HUMAN EXPOSURE TO MERCURY MAY DECREASE AS ACIDIC DEPOSITION INCREASES

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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, and increased sulphate ion concentration ($[SO_4^{2-}]$), 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. Further analysis of these fish data found that fish [Hg] was most significantly (positively) associated with lake water concentrations of dissolved organic carbon (DOC), not pH, alkalinity or $[SO_4^{2-}]$. Lake DOC levels are lower in regions of greater acidic deposition. We propose an alternate hypothesis whereby human Hg exposure declines with increasing acidic deposition. In particular, we propose that increasing sulphate deposition leads to reduced lake DOC levels, which in turn leads to lower Hg in fish, ultimately reducing human Hg exposure via fish consumption.

1. Introduction

It has been hypothesized that exposure to mercury (Hg) by persons who consume freshwater fish increases due to acidic deposition (Goyer *et al.*, 1985; Gilmour and Henry, 1991). However, this hypothesis has never been tested. This association has been inferred from two independent observations. First, sulphate deposition leads to lake acidification, most notably to reduced pH and alkalinity (Neary and Dillon, 1988), and increased sulphate ion concentration ($[SO_4^{2-}]$) (Sullivan *et al.*, 1988). Secondly, reduced lake pH and alkalinity have been associated with increased Hg contamination in fish (Scheider *et al.*, 1979; Wiener *et al.*, 1990), and increased sulphate ion concentration has been linked to increased Hg biomethylation (Gilmour *et al.*, 1992) which should also lead to increased Hg in fish. These Hg-contaminated fish would thereby pose an increased health risk to persons who consumed them.

In the present study, we examine three hypotheses: first that Hg in the hair of Amerindians from Ontario, Canada is higher in regions of higher acidic deposition; second that the Hg contamination in fish species commonly consumed by these Amerindians is higher in regions of higher acidic deposition; and third, that lake water pH, alkalinity and/or $[SO_4^{2-}]$ are the most significant correlates of [Hg] in the fish species being investigated. We will show that all three of these hypotheses are false.

2. Methods

We examined the first hypothesis using Hg exposure data for 3,187 Amerindians, aged 10 to 90 years, residing in 55 reserves located across Ontario (Figure 1). MeHg

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exposure in this population results primarily from consumption of freshwater fish (Clarkson, 1990; WHO, 1990). Data were excluded for individuals on reserves located on rivers or lakes impacted by industrial Hg contamination, and on reserves adjacent to urban centers where fish is commonly purchased from commercial sources. Reserves were ascribed sulphate deposition rates according to their location within sulphate deposition zones of Ontario, as described by Neary *et al.* (1990).

Data relating to total Hg concentrations in hair ($\mu g/g$) (which is directly related to Hg ingestion (WHO, 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). Samples were collected and analysed between 1976 and 1990. 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.

Hg 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. Hair grows at a rate of 1 cm per month (Pelifini *et al.*, 1969) and, therefore, the month of Hg exposure could be deduced from the available data. Hg exposure, and thereby fish consumption rate, 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 (geometric mean [Hg] = 2.95 $\mu g/g$ for females and 2.94 $\mu g/g$). These data had no relationship to year of sampling ($r=0.00$, $p>0.3$, $n=3,187$). Details of hair sample collection and Hg analytical methods are described elsewhere (HWC, 1987; Farant *et al.*, 1981).

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

To test the second hypothesis, that fish Hg contamination is higher in regions of higher sulphate deposition, we obtained unpublished data from the Ontario Ministries of Natural Resources (OMNR) and Environment and Energy (OMOEE) on total fish length and the concentration of Hg in 7,337 walleye (*Stizostedion vitreum vitreum*) from 375 lakes, 5,914 northern pike (*Esox lucius*) from 396 lakes, and 4,125 lake trout (*Salvelinus namaycush*) from 235 lakes (see Figure 2). These species are those most commonly used as food by Amerindians across Ontario (Coad, 1993; Hopper and Power, 1991; Lawn, 1989). Fish were collected between 1970 and 1989, matching well with the hair [Hg] data. Fish collection and Hg analysis methods are described elsewhere (McMurtry *et al.*, 1989; Wren *et al.*, 1991). Again, lakes were ascribed sulphate deposition rates according to their location within sulphate deposition zones of Ontario, as described by Neary *et al.* (1990).

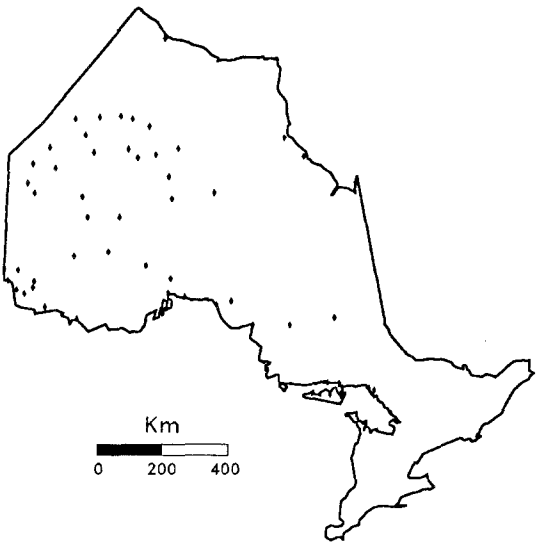

Fig. 1. Location of Amerindian reserves across Ontario from which residents supplied hair samples for Hg analysis.

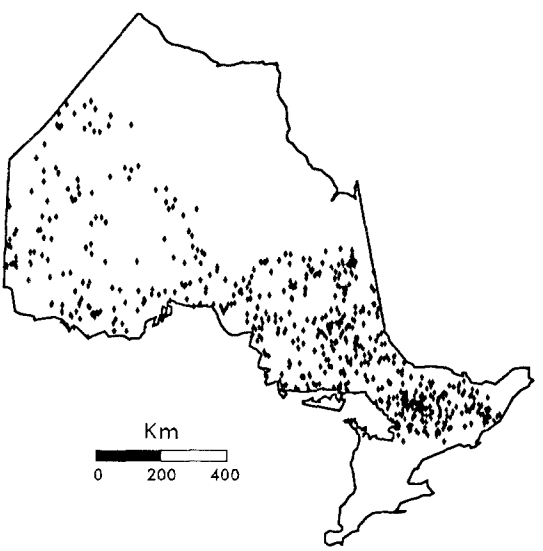

Fig. 2. Location of lakes across Ontario from which fish (lake trout and/or walleye and/or northern pike) were collected.

Fish [Hg] is influenced by a variety of factors which will confound the association between fish [Hg] and acidic deposition. Firstly, [Hg] in fish increases with fish length (Huckabee *et al.*, 1979). Lake and watershed morphometry will also confound this analysis (Bodaly *et al.*, 1993). In particular: 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 is an index of the relative loading rates from terrestrial runoff *vs* direct aerial deposition; lake volume determines the degree to which acids entering the lake will be diluted. Therefore, these factors must be controlled to detect the influence of sulphate deposition on fish [Hg], independent of confounding by these variables. Finally, watershed buffering capacity (H^+ exchange capacity or acid neutralizing capacity) has a direct influence on a lake's susceptibility to acidification (NRCC, 1981), and thereby on any influence that acidic deposition may have on fish [Hg]. Data on lake surface area and lake volume were also provided by OMNR. Data on total watershed area (lake surface + drainage basin) were determined from 1:50,000 scale topographic maps. Lake buffering capacity was determined following the scheme of Cowell and Lucas (1986).

For the third hypothesis, unpublished water chemistry data (pH, alkalinity, $[SO_4^{2-}]$ and dissolved organic carbon concentration (DOC)) for these lakes were obtained from OMOEE. Sampling and analytical methods are described elsewhere (Neary *et al.*, 1990).

3. Results and Discussion

After statistically controlling Amerindian hair[Hg] data for the influences of age, isolation and sex ($R^2=0.366, p<0.0005, n=3187$), residual variation in these data were grouped by regions of sulphate deposition. Residual hair [Hg] represents an increase (>0) or decrease (<0) from the expected ($=0$) degree of contamination predicted from age, sex and degree of isolation. Although differences between regions were not significant by MANCOVA (or ANCOVA of residual [Hg]), a significant negative correlation was observed between mean residuals and the median sulphate deposition rate for each zone (Figure 3).

Fish being the source of Amerindian exposure to Hg, then fish [Hg] should also demonstrate a similar negative association with sulphate deposition. Fish [Hg] data were statistically controlled for the influences of fish length, total watershed area, the ratio of drainage basin area to lake surface area, lake volume and also watershed buffering capacity. All these variables were significant for each species of fish, explaining 15.5%, 16.1% and 41.5% of the total variation in [Hg] among individual fish for walleye, pike and lake trout, respectively. The residuals after controlling for these variables were then grouped by regions of sulphate deposition in Ontario. Residual [Hg] in all three fish species represents an increase (>0) or decrease (<0) from the expected ($=0$) degree of contamination predicted from fish length, lake morphometry and watershed buffering capacity. This residual [Hg] was significantly different among both buffering capacity and sulphate deposition zones by MANCOVA, and mean residual fish [Hg] was negatively related to sulphate deposition, although the correlation was significant only for walleye (Figure 4a,b,c). The non-significant negative relationships may or may not be consistent with the hypothesis that fish Hg is lower in regions with higher sulphate

deposition (statistical power was low, since n=6); they are unequivocally inconsistent with the hypothesis that fish Hg increases with sulphate deposition.

Neither fish [Hg] controlled only for fish length, nor residual fish [Hg], which was controlled for fish length, watershed morphometry and buffering capacity, were strongly correlated with lake pH, alkalinity or $[SO_4^{2-}]$ (Table I). The most significant correlate of fish [Hg] was lake water DOC for all three species of fish. For walleye and lake trout, neither pH, alkalinity nor $[SO_4^{2-}]$ demonstrated any significant association at all with fish [Hg]. For northern pike, residual [Hg] was significantly associated with pH, but less so than with DOC. These observations are consistent with several other reported analyses of Hg in these species (McMurtry *et al.*, 1989; Wren *et al.*, 1991; Sorensen *et al.*, 1990; Heiskary and Helwig, 1986).

The observed decrease in fish [Hg] at higher sulphate deposition rates is consistent with predictions made in a recent review of the literature (Richardson *et al.*, in press). The concentration of DOC is positively pH-dependent (Thurman, 1985), lake water DOC has been shown to decline with increasing lake acidification (Richardson and Currie in prep.; Schindler *et al.*, 1992; Neary *et al.*, 1990), and it is expected that watershed acidification will reduce the transport of DOC from the watershed to lakes via terrestrial runoff (Schindler *et al.*, 1992; de Haan, 1992). DOC plays a predominant role in transporting both inorganic and MeHg to, and binding them in, Ontario lakes (Lee and Iverfeldt, 1991; Mierle and Graham, 1991). Reduced lake water DOC due to acidic deposition would also lead to reduced Hg availability for methylation, and/or bioaccumulation by biota. Therefore, fish [Hg] is lower in regions with greater sulphate deposition, likely due to reduced bioavailability of Hg through loss of DOC-complexed Hg in the water column of these lakes.

4. Conclusions

We have tested three hypotheses related to the inferred increase in human Hg exposure with increasing acidic deposition and have found them to be false, based on available empirical evidence from Ontario Canada. An alternate hypothesis that human Hg exposure declines with increasing acidic deposition appears plausible. In this alternate hypothesis, sulphate deposition results in reduced lake water levels of DOC. 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 concentrations in fish, and reduced human Hg exposure via fish consumption.

The empirical support for this alternate hypothesis is weak, and requires further testing. 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.

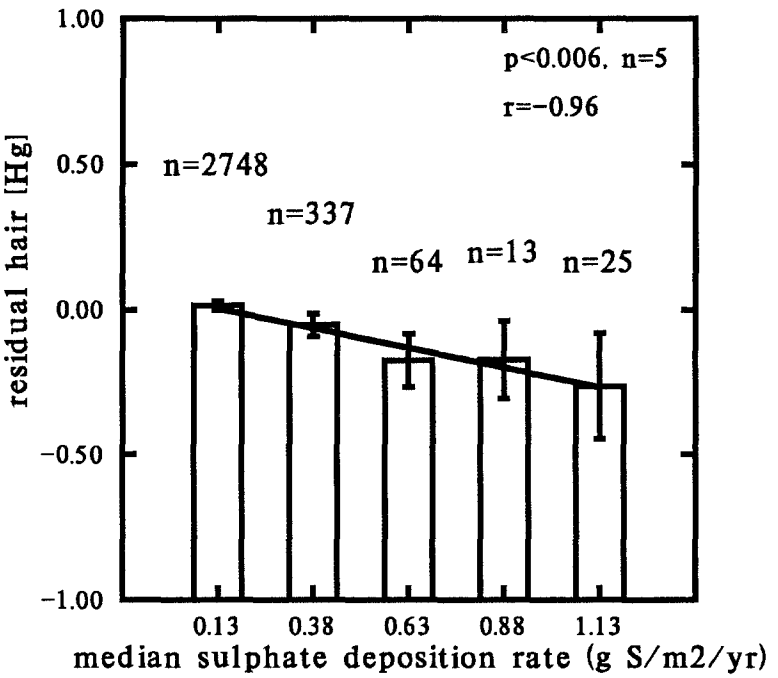

Fig. 3. Hair Hg concentration (controlled for age,sex, and degree of isolation) as a function of sulphate deposition across Ontario. Correlation results are for the association between mean residual hair [Hg] for all individuals within each sulphate deposition zone, and the median sulphate deposition rate for those zones (after Neary *et al.*, 1990). Differences between sulphate deposition zones were not significant by ANCOVA.

Table I

Pearson correlation coefficients for associations between mean ln(fish [Hg]) (controlled for fish length only) and residual fish [Hg] (also controlled for watershed morphometry and buffering capacity) and lake water chemistry parameters. Fish [Hg] was averaged by lake. Only those lakes with data for all four chemistry variables were considered. *=$p<0.01$; **=$p<0.0001$; †=$p>0.05$.

	ln[DOC]	ln[Alk+1]	ln[SO_4^{2-}]	pH
[Hg] controlled for length only				
lake trout (n=108)	+0.55**	+0.16†	-0.11†	0.10†
walleye (n=172)	+0.43**	0.00†	-0.09†	0.00†
pike (n=148)	+0.33**	-0.13†	0.00†	-0.24*
[Hg] also controlled for morphometry and buffering capacity				
lake trout (n=108)	+0.45**	+0.09†	0.00†	0.00†
walleye (n=172)	+0.42**	0.00†	-0.09†	0.00†
northern pike (n=148)	+0.32**	-0.10†	0.00†	-0.21*

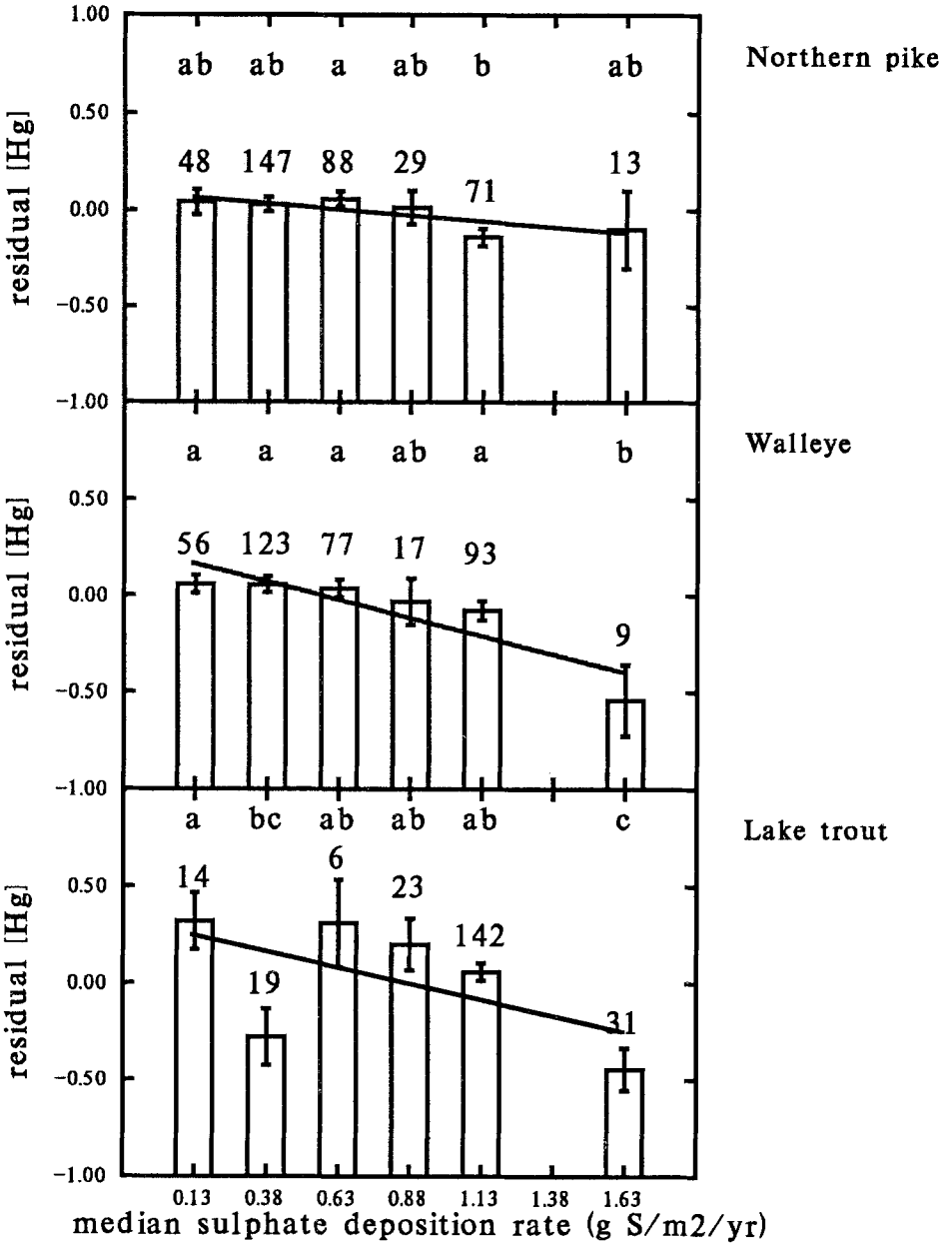

Fig. 4. Fish Hg concentration (controlled for fish length, lake and watershed morphometry and watershed buffering capacity) as a function of sulphate deposition across Ontario. Sample sizes are the number of lakes in each deposition zone. Lines drawn to illustrate general downward trend in residual fish [Hg] with increasing median sulphate deposition rate. Only for walleye was this downward trend statistically significant ($p < 0.02$). There were no data from lakes in the area receiving 1.38 g S/m^2/yr. Number of lakes varies due to availability of data.

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