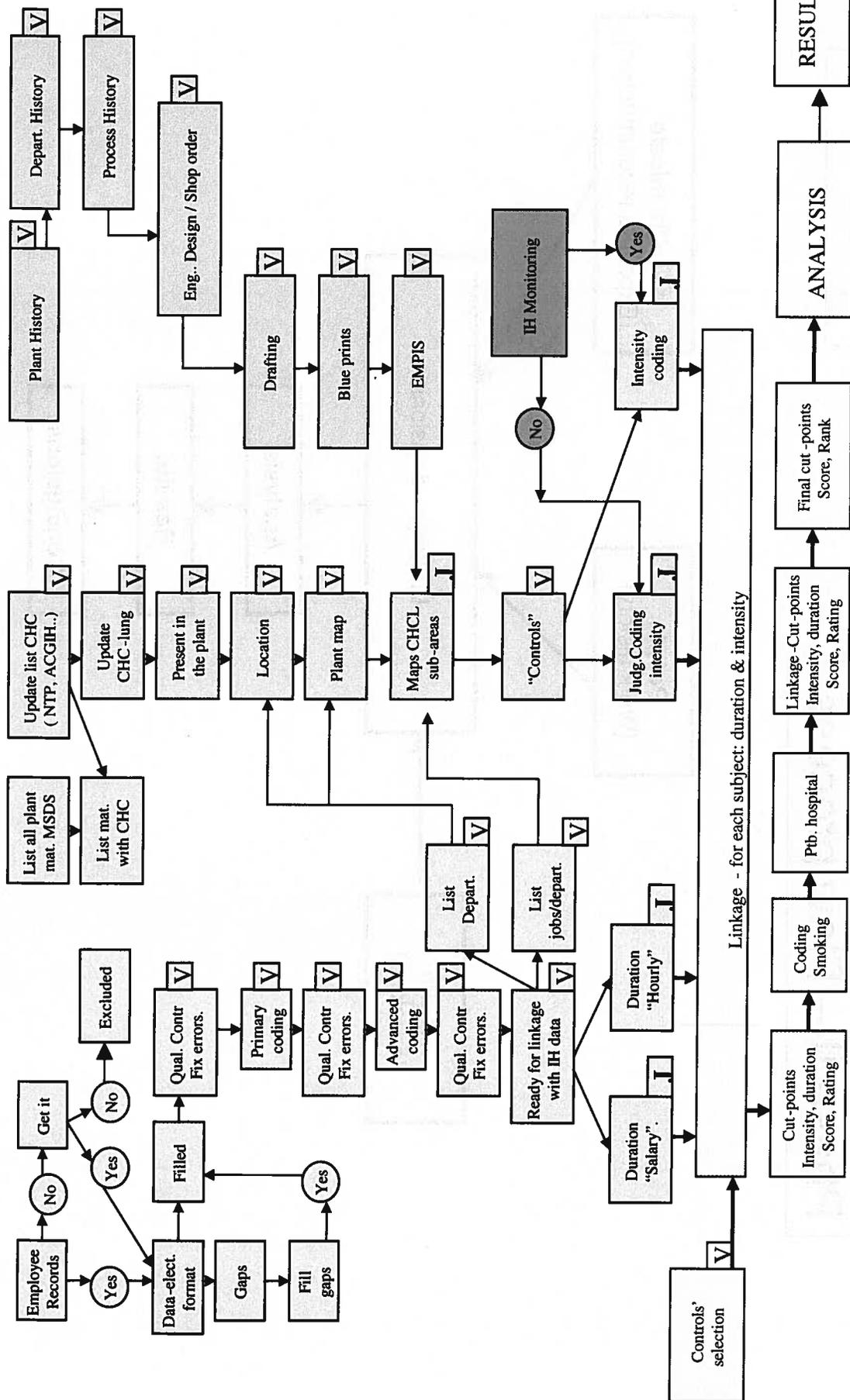
# Phase II – Flow process

## Phase II – Appendix 1



# Phase II – Flow process (detailed)

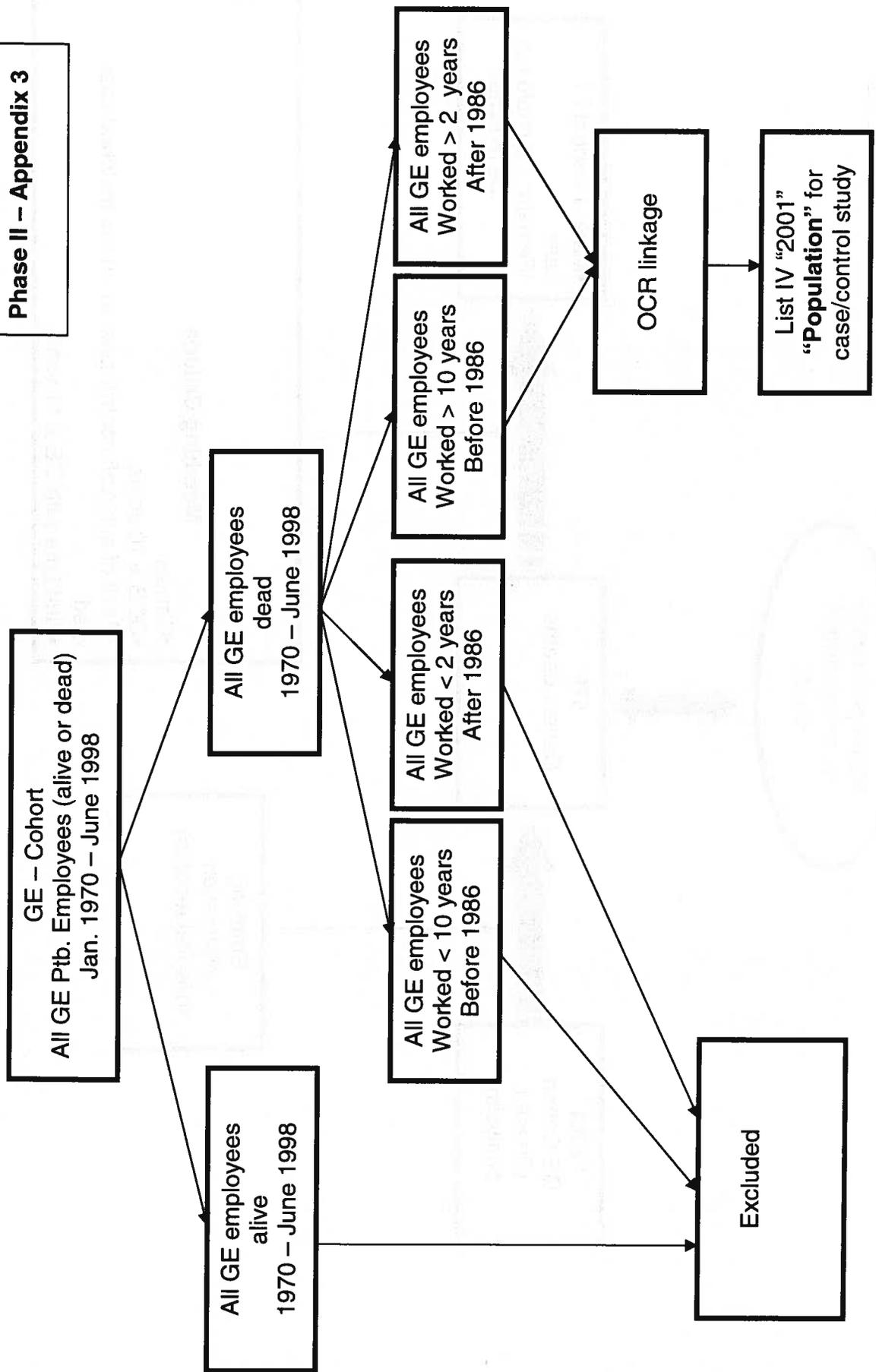
# Phase II – Appendix 2



**V** = validation

**J** = judgment

Phase II – Appendix 3



**Phase II – Appendix 4**

Study population  
for case/control  
study



**576**  
Cancer deaths

**“2001”**  
GE Cohort  
(Phase I  
analysis)



•Male: 1 control / 1 case  
•Female: 1/3 could not be matched

Smoking  
information  
(hospital records)

**Matching Criteria**

- Gender
- DOB  $\pm$  10 years
- Point of survival: control alive when the matched case died
- First time with GE  $\pm$  11 years

**Phase II – Appendix 5**

**HOURLY CLOCK NUMBERS**

200	Structural Steel
300	Machine Shop
400	Machine Shop
500	Machine Shop
600	Small IAC
700	currently Traction, previous to 94 Fractional Motors
800	"
900	currently not in use, '94 to '95 Traction, previous to 94 Tool Room
1200	Carpenter Shop
1300	Structural Steel
1400	Low Voltage Control
1500	Punch Press
1600	Small IAC
1800	Armature
1900	Switchgear (now PC&C)
2000	Switchgear (now PC&C)
2200	Barstock, salvage, maintenance
2300	Transportation (Diesel)
2400	Fractional Motors
2500	Small IAC
2600	Fractional Motors
2700	Armature (unit 616)
2800	Armature (upstairs)
2900	Main Test (unit 912)
3000	Low Voltage Control
3200	Small IAC
3300	Steam Plant
3500	Structural Steel
3600	Unwound Components
3700	Maintenance
3800	Tool Room



**Phase II -- Appendix 7**

**Primary coding**

- Separate entry for each job
- Decide job-status (H or S)

**Advanced coding**

- Development and implementation codes for Salary jobs
- Calculate job duration (2) for each entry

Start	End	Job status (H or S)	Job title

- Code 0: No exp.
- Code 1: 25% time on the floor
- Code 2: 50% time on the floor
- Code 3: 75% time on the floor
- Code 4: 100% time on the floor

**Duration 3 - calculation**

Start	End	Duration 1	Code	Duration 2	Exp. Duration %	Duration 3
1/1/1950	31/12/1952	2 y	4 (100%)	2 y	30%	8 months
1/1/1953	31/12/1955	2 y	2 (50%)	1 y	50%	6 months
1/1/1956	31/12/1958	2 y	3 (75%)	1.5 y	0 (no exp.)	0 (no exp.)

**Phase II – Appendix 8**

**Job names**



<u>Department</u>	<u>Same Job - Different Spelling</u>
Nuclear	Fuel Rod Operator - Process Group #2
Nuclear	Fuel Rod Oper - Process group #2
Nuclear	Fuel Rod Oper. - Process Group #2
Nuclear	Fuel Rod Operator- Process Group #2
Nuclear	Fuel Rod Opr - Process Group #2

**Department names**



<u>Same Department - Different Spelling</u>
Ind Cont
Ind Control
Ind. Control
Ind. Control Division
Ind. Cont.



Phase II – Appendix 9

Carcinogen	Classification Institutions *							Included in the analysis
	ACGIH	IARC	NTP	NIOSH	OSHA	EPA		
Arsenic	*	*	*	*	*	*	*	60
Asbestos	*	*	*	*	*	*	*	60
Beryllium	*	*	*	*	*	*	*	60
Cadmium		*	*	*	*	*	*	60
Chromium VI	*	*	*	*	*	*	*	60
Nickel	*	*	?	*	*	*	*	60
Silica	*	*	*	*	*	*	*	60
Uranium	*	*		*	*	*	*	60

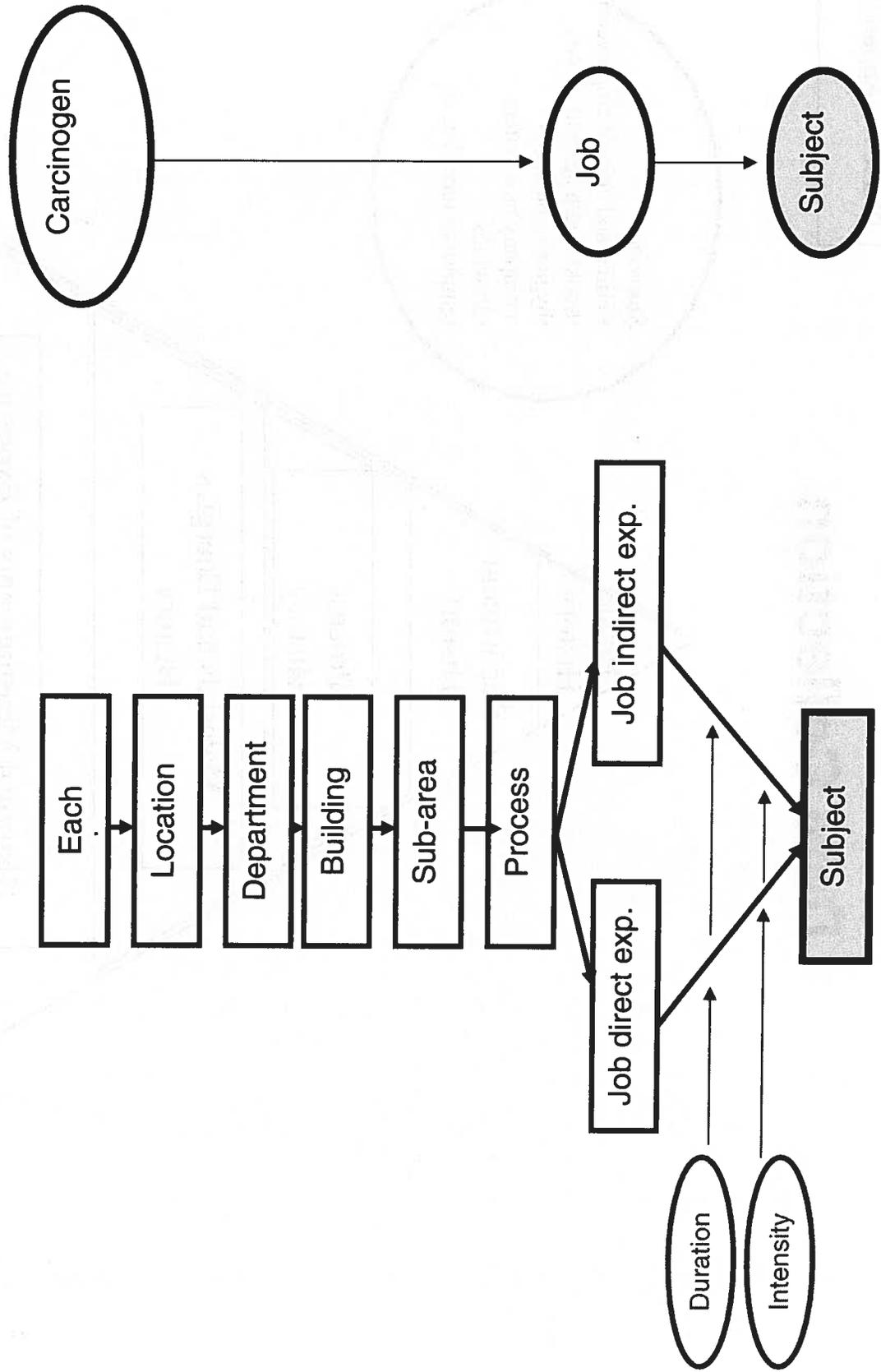
\*

- ACGIH - American Conference of Governmental Industrial Hygiene
- IARC - International Agency for Research on Cancer
- NTP - National Toxicology Program
- NIOSH - National Institute for Occupational Safety and Health
- OSHA - Occupational Safety and Health Administration
- EPA - US Environmental Protection Agency

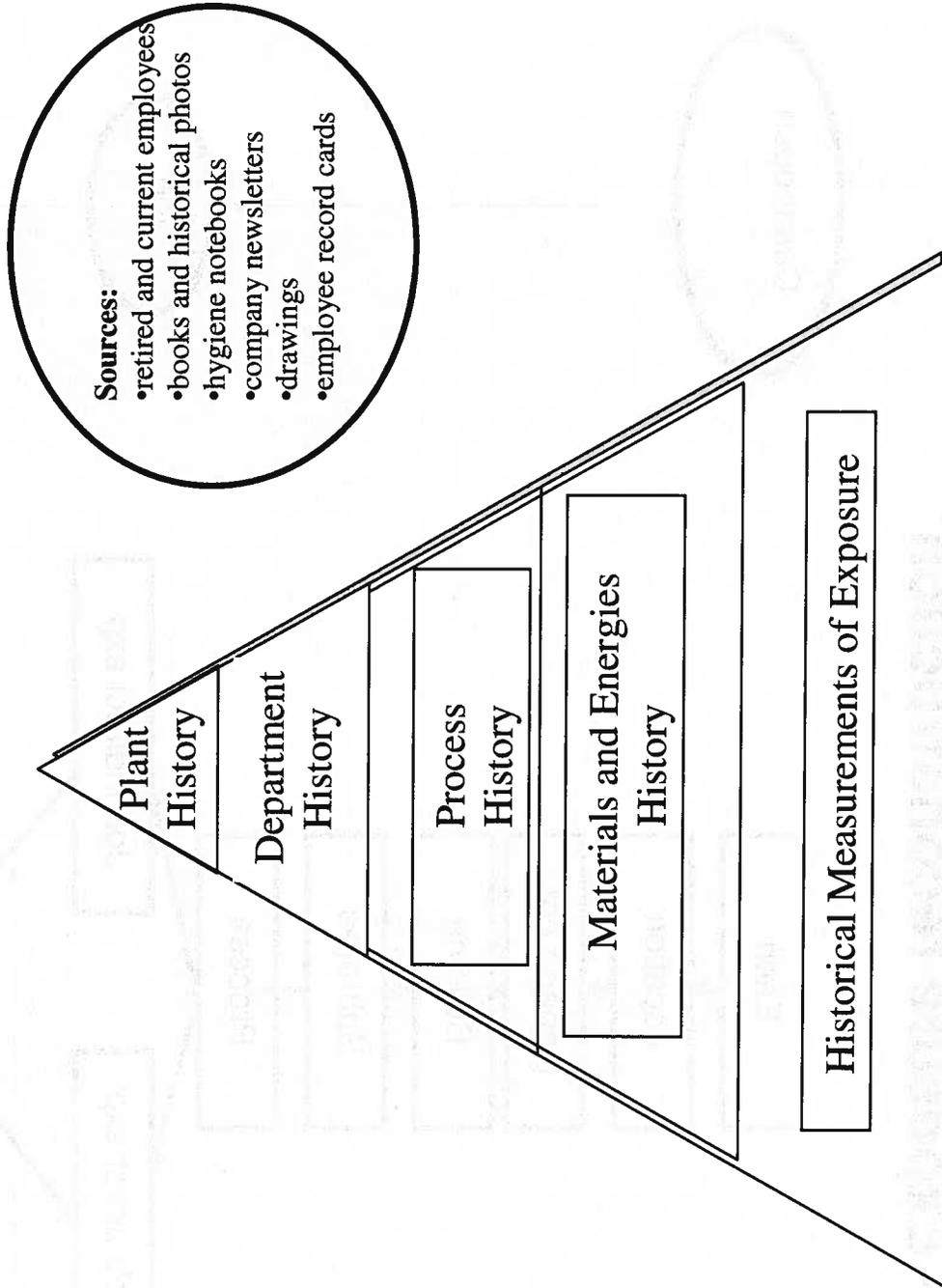
Time Weighted Average Exposure Limit (TWA)			
Carcinogen	TWA (1970)	TWA (1985)	TWA (2000)
Asbestos	5 f/cc (*)	2 f/cc (*)	0.2 f/cc (*)
Arsenic	0.5 mg/m <sup>3</sup>	0.2 mg/m <sup>3</sup>	0.01 mg/m <sup>3</sup> (*)
Beryllium	0.002 mg/m <sup>3</sup>	0.002 mg/m <sup>3</sup>	0.002 mg/m <sup>3</sup> (*)
Cadmium	0.2 mg/m <sup>3</sup>	0.5 mg/m <sup>3</sup>	0.002 mg/m <sup>3</sup> (*)
Chromium VI	1.0 mg/m <sup>3</sup> (*)	0.05 mg/m <sup>3</sup> (*)	0.01 mg/m <sup>3</sup> (*)
Nickel	1.0 mg/m <sup>3</sup>	1.0 mg/m <sup>3</sup> (*)	0.1 mg/m <sup>3</sup> (*)
Silica	0.1 mg/m <sup>3</sup>	0.1 mg/m <sup>3</sup>	0.05 mg/m <sup>3</sup> (*)
Uranium	0.2 mg/m <sup>3</sup>	0.2 mg/m <sup>3</sup>	0.2 mg/m <sup>3</sup> (*)

- f/cc = fibers / cubic centimeter of air
- mg/m<sup>3</sup> = mg of material / cubic meter of air
- ( \*) = Recognized Human Carcinogen

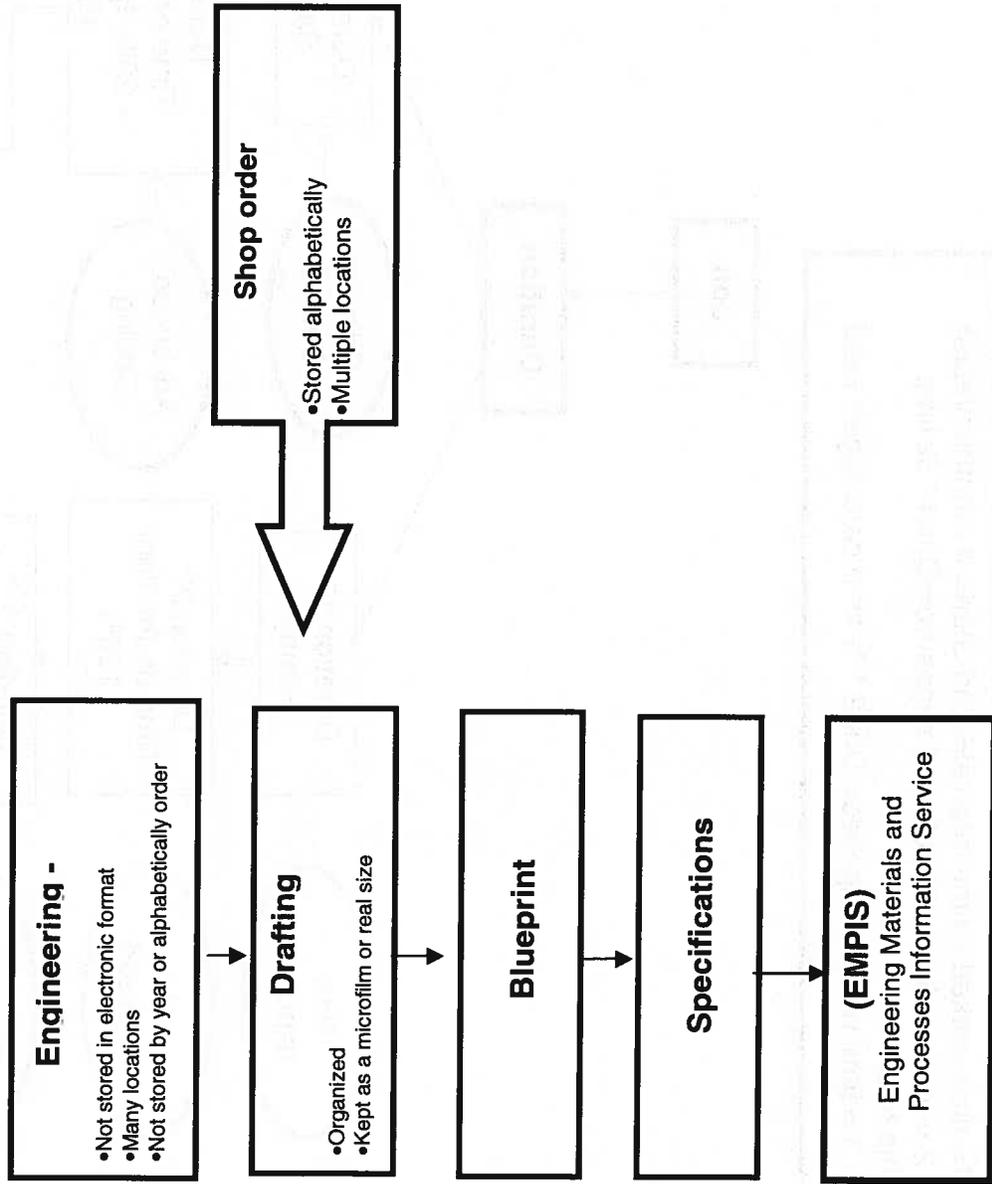
# Design – Exposure reconstruction



# Data Collection

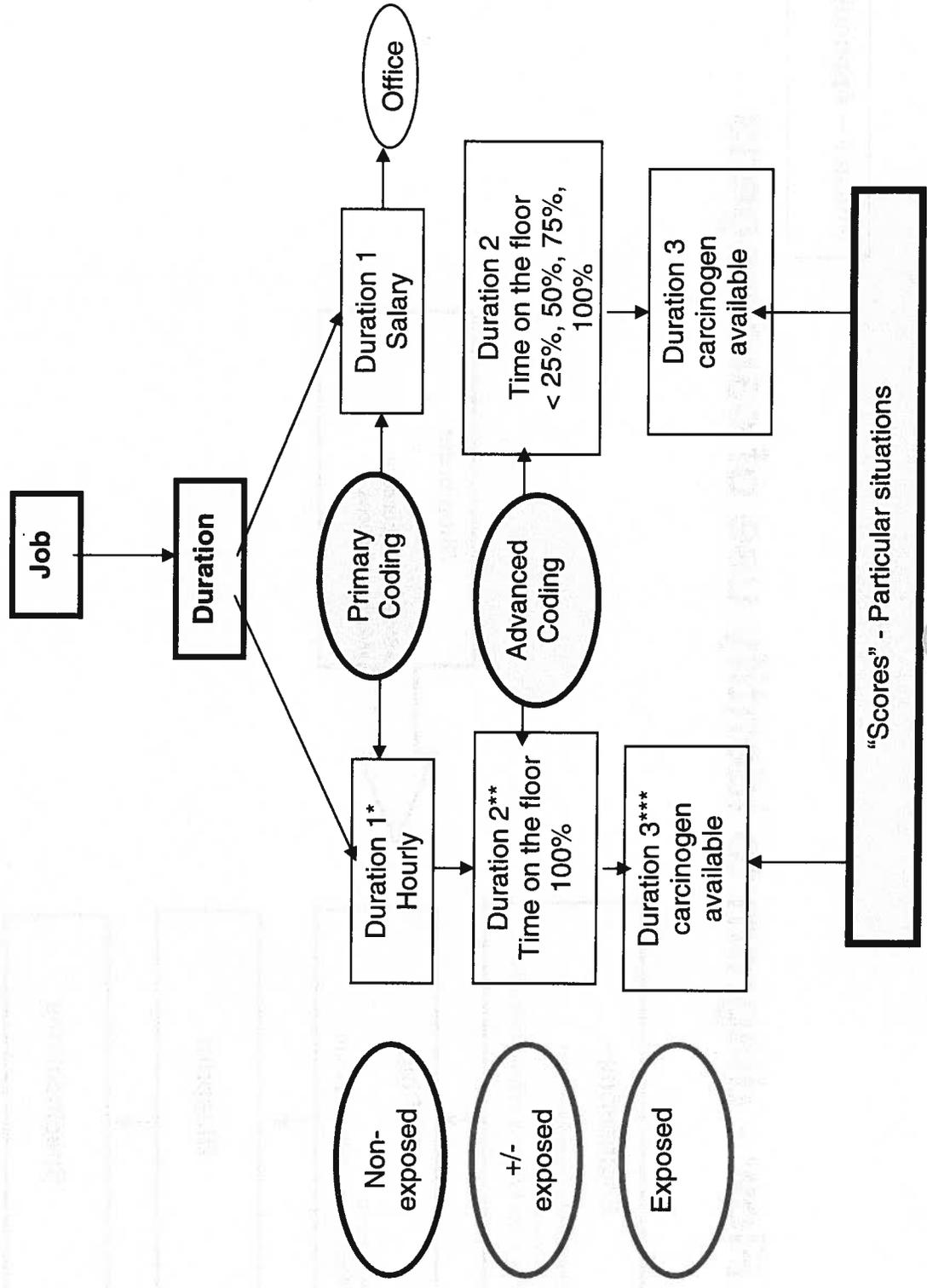


# Flow - diagram to identify use of carcinogens

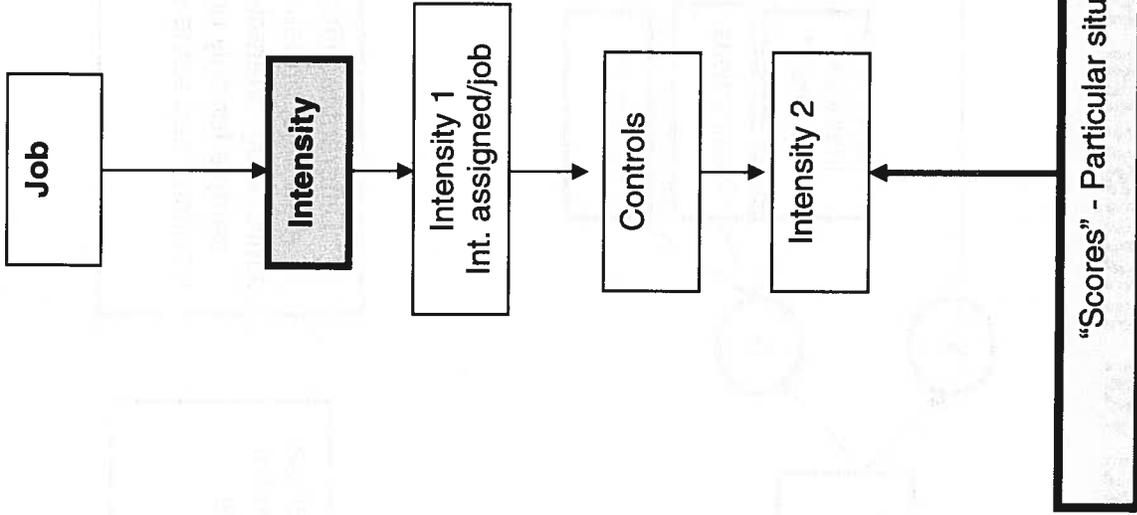


**Phase II – Appendix 14**

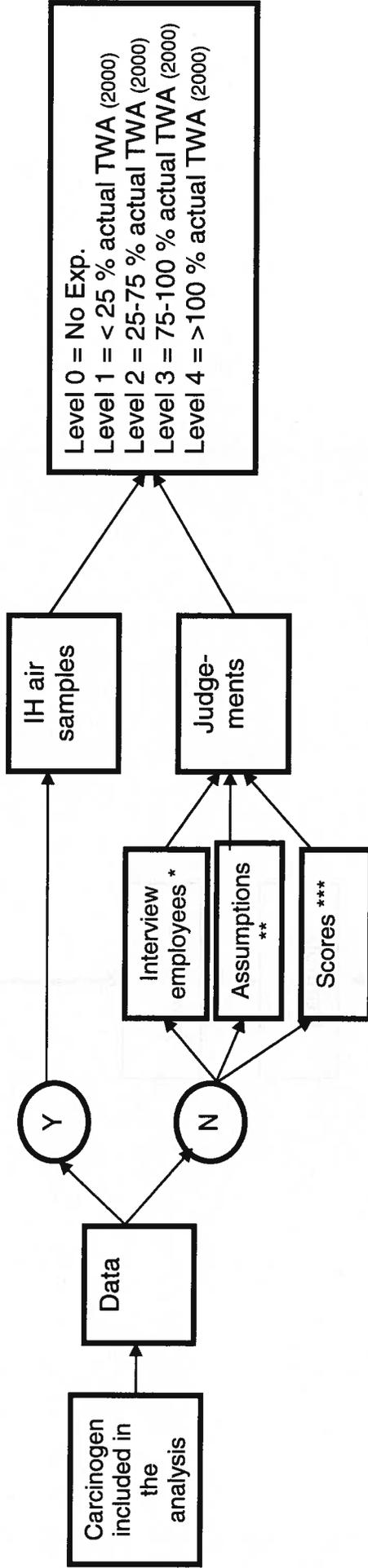
\* Duration 1 = time worked = time interval= End-Start= # months (years)  
 \*\* Duration 2 = time worked with potential exposure= Dur.1 \* % time worked on the floor  
 \*\*\* Duration 3 = time worked exposed= Dur.2 \* % time carcinogen used



**Phase II – Appendix 15**



# Exposure Intensity Criteria for Industrial Hygiene (IH) data



Level 0 = No Exp.  
Level 1 = < 25 % actual TWA (2000)  
Level 2 = 25-75 % actual TWA (2000)  
Level 3 = 75-100 % actual TWA (2000)  
Level 4 = >100 % actual TWA (2000)

\*\* Different locations for same job, materials, protective equipment, similar bldg. characteristics, with IH air sample for only one location = all locations had same exposure intensity

\* Opinions about jobs with similar exposure

\*\*\* See Particular Situations



**Phase II – Appendix 17**

Judgments

Controls

Initial Exp. Level *	Respirator	Local exhaust	General ventilation	Final Exp. Level **	Fugitive ***
1	*	*	*	1	1
		*	*	1	1
			*	1	1
2	*	*	*	1	1
		*	*	2	1
			*	2	2
3	*	*	*	1	2
		*	*	2	2
			*	3	3
4	*	*	*	1	3
		*	*	3	3
			*	4	4

\* = From work history judgments  
 \*\* = Following consideration of use of exposure control equipment  
 \*\*\* = Fugitive means exposure for those employees working in the vicinity of the operation involving the carcinogen

Air Sample

Initial Exp. Level *	Respirator	Final Exp. Level	Fugitive
1	*	1	1
		1	1
2	*	1	2
		2	2
3	*	1	3
		3	3
4	*	1	4
		4	4

\* = From I H data

# “Scores” - Particular situations

<u>Particular situations</u>	<u>A</u>	<u>B</u>	<u>C</u>
Time interval	1	1	1
Job(s)	1	1	>
Location(s)/job	1	>	1
Exp. Sources/Duration	1	>	>
Intensity Sources	1	>	>

- (C) Same time interval, multiple sources exp. (jobs), diff. Dur. exp. and Int. exp.

<u>Interval</u>	<u>Job</u>	<u>Dur. Exp.</u>	<u>Int.Exp.</u>	<u>Scores</u>
a - b	A	100%	2	200
	B (fugitive)	25%	4	100
			<b>TOTAL</b>	<b>300</b>
equivalent to level 3 exp. 100% of time				

- (B) Same time interval, same job, and multiple locations with diff. Dur. exp. and Int. exp.

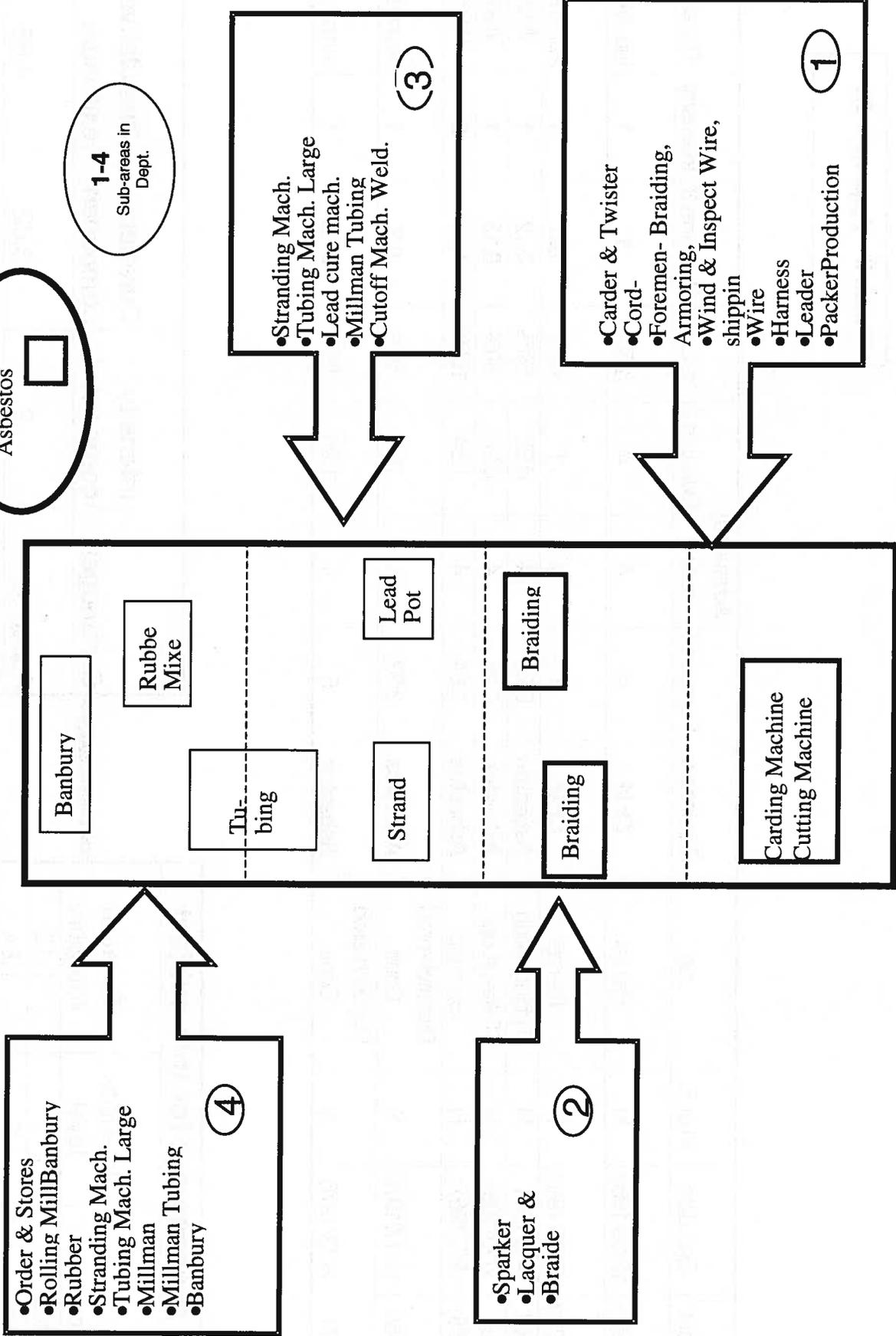
<u>Interval</u>	<u>Job</u>	<u>Location</u>	<u>Dur. Exp.</u>	<u>Int.Exp.</u>	<u>Scores</u>
a - b	A	*	50%	2	100
		**	0 (no exp.)	0 (no exp.)	0
				<b>TOTAL</b>	<b>100</b>
equivalent to level 1 exp. 100% of time					

**Phase II - Appendix 19**

**Bldg. 22 (W&C) - processes and jobs related**



1-4  
Sub-areas in Dept.



Phase II – Appendix 20

Start date	End date	H or S	Job	Carcinogen	Duration 1	Advanced coding	Duration 2	% exp.	Duration 3	Intensity	Departm.
12/26/1955	12/26/1959	H	Painter	Cr VI	4	4	4	25%	1	1	Ind. Motor
12/27/1959	12/29/1960	H	Painter	Cr VI	1	4	1	40%	0.3	1	Apparatus
1/16/1961	4/8/1961	H	Tubing mach.	Asbestos	0.23	4	0.23	50%	0.12	1	W&C
4/9/1961	7/12/1961	H	Take-off opr.	Asbestos	0.26	4	0.26	50%	0.13	1	W&C
4/5/1965	2/5/1967	H	Sparkers	Asbestos	1.84	4	1.84	100%	1.84	2	W&C
2/20/1968	12/17/1970	S	Dispatch-prod. Contr.	Asbestos	2.37	1	0.6	50%	0.3	1	Armature
2/2/1971	3/13/1976	S	Dispatch-prod. Contr.	Asbestos	5	1	1.26	50%	0.63	1	Armature

Final assessment for this subject

Carcinogen	Intensity level	Duration exposure
Asbestos	1	1.18
Asbestos	2	1.84
Cr VI	1	1.03

Carcinogen	Intensity component	Duration component	Cumulative exposure
Asbestos	2	3.02	4.86
Chromium VI	1	1.03	1.03

**Phase II – Appendix 21**

Lung	Pair	Age	Smoking	Asbestos cumulative	Arsenic cumulative	Beryllium cumulative	Cadmium cumulative	Cr VI cumulative	Ni-Cr cumulative	Uranium cumulative
1	1		1							
0	1		2							
1	2		3							
0	2									

- Lung: 0 = indicates a control, 1 = indicates a case
- Pair: there are 195 pairs of matched case/control
- Age: age at death
- Smoking: 1 = smokers, 2 = non-smokers, 3 = no information

Application package submitted to OCR  
Time: March 2000

Meeting OCR (Director: Dr. Holowaty)  
Time: May 2000

Ethics approval  
Time: 2-3 months

Ministry of Health  
Canadian Institute of Health Information  
Time: 3-4 months

OCR - List of hospitals for each name  
Time: 1 month

Wait 6-8 months  
Before you can contact  
the hospitals

Saved 6-8 months

**Peterborough**

- Ethics Cte. Approval
- Health Record Services
- Collect data

**Kingston**

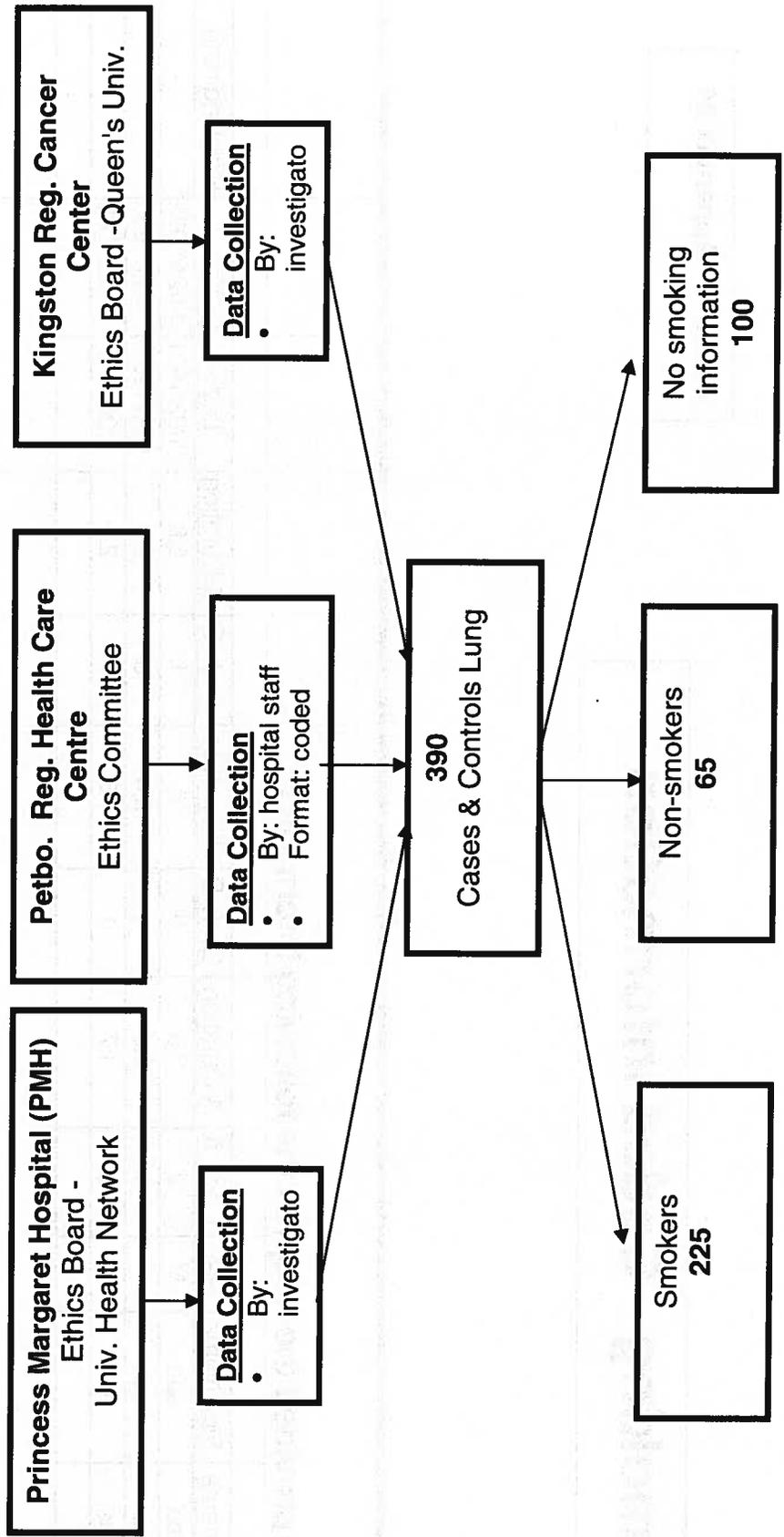
- Ethics Cte. Approval
- Health Record Services
- Collect data

**PMH**

- Ethics Cte. approval  
Time: 1 month (May)
- Cancer Registry Cte. Approval  
Time: 1 month (June)
- Health Record Services  
Time: 2 months (July-Aug)
- Collect data (Time: done in August)

# Smoking information - Data Sources

Phase II - Appendix 23



# Smoking - Coded information

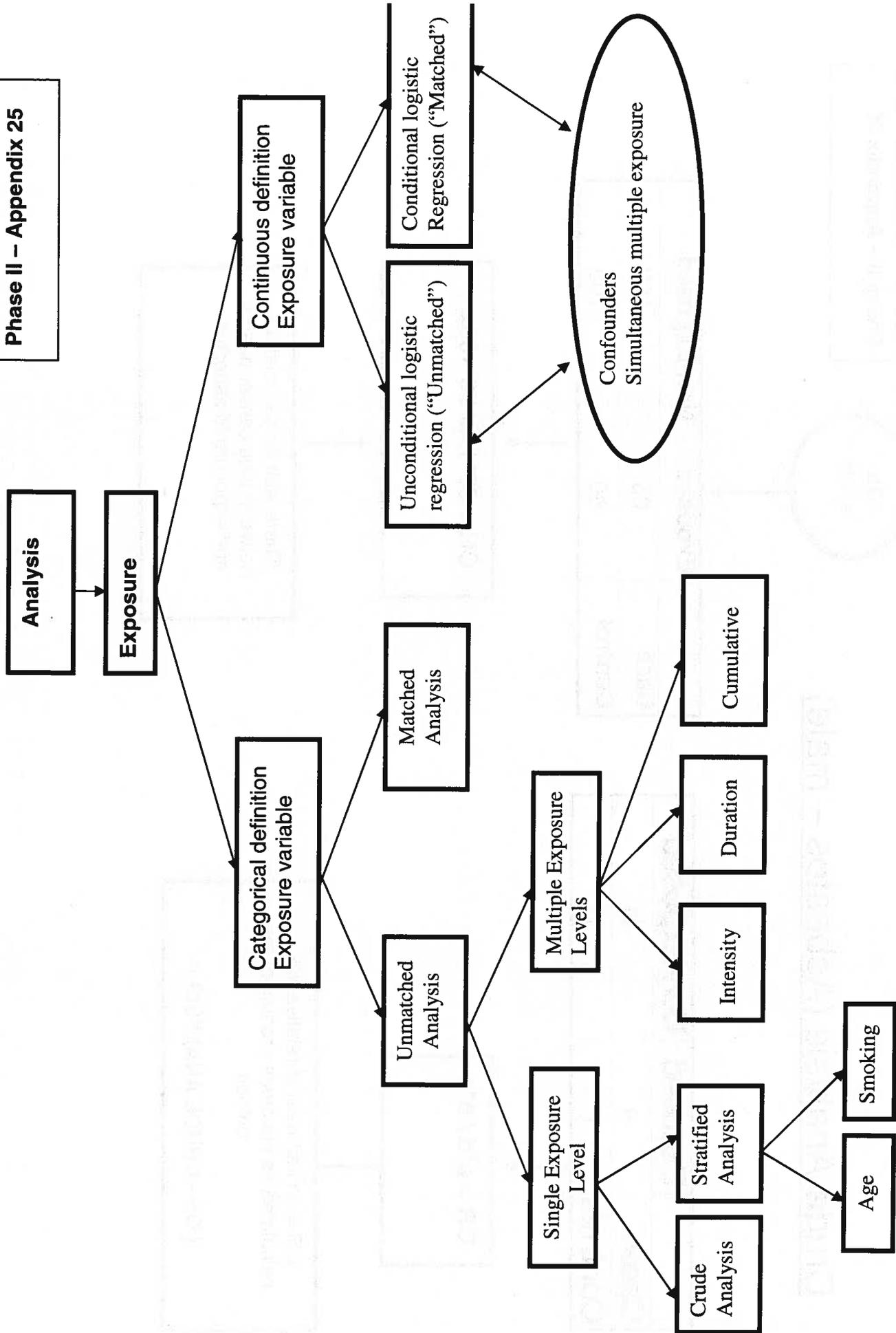
We provided the hospital the following information:

Surname	First Name	Sex	Code A	A-Duration	Code B	B-Duration	Code C	C-Duration	DOB	DOD	Smoking data
aaa	aaa	M	2	3.5	0	0	1	6.8	11/23/1921	11/24/1992	-
aaa	aaa	F	1	2.5	1	3.4	0	2.3	10/5/1905	10/7/1986	-
aaa	aaa	M	2	4.7	1	1.5	0	2.6	5/2/1910	7/12/1989	-

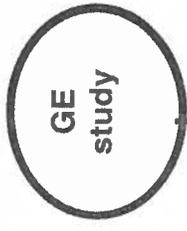
The hospital provided us the following information:

Surname	First Name	Sex	Code A	A-Duration	Code B	B-Duration	Code C	C-Duration	DOB	DOD	Smoking data
-	-	F	1	2.5	1	3.4	0	2.3	-	-	S, 20y, 2pack/day
-	-	M	2	4.7	1	1.5	0	2.6	-	-	Non-smoker
-	-	M	2	3.5	0	0	1	6.8	-	-	Ex-smoker





# Crude Analysis (Asbestos – male)



	Exposed	Non-Exposed
Cases	a	b
Controls	c	d

$$OR = a*d / b*c$$

OR = an estimate of relative risk, calculated as the cross-product of the entries  
( OR - CRUDE ANALYSIS )

	Exposed	Non-Exposed
Case	86	109
Control	86	109

$$OR = 86*109 / 86*109 = 1$$

There was no association between lung cancer deaths and exposure to asbestos.

Phase II – Appendix 27

Analysis Stratified by Age

Age group	Exposed	Non-Exp.	Exposed	Non-Exp.	OR
< 50					
50 - 59					
60 - 69					
70 - 79					
>80					

Analysis Stratified by Age and Smoking status

Age Group	Smoking status		
	Smokers	Non-smokers	No Information
< 50	cases		
	controls		
50 - 59	cases		
	controls		
60 - 69	cases		
	controls		
.....			

Age Group		No Exp.	Low	Medium	High	OR
< 50	cases					
	controls					
50 - 59	cases					
	controls					
60 - 69	cases					
	controls					
....						

Analysis  
Multiple exp. levels  
Stratified by Age



Matched Analysis



Cases	Controls	
	Exposed	Non-Exposed
Exposed	a	b
Non-Exposed	c	d

Matched OR=b/c

**Distribution of cases and controls according to intensity of exposure**

**Cut – points (Asbestos – male)**

	Highest	Highest	Lowest	Lowest
Score	# cases	# controls	# cases	# controls
Low ( 1 )	8	14	23	21
Medium ( 2,3 )	58	58	54	60
High ( 4 )	20	14	9	5

## Distribution of cases and controls according to duration of exposure

### Cut – points (Asbestos – male)

Duration (years)	# cases	# controls	Cut points
<1	47	59	Low (<1year)
1	10	0	
2	3	4	
3	2	6	
4	6	2	
5	2	3	
6	4	2	
7	3	0	
8	1	1	
9	1	1	
10	1	1	Medium (1-10)
11	1	0	
12	0	2	
13	2	0	
14	1	2	
15	0	1	
16	0	1	
17	0	1	
19	1	0	
26	1	0	High (>10)

**Distribution of cases and controls according to cumulative exposure  
Cut – points (Asbestos – male)**

Score	# cases	# controls	Cut points
< 1	37	41	Low (< 5)
1	8	10	
2	5	6	
3	4	4	
4	2	1	
5	3	2	Medium (5-10)
6	2	2	
7	3	2	
8	2	2	
9	1	0	
10	1	0	High (>10)
11	3	2	
12	1	0	
13	2	2	
14	1	3	
15	0	2	
16	1	0	
17	1	1	
18	1	1	
19	1	1	
20	1	1	
> 20	6	3	

## Intensity component of exposure – Asbestos (male)

Distribution of Cases and Controls by Age: Multiple exposure levels						
Age group	No Exposure	Low	Medium	High		
<60	Cases	17	1	10	2	
	Controls	17	4	7	5	
60-69	Cases	36	2	18	8	
	Controls	30	2	21	1	
70-79	Cases	43	3	23	8	
	Controls	50	5	22	8	
>80	Cases	13	2	7	2	
	Controls	18	3	8	0	
<b>All</b>	<b>Cases</b>	<b>109</b>	<b>10</b>	<b>64</b>	<b>12</b>	
	<b>Controls</b>	<b>109</b>	<b>23</b>	<b>56</b>	<b>7</b>	
Distrib. of Cases and Controls by Smoking: Multiple exposure levels						
Status	No Exposure	Low	Medium	High		
Smokers	Cases	71	8	42	16	
	Controls	47	6	27	8	
Non-smokers	Cases	9	0	2	2	
	Controls	28	2	20	2	
No information	Cases	29	0	14	2	
	Controls	34	6	11	4	



## Duration component of exposure – Asbestos (male)

Distribution of Cases and Controls by Age: Multiple exposure levels				
Age group	No Exposure	Low	Medium	High
<60	17	11	1	1
Controls	11	10	4	2
60-69	36	17	10	1
Controls	30	15	8	1
70-79	43	23	8	3
Controls	50	25	6	4
>80	13	6	3	2
Controls	18	9	1	1
<b>Cases</b>	<b>109</b>	<b>48</b>	<b>31</b>	<b>7</b>
<b>Controls</b>	<b>109</b>	<b>55</b>	<b>23</b>	<b>8</b>
Distrib. of Cases and Controls by Smoking: Multiple exposure levels				
Status	No Exposure	Low	Medium	High
Smokers	71	43	18	5
Controls	47	30	5	6
Non-smokers	9	4	0	0
Controls	28	14	9	1
No information	29	10	4	2
Controls	34	15	5	1

## Cumulative exposure – Asbestos (male)

Distrib. of Cases and Controls by Age: Multiple exposure levels					
Age group	No Exposure	Low	Medium	High	
<60	17	11	1	1	
Controls	11	12	0	4	
60-69	36	17	6	5	
Controls	30	16	4	4	
70-79	43	23	2	9	
Controls	50	25	4	6	
>80	13	5	2	4	
Controls	18	9	0	2	
<b>All</b>	<b>109</b>	<b>56</b>	<b>11</b>	<b>19</b>	
<b>Controls</b>	<b>109</b>	<b>62</b>	<b>8</b>	<b>16</b>	
Distrib. of Cases/Controls by Smoking: Multiple exposure levels					
Status	No Exposure	Low	Medium	High	
Smokers	71	43	10	13	
Controls	47	31	4	6	
Non-smokers	9	3	1	0	
Controls	28	14	2	8	
No information	29	10	0	6	
Controls	34	17	2	2	

<b>Crude Analysis (Ever/Never Asbestos)</b>					
	Asb. (+)	Asb. (-)	Total		
FINAL					
Cases	86	109	195		
Controls	86	109	195		
Total	172	218	N=390		
<b>Exposure Distribution of Cases and Controls by Age</b>					
	Cases		Controls		
Age group	Asb. (+)	Asb. (-)	Asb. (+)	Asb. (-)	Odds ratio
<60	13	17	16	11	0.53
60-69	28	36	24	30	0.97
70-79	34	43	35	50	1.13
>80	11	13	11	18	1.38
Total	86	109	86	109	
<b>Exposure Distribution of Cases and Controls by Smoking</b>					
	Cases		Controls		
Group	Asb. (+)	Asb. (-)	Asb. (+)	Asb. (-)	Odds ratio
Smokers	66	71	41	47	1.07
Non-Smokers	4	9	24	28	0.52
No Information	16	29	21	34	0.89
Total	86	109	86	109	

**Results - Intensity component of exposure**

**Multiple levels of exposure – Asbestos (male)**

	Crude	Crude	Stratified Age	Stratified Age	Stratified Age	Stratified Smoking	Stratified Smoking
	OR	95% limits	OR	95% limits	95% limits	OR	95% limits
<b>No Exp.</b>	1.00	-	1.00	-	-	1.00	-
<b>Low</b>	0.57	(0.23 to 1.41)	0.63	(0.29 to 1.36)	(0.29 to 1.36)	0.46	(0.21 to 1.02)
<b>Medium</b>	1.00	(0.64 to 1.57)	1.05	(0.71 to 1.55)	(0.71 to 1.55)	1.00	(0.66 to 1.51)
<b>High</b>	1.43	(0.69 to 2.95)	1.54	(0.79 to 3.0)	(0.79 to 3.0)	1.25	(0.64 to 2.43)
<b>Mantel-extension test</b>	0.5 < p < 0.75		0.5 < p < 0.75			0.75 < p < 0.9	
<b>Interpretation</b>	No trend		No trend			No trend	

## Results - Duration component of exposure

## Multiple levels of exposure – Asbestos (male)

	Crude OR	Crude 95% limits	Stratified Age OR	Stratified Age 95% limits	Stratified Smoking OR	Stratified Smoking 95% limits
No Exp.	1.00	-	1.00	-	1.00	-
Low	0.97	(0.62 to 1.52)	0.95	(0.64 to 1.4)	0.90	(0.59 to 1.35)
Medium	1.16	(0.59 to 2.25)	1.11	(0.62 to 2.0)	1.25	(0.64 to 2.44)
High	0.88	(0.31 to 2.47)	0.90	(0.37 to 2.17)	0.72	(0.29 to 1.77)
Mantel-extension test	0.9 < p < 0.95		0.95 < p < 0.975		0.75 < p < 0.9	
Interpretation	No trend		No trend		No trend	

**Results - Cumulative exposure**

**Multiple levels of exposure – Asbestos (male)**

	Crude OR	Crude 95% limits	Stratified Age OR	Stratified Age 95% limits	Stratified Smoking OR	Stratified Smoking 95% limits
No Exp.	1.00	-	1.00	-	1.00	-
Low	0.90	0.57 - 1.41	0.88	0.60 - 1.31	0.82	0.56 - 1.22
Medium	1.38	0.54 - 3.52	1.33	0.56 - 3.16	1.22	0.51 - 2.95
High	1.19	0.58 - 2.43	1.19	0.64 - 2.24	1.33	0.67 - 2.64
Mantel-extension test	0.5<p<0.75		0.5<p<0.75		0.5<p<0.75	
Interpretation	No trend		No trend		No trend	







**Response # 1** (dated: February 3<sup>rd</sup>, 2003) indicated our plans to handle for the 4 issues raised on your e-mail dated Jan. 27,2003

**Response # 2** We included changes in the text of the final report dated: February 7, 2003, as highlighted in the enclosed material (See Appendix A), which addressed 2 of the 4 issues; we thought that the other 2 would have been answered at a face-to-face meeting with the JH&S committees. This response will therefore cover all items raised.

With regards to the query to look at the issues by department, the study provided to us the ability to look at any department for exposure to human carcinogens. We showed you in detail the work done in the Wire & Cable Department, and the carcinogen map showed that the materials of concern, except for asbestos, were not present in the Armature Department.

Your Sept.13, 1995 letter raised the following concerns:

<b>Your concern</b>	<b>Response</b>
radiation	We showed that the only potential exposure was in building 21. The exposure data revealed extremely low levels that were well within the AECB limits. These data were shared previously during the industrial hygiene briefing.
asbestos	The final report included considerable coverage of asbestos use and potential exposure. The hygiene data revealed minimal exposure and the case-control analysis showed no associations.
benzene	The use of benzene was limited. Benzene is associated with leukemia.

	The PCMR analysis for leukemia was 1.18 with a 95% confidence limits (0.89-1.56), not statistically significant, thus suggesting no need for further analysis.
PCBs	This material is not a confirmed human carcinogen and its primary association is with the liver. The PCMR analysis did not show any excesses, therefore there was no need to look further.
MOCA	This material was used to a very limited extent in one small area. It is associated with bladder cancer; again, the PCMR analysis did not point us to look further at this organ site.
epoxy resin	Not a known human carcinogen
epichlorohydrin	Not used as a material, but as a co-polymer in the epoxy therefore not easily airborne. Not a known human carcinogen.

The CAW proposed that we evaluate additional cancer sites listed in paragraph 3 of its November 12, 1999 letter. However, we recommend against evaluating those sites as the likelihood of finding a meaningful and measurable excess is limited. We say this because of the wide confidence intervals found for each site-specific PCMR and the small numbers in some groups. Further investigation would be unlikely to yield information that could be further evaluated (i.e. a specific exposure problem). The specific CAW requests are addressed below in Appendix B.

Finally, the CAW requested an investigation of possible synergistic effects. This was undertaken as an appropriate step in the case-control analysis in addition to the single chemicals. No synergism was found.

**Appendix A.** Changes (in red) included in the final report

**2.1 Study Design and Data Analysis**

Advantages PCMR design	Disadvantages PCMR design
<ul style="list-style-type: none"> <li>• Most suitably used to explore for disease excesses and deficits on preliminary analysis of the available data.</li> </ul>	<ul style="list-style-type: none"> <li>• Validity depends on whether the deaths included are representative of all deaths that would be identified if complete follow-up of the full cohort.</li> </ul>
<ul style="list-style-type: none"> <li>• Good approximations to SMRs (Standard Mortality Rates) from cohort studies when all-causes combined SMR=1 (observed=expected).</li> </ul>	<ul style="list-style-type: none"> <li>• Does not directly measure the risk of dying from (e.g.) lung cancer, but the difference in mortality from other causes of death.</li> </ul>
<ul style="list-style-type: none"> <li>• Greater confidence for PCMR, since the Healthy Worker Effect less affects cancer mortality.</li> </ul>	

**3.6. Analysis**

The analysis didn't take latency into account. Latency is defined as the time interval since the person started to be exposed until he/she first developed the disease. With our collected information we can find the date when the exposure started, but we do not know when the cancer was diagnosed for the first time. The date of death cannot be used in order to calculate latency. As a consequence, more cases were included in the analysis.

**4. Conclusions**

- Using an odds ratio analysis adjusted and not-adjusted for age and smoking status (Cochran-Mantel-Haenszel, unconditional and conditional logistic regression), there was no association between lung cancer deaths and any of the carcinogens.

- There was no statistically significant trend of increasing risk of death by lung cancer with cumulative exposure, exposure intensity or duration.
- There was no increase in risk of lung cancer death with multiple carcinogens exposure.
- The only significant association for lung cancer arose when smoking was considered.
- From the Industrial Hygiene data, the exposures were predominantly within the TWA limits at the time.
- From the interviews and information collection, there was generally used personal protective equipment, and local and general ventilation in areas where carcinogens were used. In our analyses, we weighted the exposure rating to consider the level of use of the controls devices.

## **Appendix B. Response to CAW Requests Regarding Specific Sites to Evaluate**

### **Male Sites Requested:**

Digestive system; liver/gallbladder; bone; skin; prostate; kidney; brain-CNS; lymphopoietic; leukemia; lymphatic tissue

- Digestive system: there was not sufficient excess of digestive cancers to allow an analysis.
- Bone cancer: because there was only a single excess case, it does not make sense to analyze further especially since no known bone carcinogen was present.
- Kidney cancer: the kidney site was not in excess, PCMR = 0.98 (95% CI 0.53-1.82)
- Brain cancer: The brain cancer site was not in excess, PCMR = 0.73 (95% CI 0.37-1.43).
- The PCMRs of the remaining sites included on the CAW list are all very close to 1.0 and further evaluation would be unlikely to detect significant

excesses even if they existed because of limited statistical power given the background rates of the disease and exposures.

**Female Sites Requested:**

Digestive system, skin, breast, brain-CNS, leukemia, lymphatic tissue

- Additional studies were requested for 6 sites, of which 4 sites (skin, brain-CNS, leukemia, lymphatic tissue) had 2 or fewer cases. No statistical evaluation can be done because of small numbers in the categories.
- Digestive system: the digestive system was not in excess, PCMR = 0.79 (95% CI 0.47-1.33).
- Breast: there was only a minimal excess, PCMR = 1.1 (95% CI 0.66-1.84) based on 11 cases where 10 were expected. The additional evaluation is unlikely to detect an increased risk. Besides there were no known carcinogens of the breast.



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Subject: Ge peterborough report

Date: Monday January 27 2003 09:25

From: Ted Haines <hainest@mcmaster.ca>

To: "Hosein, Roland (CORP)" <roland.hosein@corporate.ge.com>, Daniela Ghiculete <daniela.ghiculete@utoronto.ca>, "Kreso G. Botic" <georgeb@caw.ca>, "caw524@ptbo.igs.net" <caw524@ptbo.igs.net>

Roland, Daniela, George and Keith

I'm sorry for the delay in sending you comments; things have been very busy.

Here I make my main observations; these are close to the ones I made at the joint meeting I attended in the fall. Additional, more minor ones could be offered but I will confine myself for the moment to the points below.

First, congratulations on and thanks for your careful and diligent work on the report.

With regard to the purpose/concern at the outset, I don't think it's accurate to describe it as non-specific.

I recall seeing a list of illnesses prepared by the local which included certain cancers that were somewhat numerous and also certain exposure categories, incidents or processes, some of which were historical. These represented hypotheses, although they may not have been very explicitly formulated. I think that additional reference to the specificities in the original concerns should be made.

We all said at the beginning that the PMR approach, focussing on deaths, had limitations in that the entire working population wasn't followed and in that non-fatal cancers would be missed.

Also, I don't believe that the analysis has taken latency into account; cancers that occur within, say, 10 years of first exposure are not likely to be work-related.

The main issue for me is whether the workers feel that their concerns have been addressed. I think we can say that some of the cancers of concern have been looked at very closely with good methods with respect to known carcinogens. I don't know whether they feel satisfied that their original concerns about exposure categories, incidents or processes have been dealt with.

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Subject: English

Date: / /

Section: / /

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Answer: / /

Explanation: / /

Conclusion: / /

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Principal: / /

Headmaster: / /

Classmate: / /

Parent: / /

