Original Article

Fish consumption and health in French Polynesia

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French Polynesians, like other remote maritime populations are intimately connected to the ocean which nourishes their daily life and culture. Their reliance on fish raises the issue of potential exposure to harmful natural and anthropogenic contaminants as well as providing essential nutrients. The purpose of this study was to assess the risks and benefits of fish consumption in French Polynesia. This cross-sectional study included 195 adults aged 18 years old and over from the Tahiti and Moorea islands. Fatty acids, selenium (Se) and mercury (Hg) blood concentrations were measured in participants and were all very high. Blood concentrations indicate that Hg, Se and omega-3 fatty acids have a common origin, i.e. fish consumption. In comparing the Polynesian group with northern populations, we found that the Polynesian group had levels of Hg similar to those observed in Inuit populations (geometric mean (range): 90.3 (15-420) nmol/L vs. Inuit: m(r): 79.6 (4-560) nmol/L). Similar results were observed with Se blood concentrations. The fatty acid concentration was also similar to that of the Inuit population even though the specific profile of fatty acids differed. For the first time, we report very high blood concentrations of mercury, selenium and omega-3 fatty acids in a fishing population from the South Pacific, comparable to those reported among fishing populations from the Northern hemisphere. Further work is ongoing to better substantiate public health nutritional policies.

Key Words: Seafood, mercury, selenium, omega-3 fatty acids, Polynesia

INTRODUCTION

Polynesians are intimately connected to the oceanic environment which nourishes their daily life and culture. They still rely on it, as an important part of their daily diet is sea food and are among the highest fish consuming nations in the world. Among the small developing island states in the Pacific and Indian oceans, as well as in the Caribbean, several countries have a per capita fish consumption of over 50 kg a year, compared to 16 kg a year for the world average. Indeed with 54 kg/year, French Polynesia is listed among the 23 countries where people consume more than 50 kg of fish per annum.[1] The consumption of high amounts of fish raises the issue of potential exposure to harmful natural and anthropogenic contaminants while providing important nutrients essential to health.[2] Balancing the risks and benefits from seafood is a burning debate not only for urban individuals, but certainly and even more so for communities who rely on this diet for their subsistence. Populations from the circumpolar region have had to face such a dilemma over the last decade.[3]

Numerous studies have reported that methylmercury present in predator fish represents a potential health threat particularly for the developing fetus. Neurobehavioral disturbances have been associated with high prenatal exposure to this seafood borne metal.

On the other hand, those populations also receive key nutrients through fish consumption which may counterbalance mercury toxicity[4] and provide nutritional benefits. Maritime populations have a generally high intake of long chain polyunsaturated fatty acids (PUFAs), the most important compounds being eicosapentanoic acid (EPA) and docosahexanoic acid (DHA). In addition, fish is an excellent source of selenium (Se).[5] It is currently believed that selenium plays a role as an antioxidant in the prevention of atherosclerotic diseases, as this essential element is an integral part of the antioxidant enzyme glutathione peroxidase.[6] Furthermore, it is proposed that selenium may exert an antagonistic effect on mercury toxicity.[7] Selenium is also extremely effective in the prevention of oxidative stress-related diseases, particularly prostate cancer.[8]

Several health organisations recommend eating fish twice a week for the general population.[9,10] Fish consumption is largely recognised as beneficial for brain development[11,12] and as being protective against cardiovascular diseases,[13-15] mental disorders [16-18] and various inflammatory conditions such as bowel diseases, asthma, and arthritis.[19]

This risk benefit dilemma is now largely debated at the global scale following reports on contaminants found in farmed salmon.[20] Excellent reviews trying to balance the risks and benefits have since been published.[21] While a lot of information is now available in the northern hemisphere

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Manuscript received 13 August 2007. Initial review completed 8 September 2007. Revision accepted 8 November 2007.

about both contaminant concentrations in fish as well as these species' nutritional value, very little has been reported for the southern part of the globe.

For example, it is generally stated that PUFAs are present in important quantities in cold water marine fish and less in tropical species in which omega-6 fatty acids are mostly present.[22]

Furthermore, anthropogenic contaminants are believed to be low in those regions located far away from industrial emissions.

French Polynesia is located in the South Pacific (Fig.1). The total population is estimated to be 275 000 scattered over 68 islands (among a total of 118). Tahiti, the main island, comprises of more than 70% of the total French Polynesian population. A rapid modernisation of society started in the 50's with increased access to imported food, a varied but excessive diet, the rapid development of obesity and the emergence of coronary heart disease (CHD). For example, in 1995, 36% and 48% of men and women respectively, had a BMI > 30.[23] In the same study the average caloric intake was estimated at 3350 and 4400 Kcal/d in women and men respectively. Little information is available on CHD morbidity but in the 80's, an average of 20 infarctions were treated each year at the cardiology department of the territorial hospital and in 2004 this number increased to 200 (personal communication G Papouin, Centre Hospitalier Mamao, Papeete). Interestingly, CHD mortality is still low in French Polynesia. In 2000, the age standardized mortality rate for CHD (ICD-10: I21 and I22) was 28.9/100 000 for males compared to 72.7/100 000 in the USA.

Considering the importance of fish consumption in Polynesia for chronic disease prevention as well as the potential risk related to the presence of mercury in seafood from a public health perspective, we recruited a group of 189 Polynesians to assess their body burden of mercury and biomarkers of seafood-based nutrient intake (PUFAs and Se). The purpose of this study was to balance the risks and benefits of fish consumption in French Polynesia using biological markers.

METHODS

Population

The following preliminary results were obtained during a study conducted in French Polynesia between March 2001 and June 1st 2004. This study compared 65 participants exposed to ciguatera disease with 130 participants who were not exposed. All participants were adults aged 18 years old and over from the Tahiti and Moorea islands.[24] In the course of this study, blood levels of Hg, Se and PUFAs were analyzed. As no significant differences were observed between exposed and non exposed participants except in gender representation and n-3/n-6 ratio, data from cases and controls were pooled for a final sample size of 195. The study was approved by the Research Ethics Committee of French Polynesia.

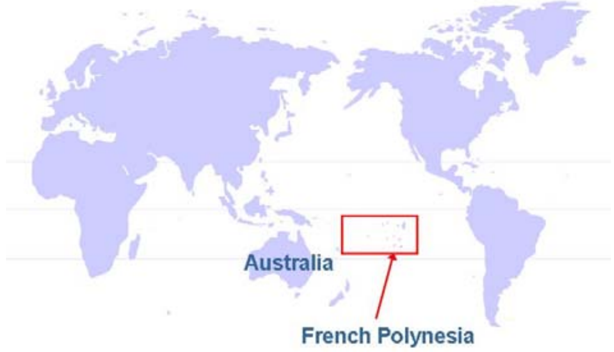

Figure 1. Map of the Pacific Ocean

Laboratory analyses

The fatty acid composition of plasma phospholipids was measured after total lipid extraction with a chloroform/methanol mixture. The separation of phospholipids was performed by thin layer chromatography and methylation of fatty acids,[25] followed by capillary gas-liquid chromatography using a DB-23 column (39m x 0.25 mm ID x 0.25 um thickness) in a HP-Packard GC chromatograph. This standard method is currently used at the Québec Lipid Research Centre. These analyses were available for only 116 participants. Selenium levels were analyzed by instrumental neutron-activation analysis. For mercury determination in blood (INSPQ method: M-109), total blood mercury concentration was determined by cold vapour atomic absorption spectrometry (Pharmacia). Samples were microwave-digested using nitric acid and an aliquot was used for the analysis. Inorganic mercury was also determined in 84 blood samples with concentrations of total mercury above 100 nmol/L. Se and Hg analyses were performed at the Laboratory of Human Toxicology of the Quebec Public Health Institute. Accuracy and precision are measured using reference material from the laboratory's Inter-laboratory Comparison Program. Periodic evaluations were also conducted by the participation of the INSPQ in the same program. The correspondence between international units and customary units is as follow: mercury 100 nmol/L = 20 μg/L; Selenium 1 μmol/L = 79 μg/L.

Statistical analysis

Results are presented as arithmetic means and standard deviations or as geometric means at 95% confidence intervals for variables that are not distributed normally. We performed Pearson correlations to quantify the linear relationship between variables. Comparisons between samples were obtained by analysis of variance (ANOVA), a conventional t-test or Fisher exact tests according to the nature of the variables. Data were analysed using SAS 9.1 version (SAS Institute Cary, NC) and the statistical significance was set at $\alpha=0.05$.

RESULTS

The sample included 115 men (mean age: 46.8±9.4 years) and 80 women (mean age: 45.1±8.7 years). Gender distribution was similar according to age, tobacco consumption and body mass index but differed for alcohol consumption ($p=0.001$).

Table 1 presents mean age, body mass index (BMI) as well as fatty acids and metal blood concentrations for the 195 participants. The mean total mercury blood concentration was high at 108 nmol/L with a maximum individual result at 420 nmol/L. Selenium was also elevated. The fatty acid profile shows high total omega-3 fatty acids in

Table 1. Age, BMI, mercury, selenium and PUFA in adults Polynesians

Variables	N	Mean†	SD	G. Mean‡	SD	Min.	Max.	Percentiles				
								p10	p25	p50	p75	p90
Age (years)	195	46.0	± 9.1	45.0	± 1.25	18.1	71.5	34.8	41.4	46.9	50.9	55.4
BMI (kg/m^2)	190	27.5	± 5.4	27.0	± 1.21	16.8	47.7	21.1	23.6	27.1	30.7	35.5
Hg-T (nmol/L)	188	108	± 67.7	90.3	± 1.85	15.0	420	41.0	59.5	93.0	142.4	205
Se (µmol/L)	187	4.47	± 2.5	3.99	± 1.57	2.10	15.5	2.40	2.80	3.80	5.10	7.80
n-3 tot %	116	7.23	± 2.20	6.93	± 1.34	3.25	16.2	4.70	5.57	6.91	8.31	10.2
EPA %	116	1.10	± 0.90	0.90	± 1.78	0.23	7.43	0.46	0.59	0.86	1.30	
DHA %	116	5.01	± 1.39	4.82	± 1.33	2.03	8.56	3.30	3.96	4.87	5.87	7.21
EPA+DHA %	116	6.11	± 2.03	5.80	± 1.37	2.45	14.6	3.78	4.62	5.75	7.09	9.0
Ratio n3/n6	116	0.27	± 0.17	0.23	± 1.71	0.08	0.87	0.13	0.16	0.21	0.32	
SAFA %	116	46.6	± 1.45	46.5	± 1.03	38.7	49.3	45.1	46.0	46.8	47.5	47.9
MUFA %	116	12.1	± 1.45	12.0	± 1.13	9.1	16.7	10.4	10.8	12.0	13.1	13.6
HUFA %	116	20.3	± 3.40	20.0	± 1.18	12.4	29.6	16.4	17.8	19.9	22.5	25.4

Hg-T: Total mercury, Se: Selenium, Σ n–3: sum of n–3 fatty acids, EPA : Eicosapentanoic acid- C20_5n3; DHA : Docohexanoïc acid C22_6n3; MUFA: monosaturated fatty acids, SAFA: saturated fatty acids, HUFA sum of n–6 and n–3 fatty acids. n3-tot=Sum (C18_3n3, C18_4n3, C20_3n3, C20_4n3, C20_5n3, C22_5n3, C22_6n3); EPA+DHA= Sum (C20_5n3, C22_6n3); Ratio n3/n6 = n3-tot/ n6-tot; MUFA = Sum (C14_1, C16_1, C18_1, C20_1, C22_1, C24_1); PUFA = Sum (n6-tot, n3-tot); HUFA = Sum (n6HUFA, n3HUFA).
† Arithmetic mean; ‡ Geometric mean.

Table 2. Correlation coefficients for biomarkers of fish consumption and age

VARIABLES	Hg-T	MeHg	Se
Hg-T	1	0.99	0.54
p	-	<0.0001	<0.0001
N	188	84	187
AA	0.23	0.35	0.31
p	0.0012	<0.01	0.0007
N	115	56	114
EPA :	0.40	0.22	0.67
p	<0.0001	0.10	<0.0001
N	115	56	114
DHA:	0.40	0.28	0.47
p	<0.0001	0.043	<0.0001
N	115	56	114

EPA: Eicosapentanoic acid - C20_5n3; DHA: Docohexanoïc acid C22_6n3; AA: arachidonic acid - C20_4n4; Hg-T: Total mercury; MeHg: Methylmercury; Se: Selenium.

plasma phospholipids (7.2%) and surprisingly, the EPA concentration was very low, only 1.1% compared to DHA (5%). The n–3/n–6 PUFA ratio was also high (0.27). As shown in table 2, blood concentrations indicate that Hg, Se and omega-3 fatty acids are all auto correlated and have a common origin, i.e. fish consumption. Furthermore, among the 84 blood samples with total mercury concentrations above100 nmol/l, methylmercury represented 87.5% of total mercury, signing the seafood origin of mercury exposure.

Blood levels of fatty acids, Hg and Se were different according to gender, men having the highest concentrations for all analyses compared to women (table 3). However, metals and fatty acids blood profiles across age strata are not as clear. The 35-49 stratum shows the highest concentrations. These results suggested a difference in fish consumption between gender and age. BMI, smoking and alcohol consumption were not associated with fish consumption biomarkers.

DISCUSSION

Distribution and sources

In comparing the Polynesian group with Arctic coastal populations, we found that the Polynesian group had levels of Hg, Se and omega-3 fatty acid similar to those observed in the Inuit population.[26,27] In 1992, Inuit adults showed a geometric mean mercury blood concentration of 79.6 nmol/L, with a maximum of 560 nmol/L, compared to 90.3 nmol/L (max. 420 nmol/L) for Polynesians.[27]

The observation that Hg, Se and n–3 PUFA were 30% lower among women is consistent with data from the 1995 dietary survey showing an average consumption of fish of 68.5 kg/pers/year for men and 44 kg/pers/year for women.[29]

In this population, like in other fish eating populations located far away from industrial emissions, the source of mercury exposure is seafood. In fish, it is estimated that 84% of mercury is methylated[28] which corresponds to what we found in the blood of participants (87.5% of Hg was in the form of methylmercury).

Unfortunately we did not have a dietary questionnaire which would have allowed us to identify the most important fish species consumed associated with mercury exposure. The Polynesian Government, in 2001-2004, measured Hg in pelagic fish species in view of exporting them and found that the concentrations for tuna and bonito (*Thunnus alalunga, Thunnus albacares*. *Thunnus obesus* and *Katsuwonus pelamis*) were all around 0.3 µg/g (n=61).[30] For a mean blood concentration of 18.6 µg/L (92.7 nmol/L), we can estimate an average daily intake of

Table 3. Hg, Se and fatty acids concentrations according to lifestyle and socio demographic variables

Variables	EPA	DHA	EPA+DHA	Ratio n3/n6	HgT	Se
GENDER						
Women	0.74 ± 0.07	4.57 ± 1.04	5.36 ± 0.04	0.22 ± 0.07	75.2 ± 1.07	3.5 ± 0.05
Men	1.06 ± 0.07	5.00 ± 1.03	6.17 ± 0.04	0.25 ± 0.07	103 ± 1.06	4.5 ± 0.04
p value	<0.001	0.08	0.01	0.12	<0.001	<0.001
Age†						
18-34	0.57 ± 0.05	3.60 ± 0.31	4.18 ± 0.30	0.16 ± 0.16	68.0 ± 0.51	3.0 ± 0.25
35-49	0.98 ± 0.05	5.04 ± 0.24	6.11 ± 0.26	0.25 ± 0.07	92.8 ± 0.67	4.2 ± 0.48
50-74	0.84 ± 0.06	4.75 ± 0.29	5.70 ± 0.32	0.24 ± 0.08	90.0 ± 0.56	4.1 ± 0.39
p value	0.01	0.00	0.00	0.04	0.17	0.01
BMI ‡	0.01	0.00	0.00	0.00	0.02	0.02
p value	0.58	0.87	0.77	0.58	<0.001	<0.001
Smoking †						
Yes	0.90 ± 0.07	4.85 ± 0.03	5.87 ± 0.04	0.24 ± 0.06	86.5 ± 0.06	4.0 ± 0.04
No	0.90 ± 0.09	4.71 ± 0.04	5.64 ± 0.05	0.23 ± 0.08	96.5 ± 0.08	6.9 ± 0.06
p value	0.91	0.57	0.58	0.50	0.25	0.81
Alcohol †						
Yes	0.82 ± 0.09	4.90 ± 0.04	5.75 ± 0.05	0.23 ± 0.08	84.8 ± 0.08	3.60 ± 0.06
No	0.95 ± 0.07	4.76 ± 0.34	5.81 ± 0.04	0.23 ± 0.06	91.8 ± 0.05	4.14 ± 0.04
p value	0.19	0.06	0.95	1.00	0.45	0.05

† Geometric mean ; ‡ β coefficient.

approximately 20 µg/day or 7300 µg per year. Data from a health and nutrition survey conducted in 1995 [29] indicate that the average total tuna and bonito consumption was 10.5 kg (for a total of 53.3 kg with other fish species and shellfish) per year. Ten and a half kg of tuna/bonito contains on average 0.3 µg/g of mercury, and corresponds to a yearly intake of 3150 µg of mercury per person, which in turn corresponds to 50% of the mercury dose. Thus we can estimate that tuna and bonito alone represent around 50% of the Hg exposure, the rest originating from other species (low consumption, high concentration such as swordfish: 0.8 µg/g, marlin: 1.7 µg/g, shark: 2.2 µg/g and high consumption, low concentration such as mahi mahi (0.1 µg/g) [30] and lagoon fish <0.1 µg/g, preliminary data). For economic reasons, French Polynesia has developed commercial pelagic fisheries and these fish are now highly accessible for the consumer. Moreover the rapid modernisation of the society in Papeete (Tahiti) makes the consumption of tuna much easier than lagoon fish which need to be gutted and scaled.

For selenium, it is unclear why Polynesians have such high concentrations of blood selenium (4.47 µmol/L). Compared to France (foods imported to Polynesia come from France), blood concentrations are around 4 times higher in Polynesia even considering the whole blood/ plasma ratio. In France, women also had significantly lower serum Se concentrations than men (1.09 (SD 0.19) µmol/L (n=7423) and 1.14 (SD 0.20) µmol/L (n=4915).[31] Selenium is usually measured in plasma or serum; however whole blood determination is sometimes reported especially for highly exposed populations. Van der Torre et al. (1991) [32] reported that Se continued to increase while plasma Se remained stable after supplementation of a group of Dutch men. In the general population, a ratio of Se-blood/ Se-plasma is usually 1.1-1.2.[33] In comparison, a mean value for serum Se in seven European Union countries can be calculated as 1 µmol/L, and the optimum level of serum Se is estimated to be 1.27 µmol/L [34] In this study, the fact that Se was so strongly correlated with mercury and n–3 PUFA suggests that most of the exposure is linked to fish consumption.

Risk due to mercury exposure

Methylmercury is a potent neurotoxicant, especially for the developing brain of the fetus. Two well designed major studies investigated the effects of prenatal exposure to MeHg on child neurodevelopment. In the Seychelles Child Development Study, results of 46 neurobehavioral endpoints were reported in children from 6 to 108 months of age and no adverse association with prenatal mercury exposure was shown.[35-40] By contrast, in the Faroe Island cohort, prenatal mercury exposure was significantly associated with a decreased neurological optimality score at 2 weeks of age,[41] neuropsychological dysfunctions in the areas of language, attention and memory at 7 years of age,[42] longer reaction time on a continued performance task, and deficits in cued naming at 14 years of age.[43] Many hypotheses have been proposed to explain discrepancies between these two well designed cohorts, such as peaks (Faroe) vs. stable Hg exposure (Seychelles), high Se (Seychelles) vs. moderate Se levels (Faroe), as well as other differences such as fatty acids profiles and possible polychlorinated biphenyls (PCB) effects. We thus measured persistent organic contaminants (14 PCB congeners and 12 chlorinated pesticides) by high resolution gas chromatography in 3 pools of plasma samples (each of them being constituted of 4 randomly selected individual samples). Polychlorinated biphenyls 153 (the most prominent congener) averaged 80 ng/g in plasma lipids (data not shown) and was low compared to the mean concentration of 450 ng/g lipids found in pregnant women who participated to the Faroe Island cohort study.[44]

According to international guidelines, FAO/WHO (2006)[45] recently reviewed the Provisional Tolerable Weekly Intake (PTWI) for methylmercury (MeHg) of 0.46 µg/kg body weight/day for adults (0.23 µg/kg/bw/day for pregnant women). Considering that MeHg represents 70% of total mercury in fish, we can estimate that the PTWI for adults for total Hg is around 0.70 µg/kg/bw/day which corresponds to 40 µg/L in blood or 200 nmol/L. Around 50% of participants have a low risk level of Hg (< 100 nmol/L) and 50% have a blood concentration in "the at-risk zone" (100 – 500 nmol/L). In the USA, in 1999-2002, the National Health and Nutrition Study (NHANES) found that blood Hg levels in young American children and women of childbearing age were usually below levels of concern (In the USA this level of 5.8 µg/L (28.9 nmol/L) and corresponds to a maternal intake of 0.1 µg Hg/kg/bw/day, about 2 times lower than the 0.23 µg/kg/bw/day FAO/WHO guideline for pregnant women). Only 6% of women of childbearing age had levels at or above a reference dose, an estimated level assumed to be without appreciable harm (≥5.8 µg/L). However, 16% of adult female participants who self-identified as Asian, Pacific Islander, Native American, or multiracial had blood mercury levels ≥5.8 µg/L.[46] In Polynesia, almost all participants had Hg blood concentrations above this 5.8 µg/l (28.9 nmol/L) US threshold.

In Polynesia, the geometric mean Hg concentration in adults was 90.3 nmol/L (18.6 µg/L), a concentration similar to other fish eating populations. In the Nunavik Inuit population (Arctic Quebec, Canada), blood levels of mercury in adults were 82.7 nmol/L (16.6 µg/L), at least 10 times greater than those found in a population sample from southern Quebec.[26] In Inuit, cord blood levels of Hg were 92.2 nmol/L (18.5 µg/L), similar to that observed in the Faroe Islands (121 nmol/L or 24.2 µg/L);[47] and slightly lower than in the Seychelles Islands cohort.[48] It is also important to note that in our study, among the 39 female participants of childbearing age (< 45 yrs), the mean mercury concentration was 65.4 nmol/L (compared to the 105 nmol/L arithmetic mean for the entire group). Polynesian women of childbearing age are exposed to mercury doses similar to those found in Inuit, Faroese or Seychellois.

Benefits from nutrients

Blood selenium concentrations in Polynesian were very high (4.5µmol/L or 360 µg/L) just as cord blood Se levels in Nunavik Inuit were also reported to be high (3.7 µmol/L) compared to other populations: 2.6 times higher than in the Faroe Islands cohort [48] and 9 times higher than in Greenlanders,[49] even considering that these measures were performed on serum. For the general population in North America, selenium mainly comes from wheat used in bread and cereals, and from meat, poultry and fish.[50] The mean selenium intake level in Canada is known to be one of the highest in the world with Japan and Venezuela.[51] In Polynesia, we hypothesized that a high selenium intake comes from fish consumption. Analyses of lagoon and pelagic fishes for Hg and Se content are ongoing. The significance of the effect of high selenium exposure on chronic diseases such as CVD and prostate cancer, as well as on protection against mercury toxicity might be of great public health importance in Polynesia.

The majority of the Se blood concentrations (85%) in this study were below 7 µmol/L, a concentration corresponding to the individual daily maximum safe intake suggested.[52]

Polynesians have relatively high concentrations of omega-3 fatty acids in their plasma phospholipids similar to the Inuit population, however their profiles differ. It is interesting to note that the EPA plasma concentrations were much lower in Polynesians (1.1% vs. 3% in Inuit) even though DHA concentrations were similar (5% both groups). EPA is known to be cardio protective and anti-inflammatory while DHA is more essential for brain and retina functioning. Preliminary data suggest that fresh tuna is now consumed more than before, compared to lagoon fish. The fear of ciguatera poisoning is probably the most important factor which currently limits lagoon fish consumption in Polynesia. While menhaden oil contains respectively 14% and 8% of EPA and DHA, tuna oil contains very little EPA (6%) and much more DHA (26.5%). Tuna oil differs from other fish oils in the ratio of EPA to DHA, with a ratio of approximately 1:4 in tuna oil and 1:0.6 in menhaden oil.[53] This could possibly explain the surprising PUFA profile observed in Polynesians. Work is ongoing to characterize PUFA profiles of various lagoon fish species.

Finally, we also found that young Polynesians (18-34) had lower EPA+DHA (4.18%) compared to the 35-49 (6.11%) age group, reflecting a lower fish consumption among the youth.

CONCLUSION

French Polynesians consume plenty of fish, a staple of their diet and of their culture. As a result, they are exposed to risks and benefits associated with the presence of a mixture of harmful contaminants and healthy nutrients in the species consumed. These preliminary data suggest that Polynesians are exposed to high doses of mercury just as fish eating populations of the Seychelles Islands, Faroe Islands and the Arctic are. Considering the pattern of exposure (stable exposure to mercury over the year with no seasonal patterns, high selenium intake, low PCBs), it is possible that the results from the Seychelles Islands that report no health effects on the exposed child, could also apply to this South Pacific community. A cord blood surveillance program for mercury exposure is ongoing in French Polynesia. It will allow us to 1) establish prenatal exposure to mercury; 2) describe exposure among women delivering babies from all archipelagos, as it is possible that mercury exposure varies considerably according to archipelagos with varying dietary habits and geography. For example, lagoon fish is consumed in higher quantities in islands having a coral atoll. Furthermore, taboos against pelagic fish consumption by pregnant women (personal communication Ms Hinano

Murphy of the *Te pū átitiá* association. Moorea) may also have an impact on mercury exposure.

In Polynesia, pelagic fish consumption is probably the major source of Hg exposure but a project is on going to assess mercury content in lagoon fish. Tropical lagoon fish species are a major source of important nutrients such as fatty acids and selenium. In a previous paper,[54] we reported that a normal consumption of tropical reef species contains sufficient quantities of omega-3 fatty acids to meet adequate intakes. However, sources of fat were quite different compared to cold water fish as fat is located in the gut. These fat deposits make up for the relatively low levels of fat in the flesh of tropical fish. A second phenomenon is that the fat content varies considerably according to seasons.

Polynesians are already in a transition phase characterised by a shift from a traditional diet towards a more western diet, mainly among young people. This dietary transition might be followed by an emergence of chronic diseases. These data will help to promote sound nutritional advice on the importance of fish consumption for chronic disease prevention.

For pregnant women, considering the specific susceptibility of the foetus to mercury toxicity, it will be necessary to promote less contaminated fish species. These preliminary results need to be complemented by other ongoing studies in order to 1) promote fish consumption particularly among youth; 2) inform and help pregnant women select less contaminated fish for their daily consumption (mercury and ciguatera) while maintaining the nutritional benefits of eating fish.

ACKNOWLEDGEMENTS

We would like to thank all the participants for their interest and participation in the study. We also would like to thank G. Vauterin, N.L Nguyen. M Chinain. A.-M. Legrand and R. Chansin from the Institut Louis Malardé (Papeete, French Polynesia) for their help with the recruitment of participants. This study was partially funded by the USA Center for Diseases Control and Prevention as well as the Pan American Health Organization.

AUTHOR DISCLOSURES

Eric Dewailly, Ludivine Château-Degat and Edouard Suhas, no conflicts of interest.

REFERENCES

1. FAO. Fishery Information, Data and Statistics Unit (FIDI). Fishery Statistical Collections. FIGIS Data Collection. FAO - FIGIS. Updated Mon Sep 05 16:37:59 CEST 2005.
2. Bates CJ, Prentice A, Birch MC, Delves HT, Sinclair KA. Blood indices of selenium and mercury, and their correlations with fish intake, in young people living in Britain. Br J Nutr. 2006; 96: 523-531.
3. Arctic Monitoring Assessment Programme (AMAP). AMAP assessment 2002: Human health in the Arctic. Oslo, Norway, 2003.
4. Chapman L,,Chan HM. The influence of nutrition on methyl mercury intoxication. Environ Health Perspect. 2000;108: 29-56.
5. Svensson BG, Schutz A, Nilsson A, Akesson I, Akesson B, Skerfving S. Fish as a source of exposure to mercury and selenium. Sci Total Environ. 1992;126:61-74.
6. Salonen JT, Seppänen K, Lakka TA, Salonen R, Kaplan GA. Mercury accumulation and accelerated progression of carotid atherosclerosis: a population-based prospective 4-year follow-up study in men in eastern Finland. Atherosclerosis. 2000;148(2):265-73
7. WHO. (World Health Organization), Environmental Health Criteria 101 - Methyl Mercury: World Health Organization; 1990. Geneva.
8. Dewailly E, Mulvad G, Sloth PH, Hansen JC, Behrendt N, Hansen JPH. Inuit are protected against prostate cancer. Cancer Epidemiol.Biomarkers Prev. 2003;12: 926-7.
9. Harris WS. Are omega-3 fatty acids the most important nutritional modulators of coronary heart disease risk? Curr Atheroscler Rep. 2004;6:447-52.
10. Kris-Etherton PM, Harris WS, Appel LJ. AHA Scientific Statement. Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. Circulation. 2002;106: 2747-57.
11. Cunnane SC, Francescutti V, Brenna JT, Crawford MA. Breast-fed infants achieve a higher rate of brain and whole body docosahexaenoate accumulation than formula-fed infants not consuming dietary docosa-hexaenoate. Lipids. 2000;35:105-11.
12. Uauy R, Hoffman DR, Peirano P, Birch DG, Birch EE. Essential fatty acids in visual and brain development. Lipids. 2001;36:885-95.
13. He K, Song Y, Daviglus ML, Liu K, Van Horn L, Dyer AR, Greenland P. Accumulated evidence on fish consumption and coronary heart disease mortality: a meta-analysis of cohort studies. Circulation. 2004; 109:2705-11.
14. Bucher HC, Hengstler P, Schindler C, Meier G. N-3 polyunsaturated fatty acids in coronary heart disease: a meta-analysis of randomized controlled trials. Am J Med. 2002; 112:298-304.
15. Emsley R, Oosthuizen P, van Rensburg SJ. Clinical potential of omega-3 fatty acids in the treatment of schizophrenia. CNS Drugs. 2003;17:1081-91.
16. Wang C, Harris WS, Chung M, Lichtenstein AH, Balk EM, Kupelnick B, Jordan HS, Lau J. n–3 Fatty acids from fish or fish-oil supplements, but not α-linolenic acid, benefit cardiovascular disease outcomes in primary- and secondary-prevention studies: a systematic review. Am J Clin Nutr. 2006;84:5-17.
17. Logan AC. Neurobehavioral aspects of omega-3 fatty acids: possible mechanisms and therapeutic value in major depression. Altern Med Rev. 2003;8:410-25.
18. Sinclair AJ, Begg D, Mathai M, Weisinger RS. Omega 3 fatty acids and the brain: review of studies in depression. Asia Pac J Clin Nutr. 2007;16 Suppl 1:391-7.
19. Ruxton CH, Reed SC, Simpson MJ, Millington KJ. The health benefits of omega-3 polyunsaturated fatty acids: a review of the evidence. J Hum Nutr Diet. 2004;17:449-59.
20. Hites RA, Foran JA, Carpenter DO, Hamilton MC, Knuth BA, Schwager SJ. Global assessment of organic contaminants in farmed salmon. Science. 2004;9:303 (5655):226-9.
21. Mozaffarian D, Rimm E.B. Fish Intake, contaminants, and human health: evaluating the risks and the benefits. JAMA. 2006;296:1885-1899.
22. Sinclair AJ, O'Dea K, Naughton JM. Elevated levels of arachidonic acid in fish from Northern Australian coastal waters. Lipids. 1983;18(12):877-8.
23. Direction de la Santé Ministère de la Santé et de la Recherche. Enquête sur les maladies non transmissibles et Polynésie Française. Papeete, 1998; 88 p (Change to English)
24. Chateau-Degat ML, Dewailly E, Cerf N, Nguyen NL, Huin-Blondey MO, Hubert B, Laudon F, Chansin R. Temporal trends and epidemiological aspects of ciguatera in French Polynesia: a 10-year analysis. Trop Med Int Health. 2007;12 (4):485-92.

25. Lepage G, Roy CC. Direct transesterification of all classes of lipids in a one-step reaction. J Lipid Res. 1986;27:114-20.
26. Dewailly E, Ayotte P, Bruneau S, Lebel G, Levallois P, Weber JP. Exposure of the Inuit population of Nunavik (Arctic Quebec) to lead and mercury. Arch Environ Health. 2001; 56(4): 350-7.
27. Dewailly E, Blanchet C, Lemieux S, Sauvé L, Gingras S, Ayotte P, Holub BJ. n-3 Fatty acids and cardiovascular disease risk factors among the Inuit of Nunavik. Am J Clin Nutr. 2001; 74(4): 464-73.
28. Boudou A, Ribeyre F. Mercury in the food web: accumulation and transfer mechanisms. Met Ions Biol Syst. 1997;34: 289-319.
29. Bricas N, Etienne J, Mou Y. Étude sur la commercialisation et la consommation des produits vivriers, horticoles et fruitiers en Polynésie française. Résultats de l'enquête de consommation alimentaire réalisée en Polynésie française en 1995. Centre de coopération internationale en recherche agronomique pour le développement (CIRAD), Ministère de l'Agriculture - Service du Développement Rural. 2001 (Translate to English)
30. Ministère de l'agriculture de l'élevage et des forets. Service du développement rural. Note AV 7688QAAV/ SDR/MAE. Pirae, le 13 juillet 2005.(Change to English)
31. Arnaud J, Bertrais S, Roussel AM, Arnault N, Ruffieux D, Favier A, Berthelin S, Estaquio C, Galan P, Czernichow S, Hercberg S. Serum selenium determinants in French adults: the SU.VI.M.AX study. Br J Nutr. 2006;95(2):313-20.
32. van der Torre HW, Van Dokkum W, Schaafsma G, Wedel M, Ockhuizen T. Effect of various levels of selenium in wheat and meat on blood Se status indices and on Se balance in Dutch men.Br J Nutr. 1991;65(1):69-80.
33. Lloyd B, Lloyd RS, Clayton BE. Effect of smoking, alcohol and other factors on the selenium status of a healthy population. J Epidemiol Community Health. 1983;37(3):213-7
34. Rayman MP. The use of high-selenium yeast to raise selenium status: how does it measure up? Br J Nutr. 2004;92: 557-73.
35. Davidson PW, Myers G J, Cox C, Shamlaye CF, Marsh DO, Tanner MA, Berlin M, Sloane-Reeves J, Cernichiari E, Choisy O. Longitudinal neurodevelopmental study of Seychellois children following in utero exposure to methylmercury from maternal fish ingestion: outcomes at 19 and 29 months. Neurotoxicology. 1995;16:677-88.
36. Davidson PW, Myers GJ, Cox C, Axtell C, Shamlaye C, Sloane-Reeves J, Cernichiari E, Needham L, Choi A, Wang Y, Berlin M, Clarkson TW. Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: outcomes at 66 months of age in the Seychelles child development study. JAMA. 1998;280:701-7.
37. Davidson PW, Palumbo D, Myers GJ, Cox C, Shamlaye CF, Sloane-Reeves J, Cernichiari E, Wilding GE, Clarkson TW. Neurodevelopmental outcomes of Seychellois children from the pilot cohort at 108 months following prenatal exposure to methylmercury from a maternal fish diet. Environ Res. 2000;84:1-11.
38. Myers GJ, Davidson PW, Cox C, Shamlaye CF, Tanner MA, Marsh DO, Cernichiari E, Lapham LW, Berlin M, Clarkson TW. Summary of the Seychelles child development study on the relationship of fetal methylmercury exposure to neurodevelopment. Neurotoxicology. 1995;16:711-6.
39. Myers GJ, Marsh DO, Davidson PW, Cox C, Shamlaye CF, Tanner M, Choi A, Cernichiari E, Choisy O, Clarkson TW. Main neurodevelopmental study of Seychellois children following in utero exposure to methylmercury from a maternal fish diet: outcome at six months. Neurotoxicology. 1995;16: 653-64.
40. Myers GJ, Davidson PW, Palumbo D, Shamlaye C, Cox C, Cernichiari E, Clarkson TW. Secondary analysis from the Seychelles child development study: the child behavior checklist. Environ Res. 2000;84:12-19.
41. Steuerwald U, Weihe P, Jorgensen PJ, Bjerve K, Brock J, Heinzow B, Budtz-Jorgensen E, Grandjean P. Maternal seafood diet, methylmercury exposure, and neonatal neurologic function. J Pediatr. 2000;136:599-605.
42. Grandjean P, Weihe P, White RF, Debes F, Araki S, Yokoyama K, Murata K, Sorensen, N, Dahl R, Jorgensen PJ. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicol Teratol. 1997;19:417-28.
43. Debes F, Budtz-Jorgensen E, Weihe P, White RF, Grandjean P. Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years. Neurotoxicol Teratol. 2006;28:363-75.
44. Grandjean P, Weihe P, Burse VW, Needham LL, Storr-Hansen E, Heinzow B, Debes F, Murata K, Simonsen H, Ellefsen P, Budtz-Jørgensen E, Keiding N, White RF. Neurobehavioral deficits associated with PCB in 7-year-old children prenatally exposed to seafood neurotoxicants. Neurotoxicol Teratol. 2001;23(4):305-17.
45. FAO/WHO Summary and conclusions of the sixty-seventh meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), 20-29 June 2006; JECFA 67/SC
46. Hightower JM, O'Hare A, Hernandez GT. Blood mercury reporting in NHANES: identifying Asian, Pacific Islander, Native American, and multiracial groups. Environ Health Perspect. 2006;114(2):173-5.
47. Grandjean P, Weihe P, Jorgensen PJ, Clarkson T, Cernichiari E, Videro T. Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. Arch Environ Health. 1992;47:185-95.
48. Muckle G, Ayotte P, Dewailly E, Jacobson SW, Jacobson J L. Prenatal exposure of the northern Quebec Inuit infants to environmental contaminants. Environ Health Perspect. 2001b;109:1291-9.
49. Bjerregaard P, Hansen JC. Organochlorines and heavy metals in pregnant women from the Disko Bay area in Greenland. Sci Total Environ. 2000;245:195-202.
50. Rayman, MP. The importance of selenium to human health. Lancet. 2000;356:233-41.
51. Rayman MP. Selenium in cancer prevention: a review of the evidence and mechanism of action. Proc Nutr Soc. 2005;64: 527-42.
52. Yang G, Yin S, Zhou R, Gu L, Yan B, Liu Y. Studies of safe maximal daily dietary Se-intake in a seliniferous area of China. Part 2. Relation between Se-intake and the manifestations of clinical signs and certain biochemical alterations in blood and urine. J Trace Elem Electrolytes Health Dis. 1989;3:123–30.
53. FDA (Food and Drug Administration), Center for Food Safety and Applied Nutrition, Office of Food Additive Safety, December 4, 2002. Agency Response Letter. GRAS Notice No. GRN 000109
54. Rouja PM, Dewailly E, Blanchet C, Bardi Community. Fat, fishing patterns, and health among the Bardi people of north Western Australia.Lipids. 2003;38(4):399-405.

Original Article

Fish consumption and health in French Polynesia

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法屬玻里尼西亞的魚類攝取與健康

法屬玻里尼西亞人像其他偏遠的濱海族群一樣，與養育他們的日常生活和文化的海洋有密切關係。他們依賴的魚類，在提供營養素的同時，也引發了潛在暴露於有害的自然和人造污染物的問題。本研究的目的是要評估在法屬玻里尼西亞攝取魚類的風險及益處。這個橫斷性研究包括了 195 位來自大溪地及茉莉亞島 18 歲以上的成人。參與者所測出血液中脂肪酸、硒及汞的濃度皆非常高。血液濃度指出硒、汞及ω–3 脂肪酸有一個共同的來源，那就是攝取魚類。比較玻里尼西亞人和北方人，我們發現玻里尼西亞人血中汞的濃度和因紐特人相似(幾何平均值：90.3 (15-420) nmol/L vs. 紐特人：79.6 (4-560) nmol/L)。血液中硒的濃度和脂肪酸濃度也有相似結果，儘管其脂肪酸的組成有所不同。我們第一次報告南太平洋捕魚的種族跟北半球捕魚種族血液一樣，含有非常高濃度的汞、硒和ω–3 脂肪酸。進一步的研究持續中，以能更具體化公共衛生營養政策。

關鍵字：海鮮、汞、硒、ω–3 脂肪酸、玻里尼西亞。