

# Mercury Science Fact Sheet

(Center for Science and Public Policy)

## A. What is Mercury?

Mercury (Hg) is an element that has existed (and will continue to exist) **naturally** since the earth was formed 4.5 billion years ago. The oceans alone contain millions of tons of mercury.<sup>1</sup>

There are two major forms of mercury emitted during fossil fuel combustion:

- Oxidized, which is water soluble and can be washed out of the air into rivers, lakes, and streams.
- Elemental, which is not water soluble and moves around in a global mercury cycle.

A small fraction (about one one-thousandth) of the oxidized mercury that ends up in waterways may be changed into an organic form called methylmercury (**MeHg**) which is the kind of mercury with which EPA is concerned. This type of mercury can be eaten by tiny organisms that are then eaten by small fish, resulting in possible bioaccumulation in larger fish eaten by humans.

Methylmercury is not emitted directly from fossil-fuel-fired power plants. It is produced and accumulated within the biosphere by a myriad of mercury transformation processes that **do not** depend upon the amount of inorganic mercury emitted from man-made sources.<sup>2</sup> The natural cyclical *production and destruction* is controlled by environmental factors and ecosystem processes that are largely beyond human control or intervention.

## B. Where does Hg come from? – A Mostly *Natural*, World-wide Phenomena

The largest source of annual **air-borne** Hg is from natural sources such as volcanoes, forest fires, and oceans.<sup>3</sup> Emissions from Yellowstone National Park, for example, likely exceed that of all Wyoming coal-fired power plants combined.<sup>4</sup> Under current estimates of total annual **air-borne** sources of Hg into the world cycle, US power plant emissions account for as little as 0.5%.<sup>5</sup>

The natural mercury emission from Earth's crust is an important factor controlling mercury distribution in marine waters and the atmosphere, along with anthropogenic emissions. Main sources of mercury to seas are submarine volcanoes, mud volcanoes and cold gas vents. All current global estimates refer to atmospheric emissions *only*. **Geological processes** supplying Hg to soil, oceans, and inland water bodies also need to be quantified and considered in the global mercury budget and its natural cycling. When estimates of **all natural sources** are considered, including geothermal events under oceans and lakes, **US power plants may account for as little as 0.02% of the entire annual world mercury emissions budget.**<sup>6</sup>

## C. Are Mercury levels changing?

From 1990 – 2000, total US anthropogenic Hg emissions **decreased** by 69 tons (to current level of about 107 tons), while Asia increased by over 500 tons (to current level of about 1204 tons) and Africa increased by about 230 tons (to about 407 tons).

In the *Atlantic Ocean*, comparisons of deep-sea fish (i.e., blue hake at depths of 1,000-3,000 m) found **no change** in tissue mercury concentration from 1880s to 1970s. The authors concluded: "The result supports the idea that the relatively high concentrations of mercury found in marine fish that inhabit the surface and deep waters of the open ocean **result from natural processes, not 20<sup>th</sup> century industrial pollution.**"<sup>7</sup>

In the *Pacific Ocean*, Princeton researchers found **no increase** in fish tissue mercury levels after comparing tuna samples from 1971 and 1998 (there was actually a minor **decline**). They expected to find a 9-26% increase. Those authors concluded the likely source of the mercury was deep ocean waters and sediments around geothermal vents: "Our findings that the concentration of mercury in tuna...has not changed over a period of time, during which anthropogenic mercury inputs...have increased, supports the idea that the **source of methylmercury in tuna is not in surface waters.** [This] provides prima facie **evidence that this concentration is not responding to anthropogenic emissions** irrespective of the mechanisms by which mercury is methylated in the oceans and accumulated in tuna."<sup>8</sup>

As for **human exposure** to mercury (Hg and MeHg) through fish consumption, there is evidence of micro-traces of mercury (equal to and greater than modern levels) present in humans **as long ago as 400AD**. For example, eight 550-year old mummies from Alaska had mercury levels **twice as high** as pregnant women in Alaska today.<sup>9</sup>

#### **D. What is the safe level (reference dose) of MeHg consumption according to EPA?**

A reference dose (**RfD**) is the amount of a substance that can be consumed each day for a lifetime (70 years) without harm. The EPA mercury RfD is based on inappropriate studies of people who consume **whale meat and blubber** (a unique diet no one in the US has) containing **multiple chemicals** (PCBs, cadmium, pesticides, persistent organic pollutants, DDT, etc.) of which mercury is only one. The owners of the raw data **refused to release** it for scientific review. EPA downplayed studies that found no harm.

EPA's RfD is the **most restrictive in the world - 10 times lower** than the lowest level of concern. EPA is **scaring people**, especially pregnant women, from eating fish and benefiting from vital nutrition.<sup>10</sup>

#### **E. What health benefits are lost from NOT eating fish due to concerns about mercury?**

The best study in the world, the *Seychelles Island Study* (Myers et al.) in the Indian Ocean, found benefits to children from mothers eating large quantities of fish (12-14 meals per week). These benefits included better eyesight and less hyperactivity in children.<sup>11</sup>

The *Bristol England study* found that high fish intake by pregnant women and young children was associated with **higher child mental development scores**, better eyesight and **no** adverse developmental effects associated with mercury.<sup>12</sup>

A Norwegian Study (Helland et al.) found that the nutrients in fish oil ingested during pregnancy and lactation **improves the intelligence** of children at 4 years of age.<sup>13</sup>

High fish or omega-3 fatty acids consumption may reduce risk for these conditions:

- (a) cardiovascular disease + coronary heart disease (CHD) + sudden deaths
- (b) breast cancer
- (c) prostate cancer
- (d) endometrial (inner lining of uterus) cancer
- (e) Alzheimer disease
- (f) type 2 diabetes in women and CHD in type 2 diabetic women
- (g) pre-term delivery and low birth weights, physiological and mental development of infants, and postpartum depression<sup>14</sup> and suicidal ideation.

<sup>1</sup> E.g., Carmago (1993) *Nature*, vol. 365, 302

<sup>2</sup> *Making Sense of State Fish Advisories*, March 2005

([http://ff.org/centers/csspp/pdf/20050228\\_hgfishadvisories.pdf](http://ff.org/centers/csspp/pdf/20050228_hgfishadvisories.pdf))

<sup>3</sup> Pacyna et al. (2003) *Atmos Environ*, vol. 37, S109-S117; Friedli et al., (2003) *Atmos Environ*, vol. 37, 253-267

<sup>4</sup> [http://newsdesk.inel.gov/press\\_releases/2003/10-21mercury\\_testing.htm](http://newsdesk.inel.gov/press_releases/2003/10-21mercury_testing.htm)

<sup>5</sup> see Pyle and Mather (2003) *Atmos Environ*, vol. 37, 5115-5124 [also see:

<http://ff.org/centers/csspp/docs/20050103EPANODACComments.doc>]

<sup>6</sup> Rasmussen (1994) *Environ Sci & Tech*, vol. 28, 2233-2241

<sup>7</sup> Barber et al. (1984) *Environ Sci & Tech*, vol. 18, 552-555.

<sup>8</sup> Kraepiel et al. (2004) *Environ Sci & Tech*, vol. 38, 4048 and see also Kraepiel et al., (2003), *Environ Sci & Tech*, vol. 37, 5551-5558.

<sup>9</sup> see Middaugh on pp 53-68 of July 24, 2002's FDA's Food Advisory Committee on MeHg

(<http://www.fda.gov/OHRMS/DOCKETS/ac/02/transcripts/3872t2.htm>) and also Arnold and Middaugh (2004) in *Use of Traditional Foods in a Healthy Diet in Alaska: Risks in Perspective* (available at <http://www.epi.hss.state.ak.us/bulletins/catlist.jsp?cattype=Mercury>)

<sup>10</sup> Oken et al. (2003), *Obstetrics & Gynecology*, vol. 102, 346-351

<sup>11</sup> Myers et al. (2003), *Lancet*, vol. 361, 1686-1692

<sup>12</sup> Daniels et al. (2004), *Epidemiology*, vol. 15, 394-402

<sup>13</sup> Helland et al. (2003), *Pediatrics*, vol. 111, e39-e44

<sup>14</sup> Hibbeln (2002) *Journal of Affective Disorders*, vol. 69, 16-29