Public Health Assessment

Final Release

FORMER CELOTEX INDUSTRIAL PARK PETITION SITE

EDGEWATER, BERGEN COUNTY, NEW JERSEY

EPA FACILITY ID: NJD000606442

Prepared by the
New Jersey Department of Health and Senior Services

FEBRUARY 9, 2011

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333
THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR’s Cooperative Agreement Partner pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR’s Cooperative Agreement Partner has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR’s Cooperative Agreement Partner addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR’s Cooperative Agreement Partner which, in the agency’s opinion, indicates a need to revise or append the conclusions previously issued.

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Prepared by:
New Jersey Department of Health and Senior Services
Public Health Services
Consumer, Environmental and Occupational Health Service
Environmental and Occupational Health Surveillance Program
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry
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Summary

Introduction
A resident living near the former Celotex Industrial Park site in Edgewater, Bergen County petitioned the federal Agency for Toxic Substances and Disease Registry (ATSDR) about health impacts including lymphoma and breast cancer from exposures associated with the site. According to the petitioner, dust generated during the construction activities seeped into the apartments on a daily basis. Foul odors emanating from the site were also reported.

In response, the New Jersey Department of Health and Senior Services (NJDHSS) in cooperation with the ATSDR prepared the following Public Health Assessment (PHA) to address the past inhalation exposures. Other exposures pathways were evaluated elsewhere.

ATSDR’s and NJDHSS’s top priority is to ensure that the community around the site has the best information possible to safeguard its health.

Conclusions
NJDHSS and ATSDR have reached four conclusions in this PHA on the Celotex Industrial Park site:

Conclusion 1
The NJDHSS and ATSDR conclude that currently there are no site related contaminant exposure pathways that can harm people’s health.

Basis for Conclusion
Since construction activities near the Promenade have been completed, residents are not being exposed to any contaminants.

Conclusion 2
The NJDHSS and ATSDR conclude that exposures to ambient and indoor airborne dust and dust containing metals are not likely to have harmed people’s health.

Basis for Conclusion
Since indoor air contaminant concentrations were not measured, metal concentrations in the ambient air were used to estimate the indoor air contaminant levels. An evaluation of exposure to ambient dust at the Celotex site indicates that health effects are not likely to occur. The calculated indoor air chromium concentration exceeded the health-based screening value, however, based on average estimated indoor air arsenic and chromium concentrations, the potential for non-cancer adverse health effects for children and adults was found to be unlikely via the inhalation and ingestion routes. For cancer, the child and adult cumulative lifetime excess cancer risks were approximately one case per 10,000 and two cases per 100,000
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Statement of Issues

In December 2003, a resident living in the neighborhood of the former Celotex Industrial Park site in Edgewater, Bergen County, New Jersey requested that the Agency for Toxic Substances and Disease Control (ATSDR) evaluate potential health concern including lymphoma and breast cancer allegedly associated with the former Celotex Industrial Park site. The petitioner stated that during redevelopment activities, dust plumes were generated that seeped into the petitioner’s apartment and the heat and ventilation system on a daily basis. The petitioner also specified foul odors emanating from the former Celotex Industrial Park site during construction activities. The petition was accepted by the ATSDR in March 2004.

In response to this request, the New Jersey Department of Health and Senior Services (NJDHSS) in cooperation with the ATSDR prepared the following public health assessment. The public health assessment identified human exposure pathways and evaluated the possible public health implications of exposures to environmental contaminants associated with the former Celotex Industrial Park site. The NJDHSS and ATSDR are aware of the fact that it took nearly seven years to complete this document. The delays is partially attributed to the long review period taken due to the presence of asbestos in the soil, several revisions to the methods developed for characterizing asbestos exposures from soil (by the regulating agencies) and competing public health priorities.

A separate health consultation, to evaluate the potential health impact of exposure to Libby asbestos and to propose appropriate actions at the former Celotex Industrial Park site, was prepared by the NJDHSS (ATSDR 2006).

Background

Site Description

The former Celotex Industrial Park site, hereinafter referred to as the “Celotex site”, is located at 1 River Road, Edgewater, Bergen County, New Jersey (see Figure 1). The 29.5 acre site is located in a mixed industrial, commercial, and residential zoned area.

The Celotex site is located in a former heavily industrialized area of Bergen County, on the west bank of the Hudson River (see Figure 2). This area has been undergoing a major revitalization with apartment and condominium complexes, shopping malls, and movie theaters. The site is bordered by the “new” River Road to the west. The Multiplex Cinemas and the Independence Harbor, a residential waterfront development of
approximately 500 units, is located to the north. The Quanta Resources Corporation site is located to the south of the site. The Quanta Resources Corporation site, the location of a former coal tar distillation operation, was added to the National Priorities List (NPL) on September 4, 2002. The area west of River Road is residential.

The Promenade, a 162-unit complex of condominiums and apartments, is located on the eastern portion of the Celotex site on the pier extending over the Hudson River (see Photograph 1). A 331-unit multi-story residential and commercial development, City Place, is located in the western portion of the Celotex site.

Site History

The Celotex Corporation operated a gypsum board manufacturing plant at the site from the 1950s to the late 1970s. The facility disposed of waste including gypsum board waste at a former on-site landfill that was adjacent to the Hudson River (see Figure 2). Records indicated that the Celotex Corporation received vermiculite (which contained naturally occurring asbestos fibers) mined from Libby, Montana during 1967 to 1969 to produce gypsum board (ATSDR 2002a). A coal tar roofing plant operated at the southern part of the Celotex site (bordering Quanta Resources Corporation) from 1896 to approximately 1974. After 1974, a metal reclaiming/refinishing plant operated at the southern part of the Celotex site. Manufacturing operations at the site ceased some time in the late 1970s (NJDEP 2000). During the 1980s, several tenants including a hazardous waste recycling company, a chemical supply company and a photographic film processor operated on site under leasing arrangements. It was sold in 1981 to Edgewater Associates (EA). Gradually EA demolished all on-site buildings and raised the grade by approximately eight to twelve feet across the entire site (NJDEP 2000). No information is available regarding the source and quality of the fill material used.

The Celotex site is currently owned by Edgewater Enterprises LLC. In the 1990's, the northeastern portion (located to the north of the landfill) of the Celotex site was redeveloped for residential use with the construction of The Promenade (see Figure 2). The gypsum landfill was capped prior to the Promenade being occupied by residents. This was necessary because the only access to the parking area for the pier community included a driveway and guard shack on the landfill. During the re-grading of the landfill prior to the building of the driveway and the guard shack excess waste material from the landfill was pushed to an area west of the landfill. The NJDEP required that the excess waste material be removed off-site to a permitted solid waste facility. Excavation began on October 28, 2001 and ceased on November 28, 2001. All stockpiled material was transported off-site and the excavation backfilled by December 13, 2001. The Promenade apartments were occupied during this time period. Air sampling conducted at four locations along the perimeter of the landfill did not monitor or analyze for asbestos (Turner 2001). The residents of the Promenade were notified a week prior to the excavation in anticipation of potential odor problems (Heller 2001).

As part of the waterfront development construction permit for the City Place, a storm water retention basin was required for the collection of on-site surface water runoff.
(see Photograph 1). Currently, the City Place spans five square blocks along the Hudson River waterfront and contains 331 apartments and street-level retail businesses. In order to accommodate the construction of the storm water retention basin, the area to the east of the City Place was excavated and the excavated gypsum material was bulldozed west (toward River Road) onto the City Place construction site. Since the western boundary of the landfill is unknown, the dust generated during the construction of the City Place may have originated from the gypsum material. The major ground intrusive construction activities at the City Place were completed in October 2003 (Kathuria 2004).

A site visit of the gypsum landfill and adjacent areas conducted by the NJDEP in early 2001 determined that the majority of the public river walkway was constructed with insufficient underlying cap, i.e., paver blocks resting on six inches or less of dense-graded aggregate and two inches of leveling sand; some paver blocks were laid directly onto gypsum waste. This was further substantiated by field investigations conducted in December 2001 and January 2002 which revealed that portions of the remaining landfill, particularly near a sidewalk and public walkway, did not have the required cap thickness (EWMA 2002).

Demography

Based on 2000 United States Census data, the ATSDR estimates that there are approximately 37,000 individuals residing within a one mile radius of the Celotex site (see Figure 3).

Past NJDHSS/ATSDR Involvement

As part of the Public Health Assessment for the adjacent Quanta Resources Corporation site, the NJDHSS, in conjunction with the ATSDR, reviewed and analyzed environmental data collected from the Quanta site and neighboring properties, including the Celotex site. Neighboring properties had contaminant levels comparable to and sometimes higher than those detected on the Quanta site (ATSDR 2002b).

The National Asbestos Exposure Review, an ATDSR project to evaluate public health impacts at sites that processed Libby vermiculite, identified the former Celotex Corporation as one of seven New Jersey facilities that received vermiculite ore from the Libby mine. The USEPA requested that the ATSDR provide technical assistance in evaluating potential public health impacts from Libby asbestos. A separate health consultation addresses those findings (ATSDR 2006).

The NJDHSS and ATSDR also evaluated cancer incidence potentially related to the Celotex site. Using an ATSDR protocol for evaluating asbestos-related and non-asbestos-related health outcomes (ATSDR 2001), NJDHSS reviewed select disease incidence and mortality. Standardized incidence ratios (SIR), standardized mortality ratios (SMR) and corresponding 95% confidence intervals were used to compare the observed occurrence of disease or death to the expected occurrence of disease or death in the community. The results did not indicate that asbestos-related outcomes were
impacted by potential asbestos exposure from the Celotex gypsum board manufacturing
operation in Edgewater (NJDHSS 2005).

Site Visit

On April 28, 2005, staff performed a site visit of the former Celotex Industrial
Park site. Representatives from the NJDHSS, ATSDR and NJDEP were present. The
NJDEP representative stated that the developer had planned to construct seven buildings
and six of these have been constructed. At the time of the site visit, construction on
Building 7 was underway (see Photograph 2), close to the access road built on the Quanta
property.

The former gypsum landfill area was visited. The former landfill was a grass
covered area bordered by a paved walkway along the Hudson River (see Photograph 3).
Some hydrogen sulfide odors were noted when walking along the walkway; there were
markers placed on the grass to denote places where the cap needs repair (see Photographs
4 and 5). The vegetation appeared stressed in some areas. According to the NJDEP
representative, the cap thickness on the landfill was compromised in a few places
(evidenced by stressed vegetation and settlement of some pavement blocks), leading to a
foul odor (caused by off-gassing of sulfurous gases from the decomposition of the
gypsum board) in areas close to The Promenade (see Photograph 6).

It was also noted that the location of The Promenade was only a few yards away
from the former landfill. When asked about the dust levels reported by The Promenade
residents during remediation of the landfill, the NJDEP representative stated that the
construction management company had used dust suppression measures (water spraying)
and that the NJDEP representative was unaware of any excessive dust issues.

During the site visit, it was apparent that the residences housed young children as
evidenced by strollers and tricycles in the balconies. Numerous residences had patio
furniture on their balconies. The visit continued next to the area where the last building
(Building 7) was being constructed close to the Quanta property line. The construction
involved ground intrusive work, i.e., it was observed that dust was blowing.

As of 2006, all construction activities at the site (i.e., “City Place”) have been
completed (R. Hayton, NJDEP, personal communication, 2006).

Community Health Concerns

According to the NJDEP representative, a common community concern regarding
the Celotex site involves rotten egg odors resulting from gypsum board debris coming in
contact with river water.

In October 2002, the NJDHSS received a telephone call from a former Cliffside
resident concerned with a variety of cancers (including lung) experienced by this
individual and several family members. This individual stated that during the years of its operation, the plant constantly emitted dust that had an odor comparable to rotten eggs.

Also in October 2002, the NJDHSS, received a telephone call from an individual on behalf of her spouse. In the mid 1980s, the spouse was employed as a security guard and was stationed in a “telephone booth-type” structure located outdoors near the entrance to the Celotex site. The spouse, who was in his mid 50’s and never smoked, was reportedly diagnosed in August 2001 with “asbestos pleural disease which may develop into mesothelioma.”

Environmental Contamination

Environmental Guideline Comparison

Typically, an evaluation of site-related environmental contamination consists of a two tiered approach: 1) a screening analysis; and 2) a more in-depth analysis to determine the public health implications of site-specific exposures (ATSDR 2005). First, maximum concentrations of detected substances are compared to media-specific environmental guideline comparison values (CV). If substance concentrations exceed the environmental guideline CV, these substances, referred to as contaminants of concern (COC), are selected for further evaluation. The subsequent evaluation is conducted by comparing estimated exposure doses, derived from site-specific exposure conditions, to dose-based health guideline CVs.

Since environmental and health guideline CVs are the same for air (ATSDR 2005), contaminant levels in air were compared directly with health guideline CVs.

Contaminants in Ambient Air

**Ambient Dust and Metals:** The on-site sub-surface soil at the Celotex site is contaminated with volatile organic contaminants (VOCs), polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), petroleum hydrocarbons, metals (arsenic, lead, beryllium, cadmium, copper, mercury, selenium, thallium and zinc) and cyanide (EWMA 2000; Enviro-Sciences 1997). Although “source materials” and “hot spot” areas were remediated, post-excavation soil sampling results indicated that significant contamination still remains on-site. For example, post-removal arsenic concentration detected in the soil at one location (PEC79-4) was 7,300 milligrams of arsenic per kilogram of soil (mg/kg). The estimated quantity of contaminated material excavated from these hot spots areas was 450 cubic yards (EWMA 2000).

During ground intrusive activities associated with the construction of Buildings 1 through 6, perimeter air monitoring (see Figure 4 for location of the perimeter air monitoring stations) was performed for organic vapors, oxygen, combustible gases and vapors and airborne total suspended particulates (TSP) (Kathuria 2004). The activities included building knock-down, building/trailer demolition, stone removal/spreading.
debris/foliage removal, ramp construction/cleanup, trench excavation/backfilling, roadway capping, digging/pad installation and catch basin installation. Ambient TSP concentrations during demolition/construction were monitored continuously using a MiniRAM TSP monitor or equivalent. Due to close proximity of residences (i.e., The Promenade) to the construction areas, air sampling data from two adjacent stations (D and E) were used to estimate the ambient contaminant concentration that the residents may have been exposed to. The results of air sampling for the two stations are summarized in Table 1. The air concentrations are expressed as milligrams of TSP per cubic meter of air (mg/m$^3$). The table also presents the appropriate upper confidence limit$^1$ (UCL) of the arithmetic mean of ambient dust concentrations using ProUCL.$^X$.

Air samples collected from the construction area boundary were also analyzed for arsenic, cadmium, chromium and lead. As indicated earlier, air sampling results from two adjacent stations (D and E) were used to estimate the ambient contaminant concentration that the residents may have been exposed to. Metals detected in the ambient air were arsenic and chromium; all cadmium and lead data were at or below the detection limit (DL) (Kathuria 2004). For lead, there was one sample detected above the DL. ProUCL.$^X$ notes that the half the DL method (with non-detects replaced by DL/2) does not perform well even when the percentage of non-detects is only 5%-10%. It is strongly suggested to avoid the use of DL/2 method for estimation and hypothesis testing approaches used in various environmental applications. ProUCL.$^X$ suggests that when most (e.g., > %95) of the observations for a contaminant lie below the DLs or reporting limits (RLs), the sample median or the sample mode (rather than the sample average which cannot be computed accurately) may be used to estimate the average exposure for the contaminant and area under consideration. However, when the majority of the data are non-detects, the median and the mode will also be a non-detect. The uncertainty associated with such estimates will be high. Therefore only the detected concentrations of arsenic and chromium in air were subjected to ProUCL.$^X$ analyses$^2$. The UCLs of the arithmetic mean of arsenic and chromium detected in the ambient air were also estimated and presented in Table 2 using ProUCL.$^X$. Using the UCL of arithmetic mean of metal and TSP concentrations in the ambient air, metal concentrations in ambient dust was calculated (see Table 2).

On March, 2007 the NJDHSS and NJDEP performed an indoor settled dust sampling event to determine if contaminants in the ambient air were impacting the Palisades Child Care Center (NJHSS 2007). The results revealed the presence of contaminants at low levels. Risk assessment calculations conducted on analytical results for metals indicated no increased exposure risks.

**Asbestos.** On April 4, 2000 representatives of the USEPA and NJDEP collected

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$^1$In 1992, the USEPA recommended that the 95 percent UCL of the arithmetic mean should be used as the exposure point concentration (EPC) (USEPA 1992). Subsequently, USEPA developed a software package, ProUCL$^®$ (USEPA 2002, USEPA 2004, USEPA 2007) that uses rigorous parametric and nonparametric statistical methods on site data sets to estimate risk assessment parameters of interest, such as the EPC. For the Celotex site, the ProUCL 4.0 was used to analyze and estimate the EPCs. In the case of inadequate data (small number), an arithmetic mean is used to estimate the EPC.

$^2$This is likely to produce an overestimate of actual exposure concentration.
12 discrete grab samples along the path and a pier located to the south of the gypsum landfill (see Figure 5). The samples were composed of exposed gypsum material and surrounding soil and represented the waste material within the landfill. It should be noted that this landfill waste was bulldozed west towards River Road into an approximately 20 foot high pile, as mentioned earlier. Most of the samples were surficial grab samples; a few of the samples were collected at approximately one foot depth holes (for planting trees); one sample was also collected from an erosion channel on the slope of the stormwater retention basin. Samples were analyzed for asbestos content using polarized light microscopy (PLM) method (USEPA 600/R-93/116) and the Superfund method (USEPA 540-R-97-028) (see Table 3). The sample (#12) which contained 25% chrysotile asbestos by PLM method was collected from a pile of demolished building debris on the abandoned pier (see Figure 5). The material appeared to be crumbled pieces of transite siding board (ATSDR 2002a); as such, the asbestos content of this sample was excluded from the dataset.

The results of the PLM and Superfund method are presented in Table 3. The PLM results indicated that the asbestos concentration in sample 1 through 11 were below detection level. The transmission electron microscopy (TEM) results indicated that five of the eleven surface soil samples contained chrysotile, actinolite, and/or amosite asbestos (see Table 3). The apparent disparity may be attributed to the detection limits for PLM methods (0.25-1% asbestos). It should be noted that 1% threshold was used extensively to determine if response actions for asbestos should be undertaken. As such, since PLM results indicated the absence of asbestos, air samples (ambient or indoor) during construction activities were not analyzed for asbestos.

It should be noted that 1% asbestos in soil/debris may not be protective of human health (USEPA 2004); USEPA suggested a risk-based approach through site sampling techniques that generate fibers from the soil and bulk samples. In September 2008, USEPA recommended a framework for characterizing the potential for human exposure from asbestos contaminated outdoor soil (USEPA 2008). Activity (for example, walking, gardening, playing) based breathing zone air sampling was recommended to characterize asbestos exposures from soil/debris.

**Odor:** No odor data (i.e., hydrogen sulfide) are available for review. A multi-gas combustible gas indicator (CGI) was used during all soil-disturbing activities to monitor the possible presence of flammable gases or vapors, including hydrogen sulfide. If the CGI indicated a concentration of more than 20 parts per million of hydrogen sulfide, remediation workers had to leave the area. It was reported from air monitoring

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1. Personal communication with R. Hayton, NJDEP.
2. The PLM method measures asbestos content of soil and bulk material by comparing refractive indices of minerals; it can distinguish between asbestos and non-asbestos fibers and between different types of asbestos. The method can detect fibers with lengths greater than ~1 μm, widths greater than ~0.25 μm, and aspect ratios (length to width ratios) of greater than 3.
3. The Superfund method incorporates a gentle tumbling action to separate asbestos from the remainder of the sample; the method is expected to preserve the distribution of the sizes of the releasable asbestos structures within the sample. The respirable fraction of the dust generated from the bulk sample is collected on fibers and then analyzed by transmission electron microscopy.
safety log sheets that hydrogen sulfide never exceeded this level; the maximum level detected was 1 part per million (EWMA 2001).

**Contaminants of Concern**

Since environmental and health guideline CVs are the same for air (ATSDR 2005), contaminant levels in air were compared directly with health guideline CVs in the following section. Airborne dust, arsenic, chromium, and hydrogen sulfide (potential cause of foul odors as specified by the petitioner) and asbestos are identified as the contaminants of concern (COCs).

The toxicological summaries of arsenic, chromium, hydrogen sulfide and asbestos are given in Appendix A. The health effects associated with dust are discussed in the relevant sections of the report.

**Discussion**

The method for assessing whether a health hazard exists to a community is to determine whether there is a completed exposure pathway from a contaminant source to a receptor population and whether exposures to contamination are high enough to be of health concern (ATSDR 2005). Site-specific exposure doses can be calculated and compared with health guideline CVs.

**Assessment Methodology**

An exposure pathway is a series of steps starting with the release of a contaminant in environmental media and ending at the interface with the human body. A completed exposure pathway consists of five elements:

1. source of contamination;
2. environmental media and transport mechanisms;
3. point of exposure;
4. route of exposure; and
5. receptor population.

Generally, the ATSDR considers three exposure categories: 1) completed exposure pathways, that is, all five elements of a pathway are present; 2) potential exposure pathways, that is, one or more of the elements may not be present, but information is insufficient to eliminate or exclude the element; and 3) eliminated exposure pathways, that is, one or more of the elements is absent. Exposure pathways are used to evaluate specific ways in which people were, are, or will be exposed to environmental contamination in the past, present, and future. Completed and potential pathways may be interrupted by remedial or public health interventions that disrupt the pathway.
Information provided by the petitioner regarding exposure to environmental contaminants was taken into consideration in evaluating exposure pathways for the Celotex site. The petitioner stated that during redevelopment activities, dust plumes were generated that seeped and settled into the petitioner's apartment through the heating, ventilation and air conditioning (HVAC) system on a daily basis. The petitioner in the Promenade also reported foul odors emanating from the former Celotex site during construction activities.

It should be noted that dust wipe samples collected from a nearby building indicated the presence of contaminants at low levels. Risk assessment calculations conducted on analytical results for metals indicated no increased exposure risks (NJDHSS 2007).

**Completed Pathways**

During construction/redevelopment activities, the Promenade was the closest residential building, located approximately 50 feet from the perimeter of the construction site (see Figure 4). Therefore, the contaminant levels detected at the air monitoring stations during construction was assumed to be the same as the ambient air concentration at the Promenade. Although the residents were advised to keep their windows shut during construction, the petitioner reported a dusty indoor environment ("film of dust in the apartment"). In order to estimate the contaminant levels in the indoor environment, it was assumed that the windows were likely to be kept open infrequently and air exchange between the ambient and indoor air occurred mainly through the ventilation system.

**Inhalation of dust and metals (in dust) in the Ambient Air (past):** There was a completed exposure pathway from inhalation of dust in ambient air to children and adults living in (namely, The Promenade) and around (for example, across the "new" River road) the Celotex site. Dust generated during construction activities at the site can infiltrate homes through air intakes, leaks in ducts, through construction joints or through leaks in doors and windows.

**Inhalation of metals in dust in the Indoor Air (past):** Residents living in the neighborhood of the Celotex site may have been exposed to dust (and metals in dust) generated during construction activities for two years. Although the inhalation exposures may have occurred in a residential scenario, it was assumed that the exposures only occurred during the hours of construction.

Since the residents were advised to keep their windows shut during construction activities, ventilation for the residents of Promenade was assumed to be via the HVAC system.

**Incidental ingestion of settled dust and metals in Contaminated Dust (past):** There was a completed exposure pathway from ingestion of dust associated with indoor air (i.e., settled dust) to children and adults living in (i.e., The Promenade) and around (for example, across the "new" River road) the Celotex site. Dust generated during
construction activities can seep inside the homes and subsequently settle onto surfaces. Residents including children may have been exposed to contaminated dust through hand to mouth behavior.

The conceptual model of completed past exposure pathways for the Celotex site are shown in Figure 6.

Potential Pathways

**Inhalation of asbestos in the Ambient and Indoor Air (past):** Residents living in the neighborhood of the Celotex site may have been exposed to asbestos. Asbestos fibers were likely to have been emitted from the site during construction operation. Although the data shows the presence of asbestos, perimeter air monitoring did not include asbestos analysis. As such, ambient and indoor asbestos levels could not be estimated for use in assessing off-site residential exposures.

**Inhalation of odor producing compounds in the Ambient and Indoor Air (past):** Residents living in the neighborhood of the Celotex site may have been exposed to odor producing contaminants. No site related odor data (i.e., hydrogen sulfide) are available for evaluation. Based on the type of odor (e.g., rotten-egg) reported by the residents, it was likely due to the formation of hydrogen sulfide gas from the gypsum board present under the landfill cap.

Eliminated Pathways

**Inhalation of dust (future):** Since the ground intrusive construction activities at the Celotex site have been completed, future dust inhalation exposure pathway was eliminated.

**Incidental ingestion of settled dust (future):** Dust generated from construction site may enter the homes and subsequently settle onto surfaces. It was assumed that the settled dust in homes associated with construction activities were cleaned up through routine cleaning activities for the last 7 years (between 2003 and 2010). In addition, the NJDHSS (2007) collected wipe samples of indoor settled dust in a building in the area and found no increased risks.

As such, future dust ingestion exposure pathway was eliminated.

Public Health Implications

The public health implications from exposures via inhalation of indoor and ambient air and incidental ingestion of dust containing contaminants are discussed in the following section.
Non-Cancer Health Effects

To assess the public health implications of site-specific exposures, estimated exposure doses, derived from site-specific exposure conditions, are compared to dose-based comparison values. To assess non-cancer health effects, ATSDR has developed Minimal Risk Levels (MRLs) for contaminants that are commonly found at hazardous waste sites. An MRL is an estimate of the daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of adverse, non-cancer health effects. MRLs are developed for a route of exposure, i.e., ingestion or inhalation, over a specified time period, e.g., acute (less than 14 days); intermediate (15-364 days); and chronic (365 days or more). MRLs are based largely on toxicological studies in animals and on reports of human occupational (workplace) exposures. MRLs are usually extrapolated doses from observed effect levels in animal toxicological studies or occupational studies, and are adjusted by a series of uncertainty (or safety) factors or through the use of statistical models. In toxicological literature, observed effect levels include:

- no-observed-adverse-effect level (NOAEL); and
- lowest-observed-adverse-effect level (LOAEL).

NOAEL is the highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals. LOAEL is the lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

If site-specific exposure dose estimates exceed the health guideline CV, this dose is compared to the NOAEL or LOAEL. If the site-specific exposures are well below a NOAEL that is based on a human study, the likelihood for adverse health effects in the exposed population would be low. If, however, the NOAEL is based on an animal study, exposure doses near the NOAEL could be of concern because of uncertainty in the relative sensitivity of animals as compared to humans. In the instance where the MRL is derived from a LOAEL, the likelihood of adverse health effects increases as site-specific exposures approach a LOAEL derived from either a human or animal study. For this analysis, relevant literature values and professional judgment is used in weighing what is known and unknown, including uncertainties and data limitations.

To ensure that MRLs are sufficiently protective, the extrapolated values can be several hundred times lower than the observed effect levels in experimental studies. When MRLs for specific contaminants are unavailable, other health based comparison values such as USEPA Reference Dose (RfD) or an Reference Concentration (RfC) can be used. The RfD is an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. An RfC is an estimate of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It is generally expressed in units of mg/m$^3$ or µg/m$^3$. 

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The contaminants detected in ambient air were metals in contaminated dust. Additionally, asbestos was also detected in landfill waste that was used to raise the grade of the site. Exposure to residents over a two-year period from inhalation and ingestion of dust containing arsenic, cadmium, and chromium is evaluated in the following section.

Inhalation – Ambient Air

Residents were potentially exposed to ambient dust and contaminants associated with dust generated during construction activities for two years. Since people spend more time indoors, the exposures associated with indoor air are much higher than those for ambient air. Therefore, quantitative assessment of ambient air exposures associated with air contaminants was not conducted.

**Odor:** The foul odor reported by the petitioner and the odors noted during the site visit indicated the presence of hydrogen sulfide, formed most likely by decomposing gypsum board under anaerobic conditions. Hydrogen sulfide is a colorless, flammable, toxic gas with a characteristic rotten egg odor. The reported odor threshold for hydrogen sulfide gas varies greatly, but it is generally less than 0.01 parts per million (ppm). Some people may be able to detect the odor of hydrogen sulfide in air at concentrations as low as 0.0005 ppm (ATSDR 2004a). The average hydrogen sulfide background concentration in the air in the United States is estimated to be between 0.0001 and 0.0003 ppm (ATSDR 2004a). At low exposure levels, hydrogen sulfide will primarily cause eye and respiratory tract irritation. The health effects from prolonged exposure to low concentrations have not been well studied. However, low concentrations have been associated with neurological symptoms, including fatigue, headache, nausea, dizziness, loss of appetite, irritability, impaired memory, and altered mood states (McGavran 2001). In the absence of any physical effects, the odor of hydrogen sulfide alone can be annoying and affect well being. The following table shows health effects of various hydrogen sulfide concentrations in air reported in the literature.

**Human Health Effects at Various Hydrogen Sulfide Concentrations in Air**

<table>
<thead>
<tr>
<th>Exposure (ppm)</th>
<th>Effect/Observation</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0005-0.01</td>
<td>Odor threshold</td>
<td>ATSDR 2004a, McGavran 2001</td>
</tr>
<tr>
<td>0.01-0.6</td>
<td>Increased eye symptoms; Increases in nausea; Increased headache, mental symptoms, diseases of nervous system and sense organs</td>
<td>ATSDR 2004a</td>
</tr>
<tr>
<td>2.0</td>
<td>Bronchial constriction in asthmatic individuals</td>
<td>WHO 2003, ATSDR 2004a</td>
</tr>
<tr>
<td>5.0</td>
<td>Increased eye complaints; Mild respiratory, cardiovascular, musculoskeletal, and metabolic changes</td>
<td>WHO 2003, ATSDR 2004a</td>
</tr>
<tr>
<td>3.6-21</td>
<td>Eye irritation</td>
<td>WHO 2003</td>
</tr>
<tr>
<td>20</td>
<td>Fatigue, loss of appetite, headache, irritability, poor memory, dizziness; Irritation of mucous membranes</td>
<td>WHO 2003, ATSDR 2004a</td>
</tr>
</tbody>
</table>
The USEPA (USEPA 2010) and ATSDR (2006a) used the same 10 week animal study (Brenneman et al. 2000) to obtain the reference concentration (RfC) and intermediate-duration inhalation MRL, respectively. The NOAEL, uncertainty factor, RfC and intermediate-duration MRL are as follows:

<table>
<thead>
<tr>
<th>NOAEL (µg/m³)</th>
<th>Uncertainty Factor</th>
<th>RfC or Int-MRL * (µg/m³)</th>
<th>Principal/Supporting Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>640</td>
<td>0.46</td>
<td>300</td>
<td>RfC = 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30</td>
<td>Int-MRL = 21</td>
</tr>
</tbody>
</table>

For the Celotex site, the following site-specific assumptions were used to assess the health effects associated with exposures to hydrogen sulfide:

<table>
<thead>
<tr>
<th>No. of Days of Exposure Per Year</th>
<th>Daily Exposure time (min)</th>
<th>Exposure Duration (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>156 days (4 days per week for 9 months)</td>
<td>211 *</td>
<td>2</td>
</tr>
</tbody>
</table>

*aMean Number of Minutes Spent in Outdoor Recreation (USEPA 1997)

The exposure factor (EF) for chronic and intermediate duration inhalation exposure are calculated as follows:

For chronic case, \[
EF = \frac{156 \text{ days}}{365 \text{ days}} \times \frac{211 \text{ min}}{24 \text{ hr} \times 60 \text{ min/hr}} = 0.06
\]

For int. duration, \[
EF = \frac{4 \text{ days}}{7 \text{ days}} \times \frac{211 \text{ min}}{24 \text{ hr} \times 60 \text{ min/hr}} = 0.08
\]

Since the hydrogen sulfide level detected adjacent to the excavation location never exceeded 1 ppm (EWMA 2001) or 1,390 µg/m³, the resulting chronic and intermediate-duration exposure concentrations at the excavation location are:

Chronic concentration = 0.06 \times 1,390 \mu g/m³
= 83.4 \mu g/m³

Intermediate conc. = 0.08 \times 1,390 \mu g/m³
= 111.2 \mu g/m³

Although the estimated chronic and intermediate duration exposure concentrations (83.4 µg/m³ and 111.2 µg/m³) at the excavation location exceeded the RfC (2 µg/m³) and MRL (21 µg/m³), respectively, the concentrations were about 7.7 and 5.7 times lower than the NOAEL (640 µg/m³). In addition, the concentration at the exposure point would be much lower due to atmospheric dispersion. As such non-cancer adverse health effects
from chronic or intermediate duration inhalation exposures to hydrogen sulfide in the ambient air are not expected.

It should be noted that hydrogen sulfide may have been present at levels above the odor threshold of individuals resulting in temporary nuisance conditions during construction activities.

Inhalation – Indoor Air

Residents were potentially exposed to metal contaminated dust generated during construction activities for two years. The main source of ventilation for the residents of the Promenade was assumed to be via the HVAC system. Based on a literature review, the ratio of indoor to ambient air (also known as an Indoor/Outdoor or I/O ratio) was assumed to be 0.5 (Cal/EPA 2004; CMHC 2003; Ontario Ministry of Environment 2002; and USEPA 1997).

The site-specific assumptions that were used to assess indoor inhalation exposures for children and adults are as follows:

<table>
<thead>
<tr>
<th>Media</th>
<th>Receptor Population</th>
<th>No. of Days of Exposure Per Year</th>
<th>I/O Ratio</th>
<th>Exposure Duration (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dust</td>
<td>Children and Adults</td>
<td>260 days (5 days per week)</td>
<td>0.5</td>
<td>2</td>
</tr>
</tbody>
</table>

Contaminant levels in ambient air were multiplied by the I/O ratio to estimate indoor air levels (see Table 4). Contaminant levels in air were compared with their respective health guideline CVs.

Particulate Matter

A summary of dust or total particulate matter (TSP) and metal concentration detected in the ambient air at the Celotex site is presented in Table 1. TSP refers to a wide range of solid particles and liquid droplets such as dust, smoke, mist, fumes, or smog, found in air; they can vary in size, shape, density and electrical charge with aerodynamic diameters of 25 to 40 microns or less (USEPA 2008a). Particulate matter smaller than 10 microns (PM$_{10}$) refers to the subset of TSP comprised of particles smaller than 10 microns in diameter. PM$_{10}$ can penetrate into sensitive regions of the respiratory tract and are generated from a variety of sources including combustion, windblown dust and grinding operations. Particulate matter smaller than 2.5 microns (PM$_{2.5}$), or "fine particulates," refers to the subset of TSP and PM$_{10}$ comprised of particles with aerodynamic diameters of 2.5 microns or less. They often originate from various combustion processes, power plants, and diesel engines.

The USEPA has established the National Ambient Air Quality Standard (NAAQS) to characterize ambient air quality (USEPA 2006a). The NAAQ standards are
health-based and were designed to be protective of many sensitive populations, such as people with asthma and children. Using 0.55 as the standard ratio of PM$_{10}$ to TSP (The World Bank 1997), the maximum PM$_{10}$ in the ambient dust for the Celotex site may be estimated as 0.23 mg/m$^3$ (0.42 mg/m$^3$ * 0.55 = 0.23 mg/m$^3$). It should be noted that the PM$_{10}$ to TSP ratio is dependent largely on the characteristics of the emission source (combustion, road dust). A major fraction of the TSP generated at the Celotex site is expected to be coarse in nature, given that the emission source in this case is from mechanical/construction activities, rather than combustion. Although the 8-hour maximum PM$_{10}$ (0.23 mg/m$^3$) exceeded the 24-hour NAAQS for PM$_{10}$ (0.15 mg/m$^3$), the UCL of arithmetic mean of 8-hr PM$_{10}$ level (0.0104 mg/m$^3$) was found to be 15 times lower than 24-hour NAAQS for PM$_{10}$. Since the major fraction of TSP detected is likely to be coarse in nature and the mean dust level was less than the NAAQS for dust, health effects from dust exposures at the Celotex site are not likely to occur.

Metals detected in the dust include arsenic and chromium.

**Arsenic:** The USEPA or ATSDR have not derived inhalation MRLs for arsenic due to lack of adequate toxicological data. Results of epidemiological studies of smelter worker indicated that adverse health effects can occur as a result of chronic exposure to arsenic compounds. Targets organs are the skin, respiratory, circulatory, and reproductive systems. However, interpretation of the results is difficult due to confounding by exposures to other compounds.

The California Environmental Protection Agency (Cal/EPA) has derived a chronic inhalation reference concentration (RfC) for arsenic ($3 \times 10^{-5}$ mg/m$^3$) based on animal studies (OEHHA 2008). The RfC is based on a LOAEL of 0.033 mg/m$^3$ and an uncertainty factor of 1,000; the critical effects were reduction of fetal weight, increased incidences of intrauterine growth retardation and skeletal malformations. Since the duration adjusted UCL of mean arsenic concentration (0.000012 mg/m$^3$) in the indoor air was below the inhalation RfC for arsenic (0.00003 mg/m$^3$), non-cancer adverse health effects from inhalation exposure to arsenic are not expected (see Table 4).

**Chromium:** Chromium exists in a series of oxidation states from -2 to +6 valence; the most important stable states are 0 (elemental metal), +3 (trivalent), and +6 (hexavalent). The health effects of chromium are related to the valence state of the metal at the time of exposure. Trivalent (Cr [III]) and hexavalent (Cr [VI]) compounds are thought to be the most biologically significant species. The perimeter air sample analysis at the Celotex site did not speciate the chromium; chromium detected in the air samples was reported as total chromium. Since the speciation of chromium detected in soil/dust is a function of environmental conditions, to be conservative, the total chromium was assumed to be in the more toxic chromium (VI) form.

The ATSDR has derived a chronic inhalation MRL for hexavalent chromium (ATSDR 2008). The inhalation MRL for hexavalent chromium (i.e., 0.000005 mg/m$^3$) is based on an occupational study where workers were exposed to chromic acid; the LOAEL (i.e., 0.0005 mg/m$^3$) is based on nasal irritation, mucosal atrophy, decreased
forced vital capacity and expiratory volume. An uncertainty factor of 100 was used to calculate the MRL.

Although the calculated indoor air chromium concentration (0.00003 mg/m³) exceeded the MRL (see Table 4), the concentration was 15 times lower than LOAEL. Additionally, although the chromium speciation [i.e., the distribution of Cr(VI) and Cr(III)] in air is unknown, it is likely that the total chromium was not all in the Cr⁺⁶. Therefore, non-cancer adverse health effects from inhalation exposure to chromium are unlikely.

**Incidental Ingestion - Contaminated Dust**

As discussed earlier, residents living in the Promenade and the neighborhood of the former Celotex Industrial Park site were exposed to dust generated during construction activities. This dust may settle on indoor surfaces (i.e., floors, furniture) and can be ingested by children through hand to mouth behavior. The following section evaluates the exposures resulting from ingestion of contaminated indoor dust.

Exposures doses based on ingestion of contaminated indoor dust were calculated using the following formula:

\[
\text{Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW}
\]

where, mg/kg/day = milligrams of contaminant per kilogram of body weight per day;
C = concentration of contaminant in indoor dust (mg/kg);
IR = dust ingestion rate (kg/day);
EF = exposure factor representing the site-specific exposure scenario; and,
BW = body weight (kg)

Based on the USEPA Exposure Factors (USEPA 1997) and site-specific conditions, the following assumptions were used to calculate ingestion exposure doses for children and adults:

<table>
<thead>
<tr>
<th>Media</th>
<th>Receptor Population</th>
<th>Dust Ingestion Rate(^a) (mg/day)</th>
<th>No. of Days of Exposure Per Year</th>
<th>Body Weight (kg)</th>
<th>Exposure Duration (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dust</td>
<td>Child</td>
<td>13</td>
<td>260 days (5 days per week)</td>
<td>16</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Adult</td>
<td>6</td>
<td></td>
<td>70</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)(USEPA 2003b)

Exposure doses based on estimated mean contaminant concentrations detected in indoor dust are presented in Table 5.

**Arsenic:** Arsenic is a naturally occurring element widely distributed in the earth's crust. The MRL for arsenic is set at a level meant to protect against non-cancer health
effects, specifically dermal lesions (ATSDR 2000b; USEPA 2006c). Chronic exposure to low levels of inorganic arsenic can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Skin contact with inorganic arsenic may cause redness and swelling. Organic arsenic compounds are less toxic than inorganic arsenic compounds.

Based on the calculated UCL of arithmetic mean concentration of arsenic in indoor dust, the exposure dose for children (i.e., 0.0015 mg/kg-day) exceeded the ATSDR MRL of 0.0003 mg/kg-day (see Table 5). The adult dose (0.00017 mg/kg-day) was lower than the ATSDR MRL (see Table 5). An uncertainty factor of three and a NOAEL of 0.0008 mg/kg-day were used to calculate the MRL, which is based on lesions on the skin (ATSDR 2000b). The child exposure dose was approximately two times higher than the NOAEL. However, when compared to the LOAEL for arsenic (0.014 mg/kg-day), the exposure dose for a child was about 9 times lower than the LOAEL. As such, non-cancer adverse health effects, especially in children, from incidental ingestion of arsenic contaminated dust is not expected.

Chromium: As indicated earlier, no chromium speciation data are available; chromium detected in the air samples was reported as total chromium. As the speciation of chromium in soil/dust is a function of environmental conditions, to be conservative, the total chromium was assumed to be in the more toxic hexavalent chromium form.

The chronic oral MRL for hexavalent chromium of 0.001 mg/kg-day is based on diffuse epithelial hyperplasia of the duodenum in a group of rats and mice (ATSDR 2008). An uncertainty factor of 100 and a benchmark dose of 0.09 mg/kg-day were used to calculate the MRL. The adult exposure dose (0.00045 mg/kg-day) was lower than the MRL (see Table 5). The exposure doses for children (0.0043 mg/kg-day), based on the UCL of arithmetic mean, exceeded the health guideline CV for hexavalent chromium (see Table 5). The child exposure dose was approximately 21 times lower than the benchmark dose. In addition, the calculated chromium concentration in dust is based on detected concentrations (see footnote 2) and is unlikely to be all in the hexavalent chromium form. As such, non-cancer adverse health effects from exposures by ingestion to chromium in dust are not expected.

Cancer Health Effects

Site-specific lifetime excess cancer risk (LECR) indicates the cancer potential of contaminants and is usually expressed in terms of excess cancer cases in an exposed population. The LECR indicates the cancer potential of contaminants. LECR estimates are usually expressed in terms of excess cancer cases in an exposed population in addition to the background rate of cancer. For perspective, the lifetime risk of being diagnosed with cancer in the United States is 46 per 100 individuals for males, and 38 per 100 for females; the lifetime risk of being diagnosed with any of several common types

*An exposure due to a dose of a substance associated with a specified low incidence of risk, generally in the range of 1% to 10%, of a health effect; or the dose associated with a specified measure or change of a biological effect
of cancer ranges between 1 and 10 in 100 (SEER 2005). Typically, health guideline CVs developed for carcinogens are based on one excess cancer case per 1,000,000 individuals. ATSDR considers estimated cancer risks of less than one additional cancer case among one million persons exposed as insignificant or no increased risk (expressed exponentially as $10^{-6}$). The NJDHSS uses the following cancer risk descriptions for health assessments:

<table>
<thead>
<tr>
<th>LECR</th>
<th>Risk Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$10^{-3}$ to $\geq 10^{-1}$</td>
<td>Increase</td>
</tr>
<tr>
<td>$10^{-4}$ to $&lt;10^{-3}$</td>
<td>Low increase</td>
</tr>
<tr>
<td>$10^{-6}$ to $&lt;10^{-4}$</td>
<td>No apparent increase</td>
</tr>
<tr>
<td>$&lt; 10^{-6}$</td>
<td>No expected increase</td>
</tr>
</tbody>
</table>

The United States Department of Health and Human Services (USDHHS) cancer classes for the Celotex site contaminants are presented in Tables 6 and 7. The cancer classes are defined as follows:

1 = Known human carcinogen
2 = Reasonably anticipated to be a carcinogen
3 = Not classified

**Inhalation – Ambient Air**

None of the available studies demonstrate that hydrogen sulfide causes cancer.

**Inhalation – Indoor Air**

**Metals**

The lifetime excess cancer risk associated with metal exposures from indoor air were calculated using the following formula:

$$\text{Cancer Risk} = \text{IUR} \times C$$

where $C = \text{concentration of contaminant in air (µg/m}^3\text{)}$; and, 
IUR = inhalation unit risk (µg/m$^3$)$^{-1}$.

The IUR is defined as the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/m$^3$ in air, i.e., the equation assumes a continuous exposure, 24 hours/day for a lifetime of 70 years. To calculate the site-specific LECR, the following equation was used:
Cancer Risk = IUR * C * \( \frac{EF \times ED}{AT} \)

where \( EF \) = exposure frequency (days/year);
\( ED \) = exposure duration (years); and,
\( AT \) = averaging time (70 years).

The assumptions used to calculate site-specific inhalation LECR were the same as described previously for non-cancer health effects. The USDHHS cancer class (see Appendix A) for arsenic and chromium in the air is given in Table 6. Based on the calculated UCL of arithmetic mean concentration of arsenic and chromium, the LECRs were determined to be approximately 2 cancer cases per 1,000,000 to the exposed population, including children, for each metal (see Table 6). These are considered no apparent increase in cancer risk. As previously indicated, the LECRs presented in this report are based on site-specific assumptions that may not be representative of actual individual exposures.

Incidental Ingestion - Contaminated Dust

Estimated cancer exposure oral doses were calculated using the following formula:

\[
\text{Cancer Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW} \times \frac{ED}{AT}
\]

where \( C \) = concentration of contaminant in dust (mg/kg);
\( IR \) = soil ingestion rate (kg/day);
\( EF \) = exposure factor representing the site-specific exposure scenario;
\( ED \) = exposure duration (year);
\( BW \) = body weight (kg); and,
\( AT \) = averaging time (year).

The assumptions used to calculate site-specific exposure doses were the same as described previously for non-cancer health effects. The LECR for adults was calculated by multiplying the cancer exposure dose by the cancer slope factor (CSF). The CSF is defined as the slope of the dose-response curve obtained from animal and/or human cancer studies and is expressed as the inverse of the daily exposure dose, i.e., \((\text{mg/kg-day})^{-1}\).

The CSF associated with ingestion exposure was obtained from the NJDEP. The NJDEP derived a CSF of 0.5 (mg hexavalent chromium/kg/day)\(^{-1}\) using chronic bioassay data of male mice from the 2008 NTP study and USEPA cancer assessment guidelines (NJDEP 2009). Alternatively, the California Department of Health Services has also developed a CSF of 0.42 (mg hexavalent chromium/kg/day)\(^{-1}\) for ingestion exposure based on a 1968 study by Borneff et al. (Cal/EPA 2009). Currently, the California Environmental Protection Agency (Cal/EPA) has developed a revised (draft) CSF of 0.6
(mg hexavalent chromium/kg/day)\textsuperscript{1} for ingestion exposure based on the 2008 NTP study (Cal/EPA 2009).

Based on the calculated UCL of arithmetic mean indoor dust arsenic and chromium levels, the LECR for children and adult ranged from seven excess cancer cases per 100,000 and six excess cancer cases per 1,000,000 individuals, respectively (see Table 7), which are considered by the NJDHSS a no apparent increase in cancer risk.

Assessment of Joint Toxic Action of Chemical Mixtures

Noncancer

At the Celotex site, residents may have been exposed to metals in dust via the inhalation and ingestion pathways. Exposure to multiple chemicals with similar toxicological characteristics may increase their public health impact (ATSDR 2005, ATSDR 2004b). The severity of the impact depends on the particular chemicals being ingested, pharmacokinetics, and toxicity in children and adults.

As evaluation of joint toxic action of chemical mixture (arsenic and chromium) were conducted (see Appendix B). The additive or interactive effect of chemical mixtures on potential health hazard could not be assessed due to lack of toxicologic interaction data.

Cancer

As measures of probability, individual LECRs can be added. LECRs associated with inhalation and ingestion exposures from metals (i.e., arsenic and chromium) are as follows:

<table>
<thead>
<tr>
<th>Cumulative Metal Ingestion and Inhalation LECR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child</td>
</tr>
<tr>
<td>1.31 x10\textsuperscript{-4}</td>
</tr>
<tr>
<td>Adult</td>
</tr>
<tr>
<td>1.62 x10\textsuperscript{-5}</td>
</tr>
</tbody>
</table>

Based on calculated UCLs of arithmetic mean indoor contaminant concentrations, cumulative inhalation and ingestion exposures associated with metals to children and adults indicated a cancer risk of approximately one excess cancer cases per 10,000 and two excess cancer cases per 100,000 individuals, respectively, which are considered by the NJDHSS to pose a low to no apparent increase in cancer risk.

Child Health Considerations

The NJDHSS and ATSDR recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination in their environment. Children are at greater risk than adults from certain types of exposures to hazardous substances. Their lower body weight and higher intake rate result in a greater
dose of hazardous substance per unit of body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most important, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

Non-cancer health effects from inhalation and incidental ingestion of dust containing arsenic and chromium were evaluated for children. Adverse non-cancer health effects from inhalation and incidental ingestion exposures associated with arsenic and chromium are unlikely. Based on calculated UCL of arithmetic mean indoor contaminant concentrations, cumulative inhalation and ingestion exposures associated with children indicated a cancer risk of approximately one excess cancer cases per 10,000 individuals over a lifetime, which is considered by the NJDHSS to pose a low increase in cancer risk (see Table 6 and 7).

**Evaluation of Community Health Concerns**

The petitioner raised health concerns (breast cancer, lymphoma and severe bronchial and sinus infections) that may be related to exposures associated with site contamination. These health concerns will be discussed in relation to the known or suspected toxicologic characteristics of the chemicals in completed exposure pathways that had the potential to cause non-cancer adverse health effects. The evaluation is based on the health effects reported in ATSDR’s Toxicological Profiles for arsenic and chromium presented in Appendix A.

Arsenic: Ingested inorganic arsenic is strongly associated with lung and skin cancers and may cause other cancers in organs such as the bladder, kidneys, and liver. The USEPA classifies arsenic as a Class A known human carcinogen by the oral and inhalation routes. Epidemiologic studies of people exposed to arsenic in Taiwan indicate that exposure to arsenic is associated with skin cancer. The literature does not suggest an association between arsenic exposure and breast cancer and/or lymphoma.

Chromium: Occupational exposure to chromium (VI) compounds in a number of industries has been associated with increased risk of respiratory system cancers, primarily lung and nasal. Studies of chromate production workers, who are exposed to a variety of chromium compounds both hexavalent and trivalent, and chrome pigment industries, where exposure is mainly to chromium (VI), have consistently demonstrated an association with respiratory system cancer. A study conducted on a population who resided in a polluted area near an alloy plant that smelted chromium in the People's Republic of China found increased incidences of lung and stomach cancer. No other information was provided, and it was not possible to estimate exposure levels based on the description of the pollution process. The exposed population was probably exposed by all environmentally relevant routes (i.e., air drinking water, food, soil) (ATSDR 2000a). The literature does not suggest an association between chromium exposure and breast cancer and/or lymphoma.
Dust: Recent studies have found strong associations between airborne particulate matter and adverse human health effects, including premature death, aggravation of asthma and other respiratory diseases, and decreased lung function. Breathing fine particles can also adversely affect individuals with heart disease, emphysema, and chronic bronchitis.

Respiratory problems mentioned by the petitioner include bronchial and sinus infections. An evaluation of dust exposure scenarios during construction activities indicates health effects from dust exposures at the Celotex site were unlikely. In addition, there are numerous other common triggers of respiratory infections, so any linkage to potential site exposures would have to be considered on an individual basis.

Public Comment

This public health assessment was released for public comment on August 8th, 2010 for a 60-day period. The NJDHSS did not receive any comments during the public comment period.

Conclusions

There were completed exposure pathways via inhalation and incidental ingestion of contaminated dust generated during construction activities at the Celotex site. The exposed population included residents in the vicinity of the site and individuals accessing the site (including children). Primary contaminants of concern were dust, arsenic, chromium, hydrogen sulfide and asbestos. The ATSDR and NJDHSS reached four conclusions in this PHA.

The NJDHSS and ATSDR conclude that at present there are no site-related contaminant exposure pathways that can harm people's health. Since construction activities near the Promenade have been completed, residents are not being exposed to any contaminants. Indoor dust wipe samples collected from a building in the area indicated no increased exposure risks.

The NJDHSS and ATSDR conclude that exposures to ambient and indoor airborne dust containing metals in the past are not likely to have harmed people's health. Since indoor air contaminant concentrations were not measured, metal concentrations in the ambient air were used to estimate the indoor air contaminant levels. An evaluation of exposure to ambient dust at the Celotex site indicates that health effects are not likely to occur. The calculated indoor air chromium concentration exceeded the health-based screening value, however, based on average estimated indoor air arsenic and chromium concentrations, the potential for non-cancer adverse health effects for children and adults was found to be unlikely via the inhalation and ingestion routes. For cancer health effects, the child and adult LECRs were approximately one excess cancer cases per 10,000 and two excess cancer cases per 100,000 individuals, respectively, which is
considered by the NJDHSS to pose a low to no apparent increase in cancer risk.

The NJDHSS and ATSDR conclude that past odor exposures did not harm people’s health. An evaluation of hydrogen sulfide exposures indicates that health effects are unlikely to occur. However, hydrogen sulfide may have been present at levels above the odor threshold and created temporary nuisance conditions.

The NJDHSS and ATSDR cannot conclude whether past exposures to airborne asbestos may have harmed people’s health. Although past construction activities conducted on the landfill may have released asbestos in the ambient air, perimeter air samples collected during construction were not analyzed for asbestos and a validated method to predict the release of asbestos from soil to air is unavailable.

Recommendations

Since the landfill wastes contain asbestos, the NJDEP should take appropriate measures to ensure adequate cap thickness on the landfill. Restrictions should be placed on current and future activities that have the potential for dust emission from the landfill.

Public Health Action Plan (PHAP)

Public Health Actions Undertaken by NJDHSS and ATSDR

1. The NJDHSS and ATSDR evaluated the petitioner’s concerns regarding potential health implications from exposures to dust from construction activities at the Celotex site.

2. The NJDHSS and ATSDR reviewed available environmental data and other relevant information for the Celotex site to determine human exposure pathways and public health issues.

3. In cooperation with the ATSDR and the NJDEP, a site visit was conducted of the Celotex site.

Public Health Actions Planned by NJDHSS and ATSDR

1. Copies of this public health assessment will be provided to local health, public officials and other interested parties in the vicinity of the site. Copies will also be available at the township library and/or the Internet.

2. Former and current area residents concerned about potential exposures to site-related contamination should be examined by their personal physicians. Upon request, NJDHSS will provide educational materials on environmental exposures to arsenic, chromium and asbestos to local physicians and other medical personnel to assist them in this evaluation.
References


http://www.ene.gov.on.ca/envision/techdocs/4255e_b7.pdf


July 12, 2006 at URL: http://www.epa.gov/air/criteria.html


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New Jersey Department of Health and Senior Services
Consumer, Environmental and Occupational Health Service
P.O. Box 369
Trenton, New Jersey 08625-0369
CERTIFICATION

The public health assessment for the former Celotex Industrial Park site, Edgewater, Bergen County, New Jersey was prepared by the New Jersey Department of Health and Senior Services under a cooperative agreement with the Agency for Toxic Substances and Disease Registry. It is in accordance with approved methodology and a procedure existing at the time the health assessment was initiated. Editorial review was completed by the cooperative agreement partner.

Gregory V. Ulirsch, MS, PhD
Technical Project Officer, CAT, CAPEB, DHAC
Agency for Toxic Substances and Disease Registry

The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this public health assessment and concurs with its findings.

Alan Yarbrough
Team Leader, CAT, CAPEB, DHAC
Agency for Toxic Substances and Disease Registry
### Table 1: Concentration of dust and metals in ambient air at the Celotex site

<table>
<thead>
<tr>
<th>Contaminant</th>
<th>No of Samples</th>
<th>Concentration(^a) in Ambient Air (mg/m(^3))</th>
<th>Range</th>
<th>Mean</th>
<th>UCL(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Suspended Particles (TSP)</td>
<td>228(^c)</td>
<td>0.001 – 0.42</td>
<td>0.0068</td>
<td>0.019</td>
<td></td>
</tr>
<tr>
<td>Arsenic</td>
<td>102(^d)</td>
<td>ND – 2 x10(^{-4})</td>
<td>1.14 x10(^{-4})</td>
<td>1.02 x10(^{-4})</td>
<td></td>
</tr>
<tr>
<td>Chromium</td>
<td>100(^d)</td>
<td>ND – 7 x10(^{-4})</td>
<td>3.17 x10(^{-4})</td>
<td>2.8 x10(^{-4})</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)8-hour sample from stations D and E due to proximity to residences; \(^b\)Upper Confidence Limit; \(^c\)June 2001 to December 2003; \(^d\)June 2001 to May 2002

### Table 2: Concentration of metals in ambient dust at the Celotex site

<table>
<thead>
<tr>
<th>Metal</th>
<th>UCL(^a) of Mean Metal Concentration(^b) in Ambient Air (mg/m(^3))</th>
<th>UCL of mean Ambient Dust Level (mg/m(^3))</th>
<th>Metal concentration in Ambient Dust(^c) (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>1.02 x10(^{-4})</td>
<td>0.019</td>
<td>5,473</td>
</tr>
<tr>
<td>Chromium</td>
<td>2.8 x10(^{-4})</td>
<td></td>
<td>14,736</td>
</tr>
</tbody>
</table>

\(^a\)Upper Confidence Limit; \(^b\)8-hour sample; \(^c\)Calculated by dividing metal concentration in ambient air by mean ambient dust level
Table 3: Asbestos concentration in the dust generated from the landfill surface soil at the Celotex site (April 2000)

<table>
<thead>
<tr>
<th>Sample Location</th>
<th>Sample Description</th>
<th>PLM(^a) Results (%)</th>
<th>Superfund Method Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Exposed white material, surface level, between rip rap and top soil</td>
<td>ND</td>
<td>Number(^b) and Type of Fiber (Length) Total Asbestos (s(^2)/g dust)</td>
</tr>
<tr>
<td>2</td>
<td>Exposed white material, surface level, between freshly planted pine trees</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Exposed white material, surface level, between rip rap and path</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Exposed white material, 1 foot below path level in hole of fallen tree</td>
<td>ND</td>
<td>3 Chrysotile (0.5 – 5 μm) 3.01 x10(^{10})</td>
</tr>
<tr>
<td>5</td>
<td>Exposed white material, surface level at base of 20 foot tree</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Exposed white material, surface level, between soil cap and red clay</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Exposed white material in retention basin three feet below path level in erosion vein</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Exposed white material, surface level in unlandscaped area</td>
<td>ND</td>
<td>1 Chrysotile (0.5 – 5 μm) 7.09 x10(^{9})</td>
</tr>
<tr>
<td>9</td>
<td>Exposed white material, surface level next to unfinished path</td>
<td>ND</td>
<td>3 Chrysotile (0.5 – 5 μm) 1 Chrysotile (&gt;5 μm) 6.01 x10(^{10})</td>
</tr>
<tr>
<td>Sample Location</td>
<td>Sample Description</td>
<td>PLM&lt;sup&gt;a&lt;/sup&gt; Results (%)</td>
<td>Superfund Method Results</td>
</tr>
<tr>
<td>-----------------</td>
<td>--------------------------------------------------------------</td>
<td>------------------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>10</td>
<td>Exposed white material, surface level in unfinished path</td>
<td>ND</td>
<td>1 Chrysotile (0.5 – 5 μm) 1 Chrysotile (&gt;5 μm)</td>
</tr>
<tr>
<td>11</td>
<td>Exposed white material, approx 8 feet below level of path on pier</td>
<td>ND</td>
<td>2 Chrysotile (&gt;5 μm) 1 Actinolite (&gt;5 μm) 1 Amosite (&gt;5 μm)</td>
</tr>
<tr>
<td>12</td>
<td>Exposed white material, approx 8 feet below level of path on pier</td>
<td>25</td>
<td>1 Amosite (0.5 – 5 μm)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Polarized Light Microscopy; <sup>b</sup>Number of fiber with high magnification; <sup>c</sup>Structures consist of fiber and bundles
### Table 4: Calculated metal concentration in the indoor air of residences

<table>
<thead>
<tr>
<th>Contaminants of Concern</th>
<th>UCL(^a) of Mean Metal Concentration in Indoor air(^b) (mg/m(^3))</th>
<th>Duration Adjusted Exposure Point(^c) Concentration (mg/m(^3))</th>
<th>RFC (mg/m(^3))</th>
<th>Potential for Non-Cancer Health Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>5.1 x 10(^{-5})</td>
<td>1.21 x 10(^{-5})</td>
<td>3 x 10(^{-5d})</td>
<td>No</td>
</tr>
<tr>
<td>Chromium (IV)</td>
<td>1.4 x 10(^{-4})</td>
<td>3.33 x 10(^{-5})</td>
<td>5 x 10(^{-6})</td>
<td>Yes</td>
</tr>
</tbody>
</table>

\(^a\)Upper Confidence Limit; \(^b\)adjusted for I/O = 0.5 (see Table 2); \(^c\)exposure scenario: 5 days/week, 8 hr/day and 2 year exposure duration; \(^d\)Based on Cal EPA

### Table 5: Calculated Exposure Doses resulting from ingestion of dust

<table>
<thead>
<tr>
<th>Contaminants of Concern</th>
<th>UCL(^a) of Arithmetic Mean of Indoor Dust Concentration(^b) (mg/kg)</th>
<th>Exposure Dose (mg/kg-day)</th>
<th>Health Guideline CV (mg/kg-day)</th>
<th>Potential for Non-Cancer Health Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>2,736</td>
<td>0.0015</td>
<td>0.00017</td>
<td>Yes</td>
</tr>
<tr>
<td>Chromium (IV)</td>
<td>7,368</td>
<td>0.0043</td>
<td>0.00045</td>
<td>Yes</td>
</tr>
</tbody>
</table>

\(^a\)Upper Confidence Limit; \(^b\)calculated by applying the I/O factor of 0.5 to ambient concentration presented in Table 2; \(^c\)exposure scenario for child: 5 days/week, 13 mg/day ingestion rate, 16 kg body weight and 2 year exposure duration; \(^d\)exposure scenario for adult: 5 days/week, 6 mg/day ingestion rate, 70 kg body weight and 2 year exposure duration; \(^e\)based on food ingestion; \(^f\)based on hexavalent Chromium

### Table 6: Calculated Lifetime Excess Cancer Risk (LECR) associated with inhalation of indoor air

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Duration Adjusted Exposure Point Concentration (mg/m(^3))</th>
<th>DHHS Cancer Class(^e)</th>
<th>Inhalation Unit Risk(^d) (mg/m(^3))</th>
<th>LECR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>1.21 x 10(^{-5})</td>
<td>1</td>
<td>4.3</td>
<td>1.48 x 10(^{-6})</td>
</tr>
<tr>
<td>Chromium (IV)</td>
<td>3.33 x 10(^{-5})</td>
<td>1</td>
<td>1.2(^e)</td>
<td>1.14 x 10(^{-6})</td>
</tr>
</tbody>
</table>

\(^a\)Exposure scenario for Child: 5 days/week, 8 hours/day, and 2 year exposure duration; \(^b\)Exposure scenario: 5 days/week, 8 hours/day, and 2 year exposure duration; \(^c\)Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; \(^d\)Inhalation Unit Risk; \(^e\)Based on Hexavalent chromium
Table 7: Calculated Lifetime Excess Cancer Risk (LECR) based on indoor dust ingestion at the Celotex site

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>UCL of Arithmetic Mean of Indoor Dust concentration (mg/kg)</th>
<th>Cancer Exposure Dose (mg/kg-day)</th>
<th>DHHS Cancer Class(^c)</th>
<th>CSF(^d) (mg/kg-day)(^t)</th>
<th>LECR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Child(^a)</td>
<td>Adult(^b)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arsenic</td>
<td>2.736</td>
<td>4.54 x 10(^{-5})</td>
<td>4.79 x 10(^{-6})</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>Chromium (IV)</td>
<td>7.368</td>
<td>1.22 x 10(^{-1})</td>
<td>1.29 x 10(^{-2})</td>
<td>1</td>
<td>0.5(^e)</td>
</tr>
</tbody>
</table>

\(^a\)Exposure scenario for Child: 5 days/week, 13 mg/day ingestion rate, 16 kg body weight and 2 year exposure duration; \(^b\)Exposure scenario for adult: 5 days/week, 6 mg/day ingestion rate, 70 kg body weight and 2 year exposure duration; \(^c\)Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; \(^d\)Cancer Slope Factor; \(^t\)Limited epidemiologic studies have indicated that exposure to cadmium in food or drinking water is not carcinogenic; \(^e\)Not available; \(^\)Based on draft CSF (CEPA 2009)
Figure 2: Location of the Promenade in relation to the former landfill
Figure 3: Demographic Information of Celotex site based on 2000 U.S. Census
Figure 4: Location of perimeter air monitors in relation to the Promenade
Figure 5: Location of samples analyzed for asbestos

Figure 1
SAMPLE LOCATIONS
EDGEBREW ABESOS SITE
EDGEBREW, NJ
JUNE 2000

APPENDIX A
Figure 6: Conceptual exposure model of the Celotex site
Photograph 1: Retention basin at the former landfill

Photograph 2: Construction of Building 7
Photograph 3: Location of the Promenade and the adjacent former landfill

Photographs 4: Markers on the former landfill denoting areas that need repair
Photographs 5: Markers on the former landfill denoting areas that need repair

Photograph 6: Evidence of stressed vegetation at the former landfill
Appendix A

Toxicologic Summaries
The toxicological summaries provided in this appendix are based on ATSDR’s ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html). Health effects are summarized in this section for the contaminants of concern. The health effects described in the section are typically known to occur at levels of exposure much higher than those that occur from environmental contamination. The chance that a health effect will occur is dependent on the amount, frequency and duration of exposure, and the individual susceptibility of exposed persons.

**Arsenic** Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds.

Inorganic arsenic compounds are mainly used to preserve wood. Breathing high levels of inorganic arsenic can give you a sore throat or irritated lungs. Ingesting high levels of inorganic arsenic can result in death. Lower levels of arsenic can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of “pins and needles” in hands and feet.

Ingesting or breathing low levels of inorganic arsenic for a long time can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Skin contact with inorganic arsenic may cause redness and swelling.

Organic arsenic compounds are used as pesticides, primarily on cotton plants. Organic arsenic compounds are less toxic than inorganic arsenic compounds. Exposure to high levels of some organic arsenic compounds may cause similar effects as those caused by inorganic arsenic.

Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer. The World Health Organization (WHO), the USDHHS, and the USEPA have determined that inorganic arsenic is a human carcinogen.

**Asbestos** Asbestos is a general name applied to a group of silicate minerals consisting of thin, separable fibers in a parallel arrangement. Asbestos minerals fall into two classes, serpentine and amphibole. Serpentine asbestos has relatively long and flexible crystalline fibers; this class includes chrysotile, the predominant type of asbestos used commercially. Amphibole asbestos minerals are brittle and have a rod- or needle-like shape. Amphibole minerals regulated as asbestos by OSHA include five classes: fibrous tremolite, actinolite, anthophyllite, crocidolite, and amosite.

Asbestos fibers do not have any detectable odor or taste. They do not dissolve in water or evaporate and are resistant to heat, fire, and chemical and biological degradation.

The vermiculite mined at Libby contains amphibole asbestos, with a characteristic composition including tremolite, actinolite, richterite, and winchite; this material will be referred to as Libby asbestos.
Breathing any type of asbestos increases the risk of the following health effects:

_Malignant mesothelioma_—Cancer of the lining of the lung (pleura) and other internal organs. This cancer can spread to tissues surrounding the lungs or other organs. The great majority of mesothelioma cases are attributable to asbestos exposure.

_Lung cancer_—Cancer of the lung tissue, also known as bronchogenic carcinoma. The exact mechanism relating asbestos exposure with lung cancer is not completely understood. The combination of tobacco smoking and asbestos exposure greatly increases the risk of developing lung cancer.

_Noncancer effects_—these include asbestosis, scarring, and reduced lung function caused by asbestos fibers lodged in the lung; pleural plaques, localized or diffuse areas of thickening of the pleura (lining of the lung); pleural thickening, extensive thickening of the pleura which may restrict breathing; pleural calcification, calcium deposition on pleural areas thickened from chronic inflammation and scarring; and pleural effusions, fluid buildup in the pleural space between the lungs and the chest cavity. Ingestion of asbestos causes little or no risk of non-cancer effects.

ATSDR considers the inhalation route of exposure to be the most significant in the current evaluation of sites that received Libby vermiculite. Exposure scenarios that are protective of the inhalation route of exposure should be protective of dermal and oral exposures.

The scientific community generally accepts the correlations of asbestos toxicity with fiber length as well as fiber mineralogy. Fiber length may play an important role in clearance and mineralogy may affect both biopersistence and surface chemistry. ATSDR, responding to concerns about asbestos fiber toxicity from the World Trade Center disaster, held an expert panel meeting to review fiber size and its role in fiber toxicity in December, 2002. The panel concluded that fiber length plays an important role in toxicity. Fibers with lengths <5 μm are essentially non-toxic in terms of association with mesothelioma or lung cancer promotion. However, fibers <5 μm in length may play a role in asbestosis when exposure duration is long and fiber concentrations are high. More information is needed to definitively reach this conclusion.

_Chromium_ Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms: chromium(0), chromium(III), and chromium(VI). No taste or odor is associated with chromium compounds. The metal chromium, which is the chromium(0) form, is used for making steel. Chromium(VI) and chromium(III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

Chromium enters the air, water, and soil mostly in the chromium(III) and chromium(VI) forms. In air, chromium compounds are present mostly as fine dust particles which eventually settle over land and water. Chromium can strongly attach to soil and only a small amount can dissolve in water and move deeper in the soil to underground water. Fish do not accumulate much chromium from water.
Breathing high levels of chromium(VI) can cause nasal irritation, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum. Ingesting large amounts of chromium(VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. Skin contact with certain chromium(VI) compounds can cause skin ulcers. Allergic reactions consisting of severe redness and swelling of the skin have been noted.

Several studies have shown that chromium(VI) compounds can increase the risk of lung cancer. Animal studies have also shown an increased risk of cancer. The WHO has determined that chromium(VI) is a human carcinogen. The US DHHS has determined that certain chromium(VI) compounds are known to cause cancer in humans. The USEPA has determined that chromium(VI) in air is a human carcinogen.

It is unknown whether exposure to chromium will result in birth defects or other developmental effects in people. Birth defects have been observed in animals exposed to chromium(VI). It is likely that health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults.

**Hydrogen sulfide** Hydrogen sulfide (H₂S) occurs naturally in crude petroleum, natural gas, volcanic gases, and hot springs. It can also result from bacterial breakdown of organic matter. It is also produced by human and animal wastes. Bacteria found in mouth and gastrointestinal tract produce hydrogen sulfide from bacteria decomposing materials that contain vegetable or animal proteins. Hydrogen sulfide can also result from industrial activities, such as food processing, coke ovens, kraft paper mills, tanneries, and petroleum refineries. Hydrogen sulfide is a flammable, colorless gas with a characteristic odor of rotten eggs. People can smell it at low levels.

Exposure to low concentrations of hydrogen sulfide may cause irritation to the eyes, nose, or throat. It may also cause difficulty in breathing for some asthmatics. Brief exposures to high concentrations of hydrogen sulfide (greater than 500 ppm) can cause a loss of consciousness and possibly death. No health effects have been found in humans exposed to typical environmental concentrations of hydrogen sulfide (0.00011–0.00033 ppm).

There are no reports of people poisoned by ingesting hydrogen sulfide. Pigs that ate feed containing hydrogen sulfide experienced diarrhea for a few days and lost weight after about 105 days. Little information is available on dermal effects of hydrogen sulfide exposures.

Hydrogen sulfide has not been shown to cause cancer in humans, and its possible ability to cause cancer in animals has not been studied thoroughly. The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have not classified hydrogen sulfide for carcinogenicity.
Appendix B

Assessment of Joint Toxic Action of Chemical Mixtures
Assessment of Joint Toxic Action of Chemical Mixtures

Non-Cancer

At the Celotex site, residents were exposed to metals in dust via the inhalation and ingestion pathways. Although toxicological effects associated with site-related contamination were evaluated individually, the cumulative or synergistic effects of mixtures of contaminants may increase their public health impact. This depends upon the specific contaminant, its pharmacokinetics, and toxicity in the receptor population. Research on the toxicity of mixtures indicates that adverse health effects are unlikely when the mixture components are present at levels well below their individual toxicological thresholds (ATSDR 2005).

To evaluate the risk for non-cancer adverse health effects of chemical mixtures, a hazard index (HI) for the contaminants was calculated (ATSDR 2005). The hazard index is defined as the sum of the hazard quotients (i.e., estimated exposure dose of a contaminants divided by applicable health guideline CV). If the HI is less than 1.0, it is highly unlikely that significant additive or toxic interaction would occur, so no further evaluation is necessary. If the HI is greater than 1.0, then further evaluation is necessary. For the Celotex site, based on the calculated UCL of arithmetic mean concentration of contaminants in the indoor air, the HI calculated for children and adult (7.06) for inhalation exposure was greater than 1 (see Table B1). The HI calculated for children (9.3) for ingestion exposure was greater than 1 (see Table B2); HI calculated for adults (1.0) for ingestion exposure was less than 1 (see Table B1).

For chemical mixtures with an HI greater than 1, the estimated doses of the individual chemicals are compared with their NOAELs or comparable values. If the dose of one or more of the individual chemicals is within one order of magnitude of its respective NOAEL, then potential exists for additive or interactive effects. The ratio dose/NOAEL for the contaminants were calculated (see Table B1 and B2). The exposure dose/NOAEL for the inhalation pathway is less than 0.1; adverse health effects associated with chemical mixtures in children and adult through this pathway is unlikely. Since the exposure dose/NOAEL for arsenic for the ingestion pathway is greater than 0.1, additive or interactive effects of arsenic and chromium in children is possible. Since the potential exists for additive or interactive effects of chemical mixtures from exposures to dust in children, an in-depth mixtures evaluation is required using ATSDR's Guidance Manual for the Assessment of Joint Action of Chemical Mixtures (2004b).

The flow chart in Figure B1 gives an overview of the steps involved in the decision process for the exposure-based assessment of the potential non-cancer impact of joint toxic action (ATSDR 2004). The toxicological profiles dealing with the mixture of chemicals detected in the indoor dust are unavailable, therefore, a component approach is employed (Step 2, Figure B1, Appendix B). Since the hazard quotients associated with arsenic and chromium are greater than 0.1, they are selected as components of concern (Step 3, Figure B1, Appendix B).

Physiologically-based pharmacokinetic/pharmacodynamic (PBPK/PD) model is unavailable for the mixture (Step 4, Figure F1). The critical health effects of the components of concern are as follows (Step 5, Figure F1):
<table>
<thead>
<tr>
<th>Arsenic</th>
<th>Chromium (IV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermal lesions</td>
<td>Hematological</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Hepatic</td>
</tr>
<tr>
<td>Hematological</td>
<td>Renal</td>
</tr>
<tr>
<td>Renal</td>
<td>Neurological</td>
</tr>
<tr>
<td>Neurological</td>
<td>Testicular</td>
</tr>
<tr>
<td>Cancer</td>
<td></td>
</tr>
</tbody>
</table>

The basis for the MRL or health assessment approach is bolded and italicized; other sensitive effects are bolded; and less sensitive effects in common across two or more metals, or known to be affected synergistically by another metal in the mixture, are listed without bold or italics.

Using target organ toxicity dose (TTD) method for components with different critical effects hazard indexes were then calculated (Step 6b, Figure B1). The hazard index associated with renal health effect was less than 1; the cumulative or synergistic effects of arsenic and chromium mixtures on dermal, cardiovascular and testicular health effect could not be determined due to lack of reference dose or MRL. The magnitude of the hazard index indicates the potential neurological (5.43) and hematological (3.93) health effects in children (see Table B3, Appendix B). As such, further evaluation of interaction (Step 7b, Figure B1) is warranted.

Binary weight of evidence (BINWOE) scores relevant to the route, duration, and endpoint for the two chemicals are available (ATSDR 2004b). The predicted direction of joint toxic action is greater than additive for the effect of chromium on arsenic and less than additive for the effect of arsenic on chromium for neurological and hematological effect (see Table B4). Thus, the additive or interactive effect of chemical mixtures on potential health hazard could not be estimated for neurological and hematological effects. The impact of interaction on potential health hazard is summarized as follows:

<table>
<thead>
<tr>
<th>Health Effect</th>
<th>Impact of interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological</td>
<td>Indeterminate</td>
</tr>
<tr>
<td>Hematological</td>
<td>Indeterminate</td>
</tr>
</tbody>
</table>
Cancer

The flow chart in Figure B2 gives an overview of the steps involved in the decision process for the exposure-based assessment of the potential cancer impact of joint toxic action (ATSDR 2004b). The cancer risk estimate for the inhalation and ingestion of metals are presented in Table 6 and 7. Since the estimated risks are greater or equal to 1 in one million for at least two of the individual component (Step 5, Figure B2, Appendix B), additional cancer health hazard are likely due to additivity or interaction. However, the qualitative weight of evidence (WOE) scoring protocol could not be used due to lack of an interaction profile.
### Table B1: Multiple Chemical Exposure Analysis for Child and Adult: Inhalation of Indoor Air

<table>
<thead>
<tr>
<th>Contaminant</th>
<th>Duration adjusted EPC(^a) (mg/m(^3))</th>
<th>Health Guideline CV (mg/m(^3))</th>
<th>Hazard Quotient</th>
<th>Hazard Index</th>
<th>NOAEL (mg/m(^3))</th>
<th>Dose/NOAEL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indoor Air Inhalation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arsenic</td>
<td>1.21 x 10(^{-5})</td>
<td>3 x 10(^{-5})</td>
<td>0.4</td>
<td>7.06</td>
<td>0.033</td>
<td>0.00037</td>
</tr>
<tr>
<td>Chromium (VI)</td>
<td>3.33 x 10(^{-5})</td>
<td>5 x 10(^{-6})</td>
<td>6.66</td>
<td></td>
<td>0.0005(^b)</td>
<td>0.067</td>
</tr>
</tbody>
</table>

\(^a\)Exposure Point Concentration (see Table 4); \(^b\)LOAEL was used for hexavalent chromium

### Table B2: Multiple Chemical Exposure Analysis for Child and Adult: Ingestion of Settled Dust

<table>
<thead>
<tr>
<th>Contaminant</th>
<th>UCL of Arithmetic Mean Concentration (mg/kg-day)</th>
<th>Health Guideline CV (mg/kg-day)</th>
<th>Hazard Quotient</th>
<th>Hazard Index</th>
<th>NOAEL (mg/kg-day)</th>
<th>Dose/NOAEL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Child</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arsenic</td>
<td>0.0015</td>
<td>0.0003</td>
<td>5</td>
<td>9.3</td>
<td>0.0008</td>
<td>1.875</td>
</tr>
<tr>
<td>Chromium (VI)</td>
<td>0.0043</td>
<td>0.001</td>
<td>4.3</td>
<td>0.09</td>
<td></td>
<td>0.047</td>
</tr>
<tr>
<td><strong>Adult</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arsenic</td>
<td>0.00017</td>
<td>0.0003</td>
<td>0.55</td>
<td>1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chromium (VI)</td>
<td>0.00045</td>
<td>0.001</td>
<td>0.45</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table B3: Target Organ Toxicity Dose modification of H1 Analysis: Components with different critical effects

<table>
<thead>
<tr>
<th>Contaminant</th>
<th>Exposure Dose (mg/kg/day)</th>
<th>Neurological</th>
<th>Dermal</th>
<th>Renal</th>
<th>Cardiovascular</th>
<th>Hematological</th>
<th>Testicular</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Child</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arsenic</td>
<td>0.0013</td>
<td>5.00</td>
<td>1.88</td>
<td>0.02</td>
<td>5.00</td>
<td>2.50</td>
<td>NA(^a)</td>
</tr>
<tr>
<td>Chromium (VI)</td>
<td>0.0024</td>
<td>0.43</td>
<td>NA(^a)</td>
<td>0.43</td>
<td>NA(^a)</td>
<td>1.43</td>
<td>0.86</td>
</tr>
<tr>
<td><strong>Hazard Index =</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5.43</td>
<td>1.88</td>
<td>0.45</td>
<td>5.00</td>
<td>3.93</td>
<td>0.86</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)Not available
Table B4: Matrix of BINWOE Determinations for Simultaneous Oral Exposure to Chemicals of Concern

<table>
<thead>
<tr>
<th>Neurological Toxicity</th>
<th>On Toxicity of</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Arsenic</td>
<td>Chromium(VI)</td>
</tr>
<tr>
<td>Effect of</td>
<td></td>
<td>&lt;IIIC2ii (-0.06)</td>
</tr>
<tr>
<td>Asbestos</td>
<td>Arsenic</td>
<td>&gt;IIIC (~0.10)</td>
</tr>
<tr>
<td>Chromium(VI)</td>
<td>Chromium(VI)</td>
<td>&lt;IIIC2ii (-0.06)</td>
</tr>
</tbody>
</table>

BINWOE scheme (with numerical weights in parentheses)
DIRECTION: = additive (0); > greater than additive (+1); < less than additive (-1); ? indeterminate (0)

MECHANISTIC UNDERSTANDING:
I: direct and unambiguous mechanistic data to support direction of interaction (1.0);
II: mechanistic data on related compounds to infer mechanism(s) and likely direction (0.71);
III: mechanistic data do not clearly indicate direction of interaction (0.32).

TOXICOLOGIC SIGNIFICANCE:
A: direct demonstration of direction of interaction with toxicologically relevant endpoint (1.0);
B: toxicologic significance of interaction is inferred or has been demonstrated for related chemicals (0.71);
C: toxicologic significance of interaction is unclear (0.32).

MODIFYING FACTORS:
1: anticipated exposure duration and sequence (1.0);
2: different exposure duration or sequence (0.79);
a: in vivo data (1.0); b: in vitro data (0.79);
i: anticipated route of exposure (1.0); ii different route of exposure (0.79).
Figure B2: Exposure-Based Assessment of Joint Toxic Action of Chemical Mixtures: Cancer Effects