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ABSTRACT

An empirical approach was taken to examine and test a variety of hypotheses that had been presented or postulated in the literature concerning mercury biogeochemistry in lakes. These hypotheses included: 1) mercury contamination in fish increases with increasing sulphate deposition; 2) mercury exposure in persons eating fish increases with increasing sulphate deposition; 3) mercury contamination in fish is unrelated to natural (non-anthropogenic) Hg sources. These hypotheses were all shown to be false for Ontario lakes, predominantly of the Canadian Shield.

Also examined was the spatial association of lake water chemistry with sulphate deposition across Ontario. It was determined that morphometric characteristics of the lakes, as well as watershed buffering capacity, together explained more of the spatial variation in lake water chemistry than did patterns of sulphate deposition across the province. Once these confounding influences were removed, the water chemistry variables most closely reflecting patterns of sulphate deposition were dissolved organic carbon (DOC) and sulphate ion concentration ($[\text{SO}_4^{2-}]$), not pH or alkalinity. Aluminum ion concentration ($[\text{Al}^+]$), which is postulated to increase in lakes as a result of acidic deposition, had no discernible relationship to sulphate deposition.

Based on the results of empirical analyses, including path analysis (causal modelling), a complex hypothesis explaining the interrelationships of precipitation, water chemistry, geochemistry, and fish mercury contamination was postulated. Of particular note concerning this model were the following: 1) fish mercury and human mercury exposure decrease as sulphate deposition increases; 2) DOC, not pH or alkalinity, is the water chemistry variable most significantly related to sulphate deposition; 3) DOC, not pH, is the water chemistry variable most significantly associated with fish mercury contamination; 4) causal modelling provided empirical evidence supporting the simultaneous influence of 3 different mechanisms postulated in the literature to control or influence mercury accumulation in freshwater fish.

RÉSUMÉ

L'auteur adopte une approche empirique pour examiner et vérifier diverses hypothèses, présentées ou formulées dans des publications, sur la biogéochimie du mercure dans les milieux lacustres. Ces hypothèses sont les suivantes : 1) la contamination du poisson par le mercure augmente en fonction du dépôt de sulfate; 2) l'exposition au mercure des personnes qui consomment du poisson augmente en fonction du dépôt de sulfate; et 3) il n'y a pas de rapport entre la contamination du poisson par le mercure et les sources naturelles (non anthropiques) de mercure. L'auteur démontre que ces hypothèses ne s'appliquent pas dans les lacs de l'Ontario, en particulier ceux du Bouclier canadien.

L'auteur a aussi examiné la relation spatiale entre la composition chimique de l'eau des lacs de l'Ontario et le dépôt de sulfate. Il a déterminé que les caractéristiques morphométriques de ces lacs, conjointement avec le pouvoir tampon des bassins versants, expliquent un plus grand pourcentage de la variation spatiale de la composition chimique des eaux lacustres que les régimes de dépôt de sulfate. Une fois ces sources d'erreur éliminées, il a établi que le carbone organique dissous (COD) et la teneur en ions sulfate ($[\text{SO}_4^{2-}]$) sont les variables reflétant le plus étroitement les régimes de dépôt de sulfate, et non le pH ou

l'alcalinité. En outre, il a conclu qu'il n'existe pas de relation évidente entre le dépôt de sulfate et la teneur en ions aluminium ($[Al^+]$) qui, selon une hypothèse, augmente par suite de retombées acides.

D'après les résultats des analyses empiriques, y compris une analyse des pistes causales (modélisation causale), l'auteur a formulé une hypothèse complexe expliquant les interrelations entre les précipitations, la composition chimique de l'eau, la géochimie et la contamination des poissons par le mercure. Le modèle qu'il a élaboré révèle les particularités suivantes : 1) le degré d'exposition des poissons et des personnes au mercure diminue en fonction d'une augmentation du dépôt de sulfate; 2) le COD, et non le pH ou l'alcalinité, est la caractéristique chimique de l'eau qui est liée de la façon la plus significative au dépôt de sulfate; 3) le COD, et non le pH, est la caractéristique chimique de l'eau associée de la façon la plus significative à la contamination des poissons par le mercure; 4) la modélisation causale fournit des preuves empiriques étayant l'existence de l'influence simultanée de trois différents mécanismes qui, d'après des données publiées, contrôlent l'accumulation du mercure chez les poissons dulçaquicoles ou ont une incidence sur celle-ci.

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The staff of the Map Library of the University of Ottawa deserve honourable mention for the assistance they provided. I never knew how much use a biologist could make of maps and the map library. As it turned out, I spent more time there than anywhere else for over a 9 or 10 month period of my studies.

Finally I want to extend special thanks to the support provided by my wife Annabelle and our three children - Luke, Amy and Todd. Despite my numerous periods of panic, tension, temper and depression, they managed to carry me through. All I can say is, "It's finally over!".

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1.0 GENERAL INTRODUCTION

Mercury (Hg) is perhaps the most widespread contaminant of the freshwater aquatic environment. Low levels of Hg occur naturally in air, water, soil, sediments and biota (Nriagu 1979). Natural geologic deposits or other geologic factors result in the 'natural' contamination of freshwater aquatic sediments (Friske 1985a,b) and fish (Loukola-Ruskeeniemi 1990; Varekamp and Waibel 1987). Increased (over background) contamination of fish has been attributed to anthropogenic activities such as: 1) direct industrial discharges from pulp and paper mills and chloralkali plants (Rudd et al. 1983); 2) impoundment of water for hydro-electric power generation (Bodaly et al. 1984); 3) atmospheric emissions of Hg from combustion of fossil fuels and smelting of sulphide ores, followed by atmospheric transport and deposition (Swain et al. 1992; Rada et al. 1989; Evans 1986; Meger 1986; Schindler 1988); and 4) increased Hg methylation/bioavailability/bioaccumulation resulting from altered water chemistry following lake acidification (Gilmour et al. 1992; Raloff 1991; Gilmour and Henry 1991; Winfrey and Rudd 1990; Jernelov 1986; Goyer et al. 1985; Beijer and Jernelov 1979).

An extensive body of literature has evolved on Hg in aquatic environments, and a variety of hypotheses have emerged regarding the factors that affect Hg accumulation in fish

(including Huckabee et al. 1979; Jernelov 1986; Richman et al. 1988; Winfrey and Rudd 1990; Gilmour et al. 1992; Weber 1993). However, there has been no resolution as to which abiotic factors are the most significant in terms of controlling or influencing Hg concentrations in aquatic organisms.

In Ontario, a unique opportunity existed to test a variety of these hypotheses. Extensive Hg-related research had been undertaken in the province over the past 2 to 3 decades because of the environmental distribution, physicochemical properties and human health concerns regarding this element. During the 1970's and 1980's, various government agencies undertook extensive monitoring programs to collect data on Hg in soils, lake sediments, freshwater fish and people. As well, relevant data were collected on lake water chemistry, watershed buffering capacity and sulphate deposition across the province.

The aim of this thesis was to integrate these separate data sets and to test empirically a variety of hypotheses and assumptions regarding the influence of abiotic factors on the accumulation of Hg in fish, and subsequent human Hg exposure via fish consumption. The quantification of the relative significance of several key abiotic factors in the accumulation of Hg in fish was also undertaken.

The analyses reported here have been conducted on extant data. Emphasis was placed on evaluating the empirical evidence supporting or refuting particular hypotheses. Controlled field or laboratory experiments to elucidate mechanisms were not undertaken, but for good reason. For the past decade, a variety of mechanisms underlying Hg accumulation in fish have been experimentally demonstrated. These include mechanisms for Hg absorption (Rodgers and Beamish 1983), biomethylation (Xun et al. 1987; Miskimmin et al. 1992; Gilmour et al. 1992), abiotic methylation (reviewed by Weber 1993), and Hg transport to, and binding in, lakes (Mierle and Ingram 1991; Lee and Iverfeldt 1991). The identification of these mechanisms has lead to numerous hypotheses about how acidic deposition may influence Hg levels in fish, but none of these hypotheses have been directly tested. The question I then hope to address is: To what extent do these various mechanisms explain observed trends in fish Hg accumulation, and human Hg exposure via fish consumption?

The effective management of Hg levels in Ontario's freshwater fisheries resources can not be achieved without a thorough integration and evaluation of these existing data and information. Management options implemented to limit Hg accumulation in fish, based on false assumptions, or which mitigate factors of low relative significance to the overall

problem, will be ineffective. The mechanisms underlying the various competing and/or antagonistic relationships investigated in this thesis have largely been demonstrated experimentally. However, their overall significance to the spatial variation in fish Hg contamination (and subsequent human Hg exposure via fish consumption) can not be defined because the relative importance of the effects these mechanisms underlie have not been defined. Therefore, it is of practical importance to assess the relative significance of the various interactions between precipitation chemistry, geochemistry, lake water chemistry and fish Hg contamination, in order to determine where intervention might best achieve a net reduction in fish Hg contamination, and thereby reduce human Hg exposure.

2.0 GENERAL REVIEW OF LITERATURE

Each chapter in this thesis has been prepared to stand alone. Each includes a brief abstract, a review of the relevant literature, methods, results and discussion. This section reviews the literature upon which the generalized model (Figure 2.1) was constructed but relevant parts of it will be repeated within each subsequent Chapter.

2.1 Human Exposure to Mercury

The consumption of fish is the primary, non-occupational source of exposure to methylmercury (MeHg) (WHO 1990). Annual per capita 'apparent' fish consumption (fish + shellfish, including fresh, frozen, cured and canned) in Canada was determined to be approximately 16 g/day, based on data relating to the disappearance of domestic fish supplies (CCAC 1977). A national dietary survey (HWC 1977; Conacher et al. 1989) estimated the national average rate of consumption of all fish and shellfish in Canada to be about 11 g/person/day. The national average rate of consumption of freshwater fish was much lower at only 1.2 g/person/day (Conacher et al. 1989).

The rate of fish consumption by Canada's Native peoples is greater than the national average (HWC 1977), although this has not been extensively quantified. Many communities of native peoples, particularly isolated ones, subsist on fish as a major component of their diet (HWC 1985; Wheatley and Wheatley 1981; Kinloch et al. 1992; Kuhnlein 1984; Lawn 1989; Lutra Assoc. 1989; Szathmary et al. 1987; Berkes 1990; Hopper and Power 1991). Therefore, native Canadians dependant on subsistence fishing for nutrition are the Canadian subpopulation at greatest risk from MeHg exposure.

2.2 Sources of Hg to Freshwater Aquatic Environments

A number of authors have detected Hg in precipitation (Mierle 1990; Sorensen et al. 1990; Bloom and Watras 1989; Johnson 1987; Scudato et al. 1987; Glass et al. 1986; Johnson et al. 1986; Dillon et al. 1978). Atmospheric deposition of Hg onto lake watersheds appears to account for virtually all the Hg entering remote lakes (Winfrey and Rudd 1990; Mierle 1990; Sorensen et al. 1990).

Elevated Hg concentrations in post-industrially deposited lake sediments suggest that anthropogenic activities (smelting, fossil fuel combustion) are a significant source of Hg to remote lakes, via atmospheric transport (Swain et al. 1992; Rada et al. 1989; Johnson 1987; Evans 1986; Johnson et al. 1986; Meger 1986; Ouellet and Jones 1983; Heit et al. 1981). Anthropogenic Hg originates from the same sources as the precursors of acid rain (fossil fuel combustion, smelting of sulphide ores (Schindler 1988; Nriagu 1979)). The rate of Hg deposition has been shown to parallel the rate of acidic deposition (Glass et al. 1991; Nater and Grigal 1992).

Lacustrine Hg contamination also arises from natural sources such as geologic 'hot spots' (Varekamp and Waibel 1987; Loukola-Ruskeeniemi 1990). Lake sediment strata deep enough to pre-date anthropogenic (industrial) inputs demonstrate significant spatial variation in Hg contamination

(Coker and Shilts 1979; Friske 1985a,b). In Ontario lakes on the Canadian Shield, non-anthropogenic Hg could arise from two possible independent sources: bedrock and soil. Ontario generally lacks geological formations containing economically significant deposits of cinnabar (HgS) (Jonasson and Boyle 1972). However, the bedrock in the province is rich in other sulphide ore deposits (CuS, PbS and ZnS) which also contain significant amounts of HgS (Jonasson and Boyle 1979; Coker and Nichol 1975). On the Canadian Shield, the soil overburden does not originate from the underlying bedrock, but was transported from the north-east and deposited during the last ice age (Coker and DiLabio 1987). Therefore, the soil is unrelated to the local bedrock.

2.3 Factors Which Influence Fish Hg Contamination

The single best predictor of the concentration of Hg in fish is fish length, due to its strong association with exposure duration (i.e. fish age) (Huckabee et al. 1979). As a result, most interlake comparisons of fish Hg contamination first standardize fish Hg concentration for fish length (McMurtry et al. 1989; Wren et al. 1991; Sorensen et al. 1990, for example).

Fish Hg contamination is also a (negative) function of lake productivity and/or biomass because of biomass dilution effects (Hakanson 1980; Richman et al. 1988). For interlake

comparisons, this may necessitate the standardization for factors which influence lake biomass or productivity, such as lake size and depth (Rawson 1952; Ryder 1965), lake water chemistry (Ryder 1965; Conroy and Keller 1976), or local geochemistry (Conroy and Keller 1976; Ryder 1964). These latter factors - lake morphometry, water chemistry and geochemistry - may also influence fish Hg accumulation in other ways. A number of water chemistry parameters have been linked directly to fish Hg accumulation (discussed below). Watershed geochemistry significantly influences lake water chemistry (Shilts and Kettles 1989), as does lake morphometry (Schindler 1971; Rasmussen et al. 1989).

pH has been the most studied water quality variable for its influence on Hg levels in fish. Almost all investigations have demonstrated higher fish MeHg contamination in fish of lakes with lower pH. The primary hypothesis for this effect is the increased net rate of MeHg production at lower pH (Richman et al 1988). Although a number of competing water column and sediment processes may be involved, net MeHg production appears to increase with decreasing pH (see reviews by Gilmour and Henry 1991; Winfrey and Rudd 1990; and Beijer and Jernelov 1979).

Alkalinity, acid neutralizing capacity (ANC) (which is equivalent to alkalinity for most lakes (Dillon et al. 1984)),

as well as measures of water hardness, are directly related to pH. As pH decreases (more acidic), alkalinity, ANC, etc. also decrease (Neary et al. 1990; Jefferies et al. 1986; Dillon et al. 1984; Eilers et al 1983). Greater Hg contamination in fish is also associated with lakes having lower alkalinity (Scheider et al. 1979). There are two proposed mechanisms for this relationship. First, lower alkalinity may reflect lower pH and subsequent increased net Hg biomethylation (as discussed above). Alternately, Ca is a major component of alkalinity and ANC (as CaCO₃) (Dillon et al. 1984; NRCC 1981). Hg uptake by rainbow trout (Salmo gairdneri) was found to be greater in soft water than in hard water (Rodgers and Beamish 1983), Ca possibly regulating the permeability of fish gills to MeHg.

Sulphate concentration in lake water may also be associated with increased Hg biomethylation and thereby increased fish Hg contamination (Gilmour et al. 1992). Sulphate reducing anaerobic bacteria in lake sediments co-metabolize inorganic Hg to MeHg. Both bacterial metabolism and Hg methylation increase as sulphate concentration increases in water overlying sediments (Gilmour et al. 1992).

Hg contamination of fish from drainage lakes (lakes recharged by surface runoff) has a positive association with the organic carbon content of water (dissolved organic carbon,

total organic carbon, colour) (Wren et al. 1991; McMurtry et al. 1989). Both inorganic and MeHg are transported to drainage lakes as a complex with organic carbon (Mierle and Ingram 1991; Lee and Iverfeldt 1991). If the flow of DOC from the watershed were reduced, then the bioavailability of MeHg to fish may be reduced. Also, DOC may play an important role in the abiotic methylation of inorganic Hg (Weber 1993). Reduced lake DOC would therefore result in a reduced rate of abiotic Hg methylation in the water column and lower relative Hg contamination in fish. Both of these proposed mechanisms would lead to a positive association between lake DOC and fish Hg levels.

However, DOC has been shown to inhibit the biomethylation of inorganic Hg in controlled experiments with no constant influx of new Hg (Miskimmin et al. 1992). In seepage lakes, which are not inundated with DOC-bound Hg from the watershed, lower levels of DOC in the water column may lead to an increase in the relative rate of Hg biomethylation with subsequent increased bioavailability for uptake by fish. This would explain the observed negative association between fish Hg and lake DOC levels in seepage lakes (Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990).

2.4 The Interaction of Water Chemistry Parameters

Changes or fluctuations in water chemistry variables do not happen independently of one another. For example, pH and alkalinity are strongly interrelated (Eilers et al. 1983). Therefore, a significant change in one implies some change in the other.

In lakes receiving sulphate (acidic) deposition, levels of SO_4^{2-} increase as the rate of deposition, and degree of lake acidification, increase (Sullivan et al. 1988; Neary et al. 1990; see also Howells 1990). Therefore, declining pH and declining alkalinity will be associated with a concomitant increase in SO_4^{2-} in these lakes.

The solubility of DOC is pH-dependent (Thurman 1985), declining at lower pH. DOC also has some acid neutralizing capacity (Schindler et al. 1992). Therefore, the concentration of DOC in water will decline with increasing acidification. This will be slower in lakes with higher relative alkalinity, due to the buffering effects of alkalinity, and to competition between DOC and alkalinity for ANC. Finally, DOC directly influences pH at relatively high DOC levels (i.e. dystrophic lakes) (Thurman 1985).

It has been hypothesized that Al may control DOC levels in acidified lakes through the formation and precipitation of

DOC-Al complexes (Effler et al. 1985). However, Schindler et al. (1992) have suggested that the postulated link between DOC and Al may be incorrect. Instead, it may be that [Al] simply reflects the concentration of organic ligands (such as humic and fulvic acids) with which it tends to complex in natural waters (Howells 1990).

2.5 Abiotic Factors Influencing Lake Water Chemistry

As previously mentioned, lakes receiving sulphate (acidic) deposition will reflect increased levels of SO_4^{2-} and lower pH and alkalinity (Sullivan et al. 1988; Neary et al. 1990; Neary and Dillon 1988; Howells 1990). Therefore, declining pH and declining alkalinity will be associated with a concomitant increase in SO_4^{2-} in these lakes. Aluminum concentration ([Al]) is also expected to increase with increasing sulphate deposition (NRCC 1981). It appears that sulphate deposition also affects lake water concentrations of DOC ([DOC]). Changes in lake [DOC] have been reported after experimental acidification and liming of lakes (Schindler et al. 1992; Molot et al. 1990; Bukaveckas and Driscoll 1991). Neary et al. (1990) showed that average [DOC] was lower in lakes located in regions of Ontario with higher sulphate deposition, the relationship being affected by lake size. Schindler et al. (1992) and de Haan (1992) both postulated that lower DOC in lakes receiving acidic deposition may, in

part, be caused by reduced DOC levels in acidified runoff, due to the pH dependence of DOC solubility in that runoff.

Lake water chemistry is also dependent on lake and watershed morphometry. DOC depends on lake and watershed morphometry since drainage basin runoff is the primary source of DOC to Canadian Shield lakes (Schindler et al. 1992; Rasmussen et al. 1989). Alkalinity, pH, [Al] and [SO₄²⁻] are also expected to vary with morphometric factors which influence lake retention times and flushing rates, dilution factors, and chemical loading rates from the drainage basin (Schindler 1971).

Another factor which will influence lake water chemistry is local geochemistry, particularly watershed buffering capacity. Lake water chemistry, including pH and alkalinity, is known to vary with the chemical nature of both watershed soils and bedrock (Ryder 1964; Conroy and Keller 1976; Kettles et al. 1991; Shilts and Kettles 1989). The impact of acidic precipitation on lake water chemistry will be off set by a highly buffered watershed compared to a poorly buffered one (NRCC 1981). The degree of leaching of Al by acidic deposition into lakes is also expected to be greater in watersheds with lower buffering capacity (NRCC 1981; Cronan and Scholfield 1979).

2.6 Summary

Human Hg exposure is believed to occur predominantly via consumption of freshwater fish. Fish Hg contamination is a function of atmospheric deposition of Hg from both natural and anthropogenic sources, combined with the influences of lake water chemistry on Hg transport, methylation and bioavailability to fish. Water chemistry parameters are, in turn, influenced by acidic deposition, lake morphometry and watershed geochemistry (predominantly buffering capacity), as well as other water chemistry parameters with which they are interdependent. These various relationships are summarized in Figure 2.1.

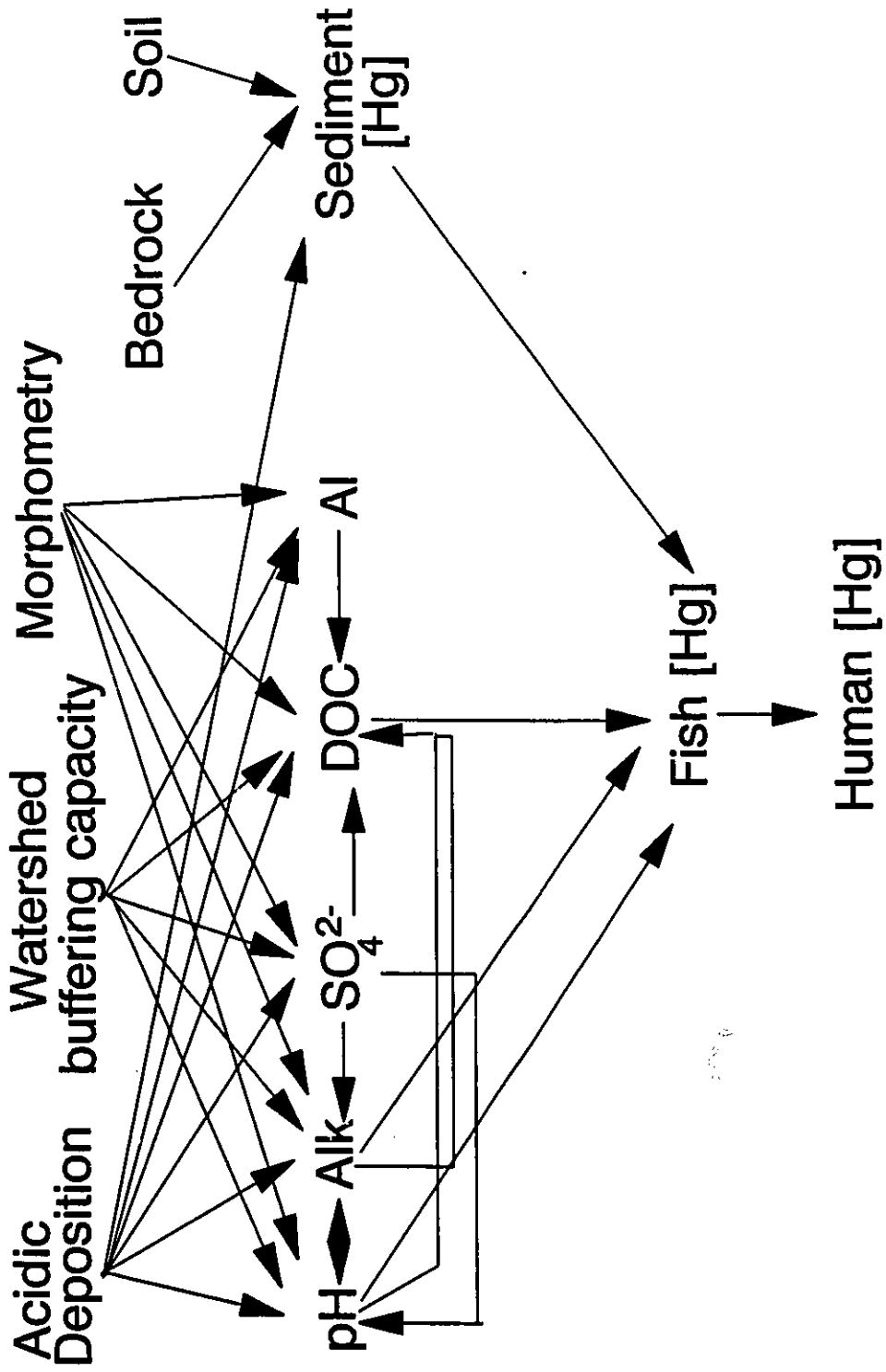
Figure 2.1 depicts the model used throughout this thesis as a basis for the derivation and testing of hypotheses. Whereas the studies upon which components of this model were based were conducted under controlled conditions, in isolation of the complex interactions which the preceding review makes apparent, this thesis integrates a wide variety of data to enable the analysis of this more complex and realistic model for Hg bioaccumulation in fish and humans.

Using empirical methods, and discussing hypothesized mechanisms presented in the extant literature, I will address the following primary questions:

1. How is lake water chemistry associated with lake morphometry and watershed buffering capacity across Ontario?
2. How is lake water chemistry associated with sulphate deposition across Ontario?
3. How are mercury levels in freshwater fish associated with sulphate deposition across Ontario?
4. How is human mercury exposure associated with sulphate deposition across Ontario?
5. How are mercury levels in freshwater fish associated with watershed geology and geochemistry across Ontario?

A variety of secondary hypotheses and analyses will be addressed in order to identify and eliminate factors which have not been controlled in previous studies of this type, and which may confound the primary associations of interest. Moreover, path analysis will be employed to quantify the relative contributions of all identified factors which appear to influence these primary associations.

Figure 2.1: Hypothetical path model depicting the interrelationships of precipitation chemistry, water chemistry, watershed geochemistry, and lake and watershed morphometrics with mercury contamination in fish and humans. Alk = lake alkalinity; $[\text{SO}_4^{2-}]$ = lake sulphate ion concentration; [DOC] = lake dissolved organic carbon concentration; [Al] = lake aluminum ion concentration; [Hg] = mercury concentration. Arrows depict hypothesized direct causal associations between predictor (independent) variables (at tail of arrow) and criterion (dependent) variables (at head of arrow).



3.0 SOURCES OF DATA

In the mid 1960's, industrial Hg pollution in the English-Wabigoon River system of northern Ontario lead to contamination of fish in this waterway (Wheatley 1979). Native communities in the area (particularly the White Dog and Grassy Narrows reserves) relied extensively on freshwater fish from this waterway for a significant portion of their dietary nutrition (Wheatley 1979). In response to the potential health risk posed by this situation, Health Canada launched a Hg monitoring program in the affected native communities. This program was later extended to most reserves across Ontario (see Wheatley 1979; Tupper 1984).

Ubiquitous natural and anthropogenic aquatic Hg pollution across Ontario, combined with the important sport fishing industry throughout the province, lead the Ontario Ministry of Environment to initiate a sport fish Hg monitoring program in the mid 1970's (OMOE 1993). Routine monitoring of various fish species collected from locations across the province was conducted to provide consumption advisories on a species-specific and location-specific basis (OMOE 1993, for example).

The Geologic Survey of Canada conducts research on mineral prospecting methods across Canada. Glaciation of the Canadian Shield has made traditional prospecting methods

ineffective in locating economically-viable ore deposits (Coker et al. 1979). As a result, the GSC launched a major program of lake sediment mineral reconnaissance throughout large areas of the Canadian Shield in Ontario (Coker et al. 1979; Friske 1985a,b). Chemical and physical weathering of ore-rich bedrock was expected to produce relatively high concentrations of minerals in the sediments of lakes and streams in close proximity to these ore deposits (Coker et al. 1979). Hg was one of several minerals for which sediment reconnaissance data were gathered (Coker et al. 1979; Friske 1985a,b).

The GSC was also interested in the potential interaction of soil and bedrock chemistry with acidic deposition (Shilts and Kettles 1989; Coker and Shilts 1979). As part of this research, data on Hg in soils (mostly glacially-deposited tills) were collected to investigate the potential for mobilization of this element by acidic precipitation (Kettles and Shilts 1983; Kettles 1988 1990).

Anecdotal reports of lake acidification lead the Ontario MOE to compile data on water chemistry in more than 6,000 of Ontario's lakes (Neary et al. 1990). These data provided a baseline for comparison of future water chemistry surveys, and also permitted an examination of water chemistry trends with

spatial patterns of acidic deposition across the province (Neary et al. 1990; Neary and Dillon 1988).

Ontario's Ministry of Natural Resources conducts research on factors which influence the productivity and health of fish populations across the province. OMNR has compiled lake morphometric data (lake depth, volume, surface area) and basic water chemistry data on over 9,000 Ontario lakes, following methods described by Dodge et al. (1989).

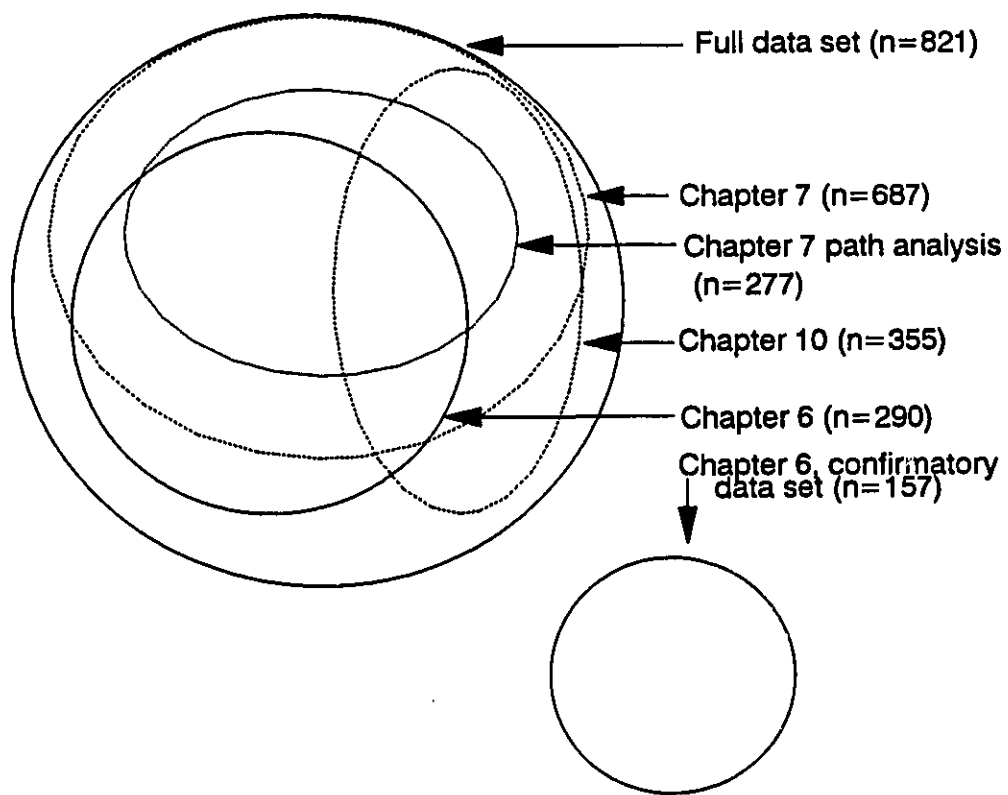
From these extensive data bases, a subset of data were extracted for investigation. Of hair [Hg] data for over 13,000 amerindians resident in 75 reserves across Ontario, a subset of 3,187 individuals in 55 reserves was identified where Hg exposure was considered free of influence by local anthropogenic Hg discharges (such as along the English-Wabigoon river system), by hydroelectric reservoirs, or by consumption of commercially-available fish (such as in reserves in close proximity to urban centres where dietary acculturation was likely). The complete data set is available through the Medical Services Branch, Health Canada, Ottawa.

A preliminary data base containing at least one datum on 12,223 lakes was also compiled, relating to lake water chemistry, morphometrics and fish Hg contamination. A subset

of 821 lakes was identified from which at least one of eight species of freshwater fish had been collected by the Ontario Ministry of Environment and analyzed for Hg content in a sample of dorsal muscle. Many of these lakes had relevant water chemistry data. A further subset of 687 lakes was identified from which lake trout (Salvelinus namaycush), walleye (Stizostedion vitreum vitreum) and northern pike (Esox lucius) were specifically collected, and for which a large proportion of all water chemistry and morphometric data were available. This data set also included 355 lakes for which [Hg] from deep (>10 cm depth) sediment samples (representing pre-industrial deposition), and 88 lakes for which [Hg] in watershed soils, were available from the GSC. How these data sets relate to one another is depicted in Figure 3.1. An independent data set of 157 Ontario lakes was also developed for comparison and confirmation of spatial patterns and associations observed for lake water chemistry (Chapter 6). No fish mercury data existed for these 157 lakes.

The lake data are variously summarized in appropriate chapters. The data relating to Hg levels in a total of 17,376 specimens of lake trout, walleye and northern pike are summarized in Table 8.1. These various data sets and subsets are not reproduced here but may be obtained from the author or from Dr. David Currie, Department of Biology, University of Ottawa.

Figure 3.1 Venn diagram indicating how the various data subsets employed throughout this thesis relate to one another. Relevant chapters are indicated. For Chapter 6, n=290 lakes for the test data set, and n=157 for the independent confirmatory data set; for chapter 7, n=687 lakes for analysis of variance (ANOVA), and n=277 lakes for the path analysis; for Chapter 10, n=355 lakes.



4.0 GENERAL METHODS

Specific methods used to collect data, and to evaluate the various hypotheses, are presented in each chapter. All data manipulation and statistical analyses were conducted using SYSTAT^R version 5.01 (Wilkinson 1991). Variables were transformed as required to stabilize variance and to linearize relationships being assessed by regression methods. Path analysis was conducted using EZ-Path^R version 1.0, a supplementary module for SYSTAT^R (Steiger 1989).

5.0 DOES ACID RAIN INCREASE HUMAN EXPOSURE TO MERCURY?

A REVIEW AND ANALYSIS OF RECENT LITERATURE

5.1 Abstract

The literature suggests that acid deposition may lead to increased mercury (Hg) contamination of fish. Employing published information, we have estimated the change in fish Hg levels associated with an increase in sulphate deposition from 0.25 to 1.25 g S/m²/yr. In seepage lakes, Hg biogeochemistry appears to be dominated by pH, which controls biomethylation. As a result, one can predict that Hg in walleye from seepage lakes, and subsequent human exposure due to consumption of these fish, would be higher at the higher rate of sulphate deposition. For drainage lakes, Hg biogeochemistry appears to be dominated by dissolved organic carbon (DOC) which is responsible for the transport of terrestrial MeHg from the watershed. Increasing acidic deposition onto drainage lakes was predicted to decrease DOC and ultimately reduce Hg accumulation in lake trout and northern pike. Subsequent human Hg exposure by consumption of these species from drainage lakes would therefore be predicted to be lower at the higher rate of acidic deposition. We concluded that the hypothesis that acidic deposition increases Hg contamination in fish, and thereby increases Hg exposure in humans via fish consumption, is likely only true for acidic deposition onto seepage lakes.

5.2 Introduction

It has been hypothesized that human exposure to methylmercury (MeHg) will increase due to acidic deposition (Gilmour and Henry 1991; Clarkson 1990; Goyer et al. 1985). This hypothesis has arisen from three separate observations. First, acidic deposition leads to reduced pH and alkalinity in lakes (Neary et al. 1990; Dillon et al. 1984; NRCC 1981). Secondly, experimental evidence indicates that the net rate of Hg methylation increases as pH declines (Xun et al. 1987), and the respiratory absorption of MeHg by fish is greater at lower alkalinity (Rodgers and Beamish 1983). Finally, many studies of the spatial variation in Hg contamination in fish report negative correlations between fish Hg contamination and pH and/or alkalinity (Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990; Suns and Hitchin 1990; Wren and MacCrimmon 1983; Scheider et al. 1979). As a result, it has been postulated that consumption of fish from acidified lakes would pose an increased health risk to humans due to expected higher MeHg intake.

MeHg exposure presents a significant human health risk (Clarkson 1990; WHO 1990), and the consumption of freshwater fish is the predominant non-occupational source of human MeHg exposure (WHO 1990). Therefore, any factor which might lead to increased levels of MeHg in fish is of concern. Fish consumption in Canada has been recently discussed by

Richardson and Currie (1993) and Coad (1993). Many Canadian amerindian communities rely on subsistence fisheries for a significant portion of their diet (Berkes 1990). This population consumes fish on a regular basis, with average fish consumption estimated at 10 to 15 times the rate of freshwater fish consumption by Canadians in general (Richardson and Currie 1993). Fish consumption by some individual amerindians probably exceeds 1 kg/day (Shephard 1976). It is apparent, therefore, that Canadian amerindians represent the population in Canada most likely to be affected by the impact of acidic precipitation on the Hg contamination in freshwater fish.

The hypothesis linking acid rain to increased exposure to MeHg has now shown up in the popularized scientific literature (Raloff 1991). However, this hypothesis has never been evaluated critically. In this chapter we review the published data and information relating to this hypothesis and present some estimates of the likely change in human MeHg exposure which might be associated with acidic deposition in Canada. The species of freshwater fish consumed by amerindian communities varies depending on local resources. However, lake trout (Salvelinus namaycush), walleye (Stizostedion vitreum vitreum) and northern pike (Esox lucius) are the species most commonly consumed by inland amerindian communities in eastern Canada (Berkes 1990). Therefore, our review relates mainly to these species except where noted.

5.3 Association between acidic deposition and water chemistry

The impacts of acidification on aquatic ecosystems have been reviewed by various authors (Dillon et al. 1984; NRCC 1981; Schindler 1988). Scheider et al. (1979) reported a decrease in the alkalinity of Clear Lake, Ontario by 75% over a 10 year period. A few other studies (Schindler 1988; Dillon et al. 1978) also report generally increased lake or surface water acidification in eastern Canada over 1 to 2 decades, although the extent of impact is variable.

Many more data exist relating differences in surface water quality to spatial variation in acidic deposition. Neary and Dillon (1988), and Neary et al. (1990) reported lower pH and alkalinity in Ontario lakes situated in regions of the province receiving progressively greater rates of sulphate deposition. Similar relationships have also been reported elsewhere (reviewed by Howells 1990).

The potential impact of acidic deposition on levels of dissolved organic carbon (DOC) in lakes has received less attention. Neary and Dillon (1988) anecdotally reported lower mean DOC levels for lakes located in regions of Ontario receiving greater sulphate deposition, although they did not document the relationship. Instead, their interest in DOC was

to demonstrate that changes in lake pH across Ontario were related to sulphate deposition and not to DOC.

Decreases in DOC content and/or colour of lakes have been linked to both sulphate deposition and lake acidification (Dillon et al. 1984; Neary et al. 1990; Stenson and Eriksson 1989). Likewise, the neutralization of acidified lakes by liming has resulted in significant increases in DOC (Molot et al. 1990; Bukaveckas and Driscoll 1991), suggesting that a direct relationship exists between lake acidification and DOC concentration. The impact of acid deposition on DOC has been attributed to the binding and precipitation of DOC with aluminum (Effler et al. 1985), which is expected to be higher in concentration under acidified conditions (see reviews by Dillon et al. 1984; Schindler 1988; Stenson and Eriksson 1989). However, the role of aluminum in this relationship has now been questioned (Schindler et al. 1992). The solubility of DOC is pH-dependent, declining at lower pH (Thurman 1985). DOC also provides some acid neutralizing capacity within lakes (Schindler et al. 1992). Therefore, the loss of DOC from acidified lakes is more likely due to hydrogen ion (H^+) absorption and subsequent precipitation, the increased H^+ arising from acidic deposition.

It is also likely that acidification of a lake's drainage basin will reduce the discharge of DOC from the watershed into

the lake (Schindler et al. 1992; de Haan 1992), this occurring as a result of the reduced solubility of DOC in acidified runoff. This would also reduce DOC levels in drainage lakes receiving sulphate deposition, and reduce the levels of inorganic and MeHg in drainage lakes by reducing the rate of complexation and transport by DOC from the drainage basin.

5.4 Associations between fish [Hg] and water chemistry

Spatial correlations between fish Hg contamination and lake pH and alkalinity are typically negative (Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990; Suns and Hitchin 1990; Wren and MacCrimmon 1983; Scheider et al. 1979). As mentioned previously, experimental evidence has demonstrated that the rate of Hg biomethylation increases as pH declines (Xun et al. 1987), and that absorption of MeHg from the water column is greater at lower alkalinity (Rodgers and Beamish 1983).

Fish Hg contamination has also been related to the organic carbon content of lake water, but this association seems dependent on lake hydrology. In drainage lakes (lakes which are recharged by surface runoff), high fish [Hg] is associated with high DOC levels (Heiskary and Helwig 1986; Sorensen et al. 1990; Wren et al. 1991; McMurtry et al. 1989).

However, in seepage lakes (lakes which are recharged by ground water and direct precipitation onto the lake surface), high fish [Hg] is associated with low [DOC] (Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990).

The predominant role for DOC in the biogeochemistry of Hg in drainage lakes is probably to transport terrestrially-generated MeHg from the drainage basin. A primary mechanism by which both inorganic and MeHg enter drainage lakes is via runoff from the terrestrial drainage basin, complexed to organic carbon (Mierle and Ingram 1991; Lee and Iverfeldt 1991). As a result, Hg concentrations in lake water relate positively to levels of DOC (Mierle and Ingram 1991; Lee and Iverfeldt 1991). DOC may also promote the abiotic methylation of Hg in the water column (Weber 1993). In contrast, experimental studies of net methylation of inorganic Hg suggest that DOC inhibits biomethylation (Miskimmin et al. 1992). However, since DOC correlates positively with fish [Hg], DOC-mediated transport of Hg from the watershed has a greater (positive) influence on the MeHg concentration in fish than DOC-mediated inhibition of inhibition of biomethylation has a negative influence.

Of particular interest in studies of drainage lakes is the observation that DOC (or total organic carbon or colour) is more strongly (and positively) associated with the Hg

content of lake trout (McMurtry et al. 1989), walleye (Wren et al. 1991) and northern pike (Heiskary and Helwig 1986; Sorensen et al. 1990; Wren et al. 1991) than are either pH or alkalinity. This suggests that DOC plays a more significant role in the bioavailability of MeHg to fish than either pH or alkalinity in drainage lakes.

The balance of biogeochemical processes in seepage lakes is different from drainage lakes (Miskimmin et al. 1992). Seepage lakes, by definition, have little terrestrial runoff, thereby limiting the role of DOC in the transport of MeHg from the drainage basin. For these lakes, the main role of DOC appears to be the inhibition of biomethylation (Miskimmin et al. 1992) and to subsequently reduce bioavailability of Hg, as evidenced by negative associations between DOC and fish [Hg]. The expected negative influence of pH on net Hg methylation rate, and thereby MeHg bioavailability to fish, is also evidenced by strong negative correlations between fish [Hg] and pH (Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990).

To summarize, we have deduced from the forgoing that decreases in lake pH and DOC have been linked to increasing sulphate deposition. We have also seen that Hg contamination in lake trout, walleye and northern pike, three commonly consumed freshwater fish species in Canada, covary strongly

with water chemistry, particularly pH and DOC. It is also apparent that there are causal links underlying the covariation of water chemistry with sulphate deposition, and underlying the covariation of Hg levels in fish with water chemistry. Therefore, we can make some preliminary predictions of fish Hg contamination as a function of changes in pH and DOC resulting from increasing sulphate deposition. These predictions can also be extended to estimate potential changes in human exposure to Hg via fish consumption.

5.5 Predicting effects of sulphate deposition on Hg levels in fish and humans

We predicted the change in pH and DOC due to increasing sulphate deposition using the data of Neary et al. (1990). These authors prepared the most extensive study to date of the spatial association between sulphate deposition and lake water chemistry. They defined sulphate deposition rate on the basis of the spatial pattern of sulphate deposition across Ontario, which ranged from <0.25 to >1.25 g S/m²/yr (see also Neary and Dillon 1988). Using these data, we derived regressions relating lake water pH and DOC to sulphate deposition rate (equations (5.1) and (5.2) - values in parentheses are standard errors). Sulphate deposition was represented as the median sulphate deposition rate for a given region of Ontario. All lakes in each region were assigned that same median deposition rate.

$$\ln(\text{DOC}) = 2.52(\pm 0.03) - 0.99(\pm 0.03) * (\text{SO}_4 \text{ deposition rate}) \quad (5.1)$$

$r^2=0.300, n=2653, p << 0.0005$

$$\text{pH} = 7.63(\pm 0.03) - 1.12(\pm 0.03) * (\text{SO}_4 \text{ deposition rate}) \quad (5.2)$$

$r^2=0.192, n=6025, p << 0.0005$

We used equations (5.1) and (5.2) to estimate mean DOC and pH for lakes receiving sulphate deposition at rates of 0.25 and 1.25 g S/m²/yr, the approximate range across Ontario (Neary et al. 1990).

We next applied these estimates for DOC and pH at the high and low sulphate deposition rates to estimate the Hg contamination of fish from both drainage and seepage lakes over this same range in sulphate deposition rate. Based on the regression of McMurtry et al. (1989) predicting [Hg] in lake trout from drainage lakes:

$$\log_{10}(\text{trout}[\text{Hg}]) = -1.072 + 0.132(\text{DOC}) \quad (5.3)$$

$r^2=0.37, p \leq 0.0001, n=61 \text{ lakes}$

we predicted that average [Hg] in standard length (44 cm) lake trout would be lower by 1.36 µg Hg/g fish tissue (approximate 98% C.I. 0.54 - 4.19 µg/g) in fish from lakes located in an area receiving the higher rate of sulphate deposition.

The same calculation can be carried out for standard length (55 cm) northern pike from drainage lakes, based on the regression of Sorensen et al. (1990):

$$\log_{10}(\text{pike}[\text{Hg}]) = 3.5(\pm 0.6) + 0.65(\pm 0.18)\log_{10}\text{TOC} - 0.21(\pm 0.07)\text{pH} \quad (5.4)$$

$r^2=0.37, \quad p<0.05, \quad n=53 \text{ lakes}$

We predict that, at the higher level of sulphate deposition, fish [Hg] is lower by 0.057 $\mu\text{g Hg/g}$ fish tissue (approximate 98% C.I. 0.007 - 0.326 $\mu\text{g/g}$) than at the lower rate of deposition.

Finally, we predicted that Hg levels in walleye (average length=44 cm; data not length-standardized) would be higher by 0.5 $\mu\text{g Hg/g}$ fish tissue (approximate 98% C.I. 0.42 - 0.61 $\mu\text{g/g}$), for walleye from seepage lakes receiving the higher rate of sulphate deposition. This prediction was based on the regression of Wiener et al. (1990):

$$\text{Walleye}[\text{Hg}] = 3.71 - 0.46(\text{pH}) \quad (5.5)$$

$r^2=0.49, \quad p<0.05, \quad n=48 \text{ specimens (from 13 lakes)}$

Assuming an average freshwater fish consumption rate of 20 g fish/day (Richardson and Currie 1993; Coad 1993), the net difference in human Hg intake rate was subsequently estimated for consumption of these species collected from either

drainage or seepage lakes located in regions of Ontario receiving sulphate deposition at rates of 0.25 and 1.25 g S/m²/yr, respectively (Table 5.1). Lower net exposure, attributable to the lower DOC levels at the higher sulphate deposition rate, were predicted for consumption of lake trout and northern pike from drainage lakes. However, we predicted a higher net human exposure rate to Hg via consumption of walleye from seepage lakes. This latter prediction was attributable to lower lake pH at the higher sulphate deposition rate.

Table 5.1. Estimated net differences in intake of mercury (μg Hg/kg body weight/day) by adults (70 kg bw) assumed to consume 20 g of fish per day. Estimates based on predicted net changes in fish [Hg] which are presented in the text. These predictions relate to consumption of fish from regions receiving sulphate deposition at rates of 0.25 and 1.25 g sulphur/m²/year. Negative values represent an estimated decrease in Hg exposure at the higher rate of deposition.

Fish species	controlling water chemistry parameter	estimated net change in Hg intake (approximate 98% CI range)
lake trout (from drainage lakes)	DOC	-0.39 (-1.197 to -0.154)
walleye (from seepage lakes)	pH	+0.15 (0.121 to 0.173)
northern pike (from drainage lakes)	DOC + pH	-0.02 (-0.093 to -0.002)

5.6 Discussion

Emphasis was placed on pH and DOC due to the availability of published regression models predicting fish [Hg] from these water quality parameters. Compared to pH and DOC, the influence of alkalinity, or other water chemistry parameters, is likely minimal since alkalinity and other parameters were examined by McMurtry et al. (1989), Sorensen et al. (1990) and Wiener et al. (1990) and did not significantly improve these authors' regression models.

The [Hg] in walleye from seepage lakes was predicted to be greater at higher acidic deposition. We suspect that this result relates more to lake hydrology than to physiological, ecological or other differences between walleye and lake trout or northern pike. For walleye from drainage lakes, the most significant predictor of [Hg] was DOC, not pH (Wren et al. 1991), and the relationship was positive, as it was for pike and lake trout. Unfortunately, these authors did not report regression models from which we could make estimates for [Hg] in walleye from drainage lakes.

The results of our review and analysis suggest, at least for consumption of fish from drainage lakes, that increasing sulphate deposition may result in a marginal decrease in Hg levels in fish species commonly consumed by humans, and thereby decrease human exposure to MeHg. Although the exact

proportion of lakes of the drainage type in eastern Canada is unknown, we examined lakes surrounding 75 Amerindian reserves in Ontario using 1:50,000 scale topographic maps and found the vast majority to be drainage lakes.

With a suggested 'safe' MeHg intake rate of 1 μg Hg/kg body weight/day (Clarkson 1990), consumption of walleye from seepage lakes, at a typical rate of fish consumption (20 g/day), should not pose a serious increase in risk. However, for those consuming greater quantities of fish (say those consuming 150 g fish/day or more), the increased Hg intake could be significant. Our estimates of exposure predicted from consumption of walleye may actually underestimate the increase in Hg intake at the higher rate of acidic deposition on seepage lakes. First, the estimate of lower pH with increasing sulphate deposition was based on all lakes, regardless of hydrological type. The hydrology of lakes could not be distinguished with available data, but the chemistry of seepage lakes is dominated by direct atmospheric input (Eilers et al. 1983), and the decline in pH for these lakes may be under-estimated in the regression model employed here. Secondly, loss of DOC from seepage lakes due to acidic deposition is expected to lead to a greater rate of biomethylation (Miskimmin et al. 1992) and subsequently greater bioaccumulation in fish from lakes with reduced DOC (Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990).

This potential effect was not discernible with the data of Wiener et al. (1990) and therefore not included in their regression model (Equation 5.5). However, any such impact would also result in greater fish Hg contamination, and thereby greater human intake via fish consumption, at greater rates of acidic deposition on seepage lakes.

DOC or pH explain 50% or less of the interlake variation in fish [Hg] (after controlling for fish length), and sulphate deposition explains only 20% and 30% of the interlake variation in pH and DOC, respectively. As a result, sulphate deposition may account for only 10% or less of total variation in Hg levels in length-standardized fish. Therefore, future quantitative analyses of the relationship between acidic deposition and fish [Hg] will require that several confounding factors be controlled. These include factors which influence:

a) lake productivity, which will cause biomass dilution of Hg contamination (Richman et al. 1988). Confounding factors include lake size, which affects the productivity of fish populations (Rawson 1952; Ryder 1965), and watershed geochemistry which influences lake primary productivity (Conroy and Keller 1976).

b) lake water chemistry, as this is influenced by watershed geochemistry (Ryder 1964) as well as acidic deposition.

c) watershed cation exchange capacity, since the degree of buffering of precipitation runoff offered by the watershed also directly influences the lakes' susceptibility to acidification (Cowell and Lucas 1986).

d) natural sources of Hg in soils and bedrock, which can result in significant lacustrine biotic Hg contamination (Loukola-Ruskeeniemi 1990; Varekamp and Waibel 1987), and the distribution of which may covary with sulphate deposition.

It is debatable whether or not the spatial relationships between sulphate deposition and water chemistry, and between water chemistry and Hg accumulation in fish, can be extrapolated temporally. However, we conclude that the generalized, simple speculation of increased risk to humans, and even wildlife (Wiener 1987), with increasing sulphate deposition (due to increased fish [Hg]) appears to be incorrect, or must be qualified for other factors which also influence fish Hg accumulation, such as lake hydrology. Furthermore, it is apparent that the role which DOC plays in influencing Hg loading, methylation and bioavailability in both drainage and seepage lakes, and the overall influence of lake and watershed acidification on DOC, require further investigation.

6.0 THE ASSOCIATION BETWEEN SULPHATE DEPOSITION AND LAKE WATER CHEMISTRY

6.1 Abstract

Data for 290 lakes were evaluated to examine the extent to which lake and watershed morphometry and watershed buffering capacity may confound the association between lake water chemistry and sulphate deposition. Lake alkalinity, pH, concentrations of sulphate ion [SO_4^{2-}], dissolved organic carbon ([DOC]) and aluminum [Al] were found to vary significantly with both morphometric and buffering capacity characteristics of lakes. These characteristics were found, in turn, to vary significantly among regions of Ontario with progressively greater rates of sulphate deposition. After controlling water chemistry data for these confounding factors, only mean lake [DOC] and [SO_4^{2-}] changed consistently with increasing rates of sulphate deposition - [DOC] was progressively lower while [SO_4^{2-}] was progressively greater. The association between DOC and sulphate deposition rate may be indirect, in so much as the statistical variation in DOC associated with sulphate deposition rate was explained by DOC's association with three other water chemistry variables - alkalinity, [SO_4^{2-}] and [Al] - thought to be influenced by acid rain. Also, there appeared to be a threshold rate of sulphate deposition of between 0.75 and 1.0 g S/m²/y below which neither average pH nor mean alkalinity appear to have been reduced. Finally, [Al] appears to have no discernible direct relationship with sulphate deposition rate.

6.2 Introduction

The qualitative impacts of sulphate deposition on three key water chemistry variables have been extensively studied (see Howells 1990 for review). Alkalinity and pH reportedly decline (OMOE 1991; Neary et al. 1990; Neary and Dillon 1988), while $[\text{SO}_4^{2-}]$ reportedly increases (Neary et al. 1990; Sullivan et al. 1988), as sulphate deposition increases. Aluminum concentration ($[\text{Al}]$) is also expected to increase with increasing sulphate deposition (NRCC 1981).

Concentrations of dissolved organic carbon (DOC) are also affected by acidic deposition. The solubility of DOC is pH dependant (Thurman 1985; Stumm and Morgan 1981) and should, therefore, decrease as lake acidification increases. Neary et al. (1990) showed that levels of $[\text{DOC}]$ were lower in lakes from regions of Ontario receiving greater rates of sulphate deposition. Changes in lake $[\text{DOC}]$ have also been reported after experimental acidification and liming of lakes (Schindler et al. 1992; Molot et al. 1990; Bukaveckas and Driscoll 1991), suggesting a direct cause-effect relationship between lake acidification and $[\text{DOC}]$.

Changes in all these water chemistry variables are hypothesized to influence fish mercury (Hg) accumulation (Figure 2.1, Chapter 5). As the influence of pH, alkalinity and $[\text{SO}_4^{2-}]$ on fish $[\text{Hg}]$ is contrary to that of $[\text{DOC}]$ (Chapter

5), then the comparative associations of these variables with sulphate deposition rate is of some practical importance to understanding and predicting the spatial impact of sulphate deposition on fish Hg accumulation.

The confounding influences of lake and watershed morphometry and watershed buffering capacity have not been controlled in previous spatial analyses of the links between acidic deposition and water chemistry. Neary et al. (1990) showed that the associations between pH, alkalinity, lake $[\text{SO}_4^{2-}]$, $[\text{DOC}]$ and sulphate deposition are qualitatively different for lakes of differing sizes. DOC is dependent on lake and watershed morphometry (Rasmussen et al. 1989), drainage basin runoff being the primary source of DOC to Canadian Shield lakes (Schindler et al. 1992). Morphometric factors which influence loading rates from the drainage basin, as well as lake retention times, flushing rates and dilution factors significantly influence lake DOC levels (Rasmussen et al. 1989). Likewise, alkalinity, pH, $[\text{Al}]$, and $[\text{SO}_4^{2-}]$ can also be expected to vary with morphometric factors which influence lake retention times and flushing rates, dilution factors, and chemical loading rates from the drainage basin (Schindler 1971).

Watershed buffering capacity can also be expected to influence lake water chemistry. Lake chemistry, particularly

pH and alkalinity, is known to vary with the chemical nature of both watershed soils and bedrock (Ryder 1964; Conroy and Keller 1976; Kettles et al. 1991; Shilts and Kettles 1989). The degree of leaching of Al by acidic deposition into lakes is expected to be greater in watersheds with lower buffering capacity (NRCC 1981; Cronan and Scholfield 1979). In many dilute lakes of the Canadian Shield, DOC provides a significant proportion of lake water acid neutralizing capacity (Dillon, pers. com. cited in Schindler et al. 1992; OMOE 1991). The concentration of DOC, therefore, would also be expected to vary with watershed buffering capacity.

These potentially confounding factors will vary spatially and may coincidentally covary with spatial patterns of sulphate deposition. It is apparent, therefore, that the empirical association between sulphate deposition rate and lake water chemistry should be controlled for these confounding variables if the extent of potential impact of acidic deposition on water chemistry is not to be over- or under-estimated.

The purpose of this chapter was to test the following hypotheses:

- 1) do lake alkalinity, pH, $[\text{SO}_4^{2-}]$, [Al] and [DOC] vary significantly with lake and watershed morphometry?

- 2) do lake and watershed morphometry vary significantly among sulphate deposition zones of Ontario?
- 3) do lake pH, alkalinity, $[\text{SO}_4^{2-}]$, $[\text{Al}]$ and $[\text{DOC}]$ vary significantly with watershed buffering capacity?
- 4) do watershed buffering capacity and sulphate deposition in Ontario significantly covary?
- 5) after statistically controlling water chemistry data for the influences of morphometry and watershed buffering capacity, do the water chemistry variables vary significantly among regions of differing sulphate deposition rate?

6.3 Methods

Water chemistry and morphometric data were obtained for 290 Ontario lakes (Figure 6.1). How these data related to other data employed in this thesis is depicted in Figure 3.1. Water chemistry data (see Table 6.1) were derived from an unpublished compilation of water chemistry surveys collected by the Ontario Ministry of Environment. Methods of water sample collection and chemical analysis are summarized by Neary et al. (1990).

Unpublished data on lake surface area, lake volume and maximum lake depth were obtained from the Aquatic Habitat Inventory of the Ontario Ministry of Natural Resources (see Table 6.1). Methods for collecting planimetry and bathometry data are described by Dodge et al. (1989). We determined drainage basin areas from 1:50,000 scale topographic maps using a polar planimeter. For drainage basin areas of non-headwater lakes, the areas of drainage basins surrounding upstream lakes were excluded so that measurements reported here represent only the area of the land draining directly into the lakes in question.

Lakes were categorized into one of seven sulphate deposition regions of Ontario, after the scheme of Neary et al. (1990) (Figure 6.2) (see also Neary and Dillon 1988). The total range in sulphate deposition across Ontario is <0.25 to >1.25 g sulphur/m²/year. Lakes were also classified into one of three acid buffering capacity categories (low, moderate and high) after Cowell and Lucas (1986). These categories are qualitative indicators of the relative acid buffering capacity of the soil and bedrock of lake watersheds across the province. Where lake watersheds were composed of 2 or more buffering capacity categories, lakes were classified to that category which comprised the greatest part of the lake's perimeter. Lakes classified with watersheds containing

Table 6.1. Geometric mean (and range) of data on Ontario lakes investigated in Chapter 6. n=290 lakes except where noted.

Parameter	Geometric mean	Range
total watershed area		
(TWA = DBA+LSA) (hectares)	2,215	80.0 - 81842.0
drainage basin area (DBA) ^a		
(hectares)	1,726	64.9 - 54150.6
lake surface area (LSA)		
(hectares)	397	8.1 - 27691.4
drainage basin area: lake		
surface area (DBA:LSA)	4.3	0.13 - 81.1
lake volume (VOL)		
(m ³ X 10 ⁴)	2,670	32.2 - 404,012
lake maximum depth (m)		
(n=270)	22.3	1.4 - 119.0
[DOC] (mg/L)	5.2	1.2 - 26.5
[SO ₄ ²⁻] (mg/L)	6.2	0.72 - 27.30
[Al] (μg/L)	22.2	2.0 - 306.0
pH	7.0 ^b	5.1 - 8.4
ALK (μeq/L)	11.2 ^c	-0.45 - 135.0

a - excludes surface area of lake;

b - arithmetic mean;

c - excludes 6 lakes with ALK values ≤ 0.

Figure 6.1: Distribution of the 290 Ontario lakes examined in Chapter 6. Single points may represent the location of more than one lake.

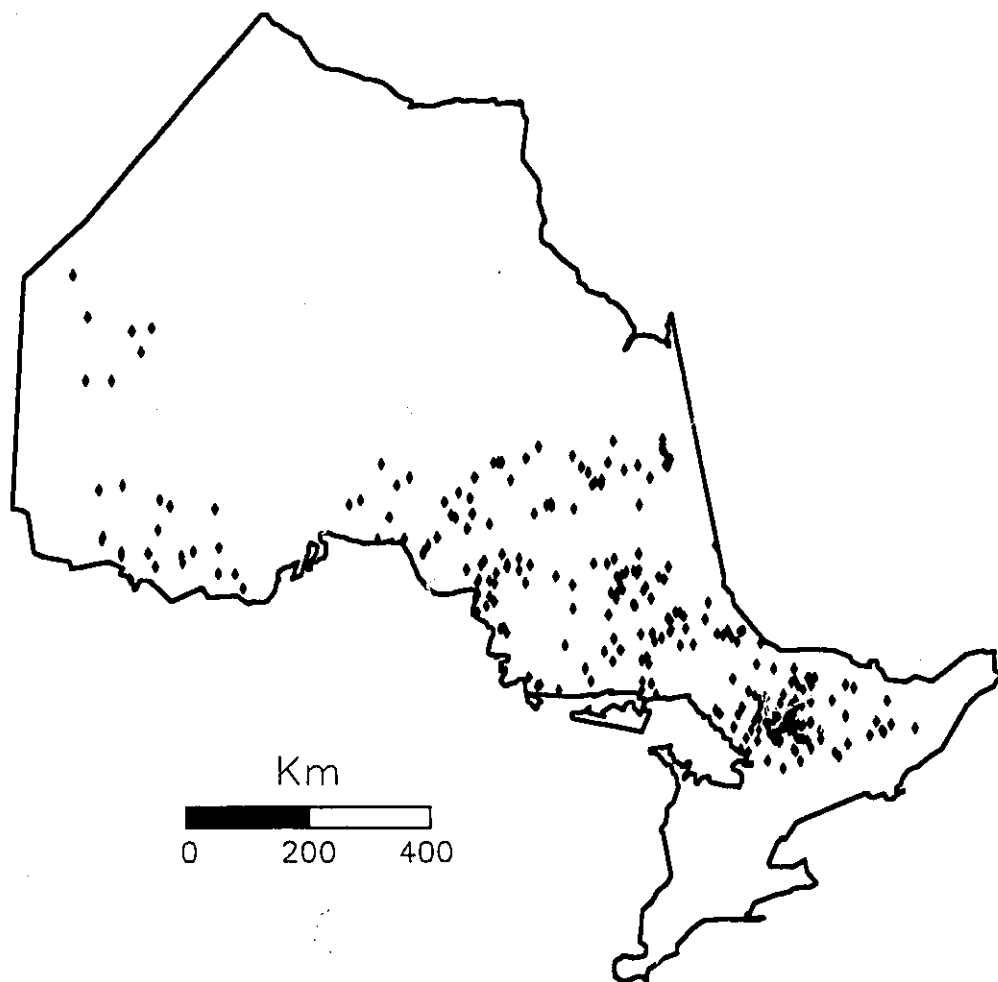
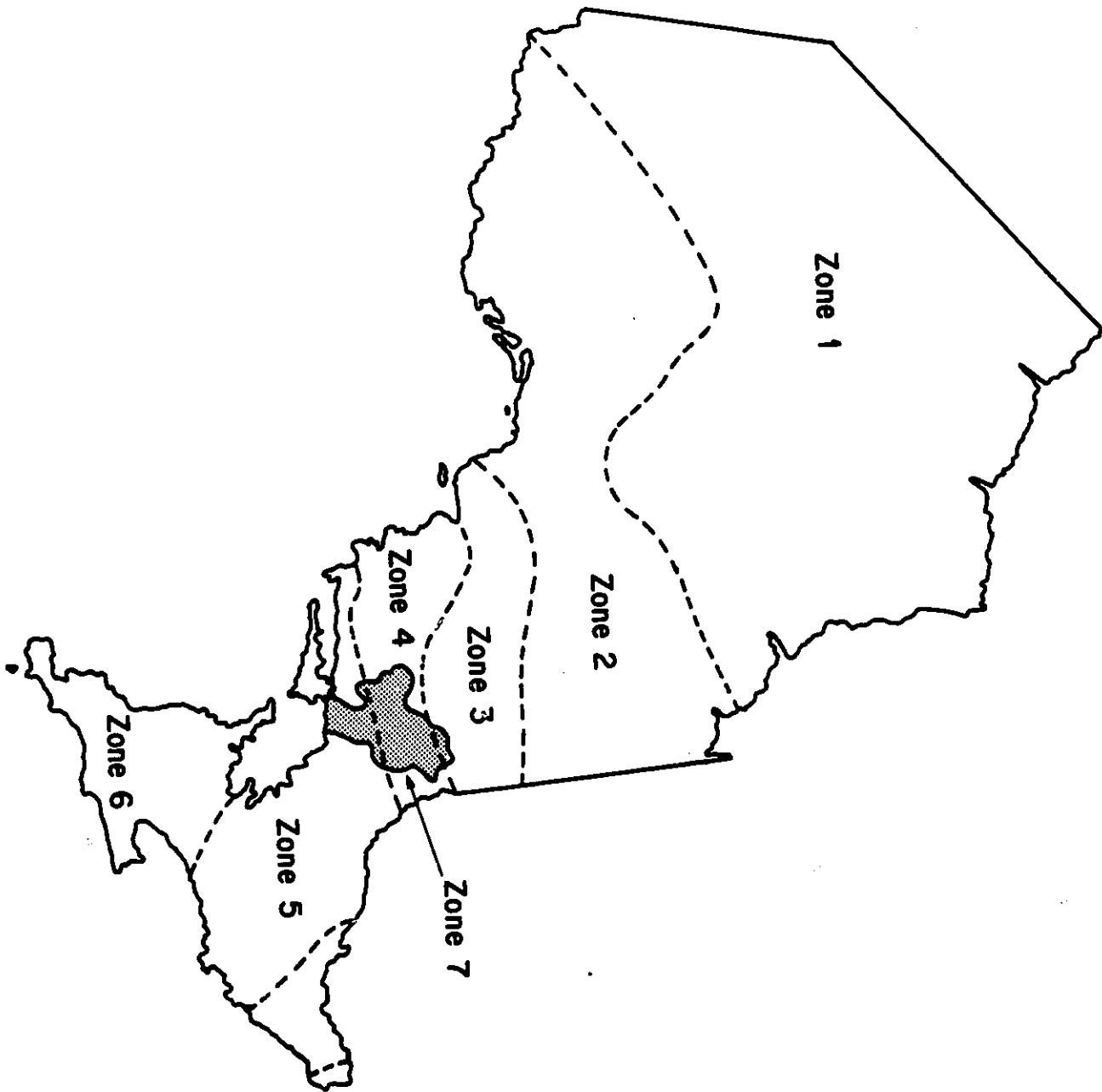


Figure 6.2: Sulphate deposition zones in Ontario, Canada (from Neary et al. 1990). Median rates of sulphate deposition are: zone 1 - 0.125 g S/m²/yr; zone 2 - 0.375 g S/m²/yr; zone 3 - 0.625 g S/m²/yr; zone 4 - 0.875 g S/m²/yr; zone 5 - 1.125 g S/m²/yr; zone 6 - 1.375 g S/m²/yr; zone 7 - 1.625 g S/m²/yr.



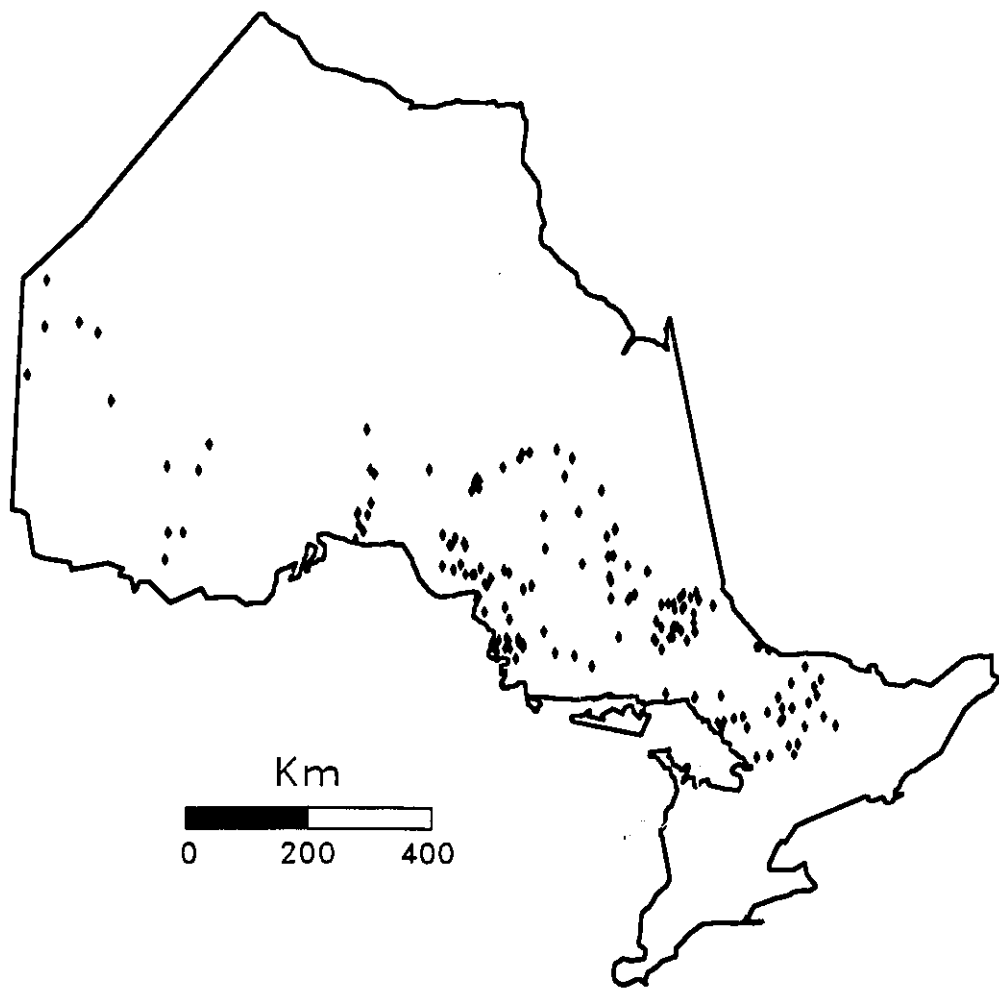
predominantly naturally acidic soils (see Cowell and Lucas 1986) were excluded from analysis because the relative buffering capacity of such watersheds is unknown.

To confirm observed relationships, a second independent data set was also compiled, with a sample size of 157 lakes. Their locations are indicated in Figure 6.3. 30 new lakes were selected at random (from those remaining with all required data) from each of five of Ontario's sulphate deposition zones, while only 7 lakes were included for the deposition zone in Ontario with the lowest rate of sulphate deposition. This was due to lack of adequate data for lakes in this remote, northwestern portion of the province. How this confirmatory data set relates to the other data employed in this thesis is depicted in Figure 3.1. Analysis of covariance (ANCOVA) was used to fit each regression model to the test and confirmatory data sets, and to the combined data set, and ANCOVA evaluated by F-test.

Multiple regression, ANCOVA and ANOVA analyses were conducted using SYSTAT^R, version 5.01 (Wilkinson 1991). All continuous variables but pH were approximately log-normally distributed and were \log_e -transformed before analysis.

Figure 6.3: Distribution of the 157 Ontario lakes composing the confirmatory data set used in Chapter 6. Single points may represent the location of more than one lake.





6.4 Results

Lake and watershed morphometry variables were found to vary significantly among regions of sulphate deposition and buffering capacity across Ontario (Table 6.2). It was also observed that all water chemistry variables were significantly correlated with lake and/or watershed morphometry (Table 6.3). The best multiple regression model explaining variation in each water chemistry variable with lake morphometry is presented in Table 6.4. The same variables - watershed area, lake volume, drainage basin area:lake surface area ratio - were found to be the best predictors for all water chemistry variables but Al. When examined by ANCOVA, there were no significant differences in these regression models for any of the chemistry variables between the experimental and confirmatory data sets (pH: $F=0.02$, $p>0.8$, $df=442,1$; alkalinity: $F=0.12$, $p>0.7$, $df=442,1$; $[SO_4^{2-}]$: $F=3.74$, $p>0.05$, $df=442,1$; [DOC]: $F=0.95$, $p>0.3$, $df=442,1$; [Al]: $F=0.49$, $p>0.4$, $df=444,1$).

Table 6.2. ANOVA results for all morphometric and water chemistry variables by zones of sulphate deposition and watershed buffering capacity. n=290 lakes except where noted. Acronyms are defined in Table 6.1. All variables were log_e-transformed but pH, sulphate deposition rate and buffering capacity. ns = not significant at p>0.05.

Variable	sulphate deposition zone			buffering capacity		
	F	p	R ²	F	p	R ²
pH	42.3	<0.0005	0.43	37.6	<0.0005	0.21
ALK+1	49.9	<0.0005	0.47	49.4	<0.0005	0.26
[SO ₄ ²⁻]	103.0	<0.0005	0.65	18.5	<0.0005	0.11
[DOC]	45.7	<0.0005	0.45	37.9	<0.0005	0.21
[Al]	2.9	<0.02	0.05	0.8	ns	<0.01
TWA	3.0	<0.02	0.05	2.4	ns	<0.02
DBA	2.6	<0.03	0.04	1.3	ns	<0.01
LSA	2.6	<0.03	0.04	6.8	<0.002	0.05
DBA:LSA	2.2	ns	<0.04	13.5	<0.0005	0.09
VOL	1.0	ns	<0.02	10.4	<0.0005	0.07
Max depth (n=270)	8.9	<0.0005	0.14	17.0	<0.0005	0.11
buffering capacity	12.9	<0.0005	0.19	--	--	--
sulphate deposition rate	--	--	--	19.7	<0.0005	0.12

Table 6.3. Pearson correlation coefficients between water chemistry variables, lake and watershed morphometrics, sulphate deposition zone and watershed buffering capacity. All continuous variables but pH were log_e-transformed. ns = not significant; all other correlations were significant at p<0.03. n=290 in all cases except where noted. Acronyms defined in Table 6.1.

	TWA	DBA	LSA	VOL	DBA:LSA	Max	sulphate	watershed
						depth	deposition	buffering
						(n=270)	rate ^a	capacity ^a
pH	0.34	0.33	0.30	0.14	ns	-0.21	-0.66	0.46
(alkalinity+1)	0.28	0.28	0.22	ns	ns	-0.30	-0.69	0.51
[SO ₄ ²⁻]	ns	ns	ns	0.12	ns	0.33	0.81	-0.34
[DOC]	0.23	0.25	ns	-0.14	0.31	-0.49	-0.67	0.46
[Al]	ns	ns	-0.21	-0.22	0.34	-0.12	0.22	ns

a results are multiple R values for analysis of variance of variance of water chemistry variables among zones of sulphate deposition or by watershed buffering capacity categories.

Table 6.4. Multiple regression models predicting water chemistry variables from lake and watershed morphometry, for 290 lakes. Acronyms are defined in Table 6.1. All variables listed were significant at $p < 0.05$. Results of ANCOVA by sulphate deposition zone and by watershed buffering capacity are presented. Partial R^2 values indicate the residual variation explained by sulphate deposition or buffering capacity after controlling for lake morphometry.

Partial R^2 : * = $p < 0.0001$; ** = $p < 0.00005$; ns = not significant.

Water chemistry variable	Regression model	---partial R^2 values---	
		sulphate deposition	buffering capacity
ln[DOC]	$= 1.09 + 0.62\ln(\text{TWA}) - 0.24\ln(\text{DBA:LSA}) - 0.49\ln(\text{VOL})$ $R^2 = 0.479, p < 0.0005$	(-) 0.358**	(+) 0.215**
ln(Alk+1)	$= 1.30 + 1.17\ln(\text{TWA}) - 0.72\ln(\text{DBA:LSA}) - 0.85\ln(\text{VOL})$ $R^2 = 0.266, p < 0.0005$	(-) 0.361**	(+) 0.210**
pH	$= 6.09 + 0.55\ln(\text{TWA}) - 0.36\ln(\text{DBA:LSA}) - 0.36\ln(\text{VOL})$ $R^2 = 0.228, p < 0.0005$	(-) 0.342**	(+) 0.176**
ln[SO ₄ ²⁻]	$= 1.52 - 0.34\ln(\text{TWA}) + 0.31\ln(\text{DBA:LSA}) + 0.31\ln(\text{VOL})$ $R^2 = 0.152, p < 0.0005$	(+) 0.612**	(-) 0.079*
ln[Al]	$= 2.40 + 0.48\ln(\text{DBA:LSA})$ $R^2 = 0.113, p < 0.0005$	(+) 0.085*	ns

pH, alkalinity, [DOC] and $[\text{SO}_4^{2-}]$ were found to vary significantly among lakes with watersheds of differing relative buffering capacity (Table 6.2). After controlling these water chemistry variables for the influence of lake morphometry, these significant variations persisted (Table 6.4). Relative watershed buffering capacity was also found to vary significantly among regions of differing sulphate deposition rate (Table 6.2), such that the lakes with the least buffered watersheds receive, on average, the greatest amount of sulphate deposition (Figure 6.4).

It was observed that morphometry, buffering capacity and sulphate deposition rate explained varying amounts of the total spatial variation of the different water chemistry variables. For [DOC], alkalinity, pH and [Al] the order of decreasing significance was:

morphometry > sulphate deposition rate > buffering capacity.

For lake $[\text{SO}_4^{2-}]$, the order was:

sulphate deposition rate > morphometry > buffering capacity.

Next, we examined the patterns of variation of water chemistry variables among regions of differing sulphate

deposition, both before (Figures 6.5 and 6.6) and after (Figures 6.7 and 6.8) controlling for lake and watershed morphometry and buffering capacity. We found that only [DOC] and lakewater $[\text{SO}_4^{2-}]$ demonstrated the expected monotonic relationship with sulphate deposition rate, although residual data for all chemistry variables did vary significantly among sulphate deposition zones (Table 6.4; Figures 6.5 to 6.8). In other words, only [DOC] and $[\text{SO}_4^{2-}]$ varied consistently with progressively greater sulphate deposition. Again, no significant differences were observed between the test and confirmatory data sets, when regression models for any of the chemistry variables included sulphate deposition or sulphate deposition and buffering capacity.

As mentioned previously, the solubility of DOC is pH-dependent and, therefore, may be associated with sulphate deposition indirectly through the influence of sulphate deposition on pH and/or possibly other water chemistry parameters. The best predictions of lake DOC were obtained by accounting for both morphometric and water chemistry parameters:

$$\begin{aligned}
 \ln[\text{DOC}] = & 0.75(\pm 0.15) + 0.26(\pm 0.04)\ln\text{TWA} - 0.07(\pm 0.04)\ln\text{DBA:LSA} \\
 & - 0.20(\pm 0.03)\ln\text{VOL} - 0.30(\pm 0.04)\ln[\text{SO}_4^{2-}] \\
 & + 0.22(\pm 0.02)\ln[\text{ALK}+1] + 0.18(\pm 0.02)\ln[\text{AL}] \qquad (6.1) \\
 & (R^2=0.739, p \ll 0.00001, n=290)
 \end{aligned}$$

where TWA = total watershed area; DBA:LSA = the ratio of drainage basin area to lake surface area; VOL = lake volume; $[SO_4^{2-}]$ = the lake concentration of sulphate ion; ALK = lake alkalinity; and [AL] = lake concentration of aluminum ion. Subsequent ANCOVA of Equation (6.1) by sulphate deposition zones showed no significant differences ($F=2.06$; $df=278,5$; $p>0.07$). In other words, variation explained by alkalinity, $[SO_4^{2-}]$ and [AL] explained the statistical variation introduced by sulphate deposition.

Figure 6.4: Covariation of relative buffering capacity and sulphate deposition rate. Letters above bars indicate differences significant by Tukey-Kramer multiple range test. Numbers over the bars indicate sample size.

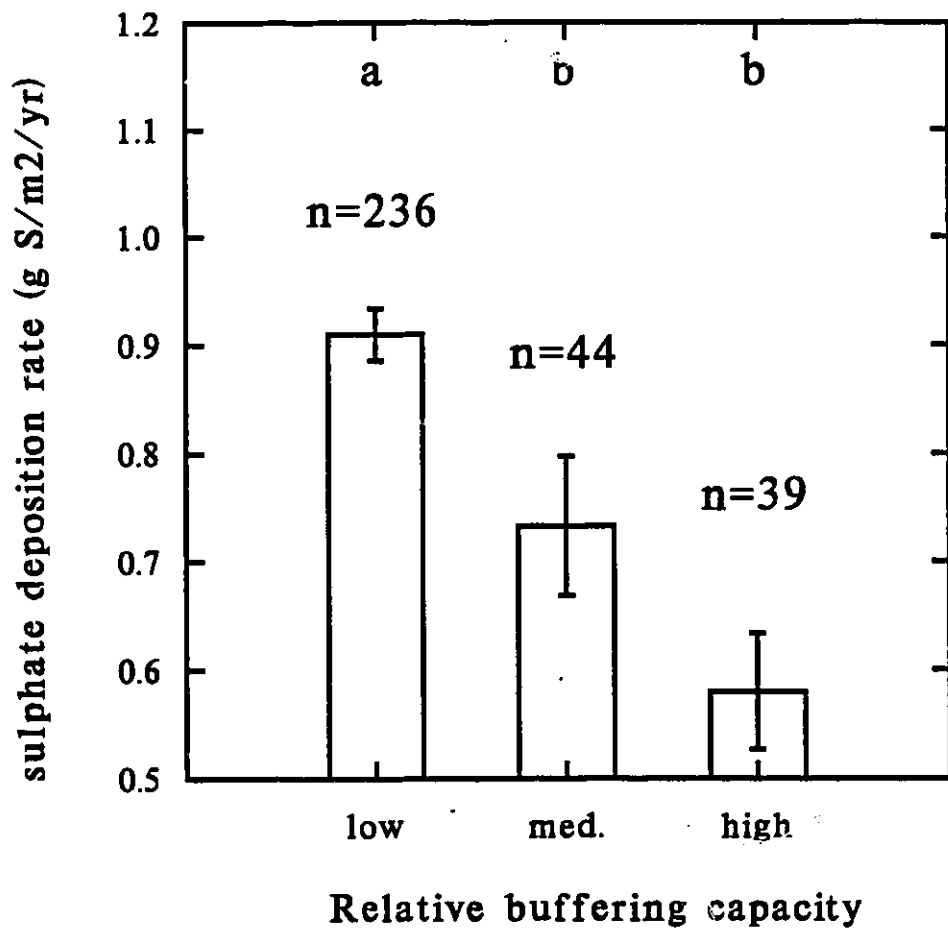
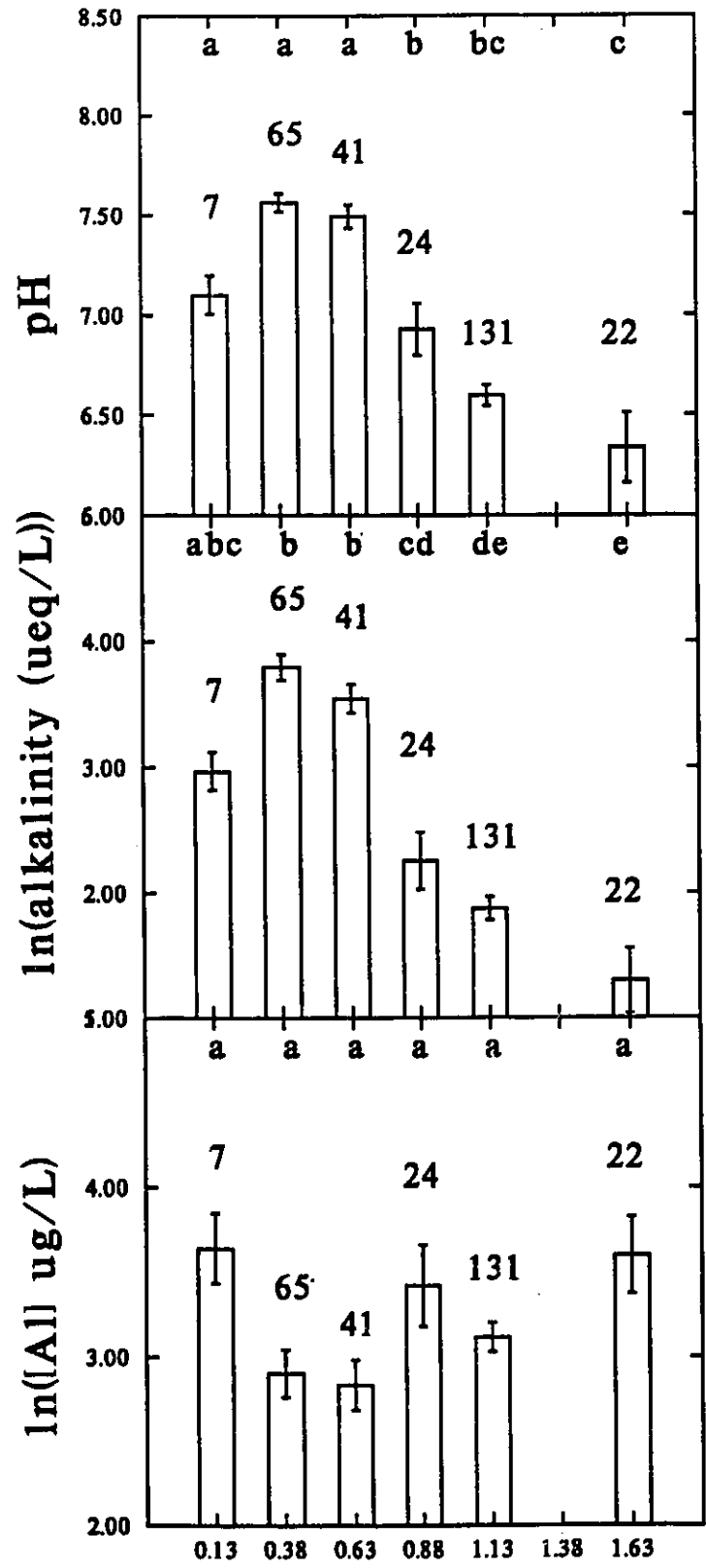
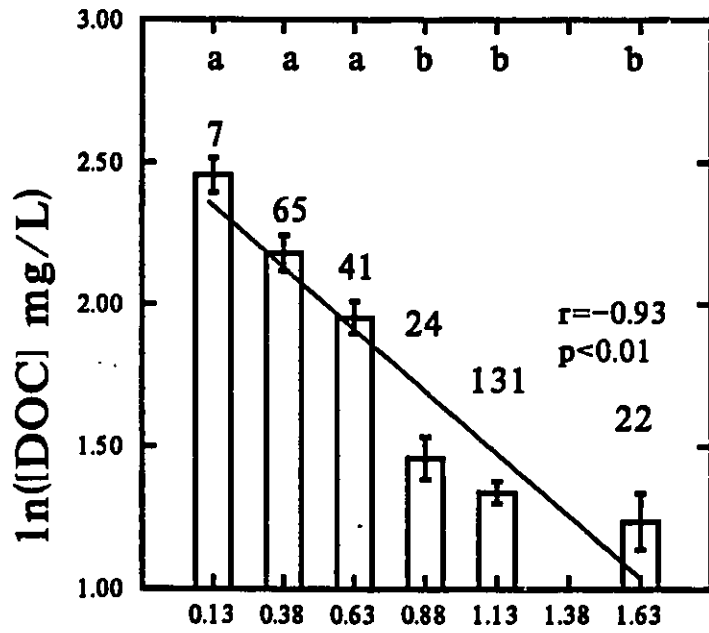
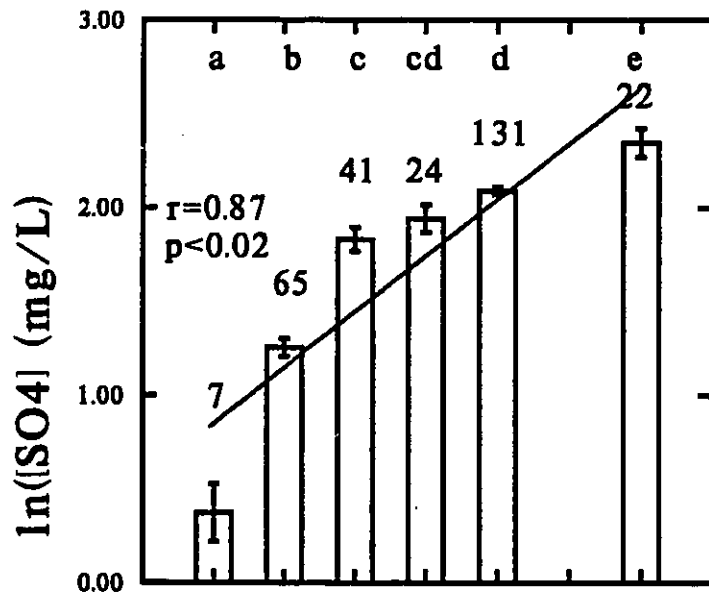


Figure 6.5: ANOVA, by sulphate deposition zone, of pH, alkalinity and [Al] without controlling for lake morphometry or watershed buffering capacity. Numbers above the bars indicate sample size. Letters indicate differences significant at $p < 0.05$ by Tukey-Kramer multiple range test.



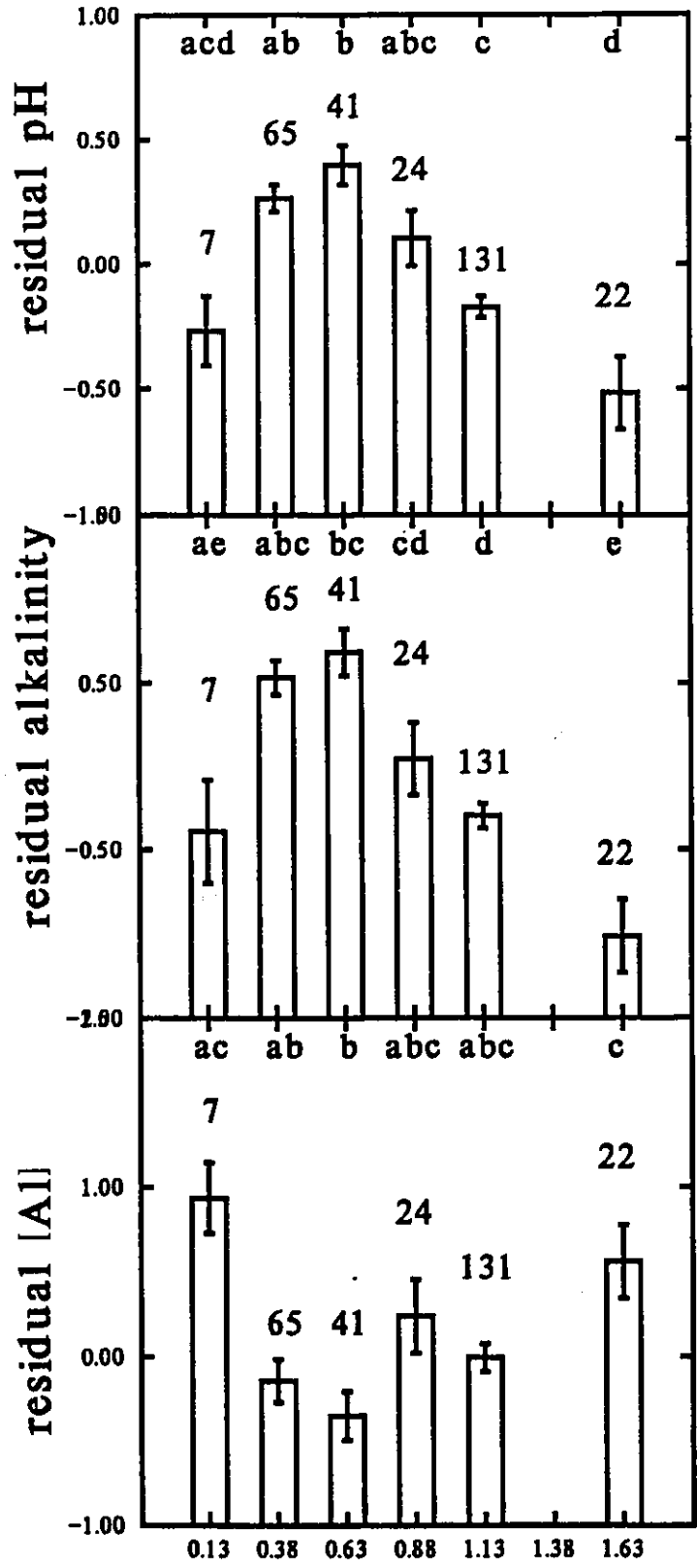
median sulphate deposition rate (g S/m²/yr)

Figure 6.6: ANOVA, by sulphate deposition zone, of $[\text{SO}_4^{2-}]$ and $[\text{DOC}]$ without controlling for lake morphometry or watershed buffering capacity. Numbers above the bars indicate sample size. Letters indicate differences significant at $p < 0.05$ by Tukey-Kramer multiple range test. The lines in the Figures represent the least squares regression line between residual $\ln[\text{SO}_4^{2-}]$ and $[\text{DOC}]$, respectively, and median sulphate deposition rate; r and p values for these curves are also given.



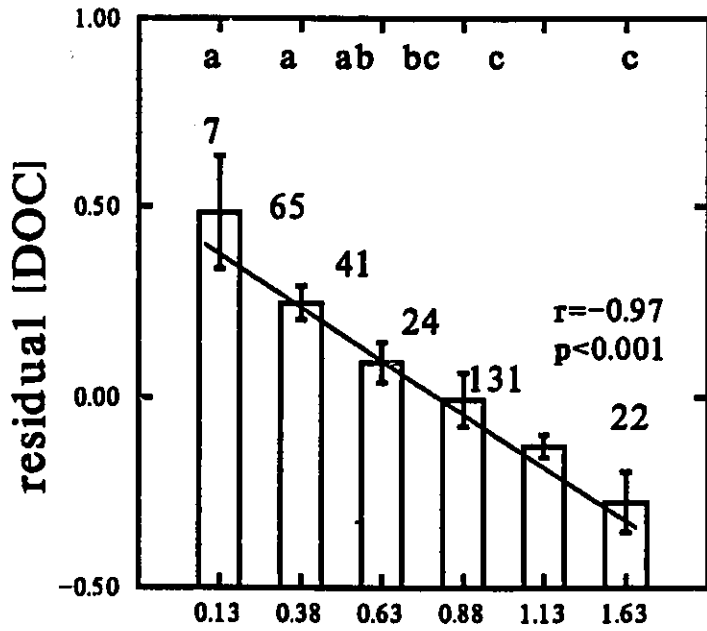
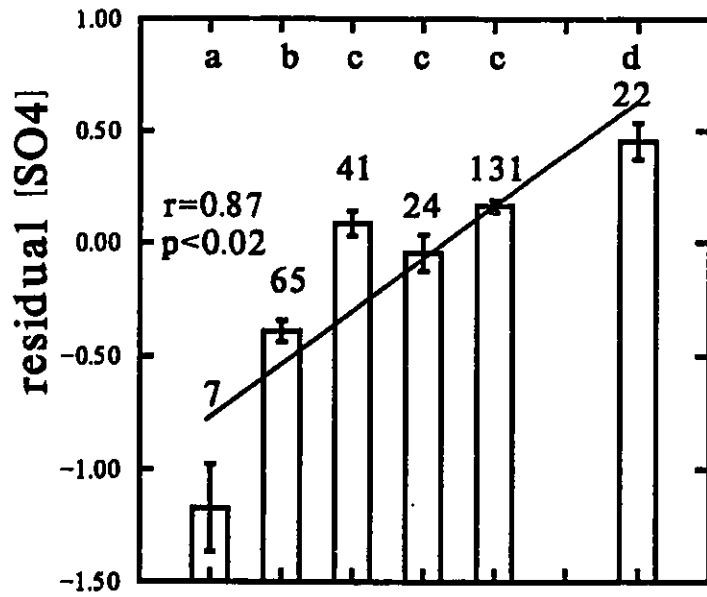
median sulphate deposition rate (g/m²/yr)

Figure 6.7: ANOVA, by sulphate deposition zone, of residual variance in pH, alkalinity and [Al] after controlling for lake morphometry and watershed buffering capacity. Alk, [Al] and lake morphometric variables were log_e-transformed prior to analysis. Numbers above the bars indicate sample size. Letters indicate differences significant at p<0.05 by Tukey-Kramer multiple range test.



median sulphate deposition rate (g S/m²/yr)

Figure 6.8: ANOVA, by sulphate deposition zone, of residual variance in $[\text{SO}_4^{2-}]$ and $[\text{DOC}]$ after controlling for lake morphometry and watershed buffering capacity. $[\text{SO}_4^{2-}]$, $[\text{DOC}]$ and lake morphometric variables were \log_e -transformed prior to analysis. Numbers above the bars indicate sample size. Letters indicate differences significant at $p < 0.05$ by Tukey-Kramer multiple range test. The lines in the Figures represent the least squares regression line between residual $\ln[\text{SO}_4^{2-}]$ and $[\text{DOC}]$, respectively, and median sulphate deposition rate; r and p values for these curves are also given.



median sulphate deposition rate (g/m²/yr)

6.5 Discussion

It is not surprising that lake and watershed morphometry, as well as watershed buffering capacity, should be found to influence the water chemistry variables evaluated here. Total watershed area represents the total surface area available to receive incident precipitation. The ratio of the terrestrial drainage basin area to lake surface area determines the relative loading of precipitation to the lake between the terrestrial and lake surface portions, indicating the degree to which incident precipitation will contact soil for potential ion exchange before entering the lake. Lake volume will determine the degree to which acids, ions and solutes entering the lake will be diluted. Finally, watershed buffering capacity has a direct influence on a lake's susceptibility to acidification, and thereby on any impact that acidic deposition may have on water chemistry. The results for lake [DOC] were in good agreement with Rasmussen et al. (1989) who assessed lake colour (a surrogate for DOC) in headwater lakes.

Lake morphometric variables as well as watershed buffering capacity varied significantly among regions of increasing sulphate deposition across Ontario (Table 6.4). As these variables explained a significant amount of the variation in the water chemistry variables, then previous analyses of the association between sulphate deposition and

these water chemistry variables (Neary and Dillon 1988; Neary et al. 1990; OMOE 1991) have likely over-estimated the extent to which water chemistry has been influenced by sulphate deposition in Ontario.

There were only minor qualitative differences in patterns of water chemistry variables among sulphate deposition zones when assessed before and after controlling for lake morphometry and watershed buffering capacity. The qualitative differences in the patterns for pH and alkalinity are perhaps of greatest significance. When assessed without controlling for lake morphometry or watershed buffering capacity, it would appear that a threshold sulphate deposition rate of about 0.5 g S/m²/yr exists below which lakes do not appear to have been affected by acidic deposition (Figure 6.5). However, once the confounding influences of morphometry and buffering capacity have been eliminated, we observed no significant differences in both mean pH and mean alkalinity among the regions of Ontario receiving the four lowest rates of sulphate deposition (Figure 6.7). This suggests a possible threshold rate of sulphate deposition of between 0.75 and 1.0 g S/m²/yr (see Figure 6.2, and Neary et al. 1990 for deposition zones) below which neither pH nor alkalinity had yet been significantly influenced by acidic precipitation in the province, at least to the date by which the data employed here were collected (up to 1988). This is contrary to the conclusion of OMOE (1991)

that identified a monotonic decline in mean pH (chemistry apparently not controlled for lake morphometry or buffering capacity) of soft water lakes (conductivity $<50 \mu\text{s/cm}$) among all regions of progressively increasing sulphate deposition rate in Ontario. An examination of the data considered here for lakes with conductivity $<50 \mu\text{s/cm}$ did not reveal the same pattern as that reported by OMOE (1991). However, the sample size used here was only 161 lakes compared to 1,180 lakes considered by OMOE (1991).

The common assumption that lake [Al] increases with increasing acidic deposition was not supported by our analysis (Figure 6.5 and 6.7). An examination of only those lakes with low watershed buffering capacity ($n=236$), and thereby those watersheds with the greatest probability of having Al leached from the terrestrial drainage basin, still failed to find any direct association between lake Al levels and increasing sulphate deposition across Ontario.

Mean DOC levels (after controlling for the influences of lake and watershed morphometry and buffering capacity) were progressively lower in regions receiving progressively greater rates of sulphate deposition. By ANCOVA, sulphate deposition accounted for 21.6% of the residual variation in DOC (after controlling for lake morphometry and buffering capacity). More interestingly, the median sulphate deposition rate for

each sulphate deposition region accounted for virtually all (93.5%) of the variation in mean DOC, after controlling for lake and watershed morphometry and watershed buffering capacity (see Figure 6.8).

Although the extrapolation of this empirical observation to a cause-effect relationship may be contentious, a variety of experimental observations and theoretical considerations support this hypothesis. Sulphate deposition is known to influence lake pH and alkalinity (see Howells 1990 for review). The general decline in lake pH and alkalinity associated with sulphate deposition should also lead to a loss of DOC from the water column due to reduced DOC solubility (Thurman 1985). Due to DOC's acid neutralizing capacity (Dillon, pers. com. as cited by Schindler et al. 1992; OMOE 1991), DOC concentration in lake water would be expected to correlate with alkalinity (a competitor for ANC) and $[\text{SO}_4^{2-}]$ (H_2SO_4 being the proton donor).

It has been hypothesized that Al may control DOC levels in acidified lakes, through the formation and precipitation of DOC-Al complexes (Effler et al. 1985). This hypothesis predicts that as [Al] increases, [DOC] would decrease. Our results, however, suggest the opposite. The correlation coefficient between Al and DOC was positive ($r=+0.17$, $p<0.003$). After controlling for all other significant

factors, the partial regression coefficient of Al on DOC was also positive. Other authors (Schindler et al. 1992) have also indicated that the postulated link between DOC and Al may be incorrect. Instead, it may be that [Al] simply reflects the concentration of organic ligands (such as humic and fulvic acids) with which it tends to complex in natural waters (Howells 1990).

Multiple regression analysis (equation 6.1) suggests that sulphate deposition may act indirectly to reduce lake DOC, by influencing other water chemistry variables which, in turn, interact with DOC. Equation 6.1 agrees with known (experimental and/or theoretical) interactions between DOC and the other water chemistry variables, and indicates the potential complexity of the geochemistry of acid-stressed lakes. The observation that pH was not a significant correlate of [DOC] in Equation 6.1 agrees with the findings of Rasmussen et al. (1989) and suggests that a direct measure of $[H^+]$ input, such as $[SO_4^{2-}]$, may be a better indicator of labile acidity from acidic deposition than is pH.

Based on the foregoing analysis, it is apparent that future spatial studies of acidic deposition and its influence on lake water chemistry must endeavour to control for confounding factors before drawing conclusions about the extent of impact that has taken place on lake water chemistry.

Assuming that the spatial analysis presented here can be extrapolated to temporal trends in acidic deposition, then DOC could be an important, diagnostic characteristic of acidifying lakes, in conjunction with other chemical indicators. Also, the strong negative relationship between sulphate deposition and lake [DOC] that was observed here suggests that Hg levels in fish, which correlate positively with lake [DOC] (McMurtry et al. 1989; Wren et al. 1991; Sorensen et al. 1990; Heiskary and Helwig 1986), may indirectly decline with increasing sulphate deposition.

7.0 MERCURY CONCENTRATIONS IN FISH ARE LOWER IN REGIONS OF ONTARIO WITH HIGHER SULPHATE DEPOSITION RATES

7.1 Abstract

It has been hypothesized that concentrations of mercury (Hg) in fish increase with increasing acidic deposition. Our investigation suggests that this hypothesis may be false for at least three species of fish from drainage lakes. Across Ontario, slightly lower average concentrations of Hg in lake trout, walleye and northern pike, controlled for fish length as well as lake morphometry and watershed buffering capacity, were associated with regions receiving progressively greater rates of sulphate deposition. We also observed that lake concentration of dissolved organic carbon, not pH, alkalinity or sulphate ion concentration, was the strongest correlate of this residual fish Hg contamination. Based on these empirical observations, an alternate hypothesis is proposed whereby increasing sulphate deposition results in lower DOC levels in lakes which, in turn, cause a decline in fish Hg contamination. Path analysis also determined a net reduction in fish [Hg] at higher rates of sulphate deposition. However, this analysis indicated a far more complex web of interactions between sulphate deposition, water chemistry and fish [Hg] than simple correlation analysis could define. Path analysis also provided empirical evidence in support of several previously described mechanisms interacting simultaneously to influence fish Hg accumulation.

7.2 Introduction

It has been hypothesized that mercury (Hg) contamination in fish will increase due to acidic deposition (Goyer et al. 1985; Jernelov 1986; Winfrey and Rudd 1990; Gilmour and Henry 1991; Raloff 1991). This speculation has arisen from a number of independent observations: a) sulphate deposition leads to lake acidification, including reduced pH, reduced alkalinity and increased sulphate ion concentration (reviewed by Howells 1990); b) decreased pH is associated with increased Hg biomethylation rate (reviewed by Winfrey and Rudd 1990; Gilmour and Henry 1991); c) decreased alkalinity is associated with increased absorption of MeHg from the water column by fish (Rodgers and Beamish 1983); and d) increased sulphate ion levels in lakes may stimulate Hg biomethylation rate (Gilmour et al. 1992). Spatially, lake pH and alkalinity often correlate negatively with fish Hg contamination (reviewed in Chapter 5), adding support to this hypothesis.

However, a review of recent literature (see Chapter 5) suggested that this hypothesis may be false for predatory fish in drainage lakes. For drainage lakes, which are recharged by terrestrial runoff, Hg contamination in three fish species commonly consumed as food - lake trout (Salvelinus namaycush), walleye (Stizostedion vitreum vitreum) and northern pike (Esox lucius) - was predicted to decrease with increasing sulphate deposition.

The purpose of this chapter was to test directly the hypothesis that increasing sulphate deposition is associated with higher Hg contamination in fish from lakes across Ontario, Canada. During this analysis, we controlled for the statistical influences of physical (lake morphometry) and geochemical (watershed buffering capacity) factors which were anticipated to confound the association of interest. We specifically controlled the statistical analysis of fish Hg levels for the influences of total watershed area, lake volume, and the ratio of terrestrial drainage basin area to lake surface area. These have previously been shown to covary with sulphate deposition rate across Ontario (Chapter 6). They were expected to confound the association between sulphate deposition and fish Hg levels since sulphate deposition is thought to influence fish Hg levels indirectly via its influence on water chemistry, and these morphological variables influence lake water chemistry (Rasmussen et al. 1989; Chapter 6). We also controlled for watershed buffering capacity because of its expected influence on lake acidification rate (NRCC 1981), its covariation with sulphate deposition (Chapter 6), and its association with lake water chemistry (Chapter 6).

We also re-examined the spatial relationships between fish Hg contamination and water chemistry parameters in order to further evaluate empirical support for possible mechanisms

underlying observed associations between sulphate deposition and fish Hg levels. This was performed using standard correlation analysis as well as path analysis.

7.3 Methods

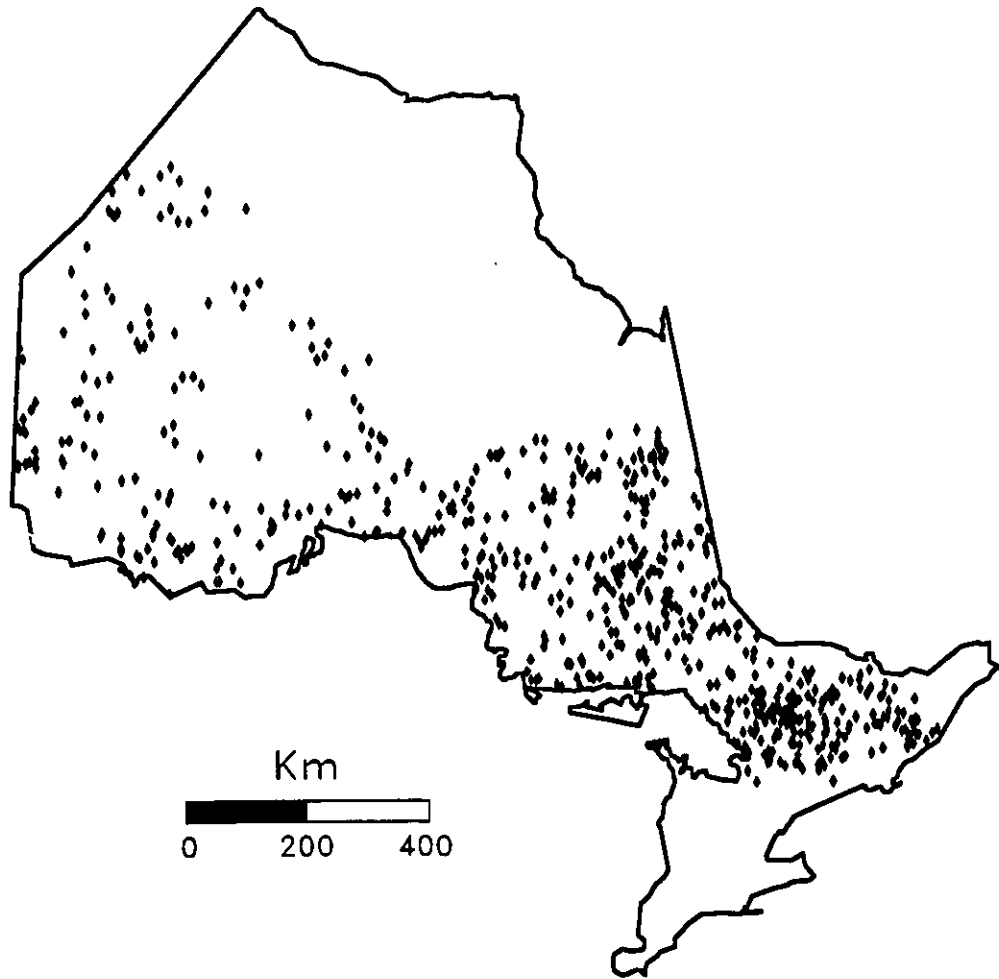
Unpublished data on fish length and mercury (Hg) concentrations in dorsal muscle for lake trout, walleye and northern pike were obtained from the Ontario Ministry of Environment (OMOE) and Ministry of Natural Resources (OMNR) (see Figure 7.1; Table 7.1). These species were selected because of their importance as food fish for recreational fishermen (OMOE 1993) and Ontario amerindians (Lawn 1989). Fish collection and analytical methods used to determine total Hg concentration in a skinless fillet of fish dorsal muscle have been described elsewhere (Wren et al. 1991; McMurtry et al. 1989). Fish collected from the English-Wabigoon River system, from the Great Lakes, and from known hydro-electric reservoirs were excluded from the analysis due to potential confounding by point source Hg contamination in fish in these lakes.

Lakes were categorized into one of seven sulphate deposition regions of Ontario, after the scheme of Neary et al. (1990) (see also Neary and Dillon 1988). The total range in sulphate deposition across Ontario is <0.25 to >1.25 g sulphur/m²/year.

Table 7.1. Summary of fish mercury data, collected from a total of 687 lakes, provided by Ontario Ministry of Environment and Ontario Ministry of Natural Resources (unpublished data).

Species	No. of specimens	No. of lakes	Geometric mean [Hg] (range) ($\mu\text{g/g}$)
lake trout	4,125	235	0.25 (0.01-10.0)
northern pike	5,914	396	0.44 (0.01-5.0)
walleye	7,337	375	0.52 (0.02-5.4)

Figure 7.1. Distribution of 687 lakes from which fish were collected. Some points represent the locations of more than one lake.



Lakes located in the extreme south western part of Ontario, which receives a median rate of sulphate deposition of 1.375 g sulphur/m²/yr (see Neary et al. 1990), were excluded because of insufficient data (only three lakes in total).

Lakes were also classified into one of three acid buffering capacity categories (low, moderate and high) after Cowell and Lucas (1986). These categories are qualitative indicators of the relative acid buffering capacity of the soil and bedrock of lake watersheds across the province. Where lake watersheds were composed of 2 or more buffering capacity categories, lakes were classified to that category which comprised the greatest part of the lake's perimeter. Lakes classified with watersheds containing predominantly naturally acidic soils (see Cowell and Lucas 1986) were excluded from analysis because the relative buffering capacity of such watersheds is unknown.

Unpublished lake water chemistry data were provided by OMOE (see Table 7.2). The distribution of lakes, and methods for the collection and analysis of water samples are described by Neary et al. (1990). Four variables were investigated for their relationship to fish mercury levels: pH, alkalinity (Alk), concentrations of dissolved organic carbon (DOC), and sulphate ion (SO₄²⁻). These were selected based on their purported role in mechanisms postulated to underlie the

hypothesized increase in fish [Hg] with increasing acidic deposition (discussed previously).

Lake morphometric data were obtained from two different sources. Data on lake surface area and lake volume were obtained from the OMNR (unpublished data). Data collection methods are summarized by Dodge et al. (1989). Total watershed areas (lake surface area + terrestrial drainage basin) were determined from 1:50,000 scale topographic maps using a polar planimeter. These data are also summarized in Table 7.2.

Hg data (\log_e -transformed) for all specimens of each fish species from all lakes were pooled and regressed as a quadratic function of fish length (\log_e -transformed), the single best predictor of fish Hg concentration for all species. The resulting regression residuals (the difference between predicted and observed values of fish [Hg]) represent the extent to which fish have more or less Hg than the average fish of comparable length. These residuals were subsequently averaged by lake and the mean residuals were regressed on morphometric and geochemical variables. The residuals of these subsequent regressions were then examined for patterns among sulphate deposition regions of Ontario, and were evaluated for significant associations with water chemistry parameters. In essence, the dependent variable in these

Table 7.2. Summary of lake water chemistry and morphometry data (geometric mean and range). Water chemistry data were not available for all lakes.

Parameter	lake trout lakes	pike lakes	walleye lakes
pH	6.7 (4.9-8.6) n=218	7.4 (5.9-9.8) n=298	7.4 (5.7-9.6) n=279
Alk ($\mu\text{eq/L}$)	5.4 ^a (-0.5-122.6) n=212	28.2 (1.6-161.2) n=288	27.5 (1.9-143.3) n=273
DOC (mg/L)	3.3 (1.2-9.9) n=108	7.4 (2.7-31.0) n=148	6.8 (1.7-26.5) n=173
SO ₄ ²⁻ (mg/L)	7.6 (2.8-22.5) n=137	4.9 (0.5-25.6) n=194	5.4 (0.5-29.0) n=197
total watershed area (h X 10 ⁴)	2072 (94.7-48503) n=235	3223 (114.0-587079) n=396	4340 (186.6-587079) n=375
DBA/LSA ^b	4.0 (0.96-25.3) n=235	5.0 (0.13-347.7) n=396	4.5 (0.13-263.0) n=375
volume (m ³ X 10 ⁴)	4398 (104.5-404007.2) n=235	2575 (35.3-495160.7) n=396	3840 (32.3-480404.0) n=375
maximum depth (m)	37.3 (6.1-119.0) n=219	16.5 (1.4-186.1) n=371	17.2 (1.0-119.0) n=352

a - excludes 9 values of alkalinity ≤ 0 .

b - DBA/LSA = (drainage basin area)/(lake surface area)

latter analyses was fish Hg concentration, averaged by lake, and free of the statistical influences of fish length, lake morphometry and watershed buffering capacity.

All continuous variables, except pH, were log_e-transformed to stabilize variance and to linearize their relationships with ln(fish [Hg]). All standard statistical analyses were performed using SYSTAT^R version 5.01 (Wilkinson 1991). Path analysis was conducted using EZ-Path^R version 1.0 (Steiger 1989), a module designed to run with SYSTAT^R.

Path analysis is an effective tool to elucidate the magnitude and directions (positive/negative) of complex interactions within a system where common regression or correlation analysis may be inadequate (Sokal and Rohlf 1981). Although most commonly employed in the social sciences (McDonald 1985), it is ideally suited to the analysis of complex environmental systems where chains or webs of interactions exist (Wright 1960). Path analysis has recently been applied to examine a variety of simultaneous influences on fish Hg contamination in Norwegian lakes (Fjeld and Rognerud 1993).

Path analysis generates a unique path coefficient (analogous to a correlation coefficient) for every postulated association between predictor and criterion variables. The

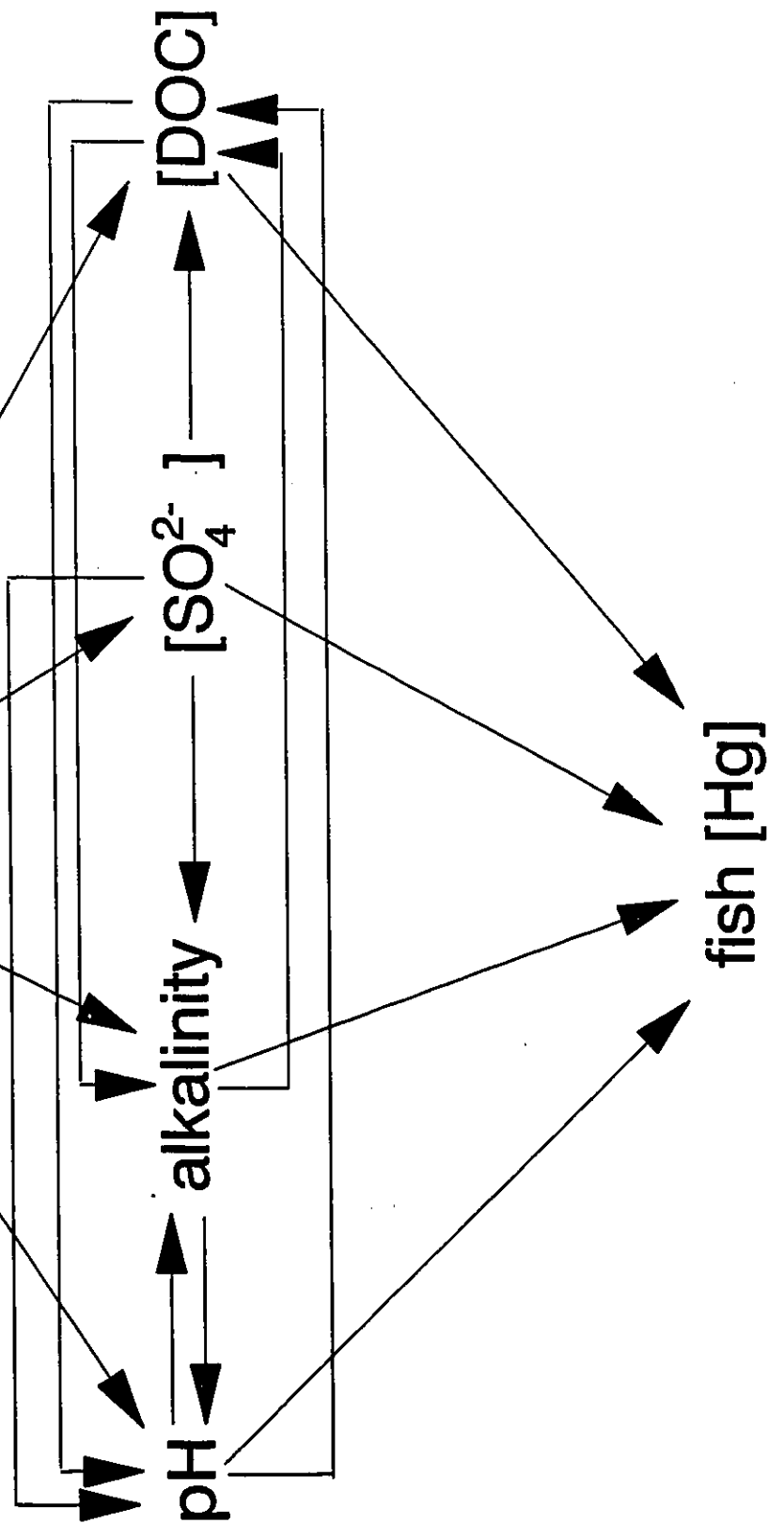
correlation between any two variables is the sum of products of the chain of path coefficients along all paths by which any two variables are directly and/or indirectly connected (Sokal and Rohlf 1981). The square of this correlation quantifies the proportion of variation in the criterion variable attributable to the predictor variable (Wright, 1960).

The statistical evaluation of path models can be complex (see Steiger, 1989), however a Chi square statistic and goodness of fit index (GFI) are typically used to evaluate how hypothesized models match the empirical data (Steiger 1989). A good fitting model will have a lower Chi square value than a poor fitting model, the associated probability indicating the likelihood of obtaining a larger Chi square statistic given that the defined model is correct. The GFI varies between 0 and 1, where a perfect fitting model has a GFI of 1. More detailed and technical discussions of path analysis are presented by Steiger (1989), MacDonald (1985), and Joreskog and Sorbom (1989).

Using subsets of 108 lake trout lakes (2,114 specimens), 148 pike lakes (1,988 specimens) and 172 walleye lakes (3358 specimens) for which all water chemistry, morphometry and buffering capacity data were available, 13,725 possible combinations and permutations of Figure 7.2 were submitted to path analysis, for each species. To determine the sulphate

Figure 7.2. Comprehensive model of hypothesized interrelationships between sulphate deposition, lake water chemistry and fish [Hg], as discussed in previous chapters. $[SO_4^{2-}]$ = sulphate ion concentration; [DOC] = dissolved organic carbon concentration; [Hg] = mercury concentration. Arrows depict hypothesized direct causal associations between predictor (independent) variables (at tail of arrow) and criterion (dependent) variables (at head of arrow).

Sulphate Deposition



deposition rate for each lake used in path analysis, lakes were first assigned to sulphate deposition zones according to the scheme of Neary et al. (1990). In general, the rate of deposition increases from north to south. Lakes could then be assigned a specific rate of sulphate deposition by adjusting for the distance north of the southern border for their zone, except for those lakes in the region impacted by smelter emissions from Sudbury for which sulphate deposition rates were based on their distance from Sudbury. This was done to create a continuous variable which is preferable for path analysis. The confounding influences of watershed buffering capacity and lake morphometry on water chemistry, and of fish length on fish Hg contamination, were controlled prior to analysis.

The most suitable models were selected in two stages. First all models with a Chi-square statistic with $p > 0.05$, and with a GFI > 0.8 were identified. Of those models with acceptable model fit, the one with the largest GFI and the fewest number of non-significant path coefficients was then selected as being the best overall model.

7.4 Results

A quadratic function of $\ln(\text{fish length})$ explained 57.1%, 32.0% and 40.1% ($p < 0.00005$ in all species) of the variation

in $\ln[\text{Hg}]$ for lake trout, walleye and northern pike, respectively. This variable was the single best predictor of individual specimen Hg levels. Lake and watershed morphometry and watershed buffering capacity together accounted for 13%, 16% and 23% of the variation in length-controlled $[\text{Hg}]$ in pike, walleye and lake trout, respectively. Length-standardized $[\text{Hg}]$ (i.e. Hg-length residuals), averaged by lake, were regressed on watershed buffering capacity (represented as dummy variables), the ratio of the terrestrial drainage basin area to lake surface area, total watershed area, and/or lake volume. The best-fitting regressions are summarized in Table 7.3.

Analysis of covariance (ANCOVA) of these multiple regressions (from Table 7.3) was then conducted among regions of sulphate deposition in Ontario. The results of these analyses are presented graphically in Figures 7.3a to c. Inclusion of sulphate deposition rate in the multiple regression model along with lake morphometry and watershed buffering capacity explained an additional 2.4%, 4.6% and 10.5% of total variation in length-controlled fish $[\text{Hg}]$ for pike, walleye and lake trout, respectively.

When residual fish Hg contamination, averaged by sulphate deposition zone, was regressed on median sulphate deposition rate for each zone, a weak but negative association was

observed for all species (Figure 7.3a to c). This association was only statistically significant ($p=0.02$) for walleye.

Average (by lake) fish Hg levels, controlled for fish length, lake morphometrics, and watershed buffering capacity, were then regressed on water chemistry variables. In all three species, the most significant correlate of mean residual [Hg] was DOC (Table 7.4). Although [Hg] in pike was negatively correlated with pH, this association was weaker, and less significant, than the positive association with DOC. In no case did $[SO_4^{2-}]$ prove to be a significant correlate of mean residual [Hg]. Weighted regressions among these variables were also performed, since sample size differed between lakes. The results were qualitatively similar: DOC was the most significant correlate of fish [Hg].

Figures 7.4 a and b present the best path model for walleye/ northern pike and lake trout, respectively. Data for walleye and northern pike were both fit by the same path model, indicating that DOC, pH and SO_4^{2-} have direct influences on fish [Hg]. The best model for lake trout, on the other hand, required an association between alkalinity and fish [Hg]. Path coefficients for the models depicted in Figure 7.4 are presented in Table 7.8.

Table 7.3. Summary of the regression models predicting mean (by lake) length-controlled fish [Hg] as functions of lake morphometry. The partial correlation of watershed buffering capacity and fish [Hg], after first controlling fish [Hg] for morphometric variables, is also shown. TWA = total watershed area; DBA = terrestrial drainage basin area; LSA = lake surface area; VOL = lake volume.

Species	Regression	partial R ² for Buffering capacity
lake	[Hg] = -1.99 + 0.08lnTWA + 0.50ln(DBA/LSA) + 0.08lnVOL	0.060 (p<0.001)
trout	R ² =0.178, p<0.000005, n=235	
walleye	[Hg] = 0.40 - 0.19lnTWA + 0.24ln(DBA/LSA) + 0.10lnVOL	0.036 (p<0.002)
northern pike	R ² =0.119 p<0.000005, n=375 [Hg] = 0.08 - 0.19lnTWA + 0.26ln(DBA/LSA) + 0.13lnVOL	0.030 (p<0.003)
	R ² =0.098, p<0.000005, n=396	

Table 7.4. Pearson correlation coefficients between residual fish [Hg] and water chemistry variables. Fish [Hg] values were lake means and were controlled for fish length, lake and watershed morphometry, and watershed buffering capacity. Only those lakes with data for all variables were included.

Fish species	ln[DOC]	pH	ln[Alk+1]	ln[SO ₄ ²⁻]
lake trout	0.481 ^{***}	ns	ns	ns
n=108				
walleye	0.411 ^{***}	ns	ns	ns
n=173				
northern pike	0.316 ^{**}	-0.210 [*]	ns	ns
n=148				

* p<0.01

** p<0.0001

*** P<0.00005

ns not significant, p>0.1

Figure 7.3. Fish Hg contamination as a function of sulphate deposition across Ontario. Fish [Hg] has been controlled for fish length, lake morphometry and watershed buffering capacity, and was then averaged by lake. Fish [Hg], fish length and lake morphometric variables were \log_{10} -transformed before analysis. Error bars are standard error of lake means averaged by deposition zone. Sulphate deposition zones after Neary et al. (1990). a) lake trout; b) walleye; c) northern pike. Letters above the bars indicate differences significant by Tukey-Kramer multiple range test. Numbers indicate sample size (number of lakes) per deposition zone. Lines represent the least squares regression curve for mean residual fish [Hg] on median sulphate deposition rate; only the regression curve for walleye is statistically significant.

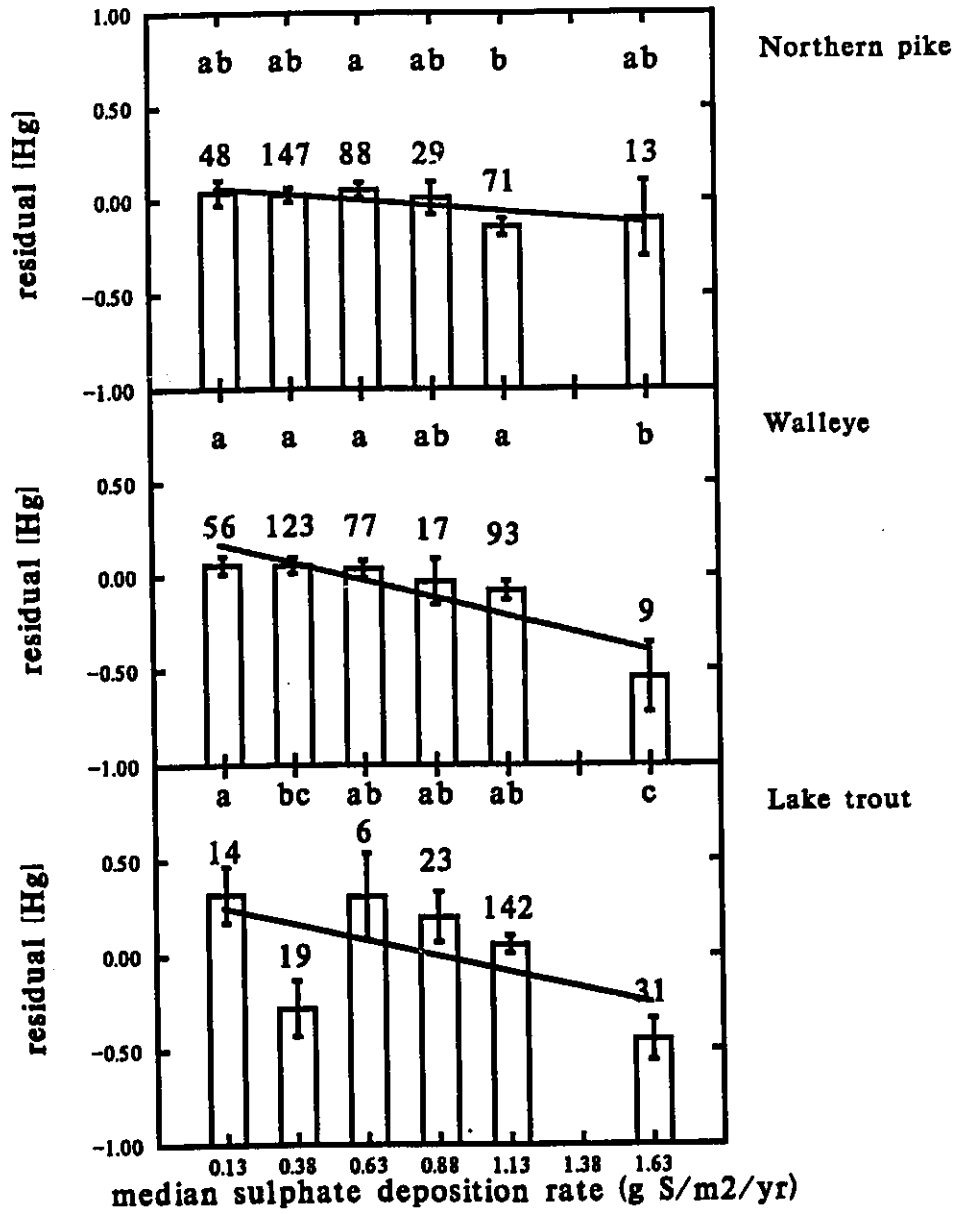


Table 7.5 Correlation matrix for lake trout from 108 Ontario lakes. Water chemistry data controlled for watershed buffering capacity and lake morphometry. Fish [Hg] data controlled for fish length.

	sulphate	pH	alkalinity	[SO ₄ ²⁻]	[DOC]
deposition					
zone					
sulphate deposition zone	1.0000				
pH	-0.5443	1.0000			
alkalinity	-0.5491	0.9105	1.0000		
[SO ₄ ²⁻]	0.6424	-0.2137	-0.2827	1.0000	
[DOC]	-0.4395	0.4291	0.5448	-0.2544	1.0000
trout [Hg]	-0.1529	0.1049	0.1469	-0.0966	0.5067

Table 7.6 Correlation matrix for northern pike from 148 Ontario lakes. Water chemistry data controlled for watershed buffering capacity and lake morphometry. Fish [Hg] data controlled for fish length.

	sulphate	pH	alkalinity	[SO ₄ ²⁻]	[DOC]
sulphate deposition zone	1.0000				
pH	-0.2954	1.0000			
alkalinity	-0.3459	0.9260	1.0000		
[SO ₄ ²⁻]	0.6669	-0.2061	-0.2716	1.0000	
[DOC]	-0.4304	0.2542	0.3236	-0.5271	1.0000
pike [Hg]	-0.0439	-0.1418	-0.0965	-0.0576	0.3052

Table 7.7 Correlation matrix for northern pike from 172 Ontario lakes. Water chemistry data controlled for watershed buffering capacity and lake morphometry. Fish [Hg] data controlled for fish length.

	sulphate	pH	alkalinity	[SO ₄ ²⁻]	[DOC]
deposition					
zone					
sulphate deposition zone	1.0000				
pH	-0.1866	1.0000			
alkalinity	-0.2627	0.9122	1.0000		
[SO ₄ ²⁻]	0.6831	-0.1349	-0.2078	1.0000	
[DOC]	-0.4912	0.2718	0.3450	-0.5509	1.0000
walleye [Hg]	-0.1486	0.0112	0.0709	-0.0637	0.3592

Figure 7.4 The best fitting path models for a) lake trout, and b) walleye and northern pike. Paths are numbered for cross reference to Table 7.8. GFI = Joreskog-Sorbom Goodness of Fit Index (see Steiger 1989; Joreskog and Sorbom 1989). To simplify presentation, unexplained variance in each parameter has not been represented. Water chemistry variables controlled for influence of lake morphometry and buffering capacity. $[SO_4^{2-}]$ = sulphate ion concentration; [DOC] = dissolved organic carbon concentration; Alk = alkalinity; [Hg] = mercury concentration. All variables but sulphate deposition rate, buffering capacity and pH were log_e-transformed.

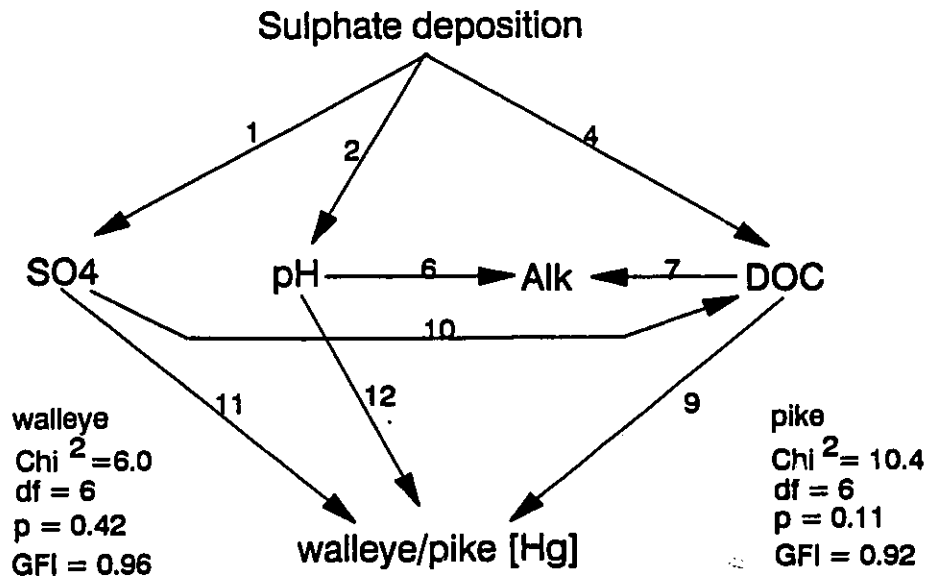
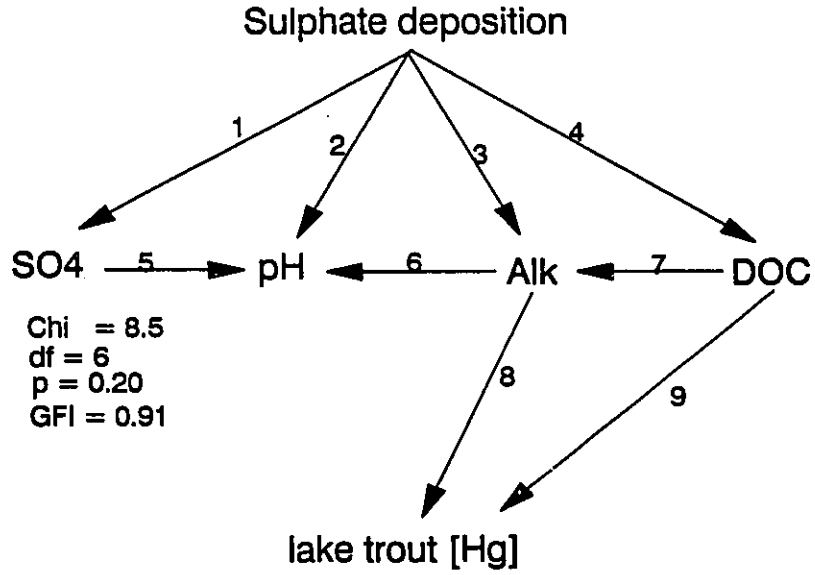


Table 7.8 Summary of path coefficients (\pm standard error) for models depicted in Figures 7.4. Path numbers correspond to those in the figures.

Path no.	lake trout	walleye	northern pike
1	0.64 \pm 0.07	0.68 \pm 0.06	0.63 \pm 0.06
2	-0.15 \pm 0.06	-0.32 \pm 0.07	-0.36 \pm 0.08
3	-0.38 \pm 0.08	--	--
4	-0.44 \pm 0.09	-0.31 \pm 0.08	-0.25 \pm 0.09
5	0.13 \pm 0.05	--	--
6	0.86 \pm 0.05	0.87 \pm 0.03	0.09 \pm 0.04
7	0.38 \pm 0.08	0.13 \pm 0.03	0.08 \pm 0.04
8	-0.13 \pm 0.10	--	--
9	0.59 \pm 0.10	0.49 \pm 0.08	0.34 \pm 0.09
10	--	-0.35 \pm 0.08	-0.32 \pm 0.09
11	--	0.20 \pm 0.09	0.10 \pm 0.09
12	--	-0.13 \pm 0.07	-0.21 \pm 0.08

The correlation matrices used for path analysis of each species are presented in Tables 7.5 to 7.7. These are reproduced so that the results of path analysis reported here can be verified, or other methods or software can be employed for comparative and confirmation analysis.

7.5 Discussion

The results reported here are inconsistent with the hypothesis that sulphate deposition leads to increased Hg contamination in walleye, northern pike or lake trout. In its simplest sense, this hypothesis would predict that the greatest level of fish Hg contamination would be observed in the region of the province receiving the greatest rate of sulphate deposition. This was not observed for any of the species examined. (see Figures 7.3a to c). Secondly, residual fish [Hg] averaged by sulphate deposition zone and regressed on median sulphate deposition rate should produce a positive association. In all three species, a negative association was observed, although this was only statistically significant in one of the species tested.

The hypothesis that fish [Hg] increases with increasing sulphate deposition predicts that one of pH, Alk or $[\text{SO}_4^{2-}]$ should be the strongest correlate of fish [Hg]. Instead, lake [DOC] was observed to be the most significant correlate of [Hg] residuals in all three species (see Table 7.4).

The results of our analysis are consistent with an alternative hypothesis whereby sulphate deposition leads to lower fish Hg contamination via an indirect mechanism involving lake [DOC]. DOC has been previously shown to have a strong negative association with sulphate deposition across Ontario (see Chapter 6). The negative association between sulphate deposition and fish [Hg] was much weaker than this, but this reflects, at least in part, low statistical power. Counteracting influences of pH and alkalinity on fish Hg levels, combined with uncontrolled influences such as food chain length (Cabana et al., in press) and perhaps primary productivity (Hakanson 1980) likely act to weaken this association.

Although Figures 7.4a and b present the best fitting path models we could define from the data examined, other models with only minor differences from those shown, did also fit the data (i.e. had Chi square statistic with $p > 0.05$ and $GFI > 0.8$). An examination of these results demonstrated the following consistencies among all models for all fish species:

1. a model must include a path from DOC to fish [Hg]. All models tested which excluded this path failed to fit the data.

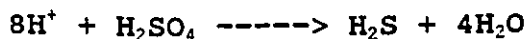
2. a model must contain at least one additional path to fish [Hg] other than from DOC. All models which excluded all paths to fish [Hg] except from DOC failed to fit the data.
3. a model must contain a direct path from sulphate deposition rate to DOC. Models excluding this association did not fit the data.
4. the net influence of sulphate deposition on fish [Hg] (i.e. sum of all paths linking sulphate deposition with fish [Hg]) was negative, indicating that fish [Hg] was lower at higher rates of sulphate deposition. This is predominantly due to the negative association (path coefficient) between sulphate deposition and [DOC], combined with the positive association between [DOC] and fish [Hg]. Although pH and/or alkalinity and/or SO_4^{2-} appeared to have an effect which counteracted that of DOC on fish [Hg], these counteracting influences were weaker.

The indirect influence of sulphate deposition on fish [Hg], via its influence on [DOC], can be determined by multiplying the path coefficient between sulphate deposition and [DOC] by the path coefficient between [DOC] and fish [Hg]. If this is repeated for all paths leading from sulphate deposition to fish [Hg], the sum of these products is then the

correlation between these variables (Wright 1960), and squaring this correlation quantifies the proportion of variance in fish [Hg] explained by sulphate deposition. Using this approach, sulphate deposition explained 3.6% of the total variation in length-controlled lake trout [Hg], 3.0% of the variation in walleye [Hg], and only 0.02% of the variation in pike [Hg]. As water chemistry was controlled for the confounding influence of lake morphometry and watershed buffering capacity prior to path analysis, then sulphate deposition likely explains even less of the overall variation in the raw, uncontrolled data.

The apparent biogeochemistry of Hg in lake trout lakes and lake trout is very different from that for pike and walleye lakes. This may relate to the differing influence of morphometric characteristics of the two lake types. Lakes inhabited by lake trout are significantly deeper but with small watersheds than those inhabited by walleye and/or pike (Table 7.2). Therefore, the balance of geochemical processes can be expected to differ. This would explain the observed differences in the relationships between fish [Hg] and lake morphometric variables among these species (Table 7.3). The fact that trout lakes are much deeper may also explain the positive path coefficient between $[\text{SO}_4^{2-}]$ and pH in Figure 7.4a. Loss of sulphate from lakes is a function of anaerobic microbial sulphate reduction (Gilmour et al. 1992). Deep

lakes will have a greater anoxic zone and, therefore, an expected greater rate of sulphate reduction. This reaction:



would reduce H^+ concentrations in these deep lakes despite the increase in H_2SO_4 input with increasing sulphate deposition, leading to a non-intuitive positive relationship between lake $[\text{SO}_4^{2-}]$ and lake pH. As anaerobic sulphate reduction fuels methanogenesis (Gilmour et al. 1992), this will also consume further excess H^+ in the breakdown of complex carbohydrates and hydrocarbons to CH_4 .

These species differences may also relate to ecological, physiological or habitat differences between species. For example, the respiratory absorption of MeHg may be more significant in lake trout than the other species, thus explaining the role of alkalinity in the trout path model (Figure 7.4 a). Also, lakes in which lake trout occur tend to be more dilute (lower alkalinity), and have lower DOC than pike or walleye lakes (see Chapters 6 and 7). This may influence the relative significance of the various mechanisms controlling Hg methylation and/or bioavailability. Further data for specimens of all three species coexisting in the same lakes would be required to better test these hypotheses.

The fact that path models for all species required a direct association between sulphate deposition rate and DOC contradicts earlier regression analyses which suggested that the influence of sulphate deposition on DOC might be indirect (see Chapter 6). These contradictory results can not be explained with the available data but do raise some questions and concerns regarding the appropriate methods for analysis of complex interrelationships as those evaluated here. Due to its ability to simultaneously evaluate parameters as both criterion (dependent) and predictor (independent) variables, path analysis is considered the superior technique for the data examined here.

This path analysis has been conducted on spatial data and its extrapolation to temporal trends in acidic deposition and lake acidification is debateable. However, such extrapolation suggests two interesting points. First, it would appear on first inspection that sulphate deposition would lead to a decrease in Hg contamination in fish, as discussed in Chapter 7. This would subsequently lead to a reduction in Hg exposure in persons eating these fish, a result which has also been discussed (see chapter 11). Secondly, assuming that the quantity of DOC in lakes is finite, then fish [Hg] might rebound, due to the subsequent influence of pH, alkalinity or SO_4^{2-} once DOC is removed from the water column. In seepage lakes, which receive little or no terrestrial DOC, it appears

that increasing sulphate deposition might be associated with increasing fish [Hg] (see Chapter 5; Wiener et al. 1990; Cope et al. 1990; Grieb et al. 1990; Miskimmin et al. 1992). Such an impact might also arise in drainage lakes, but only after DOC is removed from the water column, likely through precipitation with increasing acidification (Thurman 1985), or after the rate of DOC transport to the lake from the watershed is sufficiently reduced, due to reduced DOC solubility in acidified runoff (Schindler et al. 1992; de Haan 1992). Therefore, the conclusion that sulphate deposition may lead to reduced fish [Hg] (Chapter 8) and reduced human Hg exposure (Chapter 11) may, in part, result from the analysis of data collected prior to DOC depletion and before the alternate influence(s) of pH, alkalinity and SO_4^{2-} had been effected.

Further work in this area should include the following studies: 1) the models depicted in Figures 7.4 a,b should be tested through standard experimental techniques; 2) the hypothesis should be tested that the influence of water chemistry variables on fish Hg is dependent on the duration as well as the rate of sulphate deposition; 3) new data should be collected for the same lakes evaluated here and submitted to path analysis to determine if a water chemistry parameter other than DOC is now dominant in the associations between sulphate deposition and lake water chemistry, and between water chemistry and fish [Hg].

8.0 ESTIMATING FISH CONSUMPTION RATES FOR ONTARIO AMERINDIANS

8.1 Abstract

Estimates of fish consumption rate were determined for Ontario amerindians from data on the concentration of mercury (Hg) in the hair of 4,327 amerindians residing in 58 reserves across the province, combined with data on Hg concentrations in three commonly consumed species of fish collected from lakes surrounding these reserves. Estimated rates of fish consumption were found to differ between sexes, with males consuming a geometric mean of 19 g of fish per day, while females were estimated to consume a geometric mean of 14 g/day. Fish consumption rate was found to increase with degree of community isolation, and to increase with age. Seasonal variation was also noted, with fish consumption rates being highest during summer months and lowest in winter.

8.2 Introduction

Consumption of chemically-contaminated freshwater fish can be a significant source of human exposure to, and risk from, persistent contaminants such as mercury (Hg) (Clarkson 1990; Jacobson and Jacobson 1988; Fein et al 1984; Foran et al. 1989a,b). Chronic contamination has lead some state and provincial governments to publish recommendations regarding the rate of fish consumption which may be considered safe (OMOE 1993, for example), and to develop methods to assess chemical exposure and risk (Health and Welfare Canada, unpublished¹; U.S. E.P.A. 1986) due to fish consumption.

Annual per capita 'apparent' fish consumption (fish + shellfish, including fresh, frozen, cured and canned) in Canada was determined to be approximately 16 g/day, based on data relating to the disappearance of domestic fish supplies (CCAC 1977). A national dietary survey (HWC 1977; Conacher et al. 1989) estimated the national average rate of consumption of all fish and shellfish in Canada to be about 11 g/person/day. The national average rate of consumption of freshwater fish was much lower at only 1.2 g/person/day (Conacher et al. 1989).

¹ Health and Welfare Canada undertakes to assess risks posed by contaminants in fish, although the procedures followed are not formally published.

The rate of fish consumption by Canada's Native peoples is greater than the national average (HWC 1977), although this has not been extensively quantified. Many communities of native peoples, particularly isolated ones, subsist on fish as a major component of their diet (HWC 1985; Wheatley and Wheatley 1981; Kinloch et al. 1992; Kuhnlein 1984; Lawn 1989; Lutra Assoc. 1989; Szathmary et al. 1987; Berkes 1990; Hopper and Power 1991). Unfortunately, few studies have been performed to quantify the rate of fish consumption by these communities, and, therefore, the risk posed to them by fish-borne contaminants can not be assessed adequately.

A number of surveys of fish harvest by northern communities have been undertaken (see review by Berkes 1990; see also Lutra Assoc. 1989, and Hopper and Power 1991) from which a crude measure of fish consumption can be determined as harvest per capita. Unfortunately, such studies often report total and not edible fish weight. They also often fail to account for fish that is not consumed (used as dog food, or trap bait, for example) or for quantities given to members of neighbouring communities (Berkes 1990; Hopper and Power 1991). Therefore, estimates of fish consumption rate based on fish harvest surveys will likely overestimate consumption. A few dietary surveys have been done for communities of Canadian native peoples (Wein et al. 1991; Kuhnlein 1989; Innis et al. 1988; Kinloch et al. 1992) which make more precise

measurements of fish (and other food) consumed, usually during the 24 hours or so preceding the administration of the survey. However, the rates of fish consumption reported in these studies are only representative of the small communities upon which they were undertaken, and the day(s) during which they were conducted. There are no published sources of information which permit the determination of fish consumption rates for a relatively large segment of the Canadian native population, which could be applied to the routine assessment of exposure to fish-borne environmental chemical contaminants. Also lacking are data to help identify specific groups or communities which might be at greater risk due fish consumption at rates higher than the average for this population.

Methylmercury (MeHg), a neurotoxin, is the most toxic form of Hg in man (HWC 1986). Non-occupational exposure to MeHg via inhalation and water ingestion is insignificant compared to food (FAO/WHO 1972), and the amount found in plant produce is very small or nil (FAO/WHO 1972). MeHg is the predominant form of Hg found in fish (Huckabee et al. 1979), and consumption of fish, especially freshwater fish, is by far the most significant source of exposure to MeHg (FAO/WHO 1972; WHO 1976; Clarkson 1990).

Freshwater fish consumption comprises at least 65% of estimated dietary MeHg intake by Ontario amerindians. This is based on the data summarized by Desai-Greenaway and Price (1976) for Hg levels in fish and wildlife consumed by amerindians in that province, and rates of consumption of fish, waterfowl and other wildlife by amerindians, based on a review of literature (Coad 1993).

Virtually all of the MeHg ingested is absorbed (HWC 1986), with the halflife in the human body being about 70 days (HWC 1986). Ingested MeHg is distributed throughout the body into most tissues, including hair (WHO 1976). The concentration of Hg in hair is linearly related to the rate of daily Hg ingestion (WHO 1976; Kershaw et al. 1980). As a result, with fish consumption being the primary source of MeHg exposure, one can estimate the rate of fish consumption by an individual from data on the concentration of Hg in the fish being consumed and observed Hg levels in that individual's hair.

The release of Hg to the English-Wabigoon River system in Ontario from industrial sources resulted in the extensive contamination of fish with MeHg (Rudd et al. 1983). This lead the Canadian federal government to establish a Hg monitoring program for amerindian communities in this region, which was later expanded to other parts of the province and country

(Wheatley 1979; Tupper 1984). Samples of hair were collected and analyzed for Hg content.

The Ontario Ministry of Environment and Ministry of Natural Resources have, for some years, been collecting fish from across Ontario and analyzing them for Hg (and other contaminants). These two distinct, large data sets, when combined, provide a unique opportunity to estimate fish consumption rates for a relatively large segment of the amerindian population of Ontario.

8.3 Methods

Concentrations of total Hg in hair samples taken from 4,327 adult (age \geq 16 years) amerindian Canadians (1,789 males, 2,538 females) residing in 58 reserves in Ontario (see Figure 8.1) were obtained from Medical Services Branch, Health and Welfare Canada. Hair samples comprised a tuft of several hundred hairs, cut as close to the scalp as possible. These samples were collected between 1975 and 1990, with 82.7% of samples collected prior to 1985. Hair sample collection procedures are described by Health and Welfare Canada (1987). Methods of Hg analysis are described by Farant et al. (1981).

The raw data consisted of the total Hg concentration in 6 separate 1 cm long segments of the hair sample collected

from the 4,327 individuals. One of the six hair segments for each individual was selected at random and these subsampled data were used for further analysis. We did not use more than one sample from a given individual so that samples would be statistically independent.

A variety of other information was also available. This included the sex and date of birth of the individuals from whom the hair samples were collected, the date of sample collection, the concentration of inorganic Hg (for 1,208 samples only), the distance from the scalp of each of the six 1 cm segments of the hair sample, and the reserve of residence.

Hair grows at a rate of 1 cm per month (Pelfini et al. 1969; Dennis et al. 1972), and this rate of growth has been used repeatedly for the analysis of trends in Hg exposure (see Clarkson 1990; Al-Shahristani et al. 1976; Giovanoli-Jakubczak et al. 1974; Phelps et al., 1980; Amin-Zaki et al. 1976; and references there-in). As a result, the Hg concentration in each segment of hair sample could be ascribed to the month the Hg was deposited in the hair (i.e. the month during which Hg-contaminated fish was consumed), and an examination of seasonal patterns of fish consumption was possible.

Figure 8.1. Distribution of Indian reserves across Ontario considered in Chapter 8.

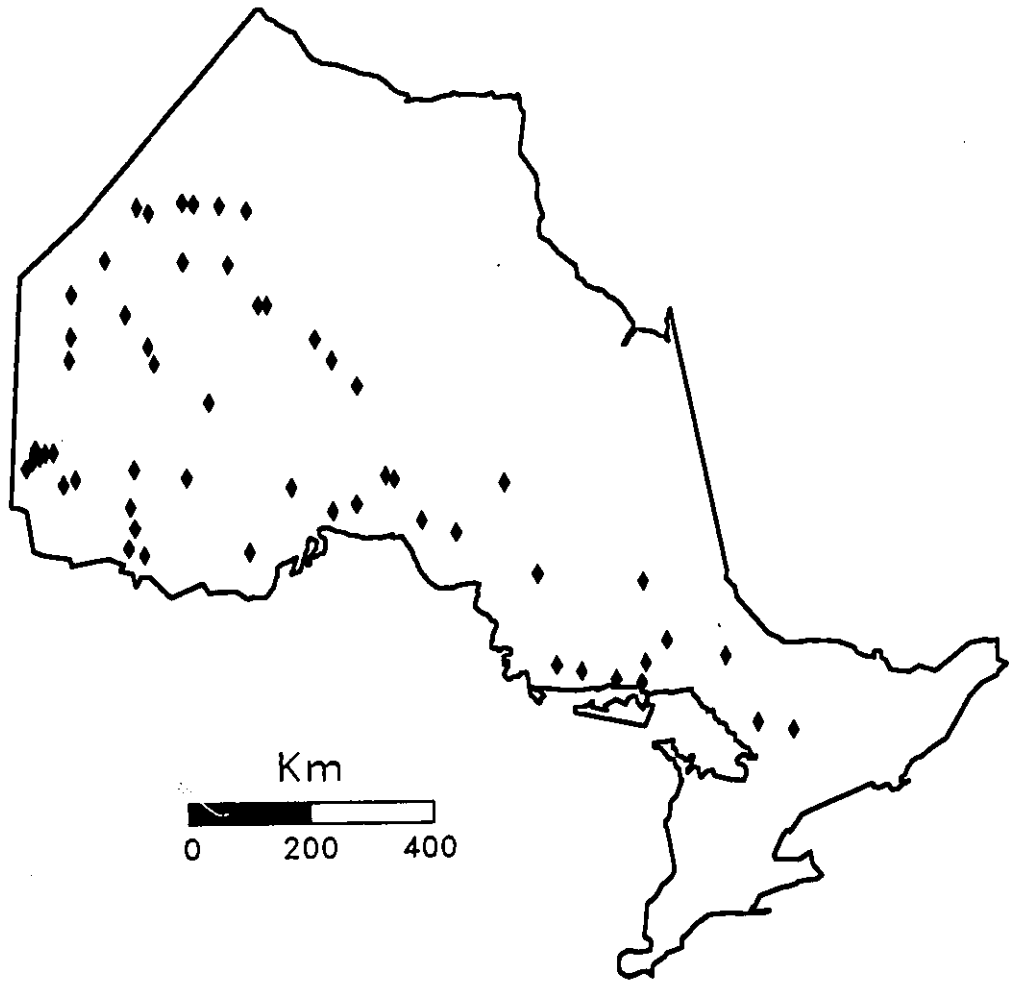
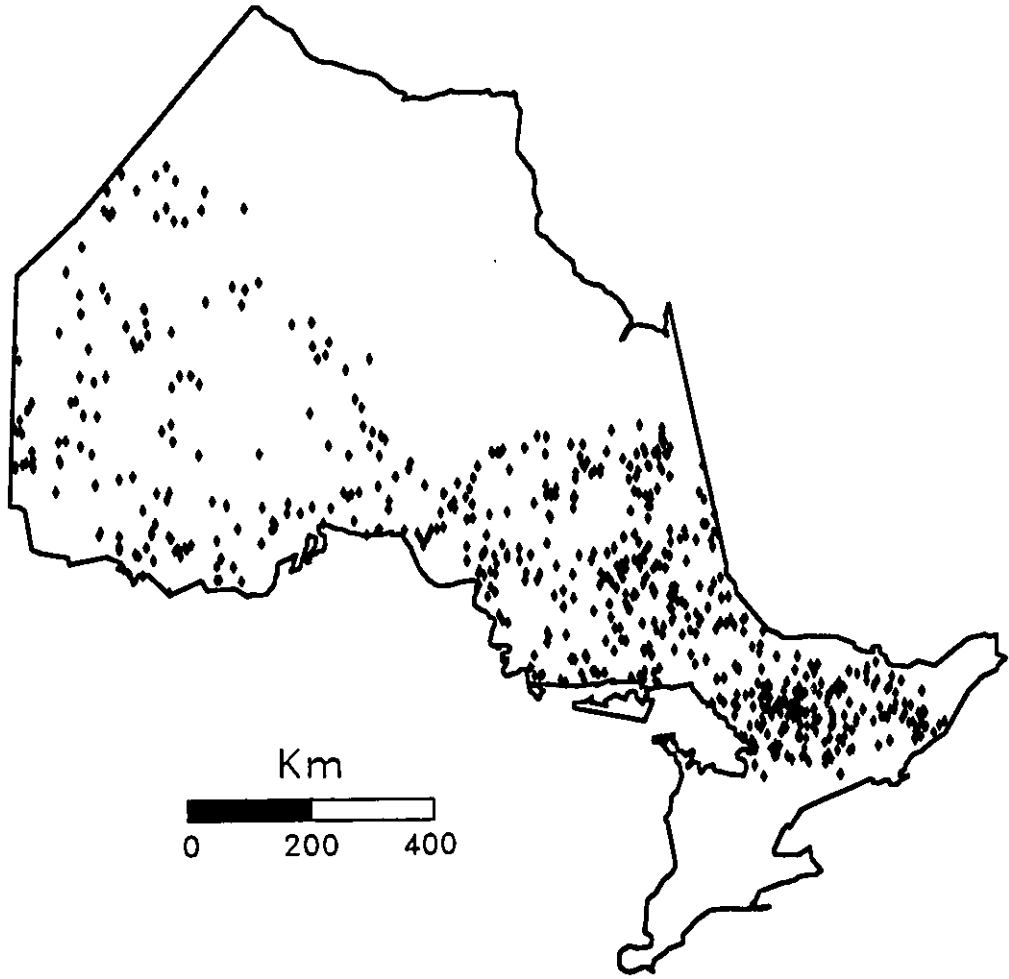


Figure 8.2. Distribution of lakes across Ontario from which lake trout, walleye and/or northern pike were collected. Some points represent locations of more than one lake.



Ontario Ministry of Environment and Ministry of Natural Resources jointly supplied unpublished data on fish length, and the Hg concentration from a sample of dorsal muscle for lake trout (Salvelinus namaycush), walleye (Stizostedion vitreum vitreum) and northern pike (Esox lucius) collected from Ontario lakes (see Figure 8.2). These species were selected for their importance as food fish for Ontario amerindians (Hopper and Power 1991; Lawn 1989; D.Dimitroff, HWC, pers. com.²). Fish were collected between 1978 and 1984, corresponding well with the period during which amerindian hair samples were collected. Fish collection and analytical methods used to determine total Hg concentration in a skinless fillet of dorsal muscle are described in Wren et al. (1991) and McMurtry et al. (1989). MeHg in fish is typically 80% to 99% of total Hg (Huckabee et al. 1979; Grieb et al. 1990). Our analyses are based on total Hg concentrations.

In order to determine a mean concentration of Hg in fish from which rates of fish consumption could be calculated, a 'zone of influence' was defined around each reserve as plus and minus 100 km of the reserves' latitude and longitude coordinates. This zone was set subjectively, but the only published report citing distance between the reserve and lakes fished (Hopper and Power 1991) indicated that a distance of up

² Dr. D. Dimitroff, Environmental Health Services, Medical Services Branch, Health and Welfare Canada, Ottawa.

to about 100 km was a reasonable assumption. A lake was assigned to as many reserves as required by the dimensions of each zone of influence.

Since fish Hg concentration data were approximately log-normally distributed, we first \log_e -transformed the data. Hg concentrations for all fish specimens (regardless of species) within a zone of influence were then averaged and the geometric mean fish [Hg] was used to estimate fish consumption rates. Fish species consumed by amerindian communities generally reflect their availability in the local environment (D.Dimitroff, pers.com.). Therefore, our estimates based on all specimens of all species combined assume that the frequency of capture by OMOE is the same as that by native fishermen. We repeated these calculations for a zone of influence of ± 50 km around each reserve, and we obtained essentially the same results as those presented below.

For adult humans, Hg absorption from the gut, its distribution in the blood, and its deposition in the hair are linear functions of the rate of Hg ingestion (WHO 1976; Kershaw et al. 1980). Therefore, at steady state, the concentration of Hg in human hair is a linear function of the level of Hg contamination in fish and the rate of fish consumption. The rate of fish consumption can therefore be estimated as follows:

$$\text{Hg intake}(\mu\text{g}/\text{day}) = \text{fish consumption rate}(\text{g}/\text{day}) * \text{fish}[\text{Hg}] (\mu\text{g}/\text{g}) \quad (1)$$

$$\text{blood} [\text{Hg}] (\mu\text{g}/\text{ml}) = a * \text{Hg intake} (\mu\text{g}/\text{day}) \quad (2)$$

$$\text{hair} [\text{Hg}] (\mu\text{g}/\text{g}) = b * \text{blood} [\text{Hg}] (\mu\text{g}/\text{ml}) \quad (3)$$

Therefore:

$$\begin{aligned} \text{hair} [\text{Hg}] (\mu\text{g}/\text{g}) &= a * b * \text{fish consumption rate} (\text{g}/\text{day}) \\ &\quad * \text{fish} [\text{Hg}] (\mu\text{g}/\text{g}) \end{aligned} \quad (4)$$

and:

$$\text{fish consumption rate} (\text{g}/\text{day}) = \text{hair} [\text{Hg}] / (\text{fish} [\text{Hg}] * a * b) \quad (5)$$

Values for *a*, the ratio of the steady-state blood Hg concentration to daily dietary intake of Hg, estimated from studies on Hg-exposed populations, vary between 0.0003 and 0.0008 (see WHO 1976 for review), however, difficulties exist in the precise determination of dietary intake in these population studies (WHO 1976). Radioactive tracer studies on volunteers, in which precise measurements of dietary intake can be determined, provide a more reliable determination, indicating a value for *a* of 0.0010 (WHO 1976; Kershaw et al. 1980). The more precise value of 0.0010 was selected by WHO (1976) for their evaluation of exposure to MeHg, and was also used here.

The ratio of hair [Hg] to blood [Hg], *b*, is about 250 in populations exposed to MeHg (reviewed WHO 1976). As with determinations of *a*, tracer studies are preferred for their ability to accurately quantify Hg ingestion. Kershaw et al. (1980) provide a more precise estimate of 292 (95% C.I. 262-

322), based on an experimental tracer study, which was used in this study.

Fish consumption rates were then estimated for individual amerindians of each sex, using equation (5). Fish consumption rate estimates were also found to be log-normally distributed. Geometric mean estimates were determined by reserve, and then tested for trends with latitude (as a surrogate for community isolation), and with proximity to urban centres, after INAC (1991). This scheme identified lakes as air access only (no roads), remote (road access >350 km), rural (road access 50 to 350 km) and urban (≤ 50 km from a city). Geometric mean values were also determined by age group to examine trends with age. Finally, geometric mean consumption rates were determined by month of Hg exposure (i.e. month preceding sample collection), and examined for seasonal variation. All data analyses were carried out using SYSTAT^R version 5.01 (Wilkinson 1991).

8.4 Results

While total Hg was measured in all samples, inorganic Hg was only measured in 28% of the samples. MeHg, the difference between total and inorganic, was $\geq 80\%$ of the total Hg in 92.8% of those samples tested, and MeHg and total Hg were strongly correlated ($r > +0.99$). Therefore, total Hg was assumed to represent MeHg.

Table 8.1 presents a summary of the Hg data for Ontario amerindians employed in this analysis, and Table 8.2 summarizes the data for the three fish species. Table 8.3 presents Ontario-wide means of \log_e -transformed fish consumption rates for male and female amerindians, and for both sexes combined. Based on these data, the geometric mean consumption rates were 18.9 g/day for males, 14.4 g/day for females and 16.2 g/day for both sexes considered together.

Reliance on 'country' foods, such as freshwater fish, is known to increase with community isolation (Szathmary et al. 1987). Also, significant subsistence fisheries exist in Native communities in Canada's northern (subarctic and arctic) regions (Hopper and Power 1991; Berkes 1990; Lutra Assoc. 1989). These practices should be reflected in our estimated fish consumption rates. To test this hypothesis, we estimated geometric mean fish consumption rates by reserve, as the locations of reserves vary with latitude. The degree of isolation (i.e. population density) also increases with latitude (EMRC 1974). Fish consumption rates increase with latitude for both males and females (Figure 8.3), although the difference between regressions for each sex is not significant ($F=0.156$, $p>0.6$, $df=1$). Weighted regressions were also performed (to account for differences in sample size between reserves) and results were very similar to those for unweighted regressions.

ANOVA of mean consumption rates by proximity of reserves to urban centres (INAC 1991) also demonstrated significantly higher consumption rates as proximity to cities decreases (Figure 8.4).

Use of 'country' foods is also thought to be age dependent, with older individuals generally consuming greater quantities of fish than younger individuals (Kinloch et al. 1992; Wein et al. 1991; Kuhnlein 1989). Figure 8.5 presents fish consumption rate estimates for males and females of three different age groups. Differences in geometric mean consumption rates between all age groups, for both sexes, were significantly different ($p < 0.0005$; Tukey-Kramer multiple range test). The geometric mean fish consumption rate for both sexes of the oldest age class (≥ 51 years) was found to be about three times that of the youngest age class (16-25 years).

Fish consumption is likely to reflect seasonal availability of the resource. Based on a hair growth rate of 1 cm/month, the distance of the hair segment from the scalp, and the date of hair sample collection, the month the Hg was deposited in the hair (i.e. month of fish consumption) was determined. Figure 8.6 shows geometric mean consumption rate estimates by month, for both males and females. It is quite apparent that consumption rates vary seasonally, generally

Table 8.1. Summary of data for Ontario amerindians. Mean age and mean hair Hg concentration are significantly different between males and females (t-test, $p < 0.0005$).

Sex	no. of individuals	geometric mean age (range) (years)	geometric mean hair [Hg] (range) ($\mu\text{g/g}$)
male	1789	31.9 (16-86)	2.7 (0.3-128.0)
female	2538	29.6 (16-89)	2.1 (0.2-38.4)

Table 8.2. Summary of mercury levels in fish sampled in Ontario lakes.

Species	no. of specimens	no. of lakes	geometric mean [Hg] (range) ($\mu\text{g/g}$)
lake trout	2,968	182	0.20 (0.01-6.13)
walleye	5,620	334	0.50 (0.01-5.42)
northern pike	4,657	360	0.45 (0.02-5.00)
all fish specimens combined	13,245	607	0.39 (0.01-6.13)

Table 8.3. Ontario-wide means for \log_e -transformed fish consumption rate (g/day) (\pm s.d.) for Amerindians age \geq 16 years. The difference between males and females is significant (t-test, $p < 0.0005$). Geometric means can be determined as the antilog_e of the mean values presented in the Table. The data are presented in this form to permit the determination of confidence limits for the sampled population (see text).

Sex	mean \log_e (consumption rate)
	(\pm s.d.)
both	2.784 (± 1.161) n=4327
M	2.937 (± 1.204) n=1789
F	2.677 (± 1.119) n=2538

Figure 8.3. Mean fish consumption rate versus latitude:
(a) males; (b) females. The solid line is the least squares
regression curve. Slopes are not significantly different
between the sexes.

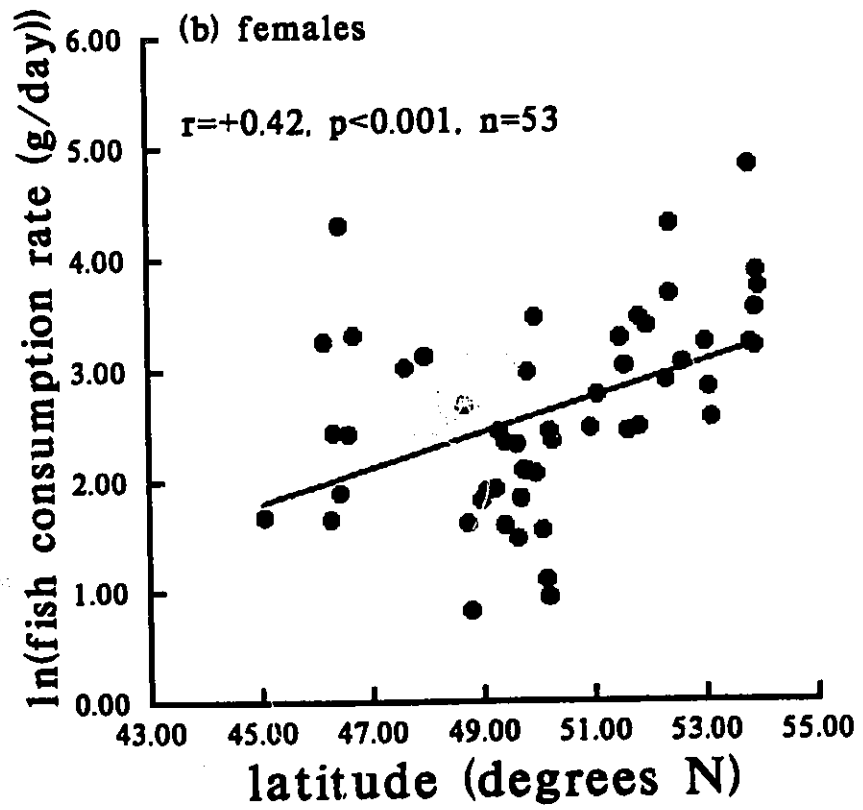
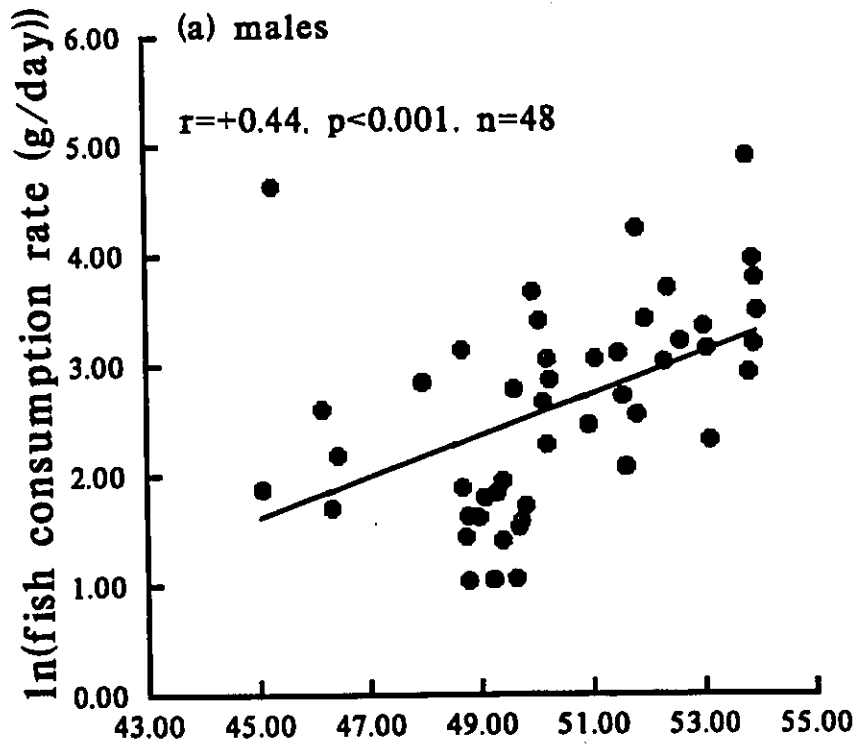
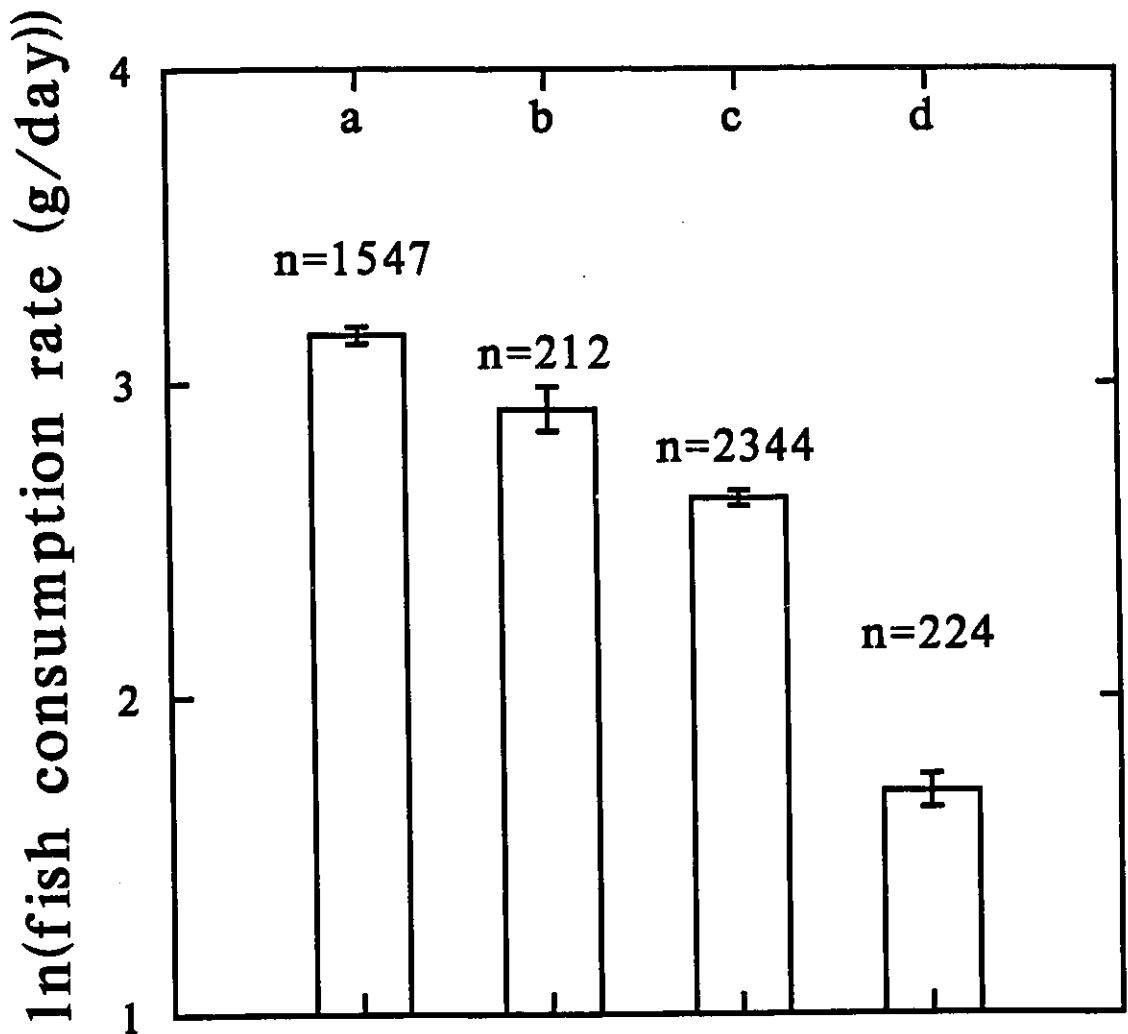


Figure 8.4. Analysis of variance of fish consumption rate with proximity to urban centres. Air=air access only; remote=road distance>350 km; rural = road distance between 50 and 350 km; urban = road distance \leq 50 km. Letters of bars indicate differences significant by Tukey-Kramer multiple range test. Numbers above the bars indicate sample size.



Relative remoteness of reserve

Figure 8.5. Mean (\pm s.e.) fish consumption rate by age group. Differences between all age groups, for each sex, are significantly different at $p < 0.0005$ (Tukey-Kramer multiple range test).

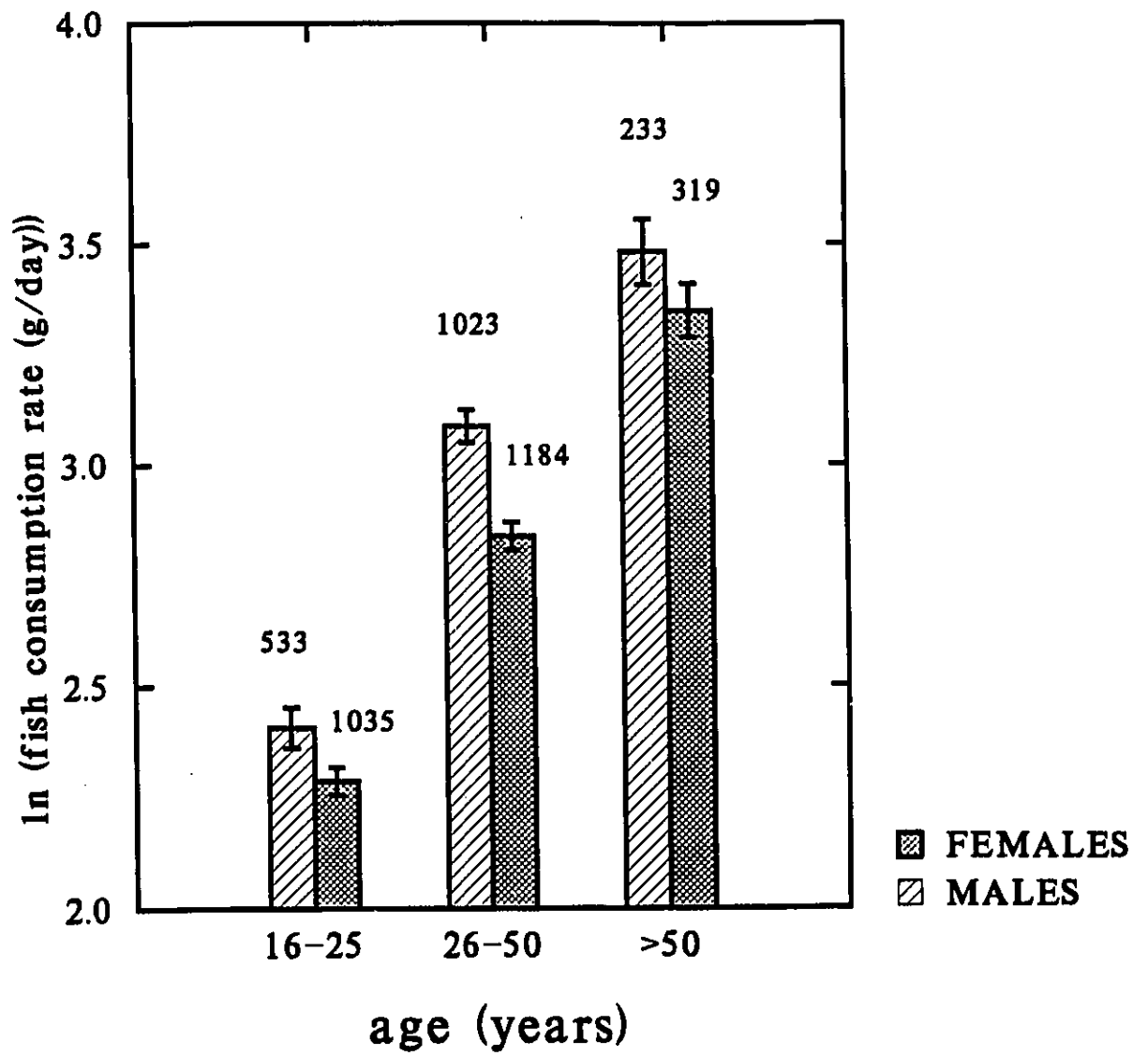
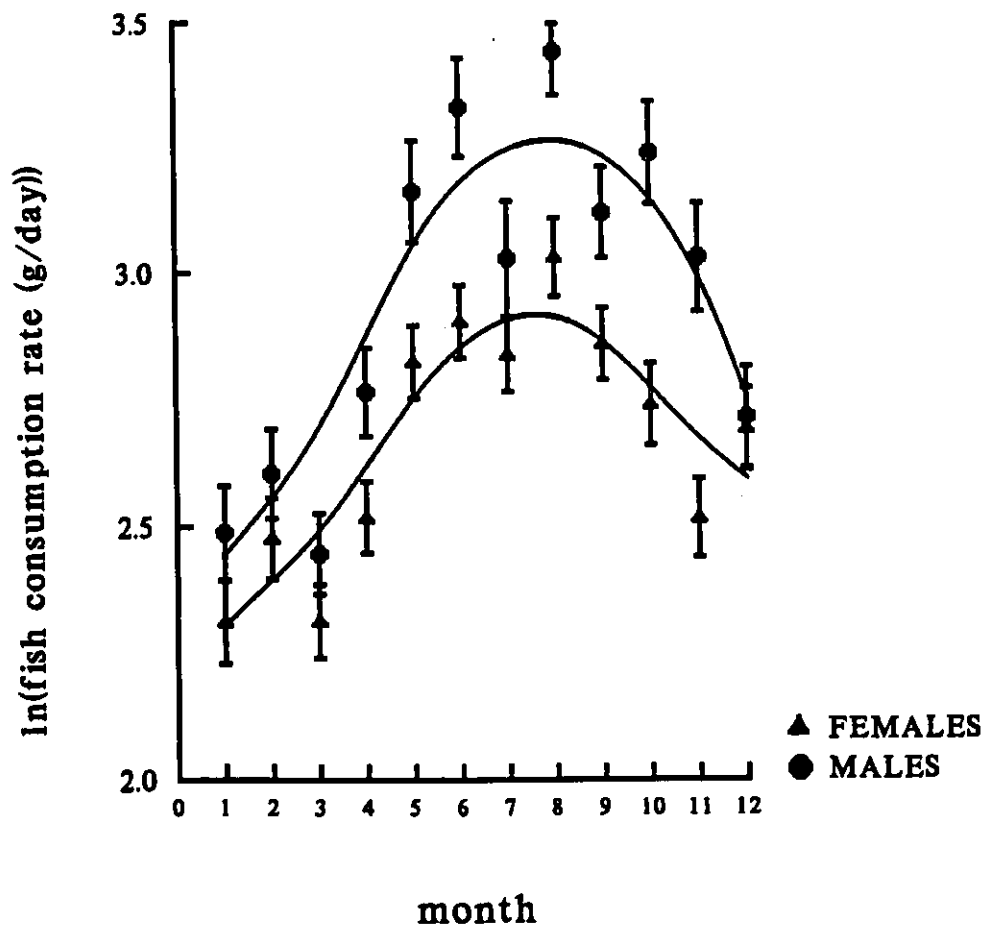


Figure 8.6. Mean estimated fish consumption rate by month of Hg exposure. The solid lines have been drawn using distance weighted least squares smoothing, and are included to better depict the cyclic nature of fish consumption patterns.



peaking in summer months and being lowest in winter. This pattern is consistent with that observed by Phelps et al. (1980), Wheatley (1979), and with the general seasonal variation in fish harvest reported by Hopper and Power (1991).

8.5 Discussion

The geometric mean fish consumption rate was estimated to be 19 g/day for males, 14 g/day for females, and 16 g/day with both sexes combined. Since hair samples were collected throughout the year, and the full data were randomly subsampled, these values represent the overall geometric mean daily fish consumption rates for this population, without respect to season, age or community isolation.

These estimated consumption rates are between 1.5 and 2 times greater than the Canadian average rate of consumption of all fish of 11 g/day (HWC 1977; Conacher et al. 1989), and about 10 to 15 times greater than the average rate of consumption of freshwater fish (Conacher et al 1989). However, they are considerably less than that reported by Lawn (1989) for residents of the Big Trout Lake and Weagamow Lake Indian Reserves, and by Hopper and Power (1991) for the Webequie Reserve in northern Ontario. Lawn (1989) estimated an average consumption rate for adults (sexes not distinguished) of 195 g/day, based on a one time survey of

frequency of use of various foods. Geometric mean estimates of fish consumption rate for these reserves, based on hair and fish Hg concentrations, were 33 g/day (n=84) for males, and 28 g/day (n=162) for females. Geometric mean consumption rates for individuals of these communities were found to be greater than that for Ontario as a whole, but still only about one sixth of the rate suggested by Lawn (1989). Estimates of fish consumption rate could not be determined for the Webequie community, for comparison to Hopper and Power (1991), due to insufficient fish Hg data.

The discrepancies between the estimates of fish consumption made here, and those of Lawn (1989) may have resulted from study design. Lawn (1989) undertook a one time survey of food use frequency, based solely on respondents' recall of food use for the previous year. Such information will be subject to considerable error and more qualitative than quantitative in nature.

Fish consumption by male amerindians is significantly greater ($p < 0.0005$) than that for females (Table 8.3). This finding is consistent with the results of Wein et al. (1991), as well as with Kinloch et al. (1992) for the Inuit of Broughton Island, NWT, and likely reflects the greater rate of food consumption observed in males generally (HWC 1977).

The influence of age on fish consumption patterns found here (Figure 8.5) agrees with other published reports (Kinloch et al. 1992; Wein et al. 1991; Kuhnlein 1989). MeHg concentrations in hair have repeatedly been shown to be a direct function of average daily dietary Hg intake (WHO 1976; Wheatley 1979; Kershaw et al. 1980; Wheatley and Wheatley 1981; Clarkson 1990). Levels in hair decrease rapidly once exposure is stopped (Phelps et al. 1980, for example), indicating that there is little time lag between Hg consumption and its deposition in the hair, and that bioaccumulation of MeHg does not occur in hair. Therefore, changes in hair Hg concentrations with age reflect changes in daily dietary intake, and not bioaccumulation.

Figures 8.3 and 8.4 present a quantitative analysis of fish consumption trends by extent of community isolation. This trend does not simply reflect increased fish [Hg] with latitude, since fish [Hg] actually decreases with latitude ($r=-0.40$, $p<<0.005$, $n=13,245$). Therefore, increased levels of Hg in humans at higher latitude must reflect increasing consumption rate.

Other published consumption rates for Canadian Native communities, based on quantitative diet survey studies, include: 69 g/day reported for female members of a community of amerindians in British Columbia, based on two 24 hour

recall surveys conducted during autumn (Kuhnlein 1989); 14 g/day (both sexes, all ages combined) reported for a community located near Wood Buffalo National Park, based on a total of four 24 hour dietary surveys conducted during at least two different seasons (Wein et al. 1991); 13 g/day for an Inuit community at Broughton Island, NWT (Innis et al. 1988), based on a single dietary recall survey conducted during the month of September; and 45 g/day for the same Inuit community studied by Innis et al. (1988), but encompassing a total of seven dietary recall surveys conducted at two month intervals over a one year period (Kinloch et al. 1992). This latter value is considered more reliable than that of Innis et al. (1988) since it encompasses seasonal variation in fish consumption.

Fish consumption rates determined in this analysis, and those reported from quantitative dietary recall surveys, appear substantially less than estimates based on fish harvest data. Based on 96 Canadian subsistence fishery harvest studies, Berkes (1990) reported an overall mean per capita fish harvest of about 42 kg edible weight per year (excludes commercial fishery catch). This equates to per capita fish consumption of approximately 115 g/day, if it is assumed that all this fish is consumed. However, Berkes (1990) points out that fish used as dog food or distributed to neighbouring communities as gifts could not be excluded from his estimate

in many cases. Therefore, the estimate of 115 g/day based on these surveys is likely high. Hopper and Power (1991) determined the per capita harvest of fish to be 210 g edible weight/day for the Ojibwa community at Webequie, Ontario, based on a year-long survey of fishery harvest. Hopper and Power (1991) indicate that some fish is used as trap bait and is also discarded in trapping areas during the non-trapping season in order to attract fur bearing animals and thereby improve trapping success. It is unclear to what extent Hopper and Power (1991) were able to discount these non-consumptive uses from their estimate of per capita harvest.

Evidence that harvest studies tend to over-estimate the rate of fish consumption as determined by quantitative diet recall surveys is given by Kinloch et al. (1992). They reported that the annual harvest of arctic charr (Salvelinus alpinus) by the Broughton Island Inuit community was 62.6 kg/resident. This equates to approximately 170 g/day/resident, almost four times the amount of 45 g/day determined from diet surveys for this community.

Based on the results of this analysis, the overall geometric mean (both sexes combined) rate of fish consumption was found to be 16 g/day. This value is the best estimate of the central tendency of the sampled population, and may be the most representative fish consumption rate for this population.

8.6 Conclusions

Fish consumption rates have been estimated for Ontario amerindians from data on concentrations of Hg in hair samples, and concentrations of Hg in fish collected from lakes surrounding amerindian reserves. Very few published reports exist which provide reliable, quantitative measures of fish consumption patterns by amerindians, and fish consumption rates based on fish harvest data appear to significantly overestimate average daily fish consumption. Results reported here are based on a significantly larger sample population (>4,000 individuals) than any previous report of fish consumption patterns, and, therefore, may provide a more statistically sound basis for determining fish consumption rates.

Based on this analysis, it was concluded that amerindian hair Hg data could be controlled, indirectly, for fish consumption rate by controlling the data for the statistical influences of age, sex and community isolation.

9.0 HUMAN EXPOSURE TO MERCURY MAY DECREASE AS ACIDIC DEPOSITION INCREASES

9.1 Abstract

It has been hypothesized that human mercury (Hg) exposure via fish consumption will increase with increasing acidic deposition. Specifically, acidic deposition leads to reduced lake pH and alkalinity, which in turn should cause increased Hg levels in fish, ultimately resulting in increased human Hg exposure via fish consumption. Our empirical test of this hypothesis found it to be false. We specifically examined Hg levels in the hair of Ontario amerindians, who are known consumers of fish from lakes across the province, and observed a weak negative association with increasing sulphate deposition. An examination of Hg levels in lake trout, northern pike and walleye, three freshwater fish species commonly consumed by Ontario amerindians, found a similar weak negative association with increasing sulphate deposition. An alternate hypothesis is proposed whereby human Hg exposure declines with increasing acidic deposition.

9.2 Introduction

The primary non-occupational source of exposure to mercury (Hg) in humans is via fish consumption (WHO 1990). Therefore, any environmental factor which influences fish Hg contamination will also influence Hg exposure in persons consuming those fish.

Across Ontario, native Canadians are known to subsist to a significant extent on freshwater fish for their dietary nutrition (see Chapter 8), and their patterns of Hg exposure strongly reflect known seasonal patterns in fish consumption (Richardson and Currie 1993). Therefore, Hg exposure in this subpopulation will likely reflect any significant spatial patterns in Hg contamination in the fish species being consumed.

It has been hypothesized that exposure to Hg by persons who consume freshwater fish increases due to acidic deposition (Goyer et al. 1985; Gilmour and Henry 1991; Clarkson 1990), and this hypothesis has now appeared in the popularized scientific literature (Raloff 1991). However, this hypothesis has never been tested. Speculation of this association arose from two independent observations. First, sulphate deposition leads to lake acidification, most notably to reduced pH and alkalinity (Neary and Dillon 1988; Neary et al. 1990; Schindler 1988; Dillon et al. 1984; NRCC 1981). Secondly,

reduced lake pH and alkalinity have been associated with increased mercury contamination in fish (Suns and Hitchin 1990; Scheider et al. 1979; Wiener et al. 1990, for example; reviewed in Chapter 5). Sulphate deposition also leads to increased lake water concentrations of sulphate ion ($[SO_4^{2-}]$) (Sullivan et al. 1988), which has also been implicated in increased Hg methylation (Gilmour et al. 1992) and subsequent fish Hg accumulation. These mercury-contaminated fish would thereby pose an increased health risk to persons who consumed them.

In the present study, we examine the hypothesis that Hg in amerindians from Ontario, Canada is higher in regions of higher acidic deposition.

9.3 Methods

We examined this hypothesis using mercury exposure data for 3,187 amerindians, aged 10 to 90 years, residing in 55 reserves located across Ontario (Figure 9.1). MeHg exposure in this population results primarily from consumption of freshwater fish (Clarkson 1990; WHO 1976 1990; Richardson and Currie 1993). Data were excluded for individuals on reserves located on rivers or lakes impacted by industrial Hg contamination, and on reserves adjacent to urban centres where fish is commonly purchased from commercial sources.

Data relating to total Hg concentrations in hair (which is directly related to Hg ingestion (WHO 1976 1990)), date of sample collection, date of birth, and reserve of residence for 1,840 female and 1,347 male amerindians were obtained from the Medical Services Branch of Health Canada (Wheatley 1979; Tupper 1984). Total [Hg] is strongly correlated ($r > 0.99$) with MeHg in these data (Richardson and Currie 1993). Since many more observations of total Hg were available, we analyzed these total Hg data.

Reserves were ascribed sulphate deposition rates according to their location within sulphate deposition zones of Ontario (after Neary et al. 1990).

Mercury analyses were performed on several 1 cm long segments of hair, of known distance from the scalp, from a tuft of several hundred hairs collected from each individual. Details of hair sample collection and Hg analytical methods are described elsewhere (HWC 1987; Farant et al. 1981). Hair grows at a rate of 1 cm per month (Richardson and Currie 1993) and, therefore, the month of Hg exposure could be deduced from the available data. Fish consumption, and thereby Hg exposure, is greatest during the summer months (Richardson and Currie 1993). Therefore, the average concentration of Hg (\log_e -transformed) in those segments of hair relating to Hg

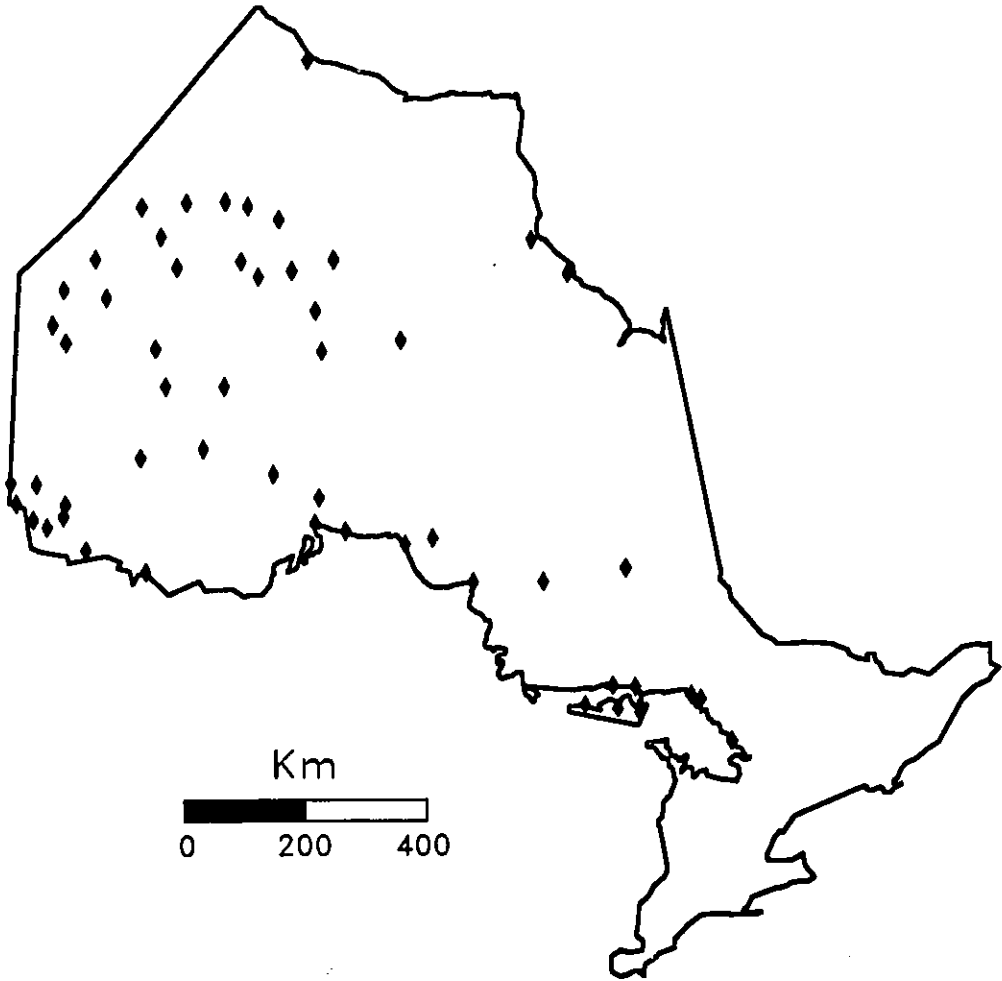
exposure during June through October were averaged and this mean summer hair [Hg] was used for further analysis.

Acidic deposition should influence human Hg exposure indirectly, through its influence on fish [Hg] levels. As human Hg exposure is a function of both fish [Hg] and fish consumption rate (Richardson and Currie 1993), it is necessary to eliminate the confounding influence of fish consumption rate. No direct data were available on the rate of fish consumption by amerindians. However, fish consumption rate can be controlled indirectly, as a function of each individual's age, sex, and the degree of isolation from urban centres (see Chapter 9; Richardson and Currie 1993), where increasing isolation leads to greater reliance on subsistence fishing for nutrition.



The analyses reported below were performed on a variety of subsamples of the available data, with qualitatively consistent results when analyzed for individuals aged ≥ 10 years, aged ≥ 16 years, or for individuals aged ≥ 20 years. Also, the results were the same whether hair [Hg] values equal to or less than the analytical detection limit (0.3 ng Hg/g hair) were equated to 0.3 ng Hg/g hair, or were eliminated from the analysis all together. Reported here are the results for all individuals aged 10 to 90 years and with hair [Hg] values at or below the detection limit equated to 0.3 ng Hg/g hair.

Figure 9.1 Distribution of 55 reserves across Ontario in which individuals providing hair samples for Hg analysis resided.

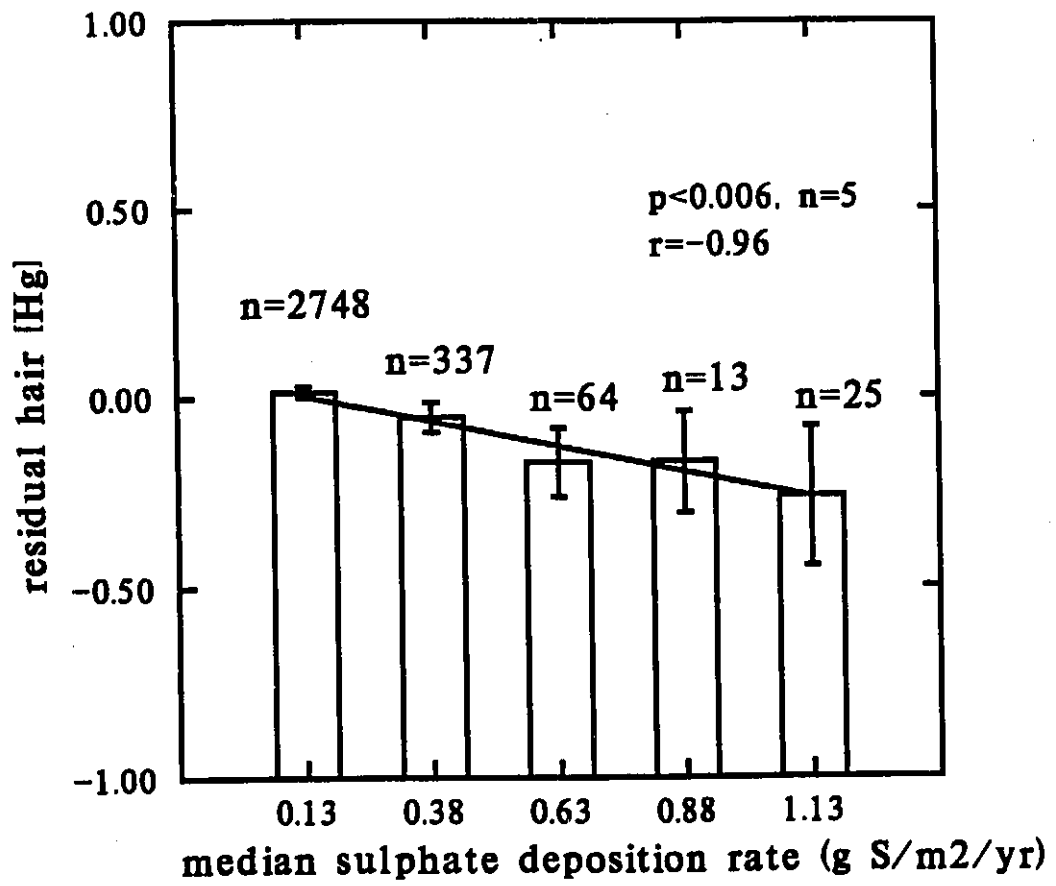


9.4 Results and Discussion

After statistically controlling hair[Hg] data for age, isolation and sex, residual variation in these data were grouped by regions of sulphate deposition. A significant negative correlation was observed between mean residuals and the median sulphate deposition rate for each zone (Figure 9.2). It was assumed that lakes from which fish were collected would have approximately the same rate of sulphate deposition as the reserve of residence, given that most fishing occurs within about 100 km of the reserve (see Richardson and Currie, 1993).

If fish Hg is the source of human Hg exposure, then fish [Hg] should also demonstrate a similar association with sulphate deposition. This association has been previously demonstrated and discussed in Chapters 7 and 8. For lake trout, walleye and northern pike, the most frequently consumed freshwater fish species for Ontario amerindians (Berkes 1990; Lawn 1989), the slope of the regression between mean, residual fish [Hg] and median sulphate deposition rate, was -0.33 for lake trout, slope=-0.38 for walleye and slope=-0.12 for pike. The average slope for all three species combined was -0.28. This is very close in value to the slope of the regression of residual human hair [Hg] on median sulphate deposition rate, slope=-0.27.

Figure 9.2: Hair Hg concentration (controlled for age, sex, and degree of isolation) as a function of sulphate deposition across Ontario. Correlation results are for the association between mean residual hair [Hg] for all individuals within each sulphate deposition zone, and the median sulphate deposition rate for those zones (after Neary et al. 1990). Data for hair [Hg] and age were \log_e -transformed prior to analysis.



Based on these observations, it was concluded that the hypothesized increase in human Hg exposure with increasing acidic deposition is false. An alternate hypothesis that human Hg exposure declines with increasing acidic deposition appears plausible. In this latter hypothesis, sulphate deposition results in reduced lake water levels of DOC (see Chapter 6). This in turn leads to a decline in lake water [Hg] (methyl and inorganic), either by reduced transport to the lake or loss from the water column as DOC-complexed Hg precipitates with increasing acidification. The reduced bioavailability of Hg for methylation and/or bioaccumulation leads to reduced fish [Hg] (see Chapter 5 and 7), and reduced human Hg exposure via fish consumption.

The empirical support for this alternate hypothesis is weak, and requires rigorous testing and examination. However, experimental and empirical evidence exist to support the purported mechanisms which underlie it. At the very least, it appears evident that Hg exposure in fish consumers, and Hg levels in the fish themselves, are not higher in regions of Ontario with greater sulphate deposition.

10.0 SIGNIFICANCE OF NATURALLY-OCCURRING MERCURY TO FISH CONTAMINATION IN REMOTE LAKES OF THE CANADIAN SHIELD

10.1 Abstract

The mercury (Hg) contamination of 8 species of fish was examined for its association with the pre-industrial (non-anthropogenic) Hg content of sediments of 355 Canadian Shield lakes in Ontario. Pre-industrial sediment [Hg] was found to correlate significantly with Hg levels in 7 of the 8 fish species collected from these lakes, explaining up to 20% of the spatial variation in fish [Hg]. Based on empirical analysis, it was concluded that local bedrock accounted for 7.5% of the variation in non-anthropogenic sediment [Hg]. Evidence supports a mechanism of atmospheric transport and deposition of natural Hg to remote lakes, and does not support a mechanism involving direct physical or chemical erosion. It was also concluded that analyses of fish Hg contamination must be controlled for their statistical association with non-anthropogenic sediment [Hg] before the contribution of anthropogenic Hg sources to biotic Hg contamination can be properly quantified.

10.2 Introduction

Studies of lake sediments indicate that mercury (Hg) deposition has increased over the past 50 to 100 years due to industrialization and subsequent atmospheric Hg emissions (Swain et al. 1992; Rada et al. 1989; Johnson 1987; Johnson et al. 1986; Evans 1986; Meger 1986; Ouellet and Jones 1983; Heit et al. 1981). Also, sediment Hg concentrations tend to increase with proximity to local atmospheric point sources (Harrison and Klaverkamp 1990; NRCC 1979). This has led to the assumption that Hg contamination in remote lakes (not affected by direct industrial discharges or impoundment) is due primarily to anthropogenic emissions. Although natural sources of Hg to lakes can be significant (Varekamp and Waibel 1987; Loukola-Ruskeeniemi 1990), the only effort generally made to control for the potential influence of natural Hg sources on lacustrine contamination is to ensure that no local geological Hg 'hot spots' exist in the watersheds of the lakes under study (Swain et al. 1992, for example).

Fish Hg contamination is known to covary with Hg sedimentation rate (measured in the water column) (Johnson 1987) and with surface sediment Hg concentration (Cope et al. 1990; NRCC 1979). Therefore, it is generally assumed that anthropogenic emissions are also the primary source of biotic Hg contamination in remote lakes.

We wished to test the assumption that fish Hg contamination is not associated with naturally-derived Hg. Hg contamination of fish from remote lakes in Ontario is spatially quite variable (McMurtry et al. 1989; Wren et al. 1990). The Hg concentrations in deep sediments of Ontario lakes, sediments deposited prior to industrialization, are also spatially quite variable (Friske 1985 a,b). This pre-industrial sediment [Hg] is a surrogate for the proportion of Hg in surficial lake sediments which arises from non-anthropogenic sources (Johnson 1987). Therefore, these two data sets offered a unique opportunity to evaluate the extent to which fish Hg contamination covaried with naturally-derived sediment [Hg] over a large geographic area. Secondly, we wanted to determine the most probable source of this natural Hg to Ontario lakes, and how it might be transported there.

10.3 Methods

Unpublished data relating to fish length and the concentration of Hg in the dorsal muscle tissue of specimens of eight species of fish collected from the lakes being studied were obtained from the Ontario Ministries of Natural Resources and Environment. These data are summarized in Table 10.1. Fish collection and analytical methods used to determine total Hg concentration in a skinless fillet of fish dorsal muscle have been described elsewhere (Wren et al. 1991; McMurtry et al. 1989; OMOE 1984). Fish collected from the

English-Wabigoon River system, from the Great Lakes, and from known hydro-electric reservoirs were excluded from the analysis due to potential confounding of Hg contamination in fish.

Fish [Hg] data (\log_e -transformed) for each fish species, from all lakes, were pooled and regressed as a quadratic function of fish length (\log_e -transformed), the single best predictor of fish Hg concentration for all species. The resulting regression residuals (the difference between predicted and observed values of fish [Hg]) represent the extent to which fish have more or less Hg than the average fish of comparable length. These residuals were subsequently averaged by lake and the mean residuals were employed for further analysis.

We obtained data from the Geologic Survey of Canada (GSC) (Friske 1985a,b) on the concentration of Hg in pre-industrial sediments and the percent of sediment sample weight lost on ignition for 355 Ontario lakes located on the Canadian Shield (see Figure 10.1). Loss on ignition (LOI) is a measure of the organic matter content of the sediment samples. GSC collects these data for the purpose of mineral exploration, particularly for sulphide ore deposits. To ensure that samples did not contain anthropogenic contamination, GSC removed the top 10 cm of sediment material before extracting

a subsample for chemical analysis (Friske 1985a), and the subsample (0.5 g wet weight) analyzed for Hg was generally selected from deeper strata within the sample (P.Friske, GSC, pers. com.). Strata below 10 cm depth predate anthropogenic contamination in Ontario Shield lakes (Evans 1986; Johnson 1987; Johnson et al. 1986).

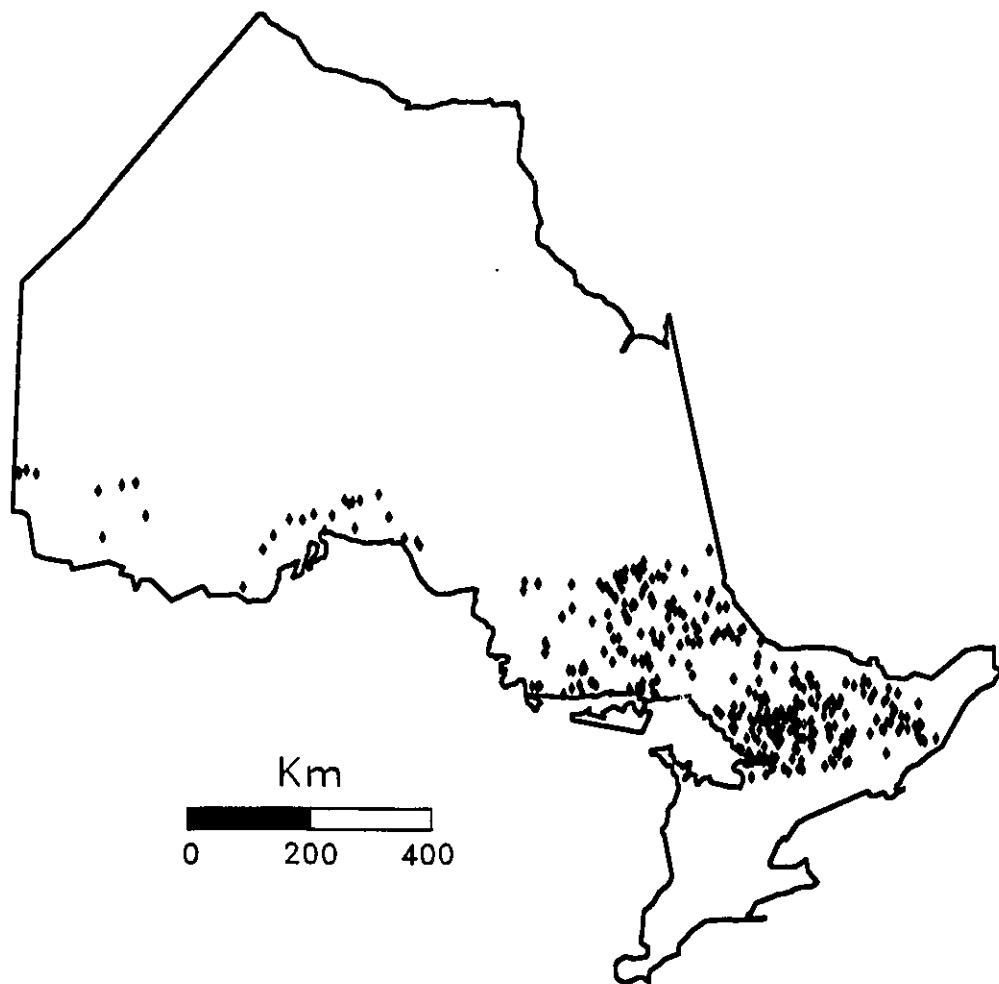
From 1 to 29 sediment samples were collected within each lake, depending on its surface area. For lakes with more than 1 sample, the arithmetic mean for [Hg] and LOI were employed for further analysis. Sampling and analytical techniques are described by Friske (1985a) and Coker et al. (1979).

The spatial patterns in deep sediment [Hg] are known to qualitatively reflect the distribution of different bedrock formations which have different concentrations of, or numbers of significant deposits of, sulphide mineral ores (Frisk 1989a; Coker and Shilts 1979). Therefore, the location of each lake on one of six basic bedrock types was also determined from the Ontario Mineral Map (OMNR 1985). The bedrock types differed in their average densities of sulphide ore mineral deposits, ranging from 2.5×10^{-4} to 7.1×10^{-3} deposits per 1,000 km². The distance from each lake to the nearest upstream sulphide ore mineral deposit within the drainage basin of each lake was also determined from the Ontario Mineral Map (OMNR 1985).

Table 10.1. Summary of fish mercury data (geometric mean and range) used in Chapter 10.

Fish species	fish [Hg] (range) ($\mu\text{g/g}$)	no. of specimens	no. of lakes
lake trout (<u>Salvelinus namaycush</u>)	0.24 (0.01-10.00)	2680	143
walleye (<u>Stizostedion vitreum vitreum</u>)	0.52 (0.02-4.30)	2556	125
northern pike (<u>Esox lucius</u>)	0.42 (0.01-5.00)	2120	138
lake whitefish (<u>Coregonus clupeaformis</u>)	0.16 (0.01-1.40)	437	31
white sucker (<u>Catostomus commersoni</u>)	0.14 (0.01-0.79)	455	28
smallmouth bass (<u>Micropterus dolomieu</u>)	0.40 (0.01-5.00)	2262	150
largemouth bass (<u>Micropterus salmoides</u>)	0.36 (0.06-3.40)	588	48
yellow perch (<u>Perca flavescens</u>)	0.15 (0.01-1.30)	1482	51

Figure 10.1: Distribution of the 355 lakes examined in chapter 10. Some points represent more than one lake.



Data were also obtained from GSC on the Hg concentration in subsoil sampled from the drainage basins of 88 of these same lakes (Kettles and Shilts 1983; Kettles 1988; Kettles 1990). On the Canadian Shield, the soil overburden is not derived from the underlying bedrock, but was transported from the north-east and deposited during the last ice age (Coker and DiLabio 1987). Therefore, the soil is unrelated to the underlying bedrock and offers a second potential source of natural Hg to lakes.

Subsoil samples consisted mostly of glacially-deposited tills and were collected from strata below signs of frost disturbance, again to ensure that samples reflected natural and not anthropogenic Hg (Kettles 1988, 1990; Kettles and Shilts 1983). Sample collection and analytical procedures are described in detail by Kettles and Shilts (1983). From 1 to 16 samples of subsoil were analyzed per watershed. For watersheds with more than 1 sample, the arithmetic mean value for soil [Hg] was employed for further analysis.

No direct data were available on Hg deposition across the province to confirm that sediment [Hg] data were independent of present-day atmospheric Hg deposition. However, the atmospheric deposition of Hg is known to parallel sulphate deposition (Glass et al. 1991; Nater and Grigal 1992). Sediment Hg data were, therefore, compared to patterns of

sulphate deposition across Ontario, after the scheme of Neary et al (1990).

Several factors may influence sediment [Hg] besides those discussed above. The mobile form of Hg in the lakes and watersheds under study is an organic complex (Mierle and Ingram 1991; Lee and Iverfeldt 1991). It follows then that sediment-borne Hg would be associated with the organic fraction (Coker et al. 1979), Hg being deposited along with the organic material to which it is bound. Therefore, the organic carbon content of the sediments will have to be statistically controlled to elucidate any association of sediment [Hg] with watershed soil or bedrock. If natural Hg is transported atmospherically, we should find significant associations between sediment [Hg] and lake and watershed morphometry, particularly the ratio of terrestrial drainage basin area to lake surface area (Mierle 1990), and likely precipitation rate.

Data on lake surface area and drainage basin area were derived from 1:50,000 scale topographic maps, using a polar planimeter. From these, the ratio of drainage basin area to lake surface area was determined. Data on annual precipitation rate for each lake were derived from the Hydrological Atlas of Canada (Fisheries and Environment Canada 1978). Table 10.2 summarizes these and other data.

Table 10.2. Summary of lake data employed in Chapter 10.
 Sample size is 355 lakes except where noted.

Parameter	geometric mean (range)
Lake area (hectares)	392 (8.1 - 27,691)
Drainage basin area (hectares)	1900 (72 - 107,377)
Drainage basin area:lake area (unitless)	4.8 (0.84 - 263.0)
annual precipitation (mm/yr)	861 (600 - 1000)
sediment [Hg] (ug/kg)	92 (5 - 280)
sediment LOI (%)	23.8 (0.5 - 72.2)
soil [Hg] (ug/kg) (n=88)	74 (2 - 845)
distance to sulphide ore deposit (km) (n=87)	3.3 (0 - 71)

Pre-industrial sediment [Hg] was regressed against all possible combinations of variables to ensure that all significant relationships would be detected. Statistical analyses were conducted using SYSTAT^R version 5.01 (Wilkinson 1991). Data were transformed as required to stabilize variance and linearize relationships.

10.4 Results

Fish Hg contamination was found to vary significantly with naturally-derived sediment [Hg]. A significant positive correlation was observed for 7 of 8 species examined (Table 10.3). It appears that non-anthropogenic sediment [Hg] may be responsible for as much as 20% of the spatial variation in log_e-transformed, length-standardized, average Hg contamination of fish in Ontario Shield lakes.

Pre-industrial sediment [Hg] was significantly associated with a number of the variables examined. A combination of sediment LOI, morphometric variables and precipitation rate explained 30% of the total variation in pre-industrial sediment [Hg]:

$$\begin{aligned} \text{Pre-industrial=} & -51.5(\pm 27.0) + 5.5(\pm 0.5)\text{LOI} - 0.07(\pm 0.01)\text{LOI}^2 \\ \text{sediment [Hg]} & + 10.7(\pm 3.0)\ln(\text{DBA:LA}) + 0.06(\pm 0.03)\text{PRECIP} \end{aligned} \quad (10.1)$$

($R^2=0.30$; $p<0.00005$)

where LOI = loss on ignition, DBA = drainage basin area, LA = lake surface area, and PRECIP = annual precipitation rate. All variables were significant at $p < 0.03$. The organic fraction to which Hg is bound, precipitation rate and DBA:LSA are the same variables associated with the present-day rate of Hg entering Ontario lakes (Mierle 1990; Lee and Iverfeldt 1991; Mierle and Ingram 1991), indicating that natural Hg also reaches Ontario lakes by atmospheric transport (i.e. the same mechanism as for present-day anthropogenic Hg).

Direct chemical erosion would not appear to contribute significantly to the natural Hg content of lake sediments. Mean sediment [Hg] did not differ between lakes with ($n=87$) and without ($n=268$) ore deposits within their watersheds ($t=0.659$, $p>0.5$). Also, for the 87 lakes identified with sulphide ore deposits upstream within their watersheds, sediment [Hg] was not significantly correlated with the distance from the lake to that deposit (Table 10.4). Again, after controlling for other variables, there was still no significant partial correlation between distance to ore deposits and sediment [Hg].

Pre-industrial sediment [Hg] was not significantly related to watershed soil [Hg] for the 88 lakes for which data were available (Table 10.4). After controlling for any and all other variables (see Table 10.2), there was still no

significant partial correlation between soil [Hg] and sediment [Hg]. Therefore, soil would not appear to be the source of natural Hg to Ontario lakes.

Deep sediment [Hg] covaried significantly with bedrock type (Figure 10.2), a consistent relationship only evident after the confounding influences of lake morphometry, precipitation rate and sediment LOI were removed. The proportion of variation in sediment [Hg] explained by bedrock geology was 7.5% (the partial R^2 for average ore deposit density, in ANOVA of Equation 10.1).

Deep sediment [Hg] appears to be independent of recent atmospheric deposition. Average sediment [Hg] does not differ among sulphate deposition zones ($F=1.43$; $df=349,5$; $p>0.2$; $n=355$), which parallel atmospheric deposition of Hg (Glass et al. 1991; Nater and Grigal 1992). After controlling for the influences of sediment LOI, lake and drainage basin area, and annual precipitation rate (i.e. ANCOVA of Equation 10.1) there were significant differences in sediment [Hg] among deposition zones but they did not systematically increase with increasing sulphate deposition rate (see Figure 10.3). Therefore, we concluded that the deep sediment [Hg] considered here did arise from natural and not anthropogenic sources.

Table 10.3. Pearson correlation coefficients between fish [Hg] and pre-industrial sediment [Hg]. The pre-industrial sediment [Hg] is assumed to represent the background, non-anthropogenic Hg input to lakes. Fish [Hg] was first controlled for the influence of fish length and then averaged by lake before being correlated with sediment [Hg]. ns = not significant at $p > 0.05$.

fish species	r	p	no. of lakes
lake trout	0.15	0.041	143
walleye	0.27	0.001	125
northern pike	0.28	<0.001	138
lake whitefish	0.45	0.006	31
white sucker	0.40	0.019	28
smallmouth bass	0.22	0.004	150
largemouth bass	0.08	ns	48
yellow perch	0.43	0.001	51

Figure 10.2: ANOVA by bedrock type of regression residuals from Equation 10.1. Bedrock is differentiated by the average density (deposits/1000 km²) of sulphide ore deposits as determined from OMNR (1985). Letters above the bars indicate differences significant at $p < 0.05$ by Tukey-Kramer multiple range test.

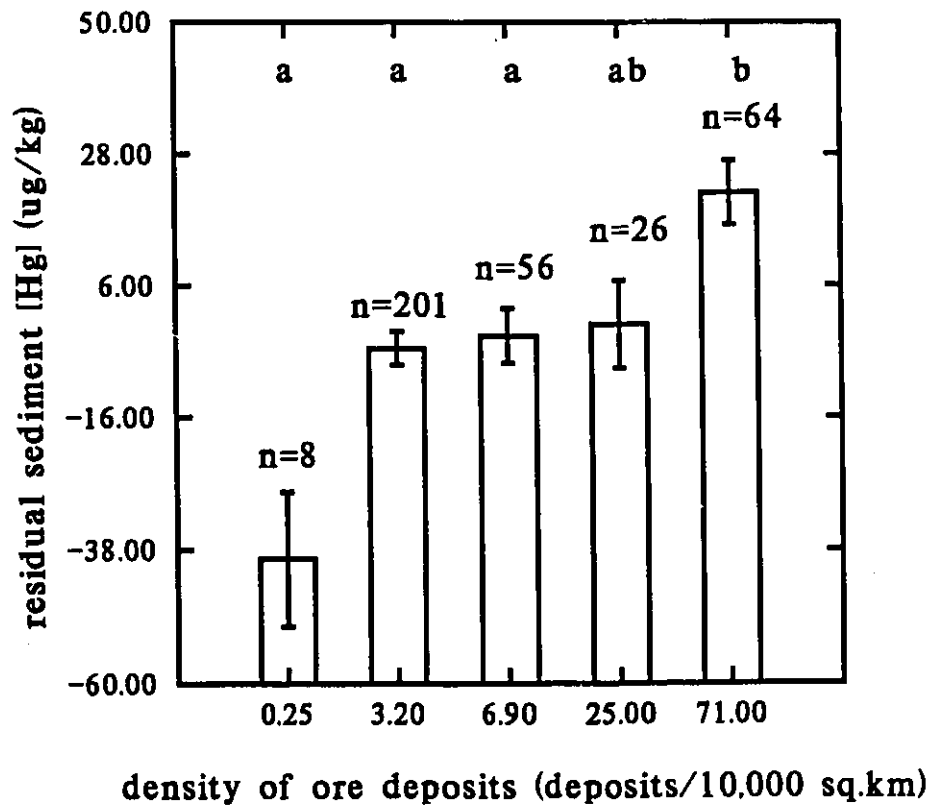


Figure 10.3: Association between sulphate deposition in Ontario and pre-industrial sediment [Hg] which has been controlled for the influence of sediment LOI, lake and watershed size, and annual precipitation rate. The dependent variable (residual [Hg]) is the residuals resulting from the regression model depicted in Equation 10.1, averaged (\pm se) by sulphate deposition zone. Letters above the bars indicate differences significant at $p < 0.05$ by Tukey-Kramer multiple range test.

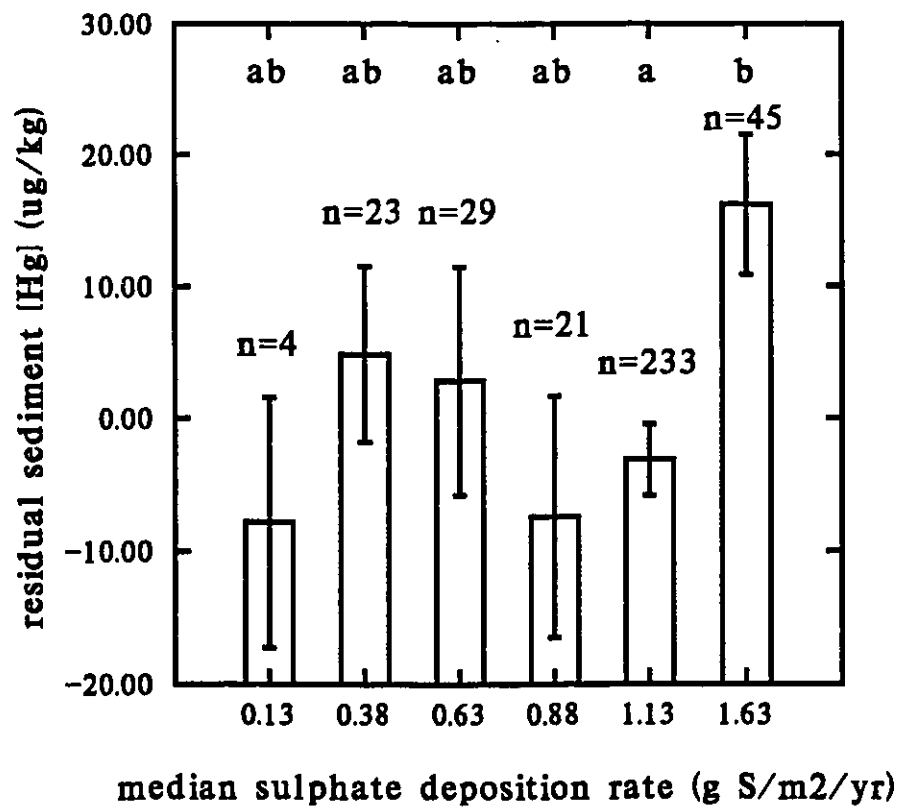


Table 10.4. Pearson correlation coefficients between pre-industrial sediment [Hg] and lake variables. Non-significant ($p>0.05$) correlations indicated as ns.

Parameter	r
sediment LOI	0.52 ^a
ln(lake area)	0.33
ln(drainage basin area)	0.24
ln(drainage basin area:lake area)	0.30
annual precipitation rate	ns
soil [Hg]	ns
distance to sulphide ore deposit	ns

a - multiple correlation coefficient; sediment [Hg] was a second degree polynomial function of sediment LOI.

10.5 Discussion

The Hg content of biota such as fish in remote lakes has received considerable attention in the literature. In many of these studies, anthropogenic pollution is identified as the cause of that contamination. Certainly the possibility that all or some portion of such biotic contamination might be natural is seldom discussed unless there is a known direct geological discharge to the lake(s) in question. We have demonstrated here that background, non-anthropogenic Hg apparently does explain a significant, albeit relatively small (2-20%), amount of the spatial variation in fish Hg contamination (Table 10.4) in lakes without known geological hot spots. However, it should be noted that surface sediment [Hg] (i.e. anthropogenic + non-anthropogenic sources) appears to explain less than 50% of the total spatial variation in fish [Hg] in Wisconsin seepage lakes (Cope et al. 1990), and therefore, natural Hg sources may be more important to lacustrine and biotic Hg contamination than our low correlation coefficients might initially suggest. Unfortunately, we lacked data on surface sediment [Hg] (i.e. current total atmospheric Hg input) to test this hypothesis directly.

The movement of minerals from bedrock to the sediments of the lakes of the Canadian Shield has been generally assumed to be controlled by chemical (i.e. solubilization) processes

(Friske 1985a,b; Coker et al. 1979). Chemical weathering of soils and bedrock is expected to solubilize Hg for subsequent physical transport via groundwater, streams and rivers in either particulate, complexed or water soluble forms (Andersson 1979; Jonasson and Boyle 1972; Friske 1985b). If this mechanism of dispersal prevailed for Hg, one would expect that lakes with significant ore deposits within the watershed would have greater deep sediment [Hg] than lakes lacking such deposits. Likewise, one would expect that sediment [Hg] would positively covary with distance to an upstream geologic Hg source (sulphide ore deposit). These relationships, however, were not observed and, therefore, chemical weathering was not considered the primary mechanism governing the movement of geologic Hg to these remote lakes.

Anthropogenic Hg is transported to remote lakes via the atmosphere (Mierle 1990). We have presented evidence to suggest that natural Hg is also transported atmospherically. Metallic Hg is volatile (Matheson 1979) and elevated concentrations have been observed in the air near mines, geologic deposits and soils containing this element (Mitra 1986; Matheson 1979). In the data examined here, pre-industrial sediment [Hg] was related to the ratio of drainage basin area to lake surface area and precipitation rate, variables typically associated with atmospheric deposition of Hg (Mierle 1990). Swain et al. (1992) also reported a

significant relationship of drainage basin area:lake surface area ratio to pre-industrial Hg contamination in sediments of remote lakes in Minnesota. It was concluded, therefore, that the most probable mechanism for transport of natural Hg to remote lakes is via the atmosphere. The diffuse, long-range nature of atmospheric Hg transport would explain the low proportion of variance in deep sediment [Hg] explained by local watershed geology.

In summary, we found that the concentration of natural (pre-industrial) Hg in lake sediments was significantly associated with the average density of ore deposits in the local bedrock. This association was only evident after controlling for other variables which are associated with a mechanism of atmospheric Hg transport, deposition to the drainage basin and subsequent runoff into the lakes - precipitation rate, ratio of drainage basin area to lake surface area, sediment organic carbon content. It is apparent, that quantitative conclusions concerning the extent to which anthropogenic Hg is contributing to Hg contamination in fish of Ontario remote lakes can not be reached without first controlling for natural sources, which make variable but significant contributions to lacustrine biotic Hg levels.

11.0 GENERAL DISCUSSION

The foregoing chapters have examined a number of commonly held or recently postulated hypotheses concerning Hg biogeochemistry in lakes, including its relation to sulphate deposition. It is apparent that the factors ultimately influencing Hg in fish, and in persons consuming fish, are interrelated in a more complex manner than previous tests and analyses of these hypotheses had acknowledged or been able to identify.

Figures 11.1a and b depict the complex interrelationships of precipitation chemistry, lake morphometry, watershed geochemistry (buffering capacity) and fish [Hg] in Ontario drainage lakes. Figure 11.1a applies to lake trout, whereas Figure 11.1b applies to walleye and northern pike. Table 11.1 presents the path coefficients (see Chapter 7 for definition) for each species, based on the same lakes and data employed for path analysis in Chapter 7.

Model fit is reasonable but not as good as observed in Figure 7.4. However the use of a categorical variable for watershed buffering capacity is expected to result in generally lower correlations than a continuous variable, thereby limiting the capacity of any path model to fit the data exceptionally well. In the previous path analyses

(Chapter 7), this problem was overcome by controlling lake water chemistry for the statistical influence of buffering capacity prior to path analysis.

A significant amount of variation in fish Hg remains unexplained by the variables employed throughout this study. In Figures 11.1a and b, some 68% of the among lake variation in length-controlled lake trout [Hg], 73% of the among lake variation in walleye [Hg] and 78% of the among lake variation in northern pike [Hg] was unexplained by the models. Regression models appear to perform better in this regard, with between 40% and 70% of the among lake variability in fish [Hg] being unexplained by multiple regression models incorporating morphometric, geochemical and water chemistry parameters (see Chapter 7). However, regression models fail to adequately reflect the complex associations between many of these variables, as regression analysis assumes that all these independent variables do not covary, an assumption known to be false. This raises concerns regarding both the quantitative and qualitative interpretation of regression analyses related to these parameters. Therefore, path analysis may be a superior analytical tool for evaluating complex associations and interactions of the sort considered in this study, despite the apparently poorer quantitative performance of this technique.

Figure 11.1 Path models describing the influence on fish Hg contamination of precipitation chemistry, lake morphometry, watershed geochemistry and lake water chemistry. Paths are numbered for cross reference to Table 11.1. GFI = Joreskog-Sorbom Goodness of Fit Index (see Steiger 1989; Joreskog and Sorbom 1989). To simplify presentation, unexplained variance in each parameter has not been represented. TWA = total watershed area; DBA\LSA = ratio of drainage basin area to lake surface area; VOL = lake volume; $[SO_4^{2-}]$ = sulphate ion concentration; ALK = alkalinity; [DOC] = dissolved organic carbon concentration; U = Unknown factors to which the unexplained variance in fish [Hg] is attributed - unknown exogenous factors for other variables in the models are not drawn to reduce clutter. TWA, DBA/LSA, VOL, $[SO_4^{2-}]$, Alk, [DOC] and fish [Hg] variables were \log_e -transformed.

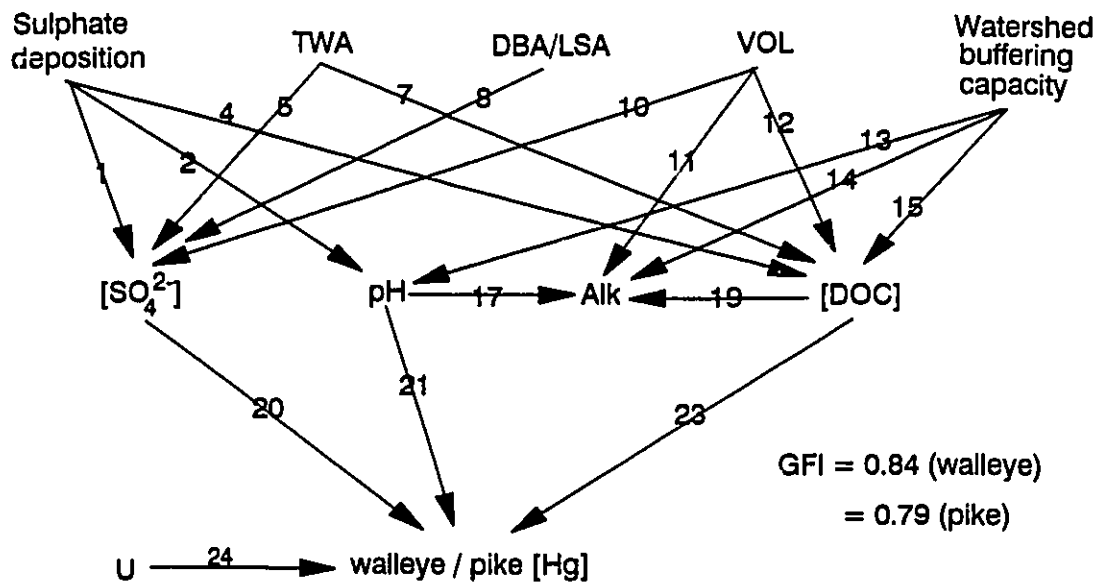
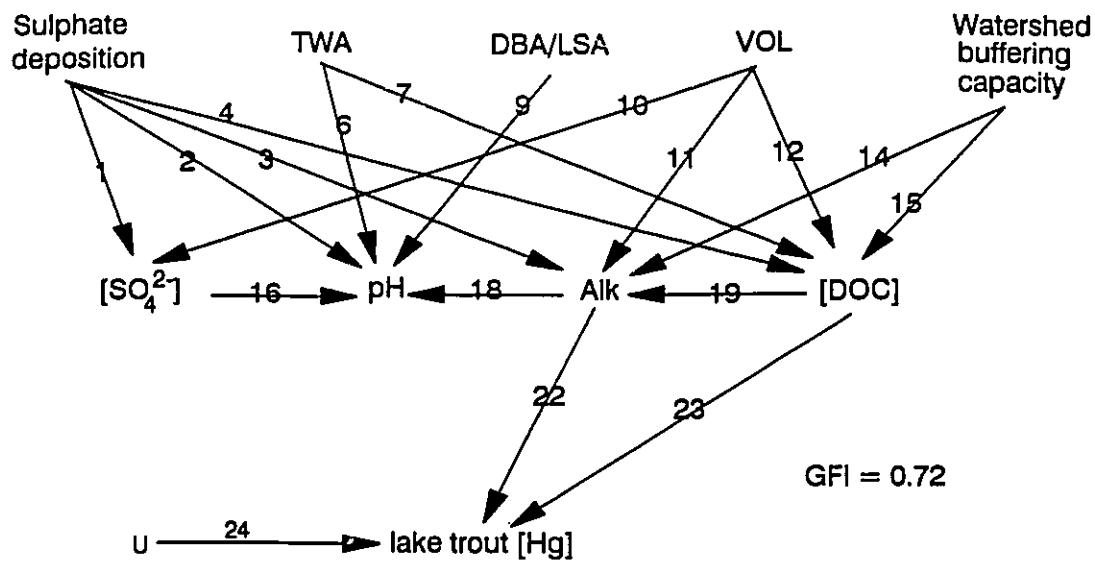


Table 11.1. Path coefficients (\pm standard errors) for models presented in Figures 11.1a and b.

Path no.	lake trout	walleye	northern pike
1	0.693 \pm 0.075	0.719 \pm 0.051	0.710 \pm 0.058
2	-0.108 \pm 0.048	-0.347 \pm 0.069	-0.420 \pm 0.075
3	-0.323 \pm 0.067	--	--
4	-0.332 \pm 0.074	-0.498 \pm 0.050	-0.417 \pm 0.057
5	--	-0.311 \pm 0.118	-0.260 \pm 0.140
6	0.133 \pm 0.038	--	--
7	1.344 \pm 0.180	0.552 \pm 0.082	0.640 \pm 0.090
8	--	0.212 \pm 0.080	0.175 \pm 0.094
9	-0.089 \pm 0.033	--	--
10	0.173 \pm 0.075	0.409 \pm 0.137	0.316 \pm 0.157
11	.0289 \pm 0.063	-0.075 \pm 0.033	-0.077 \pm 0.036
12	-1.264 \pm 0.179	-0.725 \pm 0.085	-0.750 \pm 0.094
13	--	0.243 \pm 0.069	0.141 \pm 0.075
14	0.354 \pm 0.061	0.050 \pm 0.033	0.056 \pm 0.038
15	0.132 \pm 0.072	0.170 \pm 0.050	0.207 \pm 0.059
16	0.092 \pm 0.043	--	--
17	--	0.816 \pm 0.032	0.852 \pm 0.035
18	0.807 \pm 0.042	--	--
19	0.286 \pm 0.066	0.192 \pm 0.036	0.138 \pm 0.040
20	--	0.263 \pm 0.076	0.177 \pm 0.083
21	--	-0.184 \pm 0.069	-0.253 \pm 0.079
22	-0.122 \pm 0.091	--	--
23	0.619 \pm 0.091	0.639 \pm 0.076	0.464 \pm 0.081
24	0.823 \pm 0.056	0.855 \pm 0.046	0.883 \pm 0.051

Besides the random variation that can be expected from lake to lake and specimen to specimen, a number of biotic factors may prove significant in explaining the residual variation observed here. These factors include food chain length (Cabana et al. in press) and position (planktivore versus piscivore, etc.) (Phillips et al. 1980; Mathers and Johansen 1985), and lake primary productivity (Hakanson 1980; Richman et al. 1988). A more precise measure of watershed buffering capacity would also improve the predictive strength of these models. In this study we employed a categorical variable (low, medium, high) which would have lower predictive strength than a directly measured, continuous variable.

The qualitative and quantitative differences observed for the path models of different species may relate, in part, to physiological differences between the species. Quantitative differences between species in the rates of accumulation and release of xenobiotics are always observed, being attributed to physiological, metabolic and/or biochemical differences.

Ecological differences between these species will also contribute the observed differences in the path models. As mentioned previously, ecological factors such as food chain length affect the accumulation of Hg by fish. The habitat, feeding habits and dietary preferences of lake trout, walleye and northern pike are different (Scott and Crossman 1973).

They are all piscivorous. However, lake trout are generally pelagic and tend to consume pelagic or benthic prey species. Walleye generally feed at all depths. The diet of the northern pike tends to be composed predominantly of littoral zone species of fish and amphibians.

The characteristics of the lakes from which lake trout were collected, versus those from which walleye and northern pike were collected, were very different (Table 7.2). Lake trout lakes were deeper, clearer (less DOC) and had lower pH and alkalinity on average than pike or walleye lakes. It is anticipated that these physical and chemical differences between lake types are the primary factors responsible for the qualitative (structural) differences in Figures 11.1 a and b (discussed in Chapter 7), however this could not be tested with the available data. Differences would also be expected between drainage and seepage lakes, although only drainage lakes were considered throughout this study.

Based on Figures 11.1 a and b, and the path coefficients presented in Table 11.1, variables can be ranked for their relative influence on Hg accumulation in each species. The proportion of variance explained by each factor which affects fish [Hg] directly and/or indirectly can be calculated as the square of the sum of the products of the chain of path coefficients along all of the paths by which they are

connected (Sokal and Raohlf 1981). For lake trout [Hg] the relative importance of these variables was:

TWA > VOL > [DOC] > SDR > Alk > WBC

where TWA = total watershed area, VOL = lake volume, [DOC] = dissolved organic carbon concentration, SDR = sulphate deposition rate, Alk = lake alkalinity, and WBC = watershed buffering capacity.

For walleye and northern pike, the relative influence of the various parameters was:

[DOC] > VOL > TWA > [SO₄²⁻] > pH > SDR > WBC > DBA/LSA

where [SO₄²⁻] = sulphate ion concentration, and DBA/LSA = ratio of drainage basin area to lake surface area.

Those variables not included proved to have no direct or indirect path to fish [Hg]. Sulphate deposition explained only 2.4%, 0.4% and 0.2% of the variation in [Hg] for length-controlled lake trout, walleye and northern pike, respectively. It is apparent that sulphate deposition has less relative influence on fish [Hg], and thereby on human Hg exposure via fish consumption, than DOC, and those morphometric parameters which directly influence its

concentration in drainage lakes (see Chapter 6; also Rasmussen et al. 1989). Obviously, the impact of acidic deposition will be greater on small lakes, as it would on dilute, oligotrophic lakes. From the perspective of ranking the relative influence of variables on the overall spatial variation in lake water chemistry and fish [Hg] across the province, lake morphometry appears to be contributing more to this variability than is anthropogenic sulphate deposition.

It should not be interpreted that sulphate deposition does not have significant or serious impacts on aquatic environments. Numerous reviews and studies have demonstrated conclusively that changes in water chemistry (reviewed by Howells 1990), and loss of species diversity (Matuszek and Beggs 1988; Somers and Harvey, 1984; NRCC 1981) have occurred. The net influence of sulphate deposition on [Hg] in fish from drainage lakes was observed in the present study to be negative, but other factors play vastly more significant roles than does sulphate deposition in influencing fish [Hg].

It is apparent that a portion of the variation in fish [Hg] can be predicted by lake sediment [Hg] which arises from non-anthropogenic sources. However, local geology was a poor predictor of this natural sediment Hg contamination, and poorer still for predicting fish Hg contamination (see Chapter 10). The poor association between watershed geology and deep

sediment [Hg] or fish [Hg] likely resulted from the diffuse nature of atmospheric transport and deposition of natural Hg to remote lakes. Where future studies attempt to associate extant fish or lacustrine Hg contamination with anthropogenic Hg emissions, our studies indicate that simply studying lakes with watersheds having no significant mineralization will not adequately control for non-anthropogenic Hg input. Future studies must endeavour to deduce directly the proportion of fish Hg contamination arising from non-anthropogenic sources. This might best be done by eliminating data variation explained by deep (pre-industrial) sediment [Hg]. As our study was concerned with fish Hg contamination irrespective of source, this parameter was omitted for purposes of Figure 11.1.

Despite the general increase in anthropogenic Hg emissions and sedimentation in recent decades (Swain et al. 1992; Johnson 1987; Johnson et al. 1986), the trends in fish Hg contamination observed and reported herein suggest that this has had little consequence on the level of Hg accumulation in fish from remote lakes, at least in Ontario. The independent influence of atmospheric Hg deposition on fish Hg contamination could not be tested directly due to lack of data. However, Hg deposition parallels sulphate deposition (Nater and Grigal 1992; Glass et al. 1991). Our data indicate that fish Hg levels show a negative trend with sulphate

deposition (and thereby Hg deposition) rate (Chapter 7). Data collected and analyzed by Sorensen et al. (1990) found that Hg deposition rate was not significantly related to Hg levels in northern pike. These two observations suggest that factors controlling Hg methylation and/or biotic uptake, and not the input of inorganic Hg *per se*, are the factors controlling fish Hg accumulation. These same factors also likely control human Hg exposure via fish consumption.

The hypothesis that sulphate deposition increases fish Hg contamination and Hg exposure in consumers of fish is apparently false. Evidence from extant literature, correlation analysis and path analysis provide no support for the speculation that the concentration of Hg in fish from drainage lakes in Ontario increases with increasing sulphate deposition. Nor is there any evidence that human Hg exposure is greater in regions of the province receiving greater sulphate deposition. On the contrary, these data and information suggest that sulphate deposition may lead to a decrease in fish Hg contamination and subsequent decrease in Hg exposure in Ontario amerindians, a population known to have a significant subsistence freshwater fishery. However, the evidence for a significant negative trend is weak.

This alternate hypothesis is based on spatial, not temporal, data relating to sulphate deposition, lake water chemistry and fish Hg contamination. However, the original hypothesis being tested was also based on extrapolation of spatial associations. Direct temporal data will be required to determine whether or not our alternate hypothesis holds temporally. These temporal data could be of the following forms: 1) data from the same lakes examined here, to determine if empirical associations have changed with time; 2) the controlled acidification of a lake(s), and observation of simultaneous changes in all relevant water chemistry parameters and fish Hg contamination. This latter approach must include the controlled acidification of the terrestrial watershed so that changes in DOC and Hg transport to the lake(s) can be evaluated.

Based on the empirical and path analyses performed herein, one would predict that lake and watershed acidification would lead to decreased pH, alkalinity and DOC, and an increase in SO_4^{2-} concentration within the lake(s). The rate of Hg accumulation in fish would decrease as DOC levels decreased. However, once DOC was depleted from the water column, and the rate of DOC input from terrestrial runoff was sufficiently reduced by watershed acidification, it is

possible that fish Hg accumulation might again increase, as pH, alkalinity and SO_4^{2-} influence Hg methylation and/or bioavailability.

Our findings may have significant implications for management strategies to control fish Hg contamination in remote lakes. At present, it is generally assumed that the Hg contamination of fish from remote lakes can be effectively reduced by reducing or eliminating anthropogenic Hg emissions to the atmosphere. It would seem apparent, however, that a better understanding of the biogeochemistry of Hg in remote lakes is required before their contamination with Hg can justify extensive (and expensive) remedial actions to control anthropogenic emissions of inorganic Hg. It is quite conceivable that simply reducing Hg without controlling sulphate emissions would have no effect on fish Hg contamination levels, although the input of inorganic Hg to lakes may be reduced.

In order to effect control of human Hg exposure via fish consumption, recommendations to avoid fishing in acidified lakes (such as implied by Raloff 1991) will not necessarily effect the desired health protection. Indeed, our evidence suggests that fish from acidified lakes, provided fish are still present, have lower average Hg contamination. More appropriate recommendations may include to avoid fishing in

small shallow lakes with large terrestrial drainage basins (relative to lake surface area). At present, the most effective short term control strategy remains advice to limit fish consumption, and harvest smaller size classes, as already done by OMOE (1993).

Acid rain has been investigated in Canada for some two to three decades. It has been shown to cause a variety of significant and severe ecological impacts (Schindler 1988; Somers and Harvey 1984; NRCC 1981). However, hypotheses that acid rain poses an indirect human health threat, via Hg accumulation in fish used as food, is not supported by available data and information.

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