Public Health Consultation

Potential Exposure to Asbestos in Clams

CERCLIS NO. AK8570028698

CR#A0JC

Port Heiden, Alaska

May 14, 2014

Prepared by:

Western Branch
Division of Community Health Investigations
Agency for Toxic Substances and Disease Registry

Summary of ATSDR's Findings

Breathing asbestos in air can make you sick	• Current science has found that inhalation of asbestos poses a major health risk. Ingestion of clams is expected to pose little risk associated with any asbestos that might be in them, and clams are an important source of local nutrition. • We stress that people limit their exposures to loose or damaged asbestos. It can be blown in the air and inhaled. • If you can avoid asbestos exposure, do so. Don't dig up areas with known buried asbestos or handle or use asbestos. If asbestos gets into the air, you might inhale it and get sick over time. • Because asbestos will not burn, some people still prefer it as insulation from heat sources. Reuse of old building materials from the Old Village and military areas might contain asbestos and result in exposure. We recommend you do not do this. Using asbestos as insulation could result in small pieces of asbestos becoming airborne and inhaled.
Collect clams from other beds, if possible	• Microorganisms may be in clams and shell fish near landfills, waste sites, and eroding village areas. • Collect clams in beds other than the clam beds near the landfills and waste sites, unless a regular shellfish monitoring program for microorganisms and seafood toxins is ongoing.
Eating Port Heiden clams—health effects vs. benefits	• The health benefits of clams outweigh the risk of eating small amounts of asbestos that might be found in the clam tissue. • Low asbestos exposure from eating some asbestos might not make you ill or sick, but no asbestos exposure is recommended. • We do not expect eating the small amounts of asbestos in clams to make people sick from ingestion. • Clams are rich in vitamin B_{12} and help the body absorb iron. • Vitamin B_{12} and iron both protect people from stomach illnesses caused by *Helicobacter pylori*. H. pylori infections can lead to vitamin B_{12} deficiency and low blood iron.
Preparing and cooking clams	• Microorganisms from eroding landfills and the old village remain a concern. • Many people prefer raw seafood (texture, convenience) and have been eating it for generations. We suggest that people cook the clams well. People should also look closely at the clams for any signs of unusual odor or damage.

Preventing future exposures: recommended actions for the Village in consultation with government agencies	• Reducing all potential chemical contamination levels, including asbestos contamination from landfills, beach areas and eroding village areas, is important. This will minimize potential physical hazards and potential health risks to Port Heiden residents from chemicals and microorganisms. • A regular shellfish monitoring program would be a prudent public health action.

Background

The Native Village of Port Heiden Shoreline History

Port Heiden is a small fishing village on the Bering Sea and Bristol Bay side of the Alaskan Peninsula. Most members of the Alutiiq community have a subsistence lifestyle. A part of their diet included, and still includes, fish and shellfish taken from the Port Heiden Bay village coastline.

This health consultation document responds to the villagers' concern that eating clams containing asbestos would result in cancer. Due to lack of site specific data, ATSDR evaluated the likely range of asbestos taken up in clams (non-detectable to the maximum uptake in clams).

No sampling data is available to identify levels of asbestos in past current or future clams (or other seafood) at Port Heiden. However, asbestos was used and reports associated with clean-up of the site identified that it was present in at least some buildings and pipelines [NWI 2012]. Some old building materials were also reused by the villagers. Shoreline erosion is ongoing and has revealed buried waste. Erosion of materials released to the beach and seafood harvesting areas change on a daily basis with tides and storms [USACE 2007]. Debris is currently pulled up by nets and seines in the seafood harvesting area [Personal Communication Anderson January 2014].

Figure 1 Erosion of shoreline revealing buried waste in seafood harvesting area

Photographic Source: Pat Roth USAF

Asbestos-containing material was very likely to be used in areas along the seafood harvesting area. Asbestos was used in construction of the old fish salteries, fish canneries, military buildings, as well as the piers that were built near the village seafood harvesting area [AGC 2010; USGS open file report; NBS 1933].

The eroding old village was constructed with some salvaged military building materials. The first military pier was constructed with wood from the old fish saltery. Military pier and shoreline, supply operations, as well as military and village waste disposal operations included some amount of asbestos [AGC, 2010; GAO 2009]. These historical areas that have the potential to have some amount of buried asbestos are eroding or are threatened by sea erosion.

Port Heiden residents report they eat shellfish including two clam types: cockle (Clinocardium nuttallii) and horse (Tresus nuttallii and Tresus capax). Port Heiden residents do not eat clams every day. Still, when they do eat clams, they ingest the entire clam contents. That means they eat the clam's siphuncle, or "filtering arm," which is where asbestos fibers primarily concentrate. Although no sampling has been conducted for asbestos in clams in the harvesting area, there is data to estimate how much asbestos might be in them. That data does not indicate a public health concern.

The Current Port Heiden Health Issue

Helicobacter pylorus is a bacterium which, if it enters the human body, can cause infections leading to vitamin B12 deficiency and low blood iron [Parkinson 2000].

Acu [2008] found that Alaskan native populations have high *H. pylori* prevalence which, in addition to B12 deficiency and low blood iron, can result in chronic gastritis, loss of the stomach's acid-producing potential, and damage to stomach cells. The high vitamin B12 content in clams—and clams' ability to help the human body absorb iron—suggest clams might protect Port Heiden residents against the risk of vitamin B_{12}/iron deficiency.

Thus, for Port Heiden residents the public health issue is whether the benefit of healthy B_{12}/iron levels from eating asbestos-contaminated Port Heiden clams as a part of a total diet outweighs the health risk from eating those same clams.[1]

The Port Heiden community petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to determine whether toxic substances from the Department of Defense (DoD) property posed a current public health hazard. Current health hazards including the eroding landfill and village are addressed in more detail in a separate health consult. Residents' concerns were that asbestos washed out from near-shore impoundments onto the beaches and into the clams. The residents' particular concern was for stomach illnesses.

This health consultation addresses the

- Potential for exposures to asbestos from eating clams,
- Toxicity of asbestos-contaminated clam ingestion, and
- Health-protective aspects of clam ingestion

Applicable Studies

Asbestos Toxicity

ATSDR's 2001 Toxicological Profile and the National Toxicology Program's 12th Report on Carcinogens [DHHS 2011] re-evaluated asbestos human and animal studies. The most current review in the 12th Report on Carcinogens stated that no clear association was found for cancer risk and exposure to asbestos in drinking water [NTP 2011]. It is very difficult to determine whether the excesses of cancers are due to asbestos or to other factors (exposure to other chemicals, misdiagnosis, dietary factors, alcohol intake, etc) [ATSDR 2001]. Homa et al. (1994) concluded that the results "suggested that exposure to amphibole asbestos (including amosite) maybe associated with colorectal cancer, but these findings may reflect an artifact of un-certification of cause of death" Homa et al. (1994) also concluded that "the results also suggest that serpentine (chrysotile) asbestos is not associated with colorectal cancer." Other reviewers have concluded that the available data do not establish a causal relationship between occupational exposure to asbestos and the development of gastrointestinal cancers [Doll and Peto 1985, 1987; Edelman 1988a, 1989; Goodman et al. 1999; Weiss 1995; ATSDR 2001].

ATSDR's ToxFAQs™ for Asbestos stresses that it is known that breathing asbestos can increase the risk of cancer in people. There are two types of lung cancer caused by exposure to asbestos: lung cancer and mesothelioma. Mesothelioma is a cancer of the thin lining surrounding the lung (pleural membrane) or abdominal cavity (the peritoneum). Cancer from asbestos does not develop immediately, but shows up after a number of years. Studies of workers also suggest that breathing asbestos can increase chances of getting cancer in other parts of the body (stomach, intestines, esophagus, pancreas, and

[1] Port Heiden residents get their drinking water from individual wells, not from any community water system that might use asbestos-insulated insulated pipes.

kidneys), but this is less certain [ATSDR 2001; ATSDR 2001]. The weight of evidence indicates that asbestos does not cause any significant non-carcinogenic effects in the gastrointestinal system [ATSDR 2001]. Review of asbestos drinking water studies suggest that the rather small increase in cancer incidence is related to other risk factors including smoking [ATSDR 2001]. In 2006 the Institute of Medicine re-evaluated the evidence for carcinogenicity of asbestos and found limited evidence that it caused cancer of the pharynx, stomach and colorectum [NTP 2006 in NTP 2011].

Below we discuss three categories of studies involving asbestos ingestion: 1) asbestos workers swallowing asbestos, 2) animals fed asbestos, and 3) epidemiologic or human studies of people drinking asbestos-contaminated water. Researchers intentionally elevated the amount of asbestos fed to laboratory animals. The amount of asbestos detected in worker studies and community drinking water studies was well beyond anything Port Heiden residents might encounter in clams taken from Bristol Bay. Nevertheless, each study category suggested ingesting asbestos in clams represented a low health risk.

Worker Studies

In inhalation studies much of the asbestos may reach parts of the lower respiratory tract, where it stays, and only some of the asbestos may be transported out of the respiratory tract by mucociliary clearance when it is then swallowed. It is therefore difficult to quantify what part of the dose was ultimately ingested and thus difficult to link the gastrointestinal (GI) tract health effects to a specific dose of swallowed asbestos.

Studies in which workers inhaled then swallowed asbestos showed the highest asbestos-ingestion related risk. But the evidence in these studies is weak as ingestion was secondary to mucociliary clearance[2] [Seikoff 1964, 1968, 1974; Hammond 1965, 1973; Mancuso 1965; Kleinfeld 1967; Elms 1971; Enterline 1972; McDonald 1973; Newhouse 1973; Preger 1978].

When evaluating asbestos toxicity in these studies, five points have some relevance to ingestion:

1. In addition to lung cancer, asbestos workers were found to have GI cancers. Both of these cancers result from workers inhaling and swallowing airborne asbestos [Seikoff 1964, 1968, 1974; Hammond 1965, 1973; Mancuso 1965; Kleinfeld 1967; Elms 1971; Enterline 1972; McDonald 1973; Newhouse 1973; Preger 1978].
2. Asbestos fibers can penetrate the interior walls of the stomach, intestine, and colon. Such asbestos penetration can result in lesions.
3. Some analogies can be drawn from lung tissue studies to the digestive tissues.

[2] The cilia lining the lungs are covered with mucous on top of which is a thin film that moves out of the lungs toward the mouth. The movement takes much of the trapped debris with it. The debris are either coughed out or swallowed.

4. Asbestos fibers of appropriate size can inhibit DNA repair [FDA 1973; Cook 1983; DHHS 1987]. (Note: Becomes important if DNA damage occurs[3])

Lung tissue is more sensitive than the digestive tract. Delivery of asbestos fibers from air into the lung is much more efficient than delivery of foodborne asbestos fibers into stomach tissue. This inefficiency helps lower the ingestion pathway risk. The studies discussed here, albeit limited, confirm this.

The U.S. EPA identified a potential increase of benign intestinal polyp development from asbestos-contaminated drinking water [USEPA 1991; USEPA 2010a; USEPA; 2010b]. The U.S. EPA has established a maximum contaminant level (MCL) of 7 MFL (million fibers per liter) for asbestos in drinking water [USEPA 1991; USEPA 2010a; USEPA 2010b]. A 2006 review by the Institute of Medicine (IOM) United States Committee on Asbestos found evidence suggestive—but not sufficient—to infer a causal relationship between asbestos exposure and stomach cancer as well as , colon or rectum cancer) [NAS 2006]. That said, in 2010 U.S. EPA's Safe Drinking Water Act (SDWA), National Primary Drinking Water Regulation (NPDWR), stated it did not believe a revision to the NPDWR was appropriate at that time [USEPA 2010a].

In 1973, U.S. Food and Drug Administration regulations limited the use of asbestos filters in food processing, given the *possibility* that the asbestos fibers posed a health risk [FDA 1973]. Researchers knew that asbestos from filters entered the foods themselves, but they had never found an actual risk. In fact, Cunningham [1971] showed that several kinds of popular drinks had 100 million fibers of asbestos per liter. Also at about that same time, studies began to suggest that asbestos workers had twice the amount of gastrointestinal cancers as did controls; researchers assumed these cancers were associated with ingestion [Seikoff 1964, 1968, 1974; Hammond 1965, 1973; Mancuso 1965; Kleinfeld 1967; Elms 1971; Enterline 1972; McDonald 1973; Newhouse 1973; Preger 1978]. But animal toxicity and human epidemiological studies demonstrated that these cancers and other adverse health effects resulted from swallowing asbestos that was first inhaled [Garabrant 1992; Haque 2001; DHHS 1987].

Many questions remain as to possible ways that asbestos might result in non-respiratory cancers. Research is limited with respect to determining the actual dose to gastrointestinal systems that caused these cancers.

Animal Ingestion Studies

In response to the presence of asbestos in foods and an increase in the knowledge of inhalation toxicity, several studies investigated ingestion toxicity. A study examining 45 hamsters fed 1% asbestos (including harsh chrysotile asbestos) for a lifetime showed no effects [Smith 1965]. One study showed a slight increase in stomach or colon cancers after rats were fed a lifelong diet containing 1% asbestos [McConnell 1983a 1983b, 1984]. Donham [1980] found a slight increase of colon cancer after rats were fed a lifelong (3-year) diet containing up to 20% chrysotile asbestos compared with a fiber diet

[3] In the lung, the presence of asbestos might cause various immune cells to try and ingest the asbestos fibers and result in the release of DNA reactive species.

and a normal diet. But the total number of rats (over 100) in both McConnell and Donham studies was too low to show significance. In examining the rats' colons, Donham found four tumors in those eating asbestos, three tumors in those eating a standard diet, and two tumors in those eating a high fiber diet. Because these high exposures resulted in very few cancers, Donham decided much larger populations were needed to achieve any significant results [see also DHHS 1987]. Both amosite and chrysotile asbestos have been reported in military construction materials at Port Heiden.

Chrysotile Asbestos Studies

The National Toxicology Program (NTP) testing status for chrysotile as of 2012 places emphasis on the 1985 and 1990 chrysotile asbestos rat and hamster feeding studies.

A NTP 1985 chrysotile asbestos feeding study in F344/N Rats identified no evidence of carcinogenicity in male or female rats exposed to SR chrysotile asbestos or in female rats exposed to IR chrysotile asbestos. There was some evidence of carcinogenicity in male rats exposed to IR chrysotile asbestos as indicated by an increased incidence of adenomatous polyps in the large intestine [NTP 1985].

An NTP 1990 chrysotile asbestos feeding study of Syrian golden hamsters carcinogenesis results were negative [NTP 1990a]. Under the conditions of these studies, neither short range chrysotile nor intermediate range chrysotile asbestos was carcinogenic when ingested at 1% levels in the diet by male and female Syrian golden hamsters. While there were increases in the rates of adrenal cortical adenomas in male and female hamsters exposed to intermediate range chrysotile asbestos compared to the pooled groups, these incidence rates were not different when compared with the concurrent control groups. Additionally, the biologic importance of adrenal tumors in the absence of target organ (gastrointestinal tract) neoplasia is questionable [NTP 1990].

Dietary administration of amosite or chrysotile asbestos of short or intermediate lengths did not cause tumors in female rats, but dietary exposure to the intermediate-length chrysotile asbestos resulted in benign (non-cancerous) adenomatous polyps of the large intestine of male rats. When filter material containing chrysotile was added to the diet of rats, the overall incidence of malignant tumors (including kidney, lung and liver tumors) was increased [NTP 1985; IARC 1987 in NTP 2011]. However; oral administration of amosite, tremolite, or crocidolite did not cause tumors in rats, nor did oral administration of amosite or chrysotile asbestos in hamsters [DHHS 1985; IARC 1987 in DHHS 2011].

Only one other high-dose asbestos ingestion study followed the DHHS [(1987] determination. Haque [2001] fed pregnant mice chrysotile asbestos by gavage to determine whether fibers transferred to the fetuses. The mean lung fiber count of the treated group of pups was 780 fibers/g. The mean fiber count of the liver was 214 fibers/g. No significant weight gain difference appeared between the treated and the control group of pups. Haque deemed statistically insignificant the postnatal and fetal mortality of the two groups: 8.2% for the treated group and 4.5% for the control group.

Amosite Asbestos Studies

The NTP testing status for amosite asbestos as of 2012 placed emphasis on rat and hamster feeding studies.

An NTP 1990 amosite asbestos feeding study of in F344/N Rats determined that amosite asbestos was not overtly toxic, did not affect survival, and was not carcinogenic when ingested at a concentration of 1% in the diet by male or female F344/N rats. The amosite-exposed rats showed enhanced survival compared with that of the non-exposed rats [DHHS 1990b].

A 1983 NTP amosite asbestos feeding study of Syrian golden hamsters identified the following: carcinogenesis studies of amosite asbestos were conducted by administering diets containing 1% of the asbestos in pellets from the conception of the mothers through the lifetime of male and female Syrian golden hamsters. Control groups consisted of 127 male and 126 female hamsters and the amosite asbestos groups consisted of 252 male and 254 female hamsters. No adverse effect on body weight gain or survival was observed from treatment with amosite asbestos. Neither of the amosite asbestos groups showed increased neoplasia in any organ or tissue compared to the control groups. Under the conditions of these studies, the ingestion of amosite asbestos at a level of 1% in the diet for their lifetime was not toxic and did not cause a carcinogenic response in male and female Syrian golden hamsters [DHHS 1983].

Site-specific sampling of Port Heiden clams is not available. Sampling results might change over time depending on the amount of asbestos present in eroded material in water being filtered by clams and plankton or other clam food sources. The highest asbestos concentrations in clams that Belanger [1986] examined was 1,000 fibers per mg (dry weight) of clam, or 1 fiber/g (dry weight), or 0.1 fiber/g (wet weight). This concentration is about 2,000 times lower than the Haque [2001] levels. Therefore, as compared with the occupational and drinking water studies, we expect that Port Heiden clams would have much lower asbestos levels as well.

Highest Concentration of Asbestos Found In Clam Studies

Studies involved asbestos at 1–20% of an animal's total diet over a lifetime. Compare those rates to the highest possible asbestos concentrations ingested at Port Heiden. Using Belanger's [1986] studies of the highest asbestos concentrations in clams—1,000 fibers per mg (dry weight)—and Lynch et al.'s [1970] findings of 8 fibers/ng of chrysotile asbestos, we estimate a clam might have 125 ng of asbestos for every mg of that clam's dry weight. Given that the clam's dry weight is about 10% of its wet weight, the clams would contain about 0.00125% asbestos.[4] For the clams to contain 1.25% asbestos by mass, they would have to contain 1 million asbestos fibers/mg. This asbestos level is far above anything Belanger or Lynch and colleagues found. Given that Port Heiden residents' diet does not consist exclusively of clams, the residents are not expected to reach the same dose as the animal studies that showed adverse effects.

Human Drinking Water Studies

Several human or epidemiologic studies evaluated the hazards associated with drinking water contaminated by asbestos-lined water delivery systems. Although these studies are

[4] Asbestos fibers vary in length and mass. If for example, the calculation for the average values is (1mg dry/10 mg wet) x (1000 fibers/mg clam) x (1ng wt/8 fibers) = 12.5 ng asbestos/mg clam = 0.00125%

not directly applicable to Port Heiden clams, they do provide evidence of some risk associated with asbestos ingestion.

The studies included some communities with more than 100,000 persons. They were drinking water containing over a million to more than a billion fibers of asbestos per liter of water. Despite the large populations and high asbestos concentrations, researchers observed no clear links to cancer [Marsh 1974; Harrington 1978; Sigurdson 1981, 1983; Meigs 1980; Sadler 1981].

Some argued that exposures less than 30 years in duration were too short to provide accurate results. One 40-year exposure study in California did find an association between some cancers and drinking water, with asbestos levels as high as 36 million fibers per liter of water [Kanarek 1980; Conforti 1981; Tarter 1981, 1983; Marsh 1983]. Those cancers were in the densely populated census tract area of San Francisco and were compared with the surrounding bay area—populations that may not be appropriate for direct comparison [DHHS 1987].

Three studies (one each in Florida, Washington, and Quebec) had exposures of 30–40, 40, and over 50 years respectively. These three studies, with populations of 46,123; 200,000; and 420,000, produced no statistically significant associations between exposure and excess cancer [Millette 1983; Polissar 1982; Wigle 1977].

Thus, studies have shown that ingestion of high asbestos levels—in and of itself—has not resulted in a clear cancer risk. More recent studies investigated people with colon cancer to determine whether they had been exposed to high asbestos levels. These studies also indicated little or no risk. In a recent study, 746 persons with colon cancer were compared with 746 of their noncancerous neighbors. Analyses by frequency and duration of exposure showed no association between asbestos and colon cancer risk [Garabrant 1992]. Other case studies involving people who also inhaled asbestos found weak or uncertain risks associated with colon cancer:

- Aliyu [2005] found asbestos workers who were heavy smokers had an increased risk of colorectal cancers, but that risk was much lower than the risk of pleural plaques. Indeed, one concern for studies associating asbestos and colon cancers is that lung cancer may clinically mimic other diseases [Doll and Petro 1985; Gamble 1994].
- Three papers re-evaluated studies that looked at patients that were diagnosed with gastrointestinal tumors [Doll and Petro 1985; Newhouse 1969; Seilikoff 1979]. The re-evaluation identified that half of the gastrointestinal tumors in one study were misdiagnosed and reclassified as lung cancer or mesothelioma. In another study, half of the pancreatic cancers were misclassified [Doll and Petro 1985; Newhouse 1969; Seilikoff 1979]. These misclassifications have a significant effect on gastrointestinal cancers (a factor of two) but much less effect on the more plentiful mesothelioma cases (a less than 10% increase).

The studies underscore the importance of inhalation hazards over asbestos-associated ingestion hazards. The colon or colorectal cancer risk associated with asbestos inhalation outweighs the asbestos-ingestion risk—if, in fact, any such ingestion risk exists.

While not thoroughly investigated in water and foods, inhalation hazards are highest for fibers longer than 8 microns and thinner than 0.25 microns [Davis 1978; Stanton 1981; Lipkin 1980; Platek 1985]. These observations are germane to food ingestion. Fibers found in marine environments tend to be shorter than hazardous fibers [Millet 1980, 1983].

Large populations have consumed asbestos-contaminated water for decades. Studies of such populations, however, have found little or no evidence of health risk and should provide perspective for the Port Heiden community:

- Site-specific sampling of Port Heiden clams is not available and might change over time depending on the amount of asbestos present in eroded material in the water filtered by clams and plankton or other clam food sources. Although clams accumulate asbestos, they do so at much lower levels than the asbestos levels in animal ingestion studies. Any asbestos level is a matter of concern, but the highest levels of asbestos fibers found in clams were lower than the over-a-lifetime 1%, 10%, or 20% that resulted in health effects in the worst-case animal studies.
- The amount, type and size of asbestos fibers may change over time due to materials being eroded and reworked by waves and the time between release and uptake. The average length of an asbestos fiber found in bays was less than 1 micron, with less than 0.2–2% of the fibers larger than 5 microns [Millet 1980, 1983]. The majority of the fibers Millet found in the marine environment were shorter than those considered most toxic.
- People in many parts of the United States consume water brought into their homes through asbestos-lined pipes. That water has concentrations of millions of fibers per liter, resulting in some people ingesting up to a billion fibers per day or more with no apparent health effects.

Bioaccumulation and Exposure Data — Clams and Asbestos

As previously mentioned, although no asbestos measurements were made on Port Heiden clams, we can estimate the possible levels using other studies. Some clam species can accumulate asbestos. Asbestos might reduce the clam's ability to siphon water. In an effort to study the effect of allowable levels of asbestos in water, researchers have designed a method to study asbestos accumulation in animals [Lauth and Schurr 1983, 1984; Belanger 1986a, 1986b, 1987]. Their studies found clams accumulate asbestos easily. Their studies suggest that at some sites, clams can accumulate up to the highest levels found in drinking water. The studies also note that plankton is a clam food source, and that plankton has been found to contain asbestos. Plankton, however, can only accumulate a limited amount of asbestos—excessive accumulation inhibits the plankton's swimming ability.

Clams that live on sediment surface (epifaunal) and in sediment itself (semi-infaunal) both feed on plankton through a double-tubed siphon that operates like a snorkel. One siphon obtains food, and the second siphon eliminates waste products. Clams may be harvested from different sediment and water depth depending on the species, the temperature, or the season.

Without doubt, asbestos can affect some clam species. These clam species take up and accumulate asbestos. The literature particularly reports levels and effects for the freshwater clam, *Corbicula fluminea* (Asiatic clam)[5]:

- Lauth and Schurr [1983, 1984] suggested that positively charged chrysotile fibers will attach to planktonic cells, inhibiting their swimming capacity. Because this limits the amount of asbestos plankton can accumulate, it also limits the amount of asbestos a clam can take up from plankton.
- Belanger [1986a, 1987] showed that in juvenile *Corbicula fluminea* (Asiatic clam) exposed to chrysotile fibers, siphoning activity was significantly reduced and growth and reproduction were altered, thus reducing clams' maximum asbestos intake.
- Belanger [1986b] found siphoning activity reduced by about 20% in juvenile clams exposed to 100 to 100 million fibers per liter of water for 30 days. Those clams accumulated about 150 fibers/mg of their weight.
- Belanger [1986b] also found shell growth significantly reduced at concentrations in the range of 10,000 to 100 million fibers per liter of water.
- Belanger [1987] reported clams accumulated chrysotile to a greater degree than any previously tested aquatic organism. Whole-body asbestos burdens of clams exposed to 100 million fibers per liter of water for 30 days were nearly 1,000 fibers/mg of their weight (dry weight). Field-collected clams exposed throughout their lifetime (2–3 years) to about 1 billion chrysotile fibers per liter of water accumulated as much as 650 million fibers.

Clams and Vitamin B12 and Iron

- The U.S. Department of Agriculture and the National Institute for Health state that clams have among the highest levels of vitamin B_{12}—almost 100 times more than tuna and almost 20 times more than salmon [USDA 2003].
- A serving of 1 ounce of clams represents over four times the recommended dietary intake of vitamin B_{12} [Bialostosky 2002].
- Clams are also a good source of iron and are considered to enhance iron absorption from other foods [Hurrell 2004].

This research leads to the conclusion that a balanced meal containing clams—even clams contaminated with some asbestos—along with high-iron foods (e.g., pumpkin seeds, canned beans, potatoes, and enriched pasta) will result in absorption of high quantities of iron. A diet like this especially helps people with anemia or people infected by viruses such as *H. pylori*.

[5] Any important biological differences between *Corbicula fluminea* and the clam species prevalent in Port Heiden (*Clinocardium nuttallii*, *Tresus nuttallii*, and *Tresus capax*) appear sufficiently limited as to allow the use of *Corbicula fluminea* as a biomarker for the Port Heiden species.

Conclusions

We do not expect that eating the small amounts of asbestos likely found in Port Heiden clams will result in adverse health effects.

Removal of eroding landfills and eroding village areas will help reduce chemical contamination levels, including asbestos contamination. This will minimize potential health risks to Port Heiden residents from chemicals and microorganisms, and physical hazards.

Clams are a source of vitamin B_{12} and can increase iron absorption. Both of these qualities could help protect against *H. pylori*, a risk factor associated with stomach illness in many Alaska natives.

Recommendations

- Cook the clams well to kill any potential harmful bacteria.
- If possible, harvest clams in areas free from chemical and biological contamination.
- Consider developing a shellfish monitoring program for chemicals, microorganisms and toxins, as done in other fishing communities.
- Do not eat clams with any signs of unusual odor or damage.

Report all asbestos containing material identified in the homes or in the environment to the Tribal Environmental office at (907) 837-2441or Alaska Department of Environmental Conservation, South Central and Western Facilities: Division of Environmental Health, Solid Waste Program at:

Phone: (907) 269-7622 Fax: (907) 269-7600.

Do not attempt to remove or handle asbestos. Only a trained professional should remove or handle asbestos.

Other Related Information on Asbestos and Clams

The National Institutes for Health and the U.S. Department of Agriculture provide nutritional facts on a healthy diet at: http://www.nal.usda.gov/fnic/cgi-bin/nut_search.pl. A diet that includes 3 oz. of fresh, healthy clams a day could result in a healthy dose of vitamin B_{12}.

Authors:

Charles Grosse
Geologist and Health Scientist
Western Branch, HQ Team
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Greg Zarus
Atmospheric Scientist and Geophysicist
Western Branch, HQ Team Lead
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

Reviewed By
Casandra V. Smith, Branch Chief
Supervisory Health Scientist
Western Branch
Division of Health Assessment and Consultation
Agency for Toxic Substances and Disease Registry

References

Aliyu OA, Cullen MR, Barnett MJ, et al. 2005. Evidence for excess colorectal cancer incidence among asbestos-exposed men in the Beta-Carotene and Retinol Efficacy Trial. Am J Epidemiol 162(9):868–78. Epub 2005 Sep 21

Acu RR. 2008. H. Pylori and low stomach acid: Nutritional causes, prevention, and therapies. Available at: http://www.acu-cell.com/dis-hpy.html.

AGC Army Geospatial Center, September. 2010. Port Heiden/Fort Morrow Alaska: Examination of Historical Aerial Photography.

ATSDR (Agency for Toxic Substances and Disease Registry). 2006. Asbestos: Working with patients: Diagnosis. Available at: http://www.atsdr.cdc.gov/asbestos/medical_community/working_with_patients. [accessed 2006 August 30].

ATSDR. 2001. Toxicological profile for asbestos. Atlanta: US Department of Health and Human Services US Public Health Service

ATSDR. 1989. Toxicological profile for asbestos (Draft). Atlanta: US Department of Health and Human Services US Public Health Service.

Belanger SE, Cherry DS, Cairns J. 1986a. Uptake of chrysotile asbestos fibers alters growth and reproduction of Asiatic clams. Can J Fish Aquat Sci 43:43–52.

Belanger SE, Cherry DS, Caims J. 1986b. Seasonal, behavioral and growth changes of juvenile Corbicula fluminea exposed to chrysotile asbestos. Water Res 20:1243–250.

Belanger SE, Schurr K, Allen DJ, Gohara AF. 1986c. Effects of chrysotile asbestos on Coho salmon and green sunfish: evidence of behavioral and pathological stress. Environ Res 39:74–83.

Belanger SE, Cherry DS, Cairns J, et al. 1987. Using Asiatic clams as a biomonitor for chrysotile asbestos in public water supplies. J Am Water Works Assoc 79:69–74.

Belanger SE, Cherry DS, Cairns J Jr. 1990. Functional and pathological impairment of Japanese Medaka (*Oryzias latipes*) by long-term asbestos exposure. Aquat Toxicol, 17:133–154.

Bialostosky K, Wright JD, Kennedy-Stephenson J, et al. Dietary intake of macronutrients, micronutrients and other dietary constituents: United States 1988–94. Vital Heath Stat 11(245) ed: National Center for Health Statistics; 2002.

Bogosky P, Gilson JC, Timbrell V, et al. eds. International Agency for Research on Cancer, Pub. No. 8, Lyon: 1973; p. 203–08.

Carlucci AF, Bowes PM. 1970. Production of Vitamine B12, thiamine, and biotin by Pytoplankton. J Phycol 35:1–17.

Conforti PM, Kanarek M, Jackson L, et al. 1981. Asbestos in drinking water and cancer in the San Francisco Bay Area: 1969–1974 incidence. J Chronic Dis 34:211–24.

Cook P M. 1983. Review of published studies on gut penetration by ingested asbestos fibers. Environ Health Perspect 53:121-130.

Cunningham HJ, Pontefract R. 1971. Asbestos fibers in beverages and drinking water. Nature 232: 332–33.

Davis JMG, Beckett ST, Bolton RE, et al. 1978. Mass and number of fibers in the pathogenesis of asbestos-related lung disease in rats. Br J Cancer 37:673–88.

Donham KJ, Berg JW, Will LA, et al. 1980. The effects of long-term ingestion of asbestos on the colon of F344 rats. Cancer 45:1078–084.

Doll R, Petro J. 1985. Asbestos: Effects on health of exposure to asbestos. London, UK: Health and Safety Commission.

DHHS (US Department of Health and Human Services). 1983. Lifetime Carcinogenesis Studies of Amosite Asbestos (CAS NO. 12172-73-5) in Syrian Golden Hamsters (Feed Studies). NTP TR 249. Research Triangle Park, NC: National Toxicology Program.

DHHS. 1985. Toxicology and Carcinogenesis Studies of Chrysotile Asbestos (CAS No. 12001-29-5) in F344/N Rats (Feed Studies). NTP TR 295. Research Triangle Park, NC: National Toxicology Program.

DHHS.1987. Working Group for the DHHS Committee to Coordinate Environmental and Related Programs, Subcommittee on Risk Assessment: Report on Cancer Risks Associated with the Ingestion of Asbestos. Environ Health Perspect 72:253–65.

DHHS. 1990a. Lifetime Carcinogenesis Studies of Chrysotile Asbestos (CAS No. 12001-29-5) in Syrian Golden Hamsters (Feed Studies). NTP TR 246. Research Triangle Park, North Carolina: National Toxicology Program.

DHHS. 1990b. Toxicology and Carcinogenesis Studies of Amosite Asbestos (CAS No. 12172-73-5) in F344/N Rats (Feed Studies). NTP TR 279. Research Triangle Park, NC: National Toxicology Program.

DHHS. 2011. National Toxicology Program's 12th Report on Carcinogens: National Toxicology Program

Elmes PC and Simpson MJC. 1971. Insulation workers in Belfast 3. Mortality 1940–66. Er J Int Med 28:226–36.

Enterline P, Decoufle P, Henderson V. 1972. Mortality in relation to occupational exposure in the asbestos industry. J Occ Med 14:897–03.

Food and Drug Administration. 1978. Federal Register 38:27076-27081 ().

Gamble JF. 1994. Asbestos and colon cancer: A weight of evidence review. Env Health Perspect 102:12.

Garabrant DH, Peters RK, Homa DM, et al. 1992. Asbestos and colon cancer: Lack of association in large case-control study. Am J Epidemiol 135(8):843–53.

GAO U.S. Government Accountability Office. 2009. Alaska Native Villages, Limited Progress Has Been Made on Relocating Villages Threatened by Flooding and Erosion June 3, 2009. URL http://www.gao.gov/new.items/d09551.pdf.

Hammond EC and Selikoff IJ. 1965. Neoplasia among insulation workers in U.S.A. with special reference to intra-abdominal neoplasm. Ann NY Acad Sci 132:519–25.

Hammond EC and Selikoff IJ. 1973. Relation of cigarette smoking to risk of death of asbestos-associated disease among insulation workers in the United States in biological effects of asbestos. WHO. International Agency for Research on Cancer. Pub. No. 8. Lyon FR.

Harrington JM, Craun G, Meigs JW, et al. 1978. An investigation of the use of asbestos cement pipe for public water supply and the incidence of gastrointestinal cancer in Connecticut, 1935–1973. Am. J. Epidemiol. 107: 96-103.

Haque AK, Ali I, Vrazel DM, Uchida T. 2001. Chrisotile asbestos fibers detected in the newborn pups following gavage feeding of pregnant mice. J Toxicol Environ Health 62(1):23–31.

Homa D et al., A meta-analysis of colorectal cancer and asbestos exposure, Am J Epidemiol. 1994 Jun 15;139(12):1210-22

Hurrell RF, Lynch S, Bothwell T, et al. 2004. Enhancing the Absorption of Fortification Iron: A SUSTAIN Task Force Report. Int J Vitam Nutr Res Suppl 6:387-401.

Kanarek MS, Conforti P, Jackson L, et al. 1980. Asbestos in drinking water and cancer incidence in the San Francisco Bay Area. Am J Epidemiol 112:54–72.

Kleinfield M, Messite J, Kooyman 0. 1967. Mortality experience in a group of asbestos workers. Arch Environ Health 15:177–180.

Lauth and Schurr. 1983. Some effects of crysotile asbestos on a planktonic algae (Cryptomonas erosa). Micron 14(1):93–94.

Lipkin LE. 1980. Cellular effects of asbestos and other fibers: correlations with *in vivo* induction of pleural sarcoma. Environ Health Perspect 34:91–102.

Lynch JL, Ayer HE, Johnson DL. 1970. The interrelationships of selected asbestos exposed indicies. Am Ind Hyg Assoc J 31:598-604.

Marsh GM. 1983. Critical review of epidemiologic studies related to ingested asbestos. Environ Health Perspect 54:49–56.

McConnell EE. 1984. NTP technical report on the toxicology and carcinogenesis studies of chrysotile asbestos. Research Triangle Park, NC: US Department of Health and Human Services, NTP TR 295, NIH Publication No. 84-2551, NTP-83-173.

McConnell EE, Rutter HA, Ulland BM, et al. 1983. Chronic effect of dietary exposure to amosite asbestos and tremolite in F344 rats. Environ Health Perspect 53:27–44.

McConnell EE, Shefner AM, Rust JH, et al. 1983. Chronic effect of dietary exposure to amosite and chrysotile asbestos in Syrian golden hamsters. Environ Health Perspect 53:11–26.

McDonald JC. 1973. Cancer in chrysotile mines and mills in biological effects of asbestos. Lyon, France: WHO. International Agency for Research on Cancer. Pub. No. 8. p. 189–94.

Mancuso TF. 1965. Discussion. Ann NY Acad Sci 132:589–94.

Meigs JW, Walter S, Hestbon J, et al. 1980. Asbestos-cement pipe and cancer in Connecticut, 1955–1974. Environ Res 42:187–97.

Millette JR, Craun GF, Stober JA, et al. 1983. Epidemiology study of the use of asbestos-cement pipe for the distribution of drinking water in Escambia County, Florida. Environ Health Perspect 53:91–98.

NAS National Academy of Sciences. 2006. Committee on Asbestos: Selected Health Effects, Board on Population Health and Public Health Practices. Washington DC: NA Press. Available at: www.nap.edu [©2011].

NAS National Academy of Sciences. 2006. Institute of Medicine (IOM)/United States Committee on Asbestos: Selected Health Effects: Asbestos Selected Cancers. Washington DC. Available at: www.nap.edu [©2011].

NBS National Bureau of Standards. 1933. U.S. Department of Commerce Standards Yearbook 1933, Bureau of Standards Miscellaneous Publication Number 139. {Note page 91}

NWI North Wind Incorporated. 2012. Port Heiden Remedial Investigation (June): UFP-QAPP Body of Knowledge Evaluation D-3 –D14 URL https://portheiden.northwindgrp.com/filebrowser/download/278

Newhouse ML, Wagner JC. 1969. Validation of death certificates in asbestos workers. BR J Med 26:302–07.

Parkinson A, Gold BD, Buklow RB, et al. 2000. High prevalence of Helicobacter pylori in the Alaska native population and association with low serum ferritin levels in young adults. Clin Diagn Lab Immunol 7(6)885–88.

Platek SF, Groth DH, Ulrich CE, et al. 1985. Chronic inhalation of short asbestos fibers. Fundam Appl Toxicol 5:327–40.

Polissar L, Severson RK, Boatman ES, et al. 1982. Cancer incidence in relation to asbestos in drinking water in the Puget Sound Region. Am J Epidemiol 116:324–28.

Preger L, Arai IT, Kotin P, et al. 1978. Asbestos-related disease. New York, NY: Grune and Strattion, Inc.

Sadler TD, Rom WN, Lyon JL, et al. 1981.The use of asbestos-cement pipe for public water supply and the incidence of cancer in selected communities in Utah, 1867–1976. Salt Lake City, Utah: University of Utah master's thesis.

Seilikoff IJ, Hammond EC, Saidman H. 1979. Mortality experience of insulation workers in the US and Canada 1943–1976. Ann NY Acad Sci 330:91–116.

Selikoff IJ. Epidemiology of gastrointestinal cancer. 1974. Environ Health Perspect 9:229–305.

Selikoff IJ, Churg J, Hammond EC. 1964. Asbestos exposure and neoplasia. JAMA 188:22–26.

Selikoff IJ, Hammond EC. 1968. Community effects of nonoccupational environmental asbestos exposure. Am J Pub Health 58:1658–66.

Sigurdson EE, Levy BS, Mandell J, et al. 1981. Cancer morbidity investigations: Lessons from the Duluth study of possible effects of asbestos in drinking water. Environ Res 25:50–61.

Sigurdson EE. 1983. Observations of cancer incidence in Duluth, Minnesota. Environ Health Perspect 53:61–7.

Smith WE, Miller L, Elsasser RE, et al. 1965. Tests for carcinogenicity of asbestos. Ann NY Acad Sci 132:456–88.

Stanton MF, Layard M, Tegeris A, et al. 1981. Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. JNCI 67: 965–75.

State of Alaska. Alaska Community Database—Community Information Summaries. Port Heiden. Available at: http://www.commerce.state.ak.us/dca/commdb/CIS.cfm?comm_boro_name=Port%20Heiden [updated 2012 Jun 12] [accessed 2012 Jul 3].

Stevens RH, Will LA, Cole DA, et al. 1979. Cyclic nucleotides in asbestos induced rat peritoneal mesothelioma. Environ Res 19:442–48.

Tarter ME, Cooper RC, Freeman WR. 1983. A graphical analyses of the interrelationships among waterborne asbestos, digestive system cancer and population density. Environ Health Perspect 53:78–89.

Tarter ME. 1981. Pattern recognition in the context of an asbestos cancer threshold study. In: Eddy WF, ed. Proceedings of the Thirteenth Annual Symposium on the Interface: Computer Science and Statistics. New York: Springer-Verlag. p. 105–110.

Timbrell V, Gibson JC, Webster I. 1978. UICC standard reference samples of asbestos. Int J Cancer 3:406–08.

Timbrell V, Rendall REG. 1972. Preparation of ache UICC standard reference samples of asbestos. Powder Technol 5:279–87.

USACE US Army Corps of Engineers. 2007. Alaska Baseline Erosion Assessment: Erosion Information Paper - Port Heiden, Alaska; October 24, 2007.

USEPA US Environmental Protection Agency. 1991. National Primary Drinking Water Regulations—Synthetic Organic Chemicals and Inorganic Chemicals; Monitoring for Unregulated Contaminants; National Primary Drinking Water Regulations Implementation; National Secondary Drinking Water Regulations; Final Rule. Federal Register. Vol. 56, No. 30; January 30. p. 3526.

USEPA US Environmental Protection Agency. 2010a. Federal Registry, Part II Environmental Protection Agency: National Primary Drinking Water Regulations; Announcement of the Results of EPA's Review of Existing Drinking Water Standards and Request for Public Commend and /or Information on Related Issues; Notice. 15500 Federal Register / Vol. 75, No. 59 / Monday, March 29, 2010 / Notices. Available at: http://edocket.access.gpo.gov/2010/2010-6624.htm.

USEPA US Environmental Protection Agency. 2010b. Integrated Risk Information system (IRIS): Asbestos (CASRN 1332-21-4); March 16. Available at: http://www.epa.gov/iris/subst/0371.htm [updated 2011 Mar 7].

USDA US Department of Agriculture. 2003. Agricultural Research Service. USDA Nutrient Database for Standard Reference, Release 16. Nutrient Data Laboratory

Home Page. Available at: http://www.nal.usda.gov/fnic/cgi-bin/nut_search.pl [modified 2010 Dec 2].

USGS United States Geological Survey Virta, RL. Open File report 02-149. Asbestos Geology, Mineralogy, Mining, and Uses, Open file report 02-149. last accessed February 6, 2014.

Volkheimer G. 1974. Passage of particles through the wall of the gastrointestinal tract. Environ. Health Perspect 9:215–25.

Wagner JC, Gilson JC, Berry G, et al. 1976. Epidemiology of asbestos cancers. Br Med Bull 27:71–76.

Ward JM. 1974. Morphogenesis of chemically induced neoplasms of the colon and small intestine in rats. Lab Invest 505–13.

Westlake GE, Spjut HJ, Smith MN. 1965. Penetration of colonic mucosa by asbestos particles. An electron microscopic study in rats fed asbestos dust. Lab Invest 14:2029–33.

Wigle DT. 1977. Cancer mortality in relation to asbestos in municipal water supplies. Arch Environ Health 32:185–89.