APPENDIX D

Methyl Mercury: Critical Groups and Sources of Intake

(Report from an Expert Group, 1971)

When 'mercury' is released into the environment it may appear as, or be converted into, any of the following chemical forms:

metallic mercury (Hg)
inorganic mercury (Hg^{+} and Hg^{++})
monomethyl mercury ($CH_3 - Hg^{+}$)
dimethyl mercury ($CH_3 - Hg - CH_3$)

The 'methyl mercury' discussed in this appendix is monomethyl mercury ($CH_3 - Hg^{+}$)

1. Man

(a) Adult

(i) Occupational exposure
– lab personnel
– industrial workers and farmers handling seed dressings
– workers in pulpmills and sawmills

(ii) Non-occupational exposure
– persons consuming foodstuffs with high methyl mercury levels: dressed seed, game birds, seabird eggs, fish and shellfish, meat

(b) Foetus
Placental transfer and foetal concentration (Kojima and Fufita 1973)

2. Domestic Animals

(a) Cats (Takizawa et al. 1971), from eating contaminated fish and wild birds

(b) Pigs, from eating contaminated grain (Pierce et al. 1972)

3. Wildlife

(a) Fish, living in waters above sediments contaminated with inorganic mercury

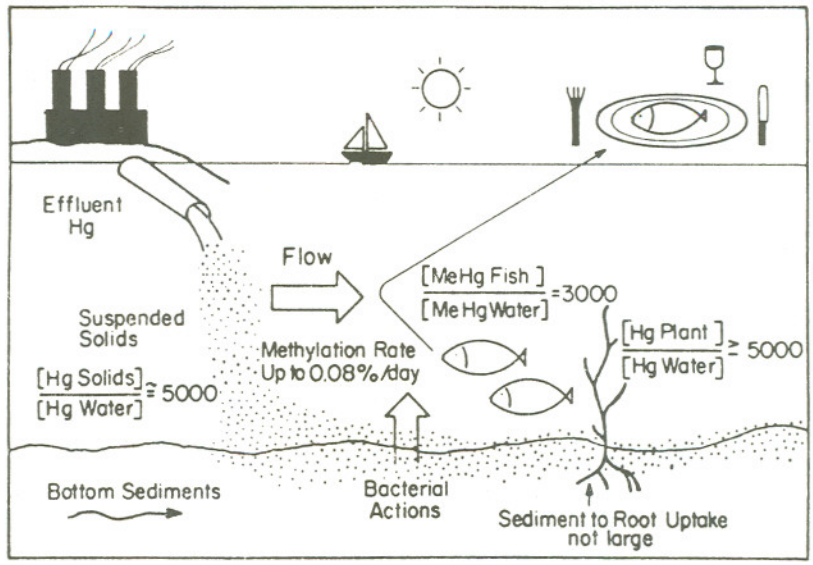

Figure 17 Aquatic food chain for mercury and methyl mercury

(b) Birds (Report from an Expert Group 1971, Borg et al. 1969)

(i) Seed-eating birds (dressed grain)

(ii) Fish-eating birds (contaminated fish)

(iii) Predatory birds (secondary intoxication from contaminated fish or birds)

(c) Mammals, both terrestrial and aquatic, with a fish diet

4. Plants

(a) Phytoplankton (Peakall and Lovett 1972)

(b) Higher plants (Mortimer and Kudo 1975)

ENVIRONMENTAL TRANSPORT

This summary on methyl mercury will not deal with the biogeochemical transport of all forms of mercury in the environment but will concentrate on the critical food chains to man and wild birds.

For seed-eating birds no model is needed since, if one knows the concentration of the organic mercury in the seeds, one can calculate the uptake [f1 = 0.9 (approx.)] and the resulting body and tissue contents (see METABOLISM section and the sub-sections General and Wild birds). This route can be monitored by analyses of feathers.

For man and fish-eating birds the most important environmental route is through contaminated fish and other edible aquatic organisms. In this route it is necessary to consider the occurrence and transport of mercury in inorganic form as well as methyl mercury since methylation of mercury is known to occur in aquatic ecosystems (Wood 1974).

In an aquatic system almost all the mercury is present in the sediments, in inorganic form. The partition coefficient for inorganic mercury between sediment and water (w/w) ranges from 1,000 for coarse sand to 5,000–50,000 for organic sediments (Ottawa River Project 1976). From this inorganic mercury in the sediments, which is retained with a half-time of 1–5 years depending on hydrological conditions, methyl mercury is continuously produced, probably by bacterial action (Wood 1974), at a rate which has been estimated (Langley 1973) at 15°C in the laboratory as 1–5 [mean = 2 (approx.)] micrograms per square meter of sediment per day. It is difficult to correlate this rate precisely with the mercury content of the sediment since the rate of net production of methyl mercury results from two or three reaction steps, some of which may be reversible (Wood 1974). For example, high concentrations of methyl mercury in the sediment may lead to some conversion back to inorganic mercury or to further methylation to the less toxic dimethyl mercury.

After release into the body of water above the sediment the methyl mercury is taken up by fish, both directly through the gills and in methyl mercury-containing food. If one knows the concentration of methyl mercury in the water one can calculate the rates of uptake by fish and the body and muscle contents at any time after the beginning of uptake (see METABOLISM section, Fish sub-section).

The proportion of total mercury in the form of methyl mercury will be magnified at each trophic level due to the more efficient uptake of methyl mercury to the blood (f1 for methyl mercury = 0.95 and f1 for inorganic mercury = 0.15) and due to the longer biological half-life of methyl mercury. For example, for the uptake from food, if F = the fraction of total mercury in the form of methyl mercury in the food, the proportion in the eater of that food

$$F_a = \frac{0.95}{0.80 + \frac{0.15}{F}} \quad \text{(see p. 212 for derivation)} \qquad \text{(E1)}$$

Thus, if invertebrates have $F = 0.3$ (found in field observations), the fish that eat them will have

$$F_a = \frac{0.95}{0.80 + \frac{0.15}{0.3}} = 0.73$$

and the fish b that eat fish a will have

$$F_b = \frac{0.95}{0.80 + \frac{0.15}{0.73}} = 0.95$$

and so on. Similar magnification factors of the proportion of mercury in the form of methyl mercury can be demonstrated for uptake by gills. These values are consistent with those found in the field and no further methylation during passage up the food chain is required to explain the increasing fractions of mercury as methyl mercury. In fact, there was an early demonstration that the analysis of fish muscle for methyl mercury could be used to estimate the level of mercury present in the water environment (Johnels et al. 1967).

Westermark and Johnels (1975) have formulated the magnification of methyl mercury concentrations in birds as a function of the biological half-lives in predator and prey. Our derivation of a comparable formula shows the magnification factor for a single stage to be

$$\frac{a_n}{x_n} \cdot F_n$$

where

a_n = g of prey $n-1$ eaten per day per g of predator n
x_n = fractional rate of loss (d^{-1}) of methyl mercury from n
F_n = fraction of the methyl mercury eaten by n absorbed to blood

Thus for a food chain of n levels the total magnification

$$= \frac{a_1}{x_1} \cdot F_1 x \frac{a_2}{x_2} \cdot F_2 x \ldots \frac{a_n}{x_n} \cdot F_n x b \qquad \text{(E2)}$$

where b = the concentration of methyl mercury in the first level (g/g).

METABOLISM

1. General

Methyl mercury is taken up through the intestines and the lungs of terrestrial animals and the intestines and gills of fish. It is also absorbed through the skin but quantitative data are lacking.

For a constant rate of uptake, I, the equation describing body content as a function of time is (Butler 1972, Nordberg et al. 1973)

$$q(t) = I \int_0^t R_s(u)\, du \qquad \text{(see p. 213 for explanation)} \qquad \text{(E3)}$$

where

$q(t)$ = the body content at time t, μg
I = the rate of uptake to blood, μg/day
$R_s(u)$ = the retention equation
= the fraction retained on day u following a single uptake

At times soon after uptake, the blood and kidneys show concentrations 10–20 times higher than the body average but at later times these decline relatively. In fish

the concentration in muscle is not greatly different from that in whole body but since skeletal muscle constitutes about half the mass of the total body, muscle is the principal storage tissue.

Since brain and nervous tissue seem to be the critical organ in mammals the deposition in these tissues is of special interest. In mouse, cat, dog, pig, and monkey the concentration of methyl mercury in the brain varies from 0.5–5 times that in blood. The rat appears exceptional in that the corresponding ratio is about 0.1.

Methyl mercury is taken up to the blood to a greater extent than inorganic mercury and methyl mercury has a much longer biological half-time in both terrestrial animals and fish than has inorganic mercury. When mercury is released to the environment a small fraction is converted to methyl mercury; so animals exposed to mercury in the environment assimilate both inorganic and methyl mercury (the latter to a greater extent). The result of the metabolic behaviour described above is that there is a preferential accumulation of methyl mercury to relatively high levels. Calculations on this topic were presented in preceding paragraphs.

Most of the methyl mercury taken up by man is excreted in the faeces. The minor fraction (as low as one-tenth) excreted in the urine also varies with time after uptake. For these reasons urinary analyses are of doubtful value for calculating body content and this has been confirmed by surveys on industrial workers contaminated internally with mercury compounds (Ladd et al. 1963) and on the farmers of Iraq who ate wheat contaminated with methyl mercury (Bakir et al. 1973).

2. Terrestrial Animals

a. Ingestion

For all animals including man, f1, the fraction taken up from the gastrointestinal tract, is 0.9–0.95 for both aqueous solutions and food.

b. Inhalation

In the Lung Model of ICRP (Bates et al. 1966) methyl mercury would be a class D compound and therefore 60% of the amount inhaled would be rapidly taken up to blood.

c. Tissue Distribution and Retention

(i) Man

The following metabolic information is given to relate diagnostic measurements on humans to the methyl mercury levels in the food they eat.

In man eating methyl mercury-contaminated fish the equilibrium levels in blood were described by the relation (Miettinen et al. 1971):

$$Y = 0.8X$$

where

Y = concentration in blood (ng/g)

X = amount ingested in fish (μg/day)

Studies of the epidemic of methyl mercury poisoning in Iraq as a result of eating contaminated wheat (Bakir et al. 1973) yielded the following results:

The blood concentration could be described by the relation

$Y = 16X$ for patients $>$ 10–15 years old

$Y = 10X$ for patients $>$ 18 years old

where

Y = blood concentration (ng/ml)

X = total intake (mg) during 40–60 days

$Cib = Cmb$ for infants nursed by methyl mercury-contaminated mothers

where

Cib = concentration in infant blood

Cmb = concentration in mother's blood

$Cmm = 0.03\ Cmb$

where

Cmm = concentration in mother's milk

Since the concentration of mercury in hair is related to the concentration of methyl mercury in the blood at the time of hair synthesis, the analysis of hair can be used to monitor the past history of blood concentrations (Bakir et al. 1973, Al-Shahristani et al. 1974). The relations between observed hair and blood concentrations have been reported (Report from an Expert Group, 1971); the hair concentration was 140–400 times (mean approx. 300) that in blood.

External counting of men after ingestion of radioactive methyl mercury indicated the following tissue distribution (Falk et al. 1970):

Cerebellum	10%
Liver	54%
Remainder of body	36%

This distribution did not change greatly during two months after uptake but the proportion in brain rose slightly due to more rapid excretion from other organs. A similar distribution was found by Aberg et al. (1969). Experiments by Miettinen et al. (1971) showed that at 100 days after a single ingestion blood contained 5–10% of the methyl mercury in the whole body. In the blood about 5–20% (mean 10%) was found to be in plasma and the remaining 80–95% in the red cells (Bakir et al. 1973, Birke et al. 1972).

Measurements of the half-time of retention in the body and tissues of man

yielded the following results:

Tissue	$T_{1/2}$ (days)	Reference
Body	71	Miettinen et al. (1971)
	72	Al-Shahristani and Shihab (1974)
	76	Falk et al. (1970)
Cerebellum	85	Falk et al. (1970)
Hair	100	Kojima and Fujita (1973)
		Miettinen et al. (1971)
Red blood cells, plasma	50	Miettinen et al. (1971)
Blood	50	Miettinen et al. (1971)
Liver	80	Kojima and Fujita (1973)
Kidney	56	Kojima and Fujita (1973)
Brain	41	Kojima and Fujita (1973)

Of the total methyl mercury excreted, the fraction in faeces is high and variable declining from about 0.95 in the first day or two after a single uptake to about 0.6 at 60 days (Aberg et al. 1969, Miettinen et al. 1971). In industrial workers chronically exposed to vapours of methyl mercury the measured concentrations in blood were from 6–50 times the concentration in urine (Lundgren et al. 1967).

(ii) Mammals and chickens

(a) Tissue distribution

	Relative concentrations						
Tissue	Mouse[a]	Rat[a,b,c]	Cat[b,d]	Dog[b]	Pig[b]	Monkey[b]	Chicken[e]
Brain	1	1	1	1	1	1	1
Blood	1	7–13	1	2	0.5	0.15	1
Kidney	1–10	5–40	2–4	2	3	1	2
Liver	4	3–4	7–24	4	5	1	2
Muscle			2				1

[a] Report from an Expert Group (1971)
[b] Kojima and Fujita (1973)
[c] Swensson and Ulfvarson (1968b)
[d] Hollins et al. (1975)
[e] Swensson and Ulfvarson (1968a)

(b) Retention equations

Rat

$$R_s(t) = 0.15e^{-(0.693/2)t} + 0.45e^{-(0.693/30)t} + 0.4e^{-(0.693/90)t} \quad \text{(E4)}$$

Cat

$$R_s(t) = 0.1e^{-(0.693/0.8)t} + 0.9e^{-(0.693/T)t} \quad \text{(E5)}$$

where

T = 117 days, including the hair
= 76 days, excluding the hair

Chicken

$$R_s(t) = e^{-(0.693/35)t} \tag{E6}$$

(iii) Wild birds

Hen pheasants with liver levels of 3–13 ppm laid eggs with 0.5–15 ppm and this relating concentrations in food, feathers, blood, brain, and whole body are urgently

Swedish work over the past decade on the methyl mercury contents of wild birds' feathers has been summarized by Westermark et al. (1975). The methyl mercury content of feathers is a function of the concentration of methyl mercury in the blood at the time of feather formation. This is reflected in variation from feather to feather and even from one part of the feather to another. Since feathers have the highest concentrations of any tissue and since these concentrations reflect the food concentration, the analysis of birds' feathers provides good information about environmental contamination. Published data on tissue distributions in wild birds are scanty. Food, carcass, and liver have been analysed for great blue heron (Dustman et al. 1970) and common tern (Fimreite 1970). Brain, pectoral muscle, and feathers have been analysed in white-tailed eagles found dead in the Archipelago of Stockholm from 1965 to 1969 (Jensen et al. 1972). Systemic data relating concentrations in food, feathers, blood, brain, and whole body are urgently required for environmental assessment.

3. Fish

The respiratory uptake of methyl mercury through the gills of freshwater fish has been shown experimentally by de Freitas and Hart (1975) to be dependent on the metabolic rate and has been formulated, for 20°C, as:

$$I(t)_{\text{resp}} = 1000 \times m^{0.8} \times C_{pw} \text{ g methyl mercury/day} \tag{E7}$$

where

$I(t)_{\text{resp}}$ = rate of uptake by gills, g/day
m = body mass, g
C_{pw} = concentration of methyl mercury in water, g/g

The uptake of methyl mercury by the ingestion of contaminated food was shown to depend on the rates of maintenance metabolism and of growth (Norstrom et al. 1975). Thus, at 20°C,

$$I(t)_{\text{ing}} = C_{pf}\left(0.025m^{0.8} + 2\frac{dm}{dt}\right) \text{ g methyl mercury/day} \tag{E8}$$

where

$I(t)_{ing}$ = rate of uptake from the intestinal tract, g/day
C_{pf} = concentration of methyl mercury in food, g/g
m = body mass, g
$\frac{dm}{dt}$ = daily increase in body mass, g/day

By analyses of whole fish, 65–90% of the retained mercury was found to be in the form of methyl mercury (Burrows and Krenkel 1973, Lockhart et al. 1972); in fish muscle the proportion was uniformly >90%. Analysis of northern pike contaminated under natural conditions (Lockhart et al. 1972) and rainbow trout dosed in the laboratory (Giblin and Massaro 1973) showed that muscle concentration was representative of the concentration in the whole body and that no tissue concentrated methyl mercury more than by a factor of 2.

The half-time of retention has been variously estimated for adults of different species as follows:

Species	Body mass(g)	$T_{1/2}$(days)	Reference
Bluegills	2.5–11	640	Burrows and Krenkel (1973)
Pike	300 (mean)	640–780	Järvenpää et al. (1970)
Flounder	180 (mean)	640–780	Järvenpää et al. (1970)
Eel	100 (mean)	1030	Järvenpää et al. (1970)

In Canadian freshwater fish (7 species) ranging in weight from 1–400 g de Freitas et al. (1975) found that the biological half-life of methyl mercury was a function of body mass regardless of species. In their notation

$$R_{pcl} = k_{cl} \cdot P \cdot W^{-0.58} \qquad \text{(E9)}$$

where

R_{pcl} = the rate of elimination from the body (μg/day)
k_{cl} = the elimination constant for a fish weighing 1 g
= 0.029
P = the body content of methyl mercury (μg)
W = the mass of the fish (g)

Thus $0.029 \cdot W^{-0.58}$ is the elimination constant (half-life = $24\ W^{+0.58}$ days) for a fish of body mass W g.

Giblin and Massaro (1973) found that in trout given a single dose of labelled methyl mercury by stomach tube half of the dose was deposited in muscle with a half-life >200 days. At 100 days after dosing 73% of the dose was retained in the whole animal. Their data suggest 80% with a long half-life, calculated to be 700 days from de Freitas' formula for 270 g fish. Their data for early loss, coupled with

the above information suggest

$$R(t) = 0.1e^{-(0.693/6)t} + 0.1e^{-(0.693/200)t} + 0.8e^{-(0.693/700)t}$$

In goldfish studied in the laboratory, de Freitas (Ottawa River Project 1976) found that increasing the uptake rate of methyl mercury from 0.05 to 0.35 micrograms per fish per day lowered the biological half-time by 50% (from 160 to 80 days).

Since water and food concentrations of methyl mercury are magnified in fish muscle by metabolic processes, the analysis of fish muscle for methyl mercury is a useful method of monitoring the hazard to man resulting from mercury contamination of the aquatic environment. If one knew the concentration of methyl mercury in water it would be a straightforward matter to calculate the concentration in fish muscle.

The body content in fish at t days after entry of methyl mercury into a river may be calculated from the equation (Butler 1972)

$$q(t) = \int_0^t I(\zeta) R_s(t - \zeta)\, d\zeta \quad \text{(See p. 213 for explanation)} \qquad \text{(E10)}$$

The rate of uptake of methyl mercury from water by the gills

$= 1000 \times m^{0.8} \times C_{pw}$, from equation (E7)

and since the uptake from food approximately equals the respiratory uptake the total uptake rate

$$I(\zeta) = 2000 \times m^{0.8} \times C_{pw}$$

$R_s(t - \zeta)$ = the fractional retention at $(t - \zeta)$ days after the uptake

$$= e^{-0.029 m^{-0.58}(t - \zeta)}, \text{ from equation (E9)} \qquad \text{(E11)}$$

$$\therefore q(t) = \int_0^t 2000 \times m^{0.8} \times C_{pw} \times e^{-0.029 m^{-0.58}(t - \zeta)} d\zeta \qquad \text{(E12)}$$

Unfortunately analytical techniques for analysing water for the very low levels of methyl mercury in natural waters, are not yet available. Failing this C_{pw} can be calculated from an observed rate of methyl mercury production and the volume of water into which it is released. For example, in the study of the Ottawa River, the velocity of flow was 17 km/d and 17 km had an area of sediment of 17×10^6 m^2. If this were all methyl mercury-producing sediment it would yield $2 \times 17 \times 10^6$ μg/day (Langley 1973). Since the volume of flow was 1.5×10^{11} l/day the resulting concentration would be 2.3×10^{-13} g/g. From equation (E7), for a 100 g fish, $I(t) = 9$ ng/day. If the uptake from ingestion approximately equals the uptake from respiration the total uptake is approximately 18 ng/day. $R_s(t)$ for a 100 g fish $= e^{-0.002t}$ so at equilibrium

$$q(\infty) = \frac{18}{0.002} = 9000 \text{ ng}$$

and for a 100 g fish the concentration = 90 ng/g.

In the Ottawa River study the observed levels were twice as high as this calculated value which could have several explanations:

– the rate of methyl mercury production (2 micrograms/m^2/day) used in the calculation was not appropriate to this section of river;

– there was an appreciable content of methyl mercury in the water entering the study area;

– the fish were feeding on plants or animals that had already concentrated methyl mercury;

– the fish were adults and had in previous years been exposed to higher concentrations of methyl mercury.

For calculations like those above it would be desirable to have more precise knowledge of the rate of methyl mercury production as a function of the mercury content of sediments. Thus monitoring of the sedimental mercury coupled with data for the flow rate of the river would permit calculation of C_{pw} and from that the amount of methyl mercury in fish according to equation (E12).

4. Aquatic Plants (Mortimer and Kudo 1975)

Laboratory experiments showed that *Elodea* has an uptake rate of 7×10^{-9} g per day (of both inorganic and methyl mercury) per g (dry weight) of plant at a water concentration of 5×10^{-12} g/g. The corresponding figure for *Utricularia* was 22 ng/g/day. In a natural river *Elodea* gave an uptake rate of 15 ng/g/day at a concentration of 5×10^{-12} g/g, in good agreement with the laboratory values.

Measurements of the rate of loss from such plants showed that the rate was independent of the state of the plant (living or dead) and that the data were fitted by the retention equation

$$R_s(t) = 0.4e^{-(0.693/140)t} + 0.6e^{-(0.693/700)t} \qquad \text{(E13)}$$

Such a slow rate of loss from the plant tissue means that there will be an almost linear accumulation of methyl mercury during the life of the plant.

CRITICAL ORGANS AND TOXIC LEVELS

Symptoms of methyl mercury poisoning are seen to involve the central nervous system, and at lower levels possibly reproductive failure. As far as it is possible to tell, the symptoms seem to be the same in man, smaller mammals, and birds. One feature of methyl mercury poisoning is the latent period; the symptoms usually appear a few weeks or months after the first exposure to contaminated food.

In man pre-natal poisoning causes mental retardation and motor disturbances resembling cerebral palsy from other causes. Post-natal poisoning is characterized by sensory disturbances, ataxia and distortion of the visual field and hearing (Report from a Expert Group 1971).

In mice the earliest sign of poisoning, i.e., loss of the ability to hold the head in a

horizontal position when the mouse is suspended by the tail, occurred at brain concentrations of 10 micrograms/g.

When rats ingested methyl mercury for 150–210 days and were examined for neurological (N) and morphological (M) lesions the following results were obtained:

Daily ingestion (mg/kg body weight)	Symptoms
1	N, M
0.5	M
0.2	0

In studies of the epidemics of methyl mercury poisoning among the fish-eating populations of Minamata and Niigata, the hair and blood of subjects showing symptoms were analysed at various times up to 1,000 days after the onset of symptoms. By extrapolating these plots back to zero time the concentrations corresponding to the appearance of symptoms could be obtained (Kojima and Fujita 1973, summarized by Skerfving 1972). In 17 cases at Niigata a blood concentration at zero time of 0.10 micrograms/g was the level below which there were no symptoms.

In the farmers of Iraq poisoned by eating bread made from methyl mercury-treated grain, it was reported (Dhahir and Clarkson 1974) that the toxic effects of methyl mercury became detectable when individuals had accumulated 0.5–0.8 mg mercury/kg body weight. This would correspond to a blood concentration in Standard Man of about 1 microgram/g. On the other hand, a plot of total neurological symptoms against blood concentration (Table 4 in Bakir et al. 1973) gave a linear relation passing through the origin. Reading off from this plot a blood level of 100 ng/ml corresponded to an incidence of 2% of minor neuromuscular disorders.

In 1969 an American family in the state of New Mexico was poisoned by eating pork from a pig fed on methyl mercury-contaminated grain (Pierce et al. 1972). In this episode it was estimated that each member of the family consumed 390 mg of methyl mercury during 100 days which, for the adults weighing 70 kg would be a daily intake of 55 micrograms/kg. Both parents remained unaffected but three children aged 8, 13, and 20 had severe neurological disorders; because of their smaller body weight their intakes would have been relatively higher. In the three children with neurological disorders the serum concentration of methyl mercury was 2–3 ppm and the hair concentration was 800 times higher.

OBJECTIVES AND STANDARDS

The following considerations and calculations show how standards for the protection of man might be derived. A blood concentration of 0.1 micrograms/g whole blood might be considered acceptable; this would correspond to 30 micrograms/g in hair (Skerfving 1972). If 10% of the methyl mercury is in the

blood (Miettinen et al. 1971) this would correspond to a whole body content of

$$0.1 \times 5500 \times 10 \simeq 5 \text{ mg}$$

Assuming $T_{½} = 72$ days this equilibrium level would be attained by the daily uptake of 50 micrograms. For a 70 kg man this is 0.75 microgram/kg/day. By applying any safety factor deemed necessary to this number an acceptable daily intake (uptake) can be calculated.

1. Food

Since the main source of intake of methyl mercury for man is dietary fish, objectives or secondary standards are usually given in terms of the concentration of methyl mercury in fish (Skerfving 1972).

Japan	'Banning' of fish is considered when it contains more than 1 ppm (micrograms/g) or when the level in the hair of fish-eaters is >50 micrograms/g
Canada and USA	0.5 ppm in fish
Italy and Germany	0.5 ppm in fish
France	0.7 ppm in fish
Sweden	1.0 ppm in fish for consumption of fish once per week

In light of the above calculations these standards appear adequate to protect average populations, but they may need modification for groups with excessive consumption of contaminated fish.

2. Air

0.01 mg/m^3 in air (Report from an Expert Group 1971)

3. Drinking Water

0.0005 mg/kg (Report from an Expert Group 1971)

ENVIRONMENTAL SIGNS

a. Of Pollution

Mercury content of bottom sediments.
Methyl mercury content of fish muscle.
Methyl mercury content of birds' feathers.
Methyl mercury content of the hair of man and animals.
Methyl mercury content of underwater aquatic plants.

b. Of Effects

Rates of death and reproduction in seed- and fish-eating birds.
Health of domestic cats.

METHODS OF ANALYSIS AND LIMITS OF DETECTION
(total mercury analysis)
(Report from an Expert Group 1971)

Method	Limit(s) of detection
(a) Dithizone methods	0.5 micrograms/10 g dry sample
(b) Atomic absorption analysis	10 ng/g
	tenths of ng/0.2 g sample
	2 ng/1 ml urine
(c) Neutron activation analysis	100–500 ng/g
	0.1–0.2 ng/g in 1 g sample
	0.02–0.03 ng/g in 10 ml sample
(d) Isotope dilution	0.04 ng/g
(e) Micrometric method	

(For methyl mercury analytical procedures, see Report from an Expert Group 1971.)

EQUATIONS

In this section are explained three of the mathematical formulations used; its purpose is to make the non-mathematical reader feel confident in the use of the formulae.

Equation (E1)

Assume that the concentration of total mercury in the food is C of which the fraction F is in the form of methyl mercury and $(1 - F)$ is in the form of inorganic mercury. If a fraction 0.95 of methyl mercury, and 0.15 of inorganic mercury, is absorbed from the intestinal tract to blood the total mass of mercury absorbed from 1 g of food is

$$0.95\,FC + 0.15(1 - F)\,C \text{ g}$$

Thus, of the total mercury absorbed into the body, the fraction in the form of methyl mercury

$$F_a = \frac{0.95FC}{0.95FC + 0.15(1 - F)C}$$

$$= \frac{0.95F}{0.95F + 0.15(1 - F)}$$

$$= \frac{0.95F}{0.95F + 0.15 - 0.15F}$$

$$\frac{0.95F}{0.8F + 0.15}$$

Dividing the numerator and denominator by F

$$F_a = \frac{0.95}{0.8 + \frac{0.15}{F}}$$

Equation (E3)

The retention function $R(t)$ is a function of time representing the fraction remaining in the body t days after uptake of a unit dose. If a continuous uniform dose rate is represented by I units per day that has continued for n days the amount remaining after n days will be

$$IR(n) + IR(n-1) + \ldots IR(1)$$

The first of these terms represents the amount remaining after n days from the uptake on the first day and so on, the last term representing the amount remaining from yesterday's uptake.

In continuous terms this sum is represented by

$$q(n) = \int_0^n IR(t)\,dt = I \int_0^n R(t)\,dt$$

where $q(n)$ = the total amount retained after n days of continuous uptake.

If the dose varies with time we can represent it as a function of time $I(t)$ and the fraction remaining after n days is $R(n-t)$ where $(n-t)$ is the number of days from the time of uptake t to the time of evaluation n.

Thus

$$q(n) = \int_0^n I(t)\,R(n-t)\,dt \quad \text{or} \quad \int_0^n I(n-t)\,R(t)\,dt$$

The two expressions are equivalent for this kind of integral which is called a convolution of I and R.

Equation (E2)

If we assume the $R(t)$ used above is described by a simple exponential decay, as is frequently the case.

$$R(t) = e^{-xt}$$

where

x is the fraction lost per day $= \dfrac{0.693}{T}$

T is the half-time of loss from the body, in days

For a constant intake as explained above,

$$q(n) = I \int_0^n R(t)\,dt$$

If n days is a much larger time than the half-time T, the amount lost from the body each day becomes equal to the amount taken up and the body content becomes constant at

$$q(\infty) = I \int_0^{\infty} e^{-xt}\, dt$$

$$= \frac{I}{x}$$

The grams of pollutant taken up by the predator each day,
where

$I = amfb$
a = g of prey eaten per g of predator
m = body mass of predator (g)
f = fraction of the pollutant in the prey absorbed from the intestines into the blood of the predator
b = concentration of pollutant in the prey (g/g)

$$\therefore a(\infty) = \frac{amf}{x} b$$

and the concentration of pollutant in the predator

$$\frac{q(\infty)}{m} = \frac{af}{x} b$$

Similarly, the predator of the first predator will have a body concentration of

$$\left[\frac{a_1 f_1}{x_1} \times \frac{a_2 f_2}{x_2} \right] b$$

and so on with one term for each step in the food chain.

If the equation describing retention in any level of the food chain is more complicated than a single exponential the algebra is slightly more complicated, e.g.,

If

$$R(t) = \alpha e^{-xt} + \beta e^{-yt} \qquad (\alpha + \beta = 1)$$

the magnification factor becomes

$$af \left[\frac{\alpha}{x} + \frac{\beta}{y} \right]$$

instead of

$$\frac{af}{x}.$$