PAPER

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Neurological abnormalities in a mercury exposed population among indigenous Wayana in Southeast Suriname

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The indigenous Wayana community of Puleowime (Apetina) in Suriname is susceptible to the effects of mercury because they consume large amounts of fish compared to mainstream communities. Small-scale and artisanal gold mining activities occur at numerous sites in eastern and southeastern Suriname placing the Wayana at risk from exposure to mercury released into the environment. A previous community-led risk assessment study showed that the Wayana were at a high lifetime risk of adverse effects from exposure to mercury. Subsequent to this earlier study, the residents of Puleowime requested assistance in a community-led follow-up research project to determine for themselves whether there were health impacts associated with exposure to mercury contamination. Neurotoxic effects consistent with methylmercury exposure were documented in an exposed population through a battery of neurological tests. Although the specific motor and cognitive batteries were not exactly the same, similar associations were observed between neurologic impairment and hair mercury concentrations compared to other studies in the Amazonia region where mean hair mercury levels were in the subacute range.

Environmental impact

Due to the informal characteristics of artisanal mining using mercury to amalgamate gold, exposure assessments of indigenous riverine populations impacted by this practice and its health effects (especially at the nervous system level) are of public health concern. This case study, which combined clinical examination and scoring of individual performance score on a battery of neurological tests in conjunction with the hair mercury data from the 2008 risk assessment and supplemented with additional exposure data in 2012, found neurologic dysfunction consistent with mercury poisoning among residents in Puleowime, Southeast Suriname. This study reveals an important health impact of actual gold mining processes in the Amazonia region of Suriname. These results must drive the attention of public health practitioners to find remedial procedures for the well-being of impacted populations and the improvement of the environment.

Introduction

The indigenous Wayana community of Puleowime (Apetina) in southeast Suriname proposes that, with assistance from outside experts, they can determine whether there were health impacts associated with exposure to mercury (Hg) contamination. In 2008, community members led a research initiative that showed the Wayana population from Puleowime was at a high lifetime risk of adverse effects from exposure to Hg.[1] After leading the risk assessment project in 2008, the appointed leader of the Wayana people in Puleowime requested further assistance performing medical assessments to determine the potential health impacts from this hazard. While many practitioners adopt participatory action research (PAR) for ethical reasons,

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the Wayana people in conjunction with a team of international public health experts adopted PAR for a very pragmatic reason which was to overcome the inadequacies of conventional research in this indigenous setting.[1–3]

There are approximately 503 Wayana people living in Suriname.[4] The Wayana people are dependent on fish as a primary source of protein.[5] Fish are also sources of polyunsaturated fatty acids, iodine, selenium and vitamin D.[6] Human populations that depend on fish as a dietary staple, such as the Wayana people in Suriname, are especially at risk of exposure to Hg.

Suriname lies north of Brazil, between Guyana and French Guiana (Fig. 1). As of 2009, the population of Suriname was approximately 520 000 people.[7] Small-scale and artisanal gold mining activities that release Hg from their operations occur at numerous sites in Eastern and Southeastern Suriname.[8,9]

The objective of this project was to continue using the guidelines for Community-Based Participatory Action Research, which was established as the norm in these communities in

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Fig. 1 Map of Suriname showing location of communities that led the community-directed mercury risk assessment study.

2008, to conduct clinical screening exams and determine whether members of their communities exhibit signs consistent with mercury poisoning on a population level. If these preliminary efforts were suggestive of mercury induced health effects, the community would be in a better position to organize a full scale study and intervention if needed.

Materials and methods

Data collection

Community leaders from Puleowime asked the Suriname Indigenous Health Fund for assistance developing the capacity to perform medical assessments that would determine whether community members show signs of neurological effects consistent with Hg exposure.[1,10]

Mode of research

The approach used was a collegiate form of Participatory Action Research (PAR) in which control and ownership of the process is relinquished to those to whom the research concerns.[11,12]

Participant selection

A Collegiate form of Community-Based Participatory Action Research methodology[12] (CBPAR) was previously adopted in 2008 during a risk assessment study[1] in which researchers and local people worked together as colleagues while local people had control over the process. The collegiate form of CBPAR was adopted for pragmatic reasons in response to the Wayana community's complaint that they have historically been over-studied, have not benefitted from past research, and wished to perform their own studies as opposed to being the subjects in someone else's research. In this study, participants were pre-selected from the participants of the 2008 risk assessment study by villagers for community members who were concerned they were experiencing neurological deficits such as ataxia, tremor or other movement disorder. Incidentally, participants comprised a range of ages weighted towards school age children which

reflected the preferences of the village. By mutual agreement, the villagers who participated in the health assessment process were also those individuals who had the highest previously measured hair mercury levels (*i.e.* >20 ppm as measured in 2008) to increase the likelihood of observing a clinical effect.

Extensive epidemiologic studies among fish-eating populations have assessed mother and child pairs for prenatal methylmercury exposure, the resulting impact on child development, and the relevance of neurological tests in children. In New Zealand and the Faroe Islands, studies showed correlations between prenatal mercury exposure and the neurological development of children.[13–17] In contrast, the Seychelles study did not show adverse effects on neurological development.[18] In this study, clinical signs and symptoms were used as the basis for assessing mercury intoxication. When the environmental history, clinical picture, and mercury levels in biological samples coincide, causal inferential associations are possible[19] and the diagnosis of mercury intoxication can be made.[20] The symptoms of chronic mercury intoxication in childhood include muscular hypotonia followed by a refusal to walk, stand, or sit, tremors, ataxia, coordination problems, as well as unspecific symptoms, such as lack of energy, tiredness, loss of appetite, weight loss, dizziness, and headaches.[20]

Index of neurological integrity

The INI was developed to integrate data collected during clinical analysis. There is no one universal INI. Attributes that are responsive to Hg impacts were combined into an index using the UNEP Health Assessment Survey index as a model.[21] Health in the Wayana communities was accomplished using a modification of the UNEP index model.

The Index of Neurological Integrity (INI) was a composite score from the neurological exam which was comprised of six metrics (G, ST, TP, RT, and FTN), the drawing test which contained four metrics, and the copying test which contained six metrics. A total score of 0–25 was possible.

Procedure for screening exam

Examinations typically required about 30 minutes each and consisted of neuro-physiological testing and a directed screening physical exam in the presence of a translator:

(1) Normal and tandem gait (G) was observed and recorded.

(2) Sensation to light touch and prick were observed and recorded (ST).

(3) Two-point discrimination (TP) was determined on the volar surface of the forearm and recorded.

(4) The Romberg (RT) and the sharpened Romberg test (SRT) was used to investigate the cause of loss of motor coordination in subjects with mild signs of neuropathology.

(5) Finger to nose (FTN) movements were observed and recorded.

(6) Each patient was assigned a Neuro-Score from 0–5 with (0/absent, 1/slight, 2/moderate, 3/marked, 4/severe, 5/extreme) based on the cumulative results of the screening exam above (G, ST, TP, RT, and FTN).

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(7) Participants used pencil and paper for drawing a Frositg test, and copying standard figures described in detail below.

Materials

A private space that permitted a 3 meter walk and a desk and chair for the physician and translator, data collection form, pencils and paper for drawing, caliper, millimeter ruler and long wooden cotton Q-tip (individually wrapped for each examinee), stop watch, reflex hammer, tuning fork, tongue depressor (individually wrapped), flashlight, recording sheet. For two severely impaired screening exams, full neurologic exams were performed in the presence of a translator (30–45 min.)

Drawing test

The drawing test is based on the Eye-Hand Coordination subtest of the Developmental Test of Visual Perception.[22] It includes four items and requires the subject to draw a line from one symbol to the other. The subject is advised to not interrupt while drawing and not touch the borders. The difficulty of the items is graded to the effect that the distances between the borders diminish. The score for each item is 0 = good, 1 = bad or 2 = very bad. Full credit (0 points) is given if the line from one symbol to the other was without interruption, if the pencil was lifted from the paper but the line continues without interruption, crutch or pointed angle or if a light angle or blur occurred in the line. One (1) point is scored if the line touched the borders (but not out of borders). Two (2) points are given if the line was interrupted (considerable interruption, crutch or pointed angle), run out of borders (a white space is visible between the boundaries and the drawn line) or the line was only adumbrated or corrected. In addition the type of error is registered: interruption (I), touch borders (T) and out of borders (B). Finally, a total drawing score from 0 to 8 points is obtained.

Copying figures test

The items of the copying figures test are taken from the Stanford–Binet (S–B) copying test.[23] For this task the subject has to draw six two-dimensional geometric designs. As well as the drawing items the items of this subtest are graded to difficulty. The original S–B copying test uses a standard scoring system to reflect whether the drawings captured the gestalt of the stimulus items. The score for each item is again 0 = good, 1 = bad or 2 = very bad. 0 points are achieved if the gestalt was captured and the drawing was as close to the original as possible. 1 point is given if the gestalt was captured, but the drawing is deficient (e.g. deformation, addition, overdrawing). 2 points are scored if the gestalt of the target was not captured. All in all a sum of 0 to 12 points is possible.

Chevrier et al.[23] acquired another scoring technique for the S–B copying test, a qualitative scoring, that is used in this study as well. The drawings were analysed with regard to their error types:

- rotation: shifting of the whole or a part of the design more than 90° from the horizontal of the page (R);
- distortion: modified from the original (e.g. angles rounds, crooked lines) (D);
- simplification: changed into a less complex one (S);
- perseveration: repeating the whole or a part of the design (P);
- overdrawing: drawing over a design several times (O);
- micro-/macrographia: very small/large drawing (M);
- tremor: appearance of shaky lines in the drawing (T).

Analysis of total Hg levels in human hair

Team members collected hair samples for analysis using methods designed to maximize sample quality and consistency and minimize cross-contamination, which emphasized the use of powderless surgical gloves and new, sterile, stainless steel scissors for each sample collected. All hair samples were collected from the lower occipital region. When long hair strands (>3 cm) were collected, the hair tips were discarded and only the proximal 1 cm were used to reduce variability and because Hg levels can decrease during hair growth under certain conditions. Hair washing procedures were not used to differentiate between airborne and internal Hg. The use of "negative controls" to detect sources of spurious causal inference was not included in this study because the investigation was not able to identify people living under comparable circumstances who have not plausibly been exposed in their lifetime to similar levels mercury through similar pathways. The lack of negative controls limits our ability to make an irrefutable causal inference therefore we limited the objective of our study to the performance of a clinical screening exam and to the determination of whether members of the Wayana communities exhibit signs "consistent" with mercury poisoning on a population level.

Each hair sample, of approximately 20 mg, was stored in a sealed, labeled envelope. The hair samples were analyzed in triplicate for total Hg (THg). Hg analysis was by the cold-vapor technique using the Portable Zeeman Lumex (RA915$^+$/RP-91C) mercury analyzer. The instrument detection level was 0.2 ng g^{-1}. All concentrations were expressed in parts per million THg (equal to μg g^{-1} THg). Measurement of THg levels in hair using the Lumex RA915$^+$/RP-91C portable analyzer had been previously confirmed by laboratory analysis using a modified National Institute for Occupational Safety and Health (NIOSH) 6009 method. In this study, the Lumex was operated in software "On Stream" mode using the procedure in the manufacturer's operation manual. NIST traceable standards #2709 for Hg at 1400 ng g^{-1} and #1633d for Hg at 141 ng g^{-1} were used to standardize the analyzer before and after each ten samples analyzed.

Statistical analyses

Statistical analyses were carried out using SPSS Version 21 (SPSS Institute Inc). Significant associations were identified at the alpha level of 0.05. Seven incomplete records were excluded. The excluded records did not differ from the study population based on age or gender. In this study, hair mercury results were summarized using simple descriptive statistics including

arithmetic mean, median, standard deviation, and range. The mean hair concentrations were evaluated by population, age and gender using the two-tailed t-test assuming equal variances ($p < 0.05$). The association between hair mercury concentrations and individual risk in 2008 and 2012 and between the index of neurological integrity and 2012 Hg mercury concentration and age was assessed by linear regression model in SPSS.

Individual risk

Individual risk is defined here as the probability of having a 5% chance of exhibiting an adverse neurological effect. It is the incremental probability that the hazard will impose an effect on some particular person.[24] It was based on the most conservative of the three dose response functions (DRFs) reported by Sullivan *et al.*[25] in which risk is correlated to the biomarker of Hg concentration in hair as a function of the amount of Hg consumed through fish. According to Sullivan, the probability of having a 5% chance of exhibiting an adverse neurological effect was estimated to be 0 for hair at 0–3 ppm Hg, 1×10^{-4} for hair at 4 ppm, 1×10^{-3} for hair at 5–6 ppm, 2×10^{-3} for hair at 7 ppm, 3×10^{-3} for hair at 8 ppm, 5×10^{-3} for hair at 9 ppm, 1×10^{-2} for hair at 10 ppm, 1×10^{-1} for hair at 11 ppm, 4×10^{-1} for hair at 12 ppm, 6×10^{-1} for hair at 13 ppm, and 9×10^{-1} for hair over 13 ppm.

R^2 value interpretation

Less than 0.04: slight, almost negligible relationship
0.04–0.16: low correlation, definite but small relationship
0.16–0.49: moderate correlation, substantial relationship
0.49–0.81: high correlation, marked relationship
0.81–1.00: very high correlation, very dependable relationship

INI score interpretation

Less than 5: no effect
6–10: few effects
11–15: moderate effects
16–20: high effects
21–25: very high effects

Human subjects review

The SIHF research team consulted with the human subjects review staff at the University of Washington and Simon Fraser University who approved the project plan. The Institutional Review Board staff found that the research design did not require full IRB review since the traditional roles of researcher and research subject did not apply. Since research subjects were co-investigators leading the research process while the Western research team acted as consulting technicians, informed consent was deemed unnecessary. Citing the CDC criteria distinguishing research from 'nonresearch' public health practice (CFR §46.102[d])[26] it was concluded that this project was aimed at a specific public health problem and it was done with the aim of preventing or promoting health, therefore it was deemed to represent nonresearch or public health practice. The IRB acknowledged that there may be secondary benefits when this

investigation yielded insights of generalizable value that merit dissemination, but the research *versus* nonresearch determination would be unchanged because it is based on the primary intent.

Results

Twenty-two individuals who had hair Hg concentrations that exceeded 20 μg g^{-1} in 2008 had repeat hair analyses for Hg and were examined clinically for signs of neuropathology (Table 1). Mean hair Hg concentrations in 2012 were significantly lower in Puleowime (13 ± 4 μg g^{-1}) than in 2008 (23 ± 6 μg g^{-1}) at the 95% confidence level (Table 2). The estimated risk of adverse neurological effects at measured levels of Hg in hair was also lower in 2012 compared to 2008 ($p < 0.01$). Results of the neurological exam indicate that there was a low correlation between neurological problems and hair mercury concentration (Table 3). As age increased the probability of having an abnormal clinical score also increased (moderate, $r^2 = 0.36$). A post-hoc power analysis indicated the chance of detecting a large effect size was greater than 95%.

Based exclusively on medical examinations of subjects the medical team diagnosed six subjects suggesting a 'tentative diagnosis' of Minamata disease (hair mercury between 9–17 ppm, ages from 20–70). The main symptoms of the six cases were disturbance in coordination, glove-and-stocking type sensory disturbance, numbness, failure in two-point discrimination, and tremor (Tables 4–6).

Discussion

This case study, which combined clinical examination and scoring of individual performance score on a battery of neurological tests in conjunction with the hair mercury data from the 2008 risk

Table 1 Criteria for the interpretation of individual risk[a] among indigenous Wayana people in Suriname exposed to mercury

Probability of having a 5% chance of exhibiting an adverse neurological effect

Hair Mercury Concentration (ppm)	Probability of Neurological Effects
0–3	0
4	1×10^{-4}
5–6	1×10^{-3}
7	2×10^{-3}
8	3×10^{-3}
9	5×10^{-3}
10	1×10^{-2}
11	1×10^{-1}
12	4×10^{-1}
>13	6×10^{-1}

[a] T. M. Sullivan, F. W. Lipfert, S. C. Morris and P. D. Moskowitz, Potential health risk reduction arising from reduced mercury emissions from coal-fired power plants, Brookhaven Science Associates, LLC for the United States Department of Energy under Contract no. DE-AC02-98CH10886, 2001.

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Table 2 Criteria for the interpretation of R^2 values and the association between hair mercury concentrations and individual risk and between the index of neurological integrity and Hg mercury concentration and age, which were assessed by linear regression

R^2 value	Interpretation
<0.04	Slight, almost negligible relationship
0.04–0.16	Low correlation, definite but small relationship
0.16–0.49	Moderate correlation, substantial relationship
0.49–0.81	High correlation, marked relationship
0.81–1.00	Very high correlation, very dependable relationship

Table 3 Criteria for the interpretation of the Index of Neurological Integrity (INI) as an indicator of the potential health impacts among individuals exposed to mercury

INI score interpretation	
INI score	Interpretation
<5	No effect
6–10	Few effects
11–15	Moderate effects
16–20	High effects
21–25	Very high effects

assessment and supplemented with additional exposure data in 2012, found neurologic dysfunction consistent with mercury poisoning among residents in Puleowime, Southeast Suriname.

The neurotoxic effects observed in this study were documented through a combined clinical examination and a scoring of individual performance on a battery of neurological tests. Although the specific motor and cognitive batteries were not exactly the same, similar associations between neurologic impairment and hair mercury concentrations were reported in the Tapajós and Pantanal regions of Brazil[27,28] where mean hair mercury levels were in the range of approximately 5 to 10 ppm, and maximum levels were near 30 $\mu g\ g^{-1}$.

Kosatsky and Foran noted in studies for which dose-response could be assessed that there was evidence of neurologic dysfunction in the range of 15 to 30 ppm in hair and there was good evidence that "chronic mercury levels up to 5 ppm in hair are without apparent neurologic effect".[29] In Puleowime, among 22 fish eaters with population mean hair mercury levels of 23 ± 6 $\mu g\ g^{-1}$ in 2008 and 13 ± 4 $\mu g\ g^{-1}$ in 2012, few neurological effects consistent with methylmercury exposure were found in eight individuals (33%), 10 individuals (42%) showed moderate effects, in four individuals (17%) the neurological effects were high and in two (8%) the neurological effects consistent with methylmercury exposure were very high.

One potential limitation of the current study is the lack of adequate control for confounding factors. Other possible explanations for symptoms such as fatigue, dizziness, and tremors found during medical examinations that could potentially introduce a false diagnosis into the clinical examination include alcohol consumption, drug use, smoking, malaria and other tropical diseases, tuberculosis, parasitosis, constant

Table 4 Mercury exposure, health assessment survey and demographic data. The Index of Neurological Integrity (INI) was a score assigned by the attending physician that combined observations from the neurological exam which was comprised of six metrics, the drawing test which contained four metrics, and the copying test which contained six metrics: gate (G), sensation to light or touch (ST), two-point discrimination test (TP), Romberg test (RT), sharpened Romberg test (SRT) and the finger to nose test (FTN). A total score of 0–25 was possible. Neurological impacts were designated as positive (+ve) for (G, ST, TP, RT, SRT and FTN)

Sample no.	2008 Hg $\mu g\ g^{-1}$	2012 Hg $\mu g\ g^{-1}$	Age	Gender	G	FTN	RT	SRT	ST	TP	Neuro score	Copying	Drawing	INI
1	16	11	70	F	+ve	+ve	0	+ve	+ve	0	4	8	8	20
2	20	12	37	F	+ve	0	0	+ve	0	0	2	5	2	9
3	24	14	6	M	0	+ve	+ve	+ve	0	0	3	5	4	12
4	28	21	32	F	0	0	0	0	+ve	0	1	2	4	7
5	25	18	41	F	+ve	+ve	0	0	0	0	2	5	8	15
6	25	18	10	F	0	0	0	0	+ve	0	1	3	8	12
7	26	14	5	F	0	0	0	0	+ve	+ve	2	7	6	15
8	18	17	71	M	+ve	+ve	+ve	+ve	0	+ve	5	10	8	23
9	22	19	9	F	0	0	+ve	0	0	0	1	3	8	12
10	21	12	57	F	+ve	0	+ve	+ve	+ve	0	4	4	4	12
11	26	14	9	F	0	0	0	+ve	0	0	1	3	4	8
12	32	18	27	F	0	+ve	0	0	+ve	0	2	5	8	15
13	20	15	6	F	0	0	+ve	0	+ve	+ve	3	7	1	11
14	12	10	70	F	+ve	+ve	+ve	+ve	+ve	+ve	6	11	2	19
15	34	10	10	M	0	0	0	0	+ve	0	1	2	6	9
16	13	17	20	F	+ve	+ve	+ve	+ve	+ve	0	5	2	8	15
17	22	9	18	F	0	0	0	+ve	0	0	1	4	8	13
18	24	9	49	F	+ve	0	+ve	+ve	+ve	+ve	4	7	8	19
19	20	11	15	F	0	+ve	0	+ve	0	0	2	2	4	8
20	30	13	37	F	0	0	0	+ve	0	+ve	2	4	4	10
21	30	10	28	F	0	0	0	0	+ve	+ve	2	6	2	10
22	24	20	6	M	0	+ve	+ve	+ve	+ve	0	2	6	1	9

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Table 5 Descriptive statistics including age, sample size, total hair mercury concentrations (μg g^{-1}), individual risk values, and index of neurological integrity results for Puleowime (Apetina) in 2008 and 2012

| | Puleowime (Apetina) | | |
	2008	2012	
Average age	24	29	
Number	158	22	
Mean hair total mercury concentration (μg g^{-1}) $\pm$ SD	23 $\pm$ 6	13 $\pm$ 4	$r^2 = 0.01$
Median hair total mercury concentration (μg g^{-1})	24	14	
Range hair total mercury concentration (μg g^{-1})	12 − 34	9 − 21	
Mean of individual risk (08 × 12)	0.86	0.57	$r^2 = 0.03$
Mean index of neurological integrity (0 good − 25 impaired)	—	13	
Median index of neurological integrity (0 good − 25 impaired)	—	12	
Range for index of neurological integrity (0 good − 25 impaired)	—	7 − 22	

Table 6 The coefficient of determination (r^2) and probability values (p) as measures of the strength of association between parameters

		R^2	p
Total mercury concentration 2008 × total mercury concentration 2012	Students t-test	—	<0.01
Index of neurological integrity × gender	Students t-test	—	0.91
Index of neurological integrity × 2012 total mercury concentration	Regression	0.01	—
Index of neurological integrity × age	Regression	0.36	—

handling of gasoline, kerosene or pesticides, epilepsy, stroke, Parkinson's disease, other health problems (kidney, blood pressure, pneumonia), stress, allergies, arthritis, diabetes, venereal disease, number of dental amalgam fillings, ingestion of selenium, or exposure to other pollutants such as PCB's.[30] The observed dependence of the clinical scores on age might have enhanced the influence of some of these confounders. However, it is possible that the participation of individuals which were weighted towards school age children and the isolation of the Wayana people living a traditional lifestyle in the Amazonian forest would moderate the importance of many of these potentially confounding factors.

The effect of diet on the toxicity of methylmercury is an emerging concern.[22] The community of Puleowime, in its attempt to control their exposure to mercury is at risk of reducing their consumption of fish which could lead to a diet deficient in essential nutrients including protein. Ironically, if this happens individuals could increase their susceptibility to the toxic effects of methylmercury and cause adverse effects that might be attributed to methylmercury (e.g. developmental delays, poorer performance on neurological tests, immunological deficiencies). The nutritional benefits of fish, which are rich in protein, in important nutrients and essential oils, and low in saturated-fat, may reduce susceptibility to the toxic effects of methylmercury.[31] However, the extent to which omega-3 fatty acids and protein influence the uptake, distribution and effects of methylmercury exposure have not been sufficiently investigated to allow a precise characterization of the relationship of a fish diet to mercury toxicity.

Among non-fish-eating communities, hair mercury concentrations reflect primarily exposure to inorganic mercury and typically are in the range of 0.2 to 0.8 μg g^{-1}.[32] In communities that consume fish on a regular basis (i.e., daily) total hair mercury levels are an order of magnitude higher and most of the mercury is in the form of methylmercury.[10] Therefore, total mercury in hair of regular fish consumers is an acceptable surrogate for methylmercury in hair.

Although the probability of having an abnormal clinical score increases with increasing Hg, the small sample size and screening nature of the design limited the study findings as evidence for a causal relationship between Hg exposure, fish consumption and neurological outcomes. A review of two literature surveys[29,33] included 13 investigations on the health effects from moderate exposure to Hg through fish consumption revealed they were similarly limited in their ability to show that the observed neurological effects were dose-dependent, i.e., increasing in magnitude with increasing hair mercury levels up to a maximum of approximately 50 μg g^{-1}.

While several studies have shown that Hg levels in hair are higher in residents of areas contaminated by mercury than in residents of uncontaminated regions, others show wide variations depending on the relative importance of fish in the diet.[8] In Puleowime, the community fills most of its dietary needs by fishing. The Wayana in Puleowime live in an isolated village on the Tapanahoni River and are considered excellent examples of members of a "fishing civilization". Although the actual exposure among indigenous populations can be highly variable, location specific, and they will depend on local fish Hg levels and individual fish consumption patterns, investigations by Frery et al.[5] show that most subjects take more than 14 fish meals per week.

Seven of the studies reviewed had no statistical analyses of the dose-response relationship nor were dose-effect relations observed between the bioindicators of exposure to mercury and

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the neurological outcomes.[34] In these studies, elevated hair mercury levels were associated with symptoms of mercury toxicity. The small sample size of these studies limited, however, the study's findings as evidence for a relationship between mercury exposure, fish consumption and neurological outcomes. Larger, rigorously controlled studies are needed, including dietary intervention trials. Six other studies from the Amazonia region showed neurotoxic effects below 50 μg g^{-1} hair-Hg. In these studies, significant dose-effect associations were reported for motor, visual and cognitive functions.[35–40]

The authors of the Tapajós region fish consumption study emphasized that the observed correlations may be related to exposures previously accumulated over their lifetime rather than the sub-acute effects of current mercury levels.[27] This observation reveals a caveat with respect to the use of hair as a biomarker. In a mouse study results showed that exposure to low levels of methylmercury produced behavioral effects that depend on the lifetime exposure to Hg.[41] The authors of the mouse study concluded that lifetime exposure should be a component of the risk assessment process for Hg neurotoxicity. Although hair is the biomarker that best integrates exposure to mercury over the longest period of time it can only estimate exposure over many months depending on the length of the sample taken and does not provide an estimate of lifetime exposure.

Conclusion

This case study, which combined clinical examination and scoring of individual performance score on a battery of neurological tests in conjunction with the hair mercury data from the 2008 risk assessment and supplemented with additional exposure data in 2012, found neurologic dysfunction consistent with mercury poisoning among residents in Puleowime, Southeast Suriname.

Acknowledgements

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