#### Perspectives on Magnola and HCB Emissions

#### McGill University School of Environment Undergraduate Student Project for course 401 (2001)

Description from the MSE website:

Métallurgie Magnola recently established a plant that uses asbestos tailings for magnesium extraction in Asbestos, Québec. Currently, no comparable facilities exist elsewhere, and local citizens are concerned about the presence of Magnola's plant. This report on our project attempts to answer the three main questions posed by the Comité de Citoyens:

- 1) Are Magnola's prevention measures of groundwater contamination and emission of hexachlorobenzene (HCB) from the tailings pond adequate?
- 2) What are the effects of Magnola's emission, most notably HCB, on human health?
- 3) How will the ecology be affected on the local and regional scales?

These questions are concerns for the local citizens because researchers are still investigating the effects of HCB exposure on human health. In addition, HCB is known to bioaccumulate in the food chain and animals are likely to be affected to some degree.

- Student's Client: "Comité de Citoyens du Project Magnola"
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- Note: This is an archived copy of a *student project* completed for work by undergraduates in the McGill School of Environment's Undergraduate Course "ENVR 401".
- In this course, students worked in an interdisciplinary team on a real-world research project involving problem definition, methodology development, ethical research approval, execution of the study, and dissemination of results to the research community and to the people affected.

#### This project has not been peer-reviewed, and was written 19 years ago.

Views expressed do not necessarily represent the opinions of McGill University, the School of Environment or any of its Faculty, or of the Supervisor (Dr. Case).

This archived copy was restored from the web archive at <a href="http://bwcase.tripod.com/Magnola\_homepage.htm">http://bwcase.tripod.com/Magnola\_homepage.htm</a>, which is 20 years old (but accessed Jan. 16<sup>th</sup> of 2020).

Please note as well that the *Appendix* referred to throughout the document is available as the separate document MagnolaAppendix.doc

### 1. Introduction

Métallurgie Magnola recently established a plant that uses asbestos tailings for magnesium extraction in Asbestos, Québec. Currently, no comparable facilities exist elsewhere, and local citizens are concerned about the presence of Magnola's plant. This report on our project attempts to answer the three main questions posed by the <u>Comité de Citoyens</u>:

1) Are Magnola's prevention measures of groundwater contamination and emission of hexachlorobenzene (HCB) from the tailings pond adequate?

2) What are the effects of Magnola's emission, most notably HCB, on human health?

3) How will the ecology be affected on the local and regional scales?

These questions are concerns for the local citizens because researchers are still investigating the effects of HCB exposure on human health. In addition, HCB is known to bioaccumulate in the food chain and animals are likely to be affected to some degree. Presently, the emission projection cannot be verified. Sources of information of this investigation include scientific literature and communications with representatives of the company and government agents. Several recommendations were made for both Magnola and the Comité de Citoyens based on our calculations and safety standards established by authorities. The plant appears to produce little observable effects on human health and ecology. However, the population risk level is not uniform, and a more rigorous monitoring program should be devised.

#### 2. Groundwater Contamination

#### Introduction

A common concern with landfills and waste containment facilities is the risk of groundwater contamination. Groundwater contamination due to inadequate or absent prevention measures has adversely affected hundreds of thousands of persons in the United States of America (EPA, 2000). As the tailings pond at the Magnola facility is intended to contain several hazardous organochlorine compounds, an evaluation of their planned groundwater contamination prevention measures is essential to an environmental assessment.

Magnola's current plan is to line the basin with an upper layer of bentonite in geotextiles, also called a geosynthetic clay liner (GCL) and a lower layer of high-density polyethylene (HDPE) (Magnola Pers., Comm.). The reliability of this design was assessed through an extensive review of the literature on geosynthetic clay liners and geotextiles.

#### Project Issue in a Broader Context / Literature Review:

Geosynthetics clay liners are a composite material consisting of bentonite and geosynthetics. Bentonite in GCLs used in North America is usually sodium bentonite (also called sodium montmorillonite), and the geosynthetics are either two geotextiles or a geomembrane. GCLs are usually 5-10 mm in thickness and come in rolls between 4-5 meters wide and 30-60 meters long (Daniel and Koerner, 1995). The bentonite paste contained between the two geosynthetics is of very low permeability, and is thus a suitable hydraulic barrier to water, leachate, or other liquids. Upon hydration the bentonite swells,

increasing the width of the hydraulic barrier, and the confining geosynthetic layers prevent the bentonite phase from becoming discontinuous (Daniel and Koerner, 1995).

The ability of GCLs to withstand high stresses, physical and chemical weathering, slope or swell shifting, punctures and other adversities common to waste containment facilities has been well documented (Koerner, 1997). The attribute of shear strength is conventionally tested in terms of the GCLs ability to withstand normal stress (stress in the vertical plane) and shear stress (stress in the horizontal plane). The aforementioned problems are important to the assessment of GCLs as a measure of preventing groundwater contamination because their occurrence compromises the lining's low permeability (Koerner, 1997).

## Research Question / Hypothesis

Magnola's plan will be considered acceptable if it fulfills the fourth condition recommended by the Environnement et Faune Québec in their environmental analysis report. Translated, this condition is: "That Métallurgie Magnola inc. arranges, subjacent with the silica-iron waste cell described in the impact assessment, a lining of bentonite of which the permeability is equal or lower than 10-6 cm/s" The durability of the GCL-HDPE configuration was also assessed after preliminary research revealed numerous problems, which compromised previous waste containment designs. The permeability, as well as the durability of the GCL configuration was assessed through a literature review of acceptance testing and criteria.

## Methodology

The ideal method for testing the GCL would have been to conduct standard quality tests as outlined by Koerner (1997) on samples of the product to be used. Monetary constraints, however limited us to conducting an extensive literature review for sources of information regarding the permeability and durability of GCLs.

Several sources were obtained regarding geosynthetics and waste containment applications in general, and only results from studies using similar or identical lining processes were used to evaluate Magnola's plan.

#### Analysis and Discussion

The current plan is to use an upper layer of bentonite in geotextiles and a lower layer of high-density polyethylene (HDPE) to prevent groundwater infiltration. Research into and experience with waste containment linings over the past two decades indicated that the application of such geosynthetic clay liners (GCLs) is reliable and effective barrier against groundwater contamination (Daniel and Koerner, 1995). Typical issues of concern during the planning of a landfill lining are: 1) shear strength behavior, 2) weathering effects, 3) interaction between the liner and the contained waste, 4) slope stability and 5) atmospheric uptake, this last issue being discussed in the next section (Daniel and Koerner, 1995). GCLs were found to be able to handle shear stresses of 325 kPa without deformation (Hewitt et al., 1996). While the density of the reprocessed tailings is not known, it should be noted that is highly unlikely that the load on the basin lining will come close to 325 kPa, which is equivalent to the pressure exerted by a water height of roughly 33 meters. In addition, studies by Merrill and O'Brien (1997) tested a bentonite GCL - HDPE configuration similar to the one planned by Magnola under three shear stresses of 138, 276 and 552 kPa, which were used to simulate 18, 36 and 76 meters, respectively, of solid refuse at inclinations under 18 degrees. These studies concluded that all tests indicated that the lining design would be adequate for the large landfill in question. It should also be mentioned that the safety criteria

used in this study included substantial seismic considerations, further substantiating the reliability of GCL - HDPE linings.

Because the reprocessed tailings will be submerged in water, the potential effects of hydration on the lining must also be considered. While hydration has been known to lower the shear strength of bentonitic blankets, a long term study by Trauger et al (1997) has shown bentonite GCLs to be resistant to displacement after long periods of time. This study subjected a bentonite GCL to a soaking period followed by four1000 hour or longer consolidation/shearing phases. With each phase the normal and shear stresses were increased, with a maximum normal stress of 389 kPa and a maximum shear stress of 135 kPa during the last phase. The maximum shear displacement of 0.5 mm occurred during the first 500 hours of the last phase, with no movement observed during the last 500 hours. Despite being subjected to over 7200 hours of consolidating and shearing, the specimen did not deform and was in good condition at the end of the experiment. Further tests by Seibken et al (1997) applied a normal stress of 621 kPa and a shear stress of 311 kPa on a GCL for 500 hours. After an initial displacement of 9.2 mm, subsequent movement (creep) measured an additional 0.6 mm over 500 hours. These studies indicate that bentonitic blankets may shift slightly (several millimeters) within the first hundred hours, after which the rate of displacement will fall to a near-zero level. Depending on the conditions, nonuniform initial displacements may cause problems such as gaps or sections of increased permeability between the geosynthetic mats in some GCLs (Daniel and Koerner, 1995). However, Mr. Alain Bergeron, Magnola's senior environmental officer, indicated that the GCLs would be thermally fused together to form one continuous barrier, making such problems extremely improbable. No problems of weakness at the seams were noted in the tests on thermally locked GCLs by Seibken et al (1997).

It should also be mentioned that bentonite GCLs have demonstrated a strong resilience against effects of punctuation, desiccation, freezing and chemical weathering damage as described by Shan and Daniel (1991). They found their bentonite GCL was able to withstand 140 kPa of normal pressure with no change in swelling behavior. At normal pressures beyond 140 kPa, the bentonite granules were observed to compact, which reduced the swelling of the bentonite layer after hydration. The level of reduction was proportional to the compressive stress applied. Furthermore, the permeability of bentonic blankets was also determined to be 2 \* 10-11 m/s at a low pressure of 14 kPa and 3 \*10-12 m/s at a high pressure of 138 kPa (Shan and Daniel, 1991).

The study also observed that bentonite GCLs responded well to punctures 12 and 25mm in diameter. Punctures of 75 mm were not adequately sealed, increasing the permeability by five orders of magnitude. Such punctures, however, are highly improbable if the GCL is properly applied (Daniel and Koerner, 1995). The bentonite layer filled the holes fully upon swelling, re-establishing a permeability to a comparable level before the occurrence of the puncture (Table 1) (Shan and Daniel, 1991).

Table 1: Effect of Punctures (modified from Shan and Daniel, 1991)

Diameter of Punctures Permeability (m/s) No punctures 2 x 10-11 12 mm 3 x 10-11 25 mm 5 x 10-11 75 mm >2 x 10-6

These tests demonstrated that bentonitic blankets have some self-healing capability. Shan and Daniel (1991) concluded that small holes or imperfections in bentonitic materials are probably of little

consequence so long as the bentonite is not impeded from swelling to fill the holes once the material is hydrated.

Further tests by Shan and Daniel (1991) also looked at the effects of freeze-thaw cycles on the permeability of bentonitic blankets. The permeability of the test specimen before freezing was 2 \* 10-11 m/s. After five freeze-thaw cycles, the permeability of the specimen was found to still be 2 \* 10-11 m/s. Under these conditions, freeze-thaw cycles had no effect on permeability.

#### **Conclusions and Recommendations**

Research regarding the performance of GCLs has indicated that it is robust against many conventional problems facing other waste containment designs. Considering that the water depth of the pond will be approximately one meter, and that the study by Merrill and O'Brien (1997) demonstrated that a GCL - HDPE configuration could bear loads of 552 kPa (the equivalent of roughly 56.3 meters of water) without significant deformation, it can be concluded that the occurrence of shearing and deformation problems is highly improbable. The tests done by Seibken et al (1997) and Trauger et al (1997) both indicate that GCLs are slope-stable and shear-stable over a long time. Shan and Daniels (1991) demonstrated that bentonite provides "self-healing" characteristics, making it robust against the effects of common punctures, desiccation and freeze-thaw cycles.

In conclusion, the majority of research findings on GCLs indicate it to be an effective long-term barrier to groundwater contamination. It therefore seems that Magnola's plan to line the tailings pond with a GCL and a HDPE will achieve its objective permeability less than 1 x 10-8 m/s.

#### Problems related to pre-placement hydration :

It should be noted that two considerations must be taken into account concerning the deployment of the lining layer. Bentonite liners have been known to increase their conductivity to organochlorines by an average of three orders of magnitude when permeated first with organochlorine solutions rather than with water (Shan and Daniel, 1991; Park et al, 1996; Didier and Comeaga, 1997). More importantly, the premature hydration of GCLs has been known to severely reduce their effectiveness in terms of both permeability (Didier and Comeaga, 1997) and stability (Daniel and Koerner, 1995). As such it is important to stress that GCLs must be covered before a rainfall or snow event. The reason for covering the GCL is that hydration before covering can cause shifting of the bentonite as a result of uneven swelling or whenever compressive or shear loads are encountered (Daniel and Koerner, 1995).

#### Monitoring

Magnola intends to monitor for groundwater pollution through a series of perforated pipes beneath the basin that will conduct groundwater to a location where it can be tested for hazardous compounds (Alain Bergeron, Pers. Comm.). Regular water sampling will also be conducted to measure the concentrations of selected compounds in the tailings pond. These measures appear adequate to determine the occurrence and magnitude of groundwater contamination (and thus the effectiveness of the GCL).

#### 3. Risks Of Volatilization

#### Introduction

Like all organochlorines, HCB has very low water solubility. Because the BAPE report and the environmental follow-up did not mention a covering for the tailings pond, volatilization of HCB to the atmosphere must be considered as another source of environmental pollution from the facility. The issue was addressed through the research of models that could approximate the rates of volatilization. Several models were found, from which the General Fugacity Model as described by MacKay and Paterson (1981) was chosen due to its simplicity and its general acceptance and frequent use in other studies. Studies concerning the volatilization or pathways of organochlorines were also researched to provide insight into the validity of the results obtained.

A major limitation to the accuracy of the predictions was the inability to determine the exact dimensions of the tailings pond. While parameters such as volume and general composition were obtained, others, such as width and length had to be approximated through aerial photographs and diagrams.

## Project Issue in a Broader Context / Literature Review

Organochlorines tend to have very low water solubilities, leading to high Henry's Law constants. Thus it is plausible to raise concerns over the potential for high quantities of HCB to volatilize out of the tailings pond and into the atmosphere. Because emissions from the tailings pond were not considered in the environmental impact assessments by Hatch (Magnola, Pers. Comm.) and Environnement et Faune Québec, or by the BAPE report (1998), the potential for a large contribution of HCB to the environment from this source could be cause for reassessment. Furthermore, as a potentially major source of HCB to the environment, the probability of this occurrence must be researched for reasons detailed in the section of potential ecological impacts.

# Research Question / Hypothesis

What will be the rate of volatilization of HCB from the tailings pond at the predicted input rate of 53.8 kg per year?

Due to a lack of information, our study was not able to determine the details of this issue. Rather, we sought to evaluate whether there was a need for a more rigorous study into the matter and whether the concerns regarding the emission of HCB from the tailings pond were plausible.

#### Methodology

The General Fugacity Model as described by MacKay and Paterson was used to approximate the potential volatility rate of HCB from the tailings pond. This model was used due to its simplicity and its general acceptance and frequent use by other studies. Several unknown parameters were estimated from information obtained through correspondence with Mr. Alain Bergeron, and, as it was predicted that a high rate of volatilization would result, both normal and best case scenarios were constructed. Studies regarding the modelling of HCBs pathways and volaticity in the Great Lakes were also researched to determine the validity of the results obtained.

#### Analysis and Discussion

The solubility of HCB in water is only 0.011 mg/L at 24 oC. This leads to numerous problems with the reprocessed tailings plan. The basin volume is known to be 850 000 m3, and the water in the pond will

therefore be able to dissolve 9.35 kg of HCB. Considering that Magnola's estimated output of HCB into the tailings pond is 53.8 kg per year, it would be plausible to forecast a high rate of volatilization. The general fugacity model (MacKay and Paterson, 1981), estimated a high rate of volatilization, predicting that almost all the HCB would volatize out of the tailings pond, with a negligibly small amount left dissolved in the water medium. Even when the depth of the pond used in the calculations was increased to a generous 10 meters to create a best-case scenario (the current plan intends for a water covering of only one meter), the same results were obtained. While both surprising and alarming, in light of the supersaturation of the tailings pond, the high surface area to volume ratio, and the low solubility of HCB, this result becomes more credible. Due to the inability of the water to dissolve HCB and the fixed, known, volume of the basin, for the pond to dissolve all 53.8 kg of HCB the depth of the water would have to be over 2500 meters.

Since the above results indicated that virtually all the HCB placed in the tailings pond would volatilize, the rate of volatilization was calculated by dividing the total inputs over one year by one year. This resulted in a rate of volatilization of 0.0061373 kg/h.

The validity of these alarming findings was determined by comparing with other studies of the volatility of HCBs. A study that assessed the pathways of several chlorinated benzene species, including HCB, found that over 80% of the HCB input to Lake Ontario left through volatilization (Oliver, 1984). This is supportive of the prediction that the vast majority of the HCBs placed into the tailings pond will rapidly volatilize into the atmosphere.

Mr. Alain Bergeron (Pers. Comm.) has revealed that Magnola's plan is based upon research of tailings ponds for mines in northern Quebec, where basins with water coverings 1 meter deep effectively prevented the volatilization of heavy metals. However, the free form of most heavy metals such as zinc is strongly held by soil and sediment particles and is available for biological uptake (Daniel, 1995). Furthermore, heavy metals such as cadmium, mercury, lead and zinc form soluble chloride complexes and complexes with oxide and humic particles (Daniel and Koerner, 1995). Thus it is no surprise that heavy metals are much more soluble in water than HCB or other organochlorine compounds. This lends further support to the notion that Magnola's plan to place large quantities of HCB in a water medium contains serious oversights.

#### Recommendations

In light of these alarming findings, it becomes evident that this issue requires closer scrutiny. Indeed, even Noranda's vice-president acknowledged this oversight, but there has yet to be mention of an alternative plan. One suggestion may be to ship the HCB contaminated wastes to a company, which will then dispose of them safely. The Norsk Hydro magnesium producing facility in Bécancour, Quebec, currently uses this disposal method. As our study lacked information that could have resulted in very different findings, we recommend that the approximated volatility rate and analysis be viewed as a preliminary study. We also highly recommend that the risk of high rates of atmospheric HCB emissions from the tailings pond be assessed further, with more complete data, and with other fugacity models.

# Monitoring

Magnola will sample air HCB levels at three stations north of the facility to determine atmospheric emissions. This measure is not sufficient, however, to determine the volatilization rate from the tailings pond because those measurements do not give any information on the individual contributions from the

tailings pond and the electrolysis plant. We therefore highly recommend that air HCB levels be monitored directly above and near the surface of the tailings pond continuously.

## 4. Health Effects

### Introduction

Two of the main emissions from Magnola's magnesium extraction plant that cause concerns are Hexachlorobenzene (HCB) and dioxins. Dioxin is a general term used to refer to all polychlorinated dibenzo-para-dioxin and dibenzofurans (PCDD/Fs) of which the most potent and extensively studied congener is 2,3,7,8-tetrachlorinated dibenzo-p-dioxin (2,3,7,8-TCDD).

Because the Comité de Citoyens expressed that the primary concern of residents around Magnola is the effect of HCB ) exposure on human health, it will be the emphasis of the health assessment.

HCB was widely used as a fungicide. Residents of Diyarbakir in southeastern Turkey consumed bread prepared from wheat treated with HCB between 1955 and 1961, and an outbreak of porphyria cutanea tarda (PCT) resulted. There were approximately 3000 patients who suffered in various degrees, and the victims were exposed to 50-200mg HCB/day for several months (Cam & Nigogosyan, 1963). It was the first major incident to demonstrate the danger of exposure to HCB. Since then, the general public has been and still is concerned about the exposure to HCB and other organochlorine compounds. This issue deserves attention because both HCB and dioxins accumulate in lipid-rich tissues of animals including humans (To-Figueras et al. 2000). Moreover, both HCB and dioxins are widely distributed. In particular, the long-distance transport and distribution of HCB via the troposphere is especially important and it makes HCB an evenly distributed semi-volatile organic compound around the world. Therefore, HCB emission has health and environmental implication at both local and global levels. Both HCB and dioxins are still being released into the environment as by-products of various industrial processes. In the United States over 4000 tons of HCB are generated annually as a waste byproduct mainly from the manufacture of chlorinated solvents (Michielsen et al. 1999). Waste incineration is another source of HCB and dioxins emission into the environment. In the magnesium extraction process, HCB and dioxins are the by-product of electrolysis.

However, there are some difficulties in our investigation. First, currently Magnola is not at its full operation capacity, and there is no real data to be collected and analyzed. Magnola's emission projection cannot be verified yet. In addition, there is little information on the effects of HCB exposure to certain subsets of population. Animal studies occasionally show species differences and when comparable human studies do not exist, it is somewhat difficult to interpret conflicting findings of animal studies.

#### Project Issue in a Broader Context / Literature Review

Potential Adverse Health Effects Caused by HCB and Dioxins

Except from the accidental contamination follow-up studies, there is little information available regarding the toxicity of HCB. In particular, knowledge of possible health effects due to chronic exposure to HCB is incomplete. However, HCB and dioxins share some common characteristics. Medical researchers are currently investigating the contribution of HCB exposure to the following five most observable health effects:

- 1. Porphyria cutanea tarda (PCT)
- 2. Increased risk of thyroid cancer and soft-tissue sarcoma
- 3. Liver dysfunction and liver cell tumors
- 4. Impaired immune system function
- 5. Adverse effects on reproduction

1. Porphyria cutanea tarda (PCT)

As mentioned previously, the HCB contaminated bread incident in Turkey demonstrated that an exposure to a large dose of HCB would cause PCT in humans. It should also be mentioned that researchers also believe an acute exposure to TCDD can trigger PCT in some individuals (Mukerjee 1998). More precisely, individuals who were exposed to either substance would develope type IV PCT. (Table 2).

Table 2: Types and descriptions of PCT (Modified from De Mola et al. 1996) Porphyria type Description

I Sporadic; deficiency of the enzyme is only in the liver

II Hereditary; autosomal dominant transmission; enzymatic defect in all cells

III Hereditary porphyria with normal uroporphyrinogen-3-decarboxylase (URO-D) activity in red blood cells

IV Toxic form due to an exogenous chemical agent (e.g. HCB)

This disorder primarily affected children under 16 years of age; in the Turkish bread incident only 10% of the patients were over 16 years of age (Courtney 1979). The interval between HCB ingestion and the development of the initial symptoms of PCT is approximately 6 months (Cripps et al., 1984). In a normal individual, porphyrins are incorporated into hemoproteins. HCB-induced porphyria is characterized by a deficiency of the enzyme uroporphyrinogen decarboxylase resulting in the accumulation of excessive porphyrins in the liver and increased urinary excretion of highly carboxylated porphyrins (Michielsen et al. 1999). Urine samples from patients suffering from PCT were red or brown. Patients had visible skin lesions, experienced loss of appetite and were irritable with cholic. The acute symptoms became less frequent 5 years after exposure to HCB. However, there are other symptoms such as small stature, small hands and painless arthritis, which are considered permanent effects (Cripps et al. 1984).

Animal study experiments indicate that HCB is a rodent carcinogen (van Birgelen, 1998). The organs that appear to be the major targets of this substance are thyroid, parathyroid and adrenal glands and liver. An investigation of effects of HCB exposure on human health conducted in Flix, Spain reveals that all subjects who are cancer patients have been workers in an electrochemical plant for some time. This study will be discussed in detail later in the report. The finding suggests that although International Agency for Research on Cancer (IARC) currently designates HCB as a class 2B carcinogen (i.e. possible carcinogen), its carcinogenic potency should be reviewed. Regarding dioxins, the International Agency for Research on Cancer (IARC) evaluated 2,3,7,8-TCDD and classified it as a Group 1 substance, which means it is carcinogenic to humans. Overall, it was found that the carcinogenicity of TCDD was for all cancers combined rather than for any specific site (McGregor et al. 1998). However, there was inadequate evidence in humans for the carcinogenicity of all other PCDDs. With regard to PCDFs it was concluded that there is inadequate evidence in humans for carcinogenicity.

#### 3. Liver Dysfunction

Liver is a major organ affected by the exposure to HCB. Only fat tissue has a higher HCB concentration than liver. In fact, porphyria can also be considered a liver disorder since the overproduction of porphyrins occurs in the liver of the affected individuals. Linear relationships exist between fat and blood HCB concentrations and between liver and blood concentrations. Den Tonkelaar et al. (1978) measured HCB concentrations in liver, kidney and brain tissues after 13 weeks of administration of HCB to pigs, and it was demonstrated that HCB concentration in liver is higher than that in kidney and brain. (Table 3)

Table 3: Mean HCB concentration in liver, kidney and brain of pigs that received HCB for 13 weeks (Modified from Den Tonkelaar et al. 1978)

HCB (mg/kg/day) HCB (mg/kg)

Liver Kidney Brain 0.5 3.3 2.17 1.95

5.0 42.3 17.9 19.9

Animal studies reveal that chronic exposure to HCB could induce liver cell tumors in rats, mice and hamsters. Furthermore, De Mola et al. (1996) reported that female PCT patients during pregnancy may have liver dysfunction.

Although a high dose of PCDD/F can cause liver damage in humans, effects of low level exposure are questionable (Triebig et al. 1998). While there is a notable 3-fold increase in liver cancer mortality based on a 22-year follow-up study in Japan, such a pattern was not found based on a 12-year follow-up study in Taiwan, where there is no increase in liver tumor incidence in the affected population (McGregor et al. 1998).

#### 4. Immune System Impairment

The effects of HCB on the immune system are species-dependent. For instance, HCB stimulates the immune response in the rat (Vos 1986). In contrast, HCB seems to suppress humoral and cell-mediated immunity in the mouse (van Loveren et al. 1990). Despite the documented experimental effects of HCB on animals, there is no systematic study of the effects of HCB in the human immunological system (Queiroz et al. 1997).

Queiroz et al. (1997) investigated the health status of former workers in a chemical plant. Impairment in neutrophil migration was observed in the exposed workers when compared to subjects in the general population.

Although Michielsen et al. (1999) stated that the mechanism by which HCB affects immune system is still unclear, Queiroz (1997) suggested HCB is biotransformed to sulphur-containing metabolites originating from the conjugation to glutathione (GSH). GSH protects cells and tissues from free radicals, functions as a detoxifying agent and regenerates immune cells. Presence of GSH will stop the replication of many intracellular pathogens. Koss et al. (1987) demonstrated a depletion of GSH level after administration of HCB to rats. The impairment of neutrophil chemotaxis observed may be attributed, at least in part, to the HCB-induced decrease in GSH levels (Queiroz et al., 1997).

In another study, Queiroz et al. (1998) also found significantly greater levels of immunoglobulin G (IgG) and IgM in HCB exposed workers when compared to non-exposed subjects (Table 4).

Table 4: Mean serum IgG, IgM levels in control subjects and HCB-exposed workers

Control subjects Exposed workers

Mean serum IgG level (mg/ml) 13.75 17.50

Mean serum IgM level (mg/ml) 1.37 1.80

IgG and IgM levels of HCB-exposed workers are similar to those of alcoholic cirrhosis patients. Therefore, the changes in IgG and IgM levels support the indication of hepatic dysfunction in the HCB exposed workers. In animal studies, Wistar rats that were exposed to HCB also show a dose-response relationship for increase in total serum IgM, IgG and IgA levels (Michielsen et al., 1999).

It has been demonstrated that HCB has a stimulating effect on the number of B-lymphocytes, particularly the B1 subset, which is responsible for the production of 50% of total serum IgM. The derangement of the B1 cell population has been associated with human autoimmune diseases, which may be frequently associated with dermal lesions, especially in the sun-exposed areas. Some studies demonstrate that HCB induces the formation of autoantibodies and causes dermal lesions, suggesting that an autoimmune mechanism may underlie HCB induced symptoms.

Moreover, oral exposure to HCB for 6 weeks suppressed Natural Killer cell (NK) activity in rat lungs in a dose-related manner (van Loveren et al., 1990). In rats that were exposed to 450mg HCB/kg food, the NK activity in the lung was one-third of that of normal rats. NK activity combats neoplastic and virus-infected cells. This may explain why HCB exposure is thought to contribute to an increase in cancer risk

and immune system impairment.

TCDD affects the immune system by affecting the B and T-lymphocytes. In in vitro studies, human B cells were reduced at TCDD concentrations as low as 10-14 mmol. The development of T cells in animals was also shown to be impaired by TCDD (Jung et al. 1998).

5. Adverse Effects on Reproduction

In animal studies, it was found that the male reproduction system was affected only by repeated exposure to very high doses of HCB (Den Tonkelaar et al., 1978). In contrast, female rhesus monkeys appear to be more sensitive since HCB exposure causes severe changes in ovarian structure. This seems plausible because males produce a large quantity of sperm continuously, whereas the number of oocytes in females is fixed and it is observed that the quality of the oocytes deteriorates as the females approach the end of the reproductive age. Exposure to HCB might accelerate the process of deterioration.

Certain dioxin congeners, for instance 2,3,4,7,8-PCDF, were reported to have reproductive effects such as lowering sperm count and alteration of female genital tracts. TCDD exposure has also been attributed to decreased sperm production (Mukerjee 1998).

## Controversy over Effects of HCB and Dioxin Exposure

Although some attribute effects described above to the exposure to compounds such as HCB and dioxins, others do not agree. For instance, Safe (2000) states that the sperm counts have not decreased over the last 60 years in North America. In addition, there are studies that show the human immune system actually uses chlorine, bromine and iodine to kill invading microorganisms (Gribble, 1994). In addition, some studies of populations that are chronically exposed to HCB do not indicate adverse health effects. For instance, Burns and Miller (1975) did not find evidence of adverse health effects due to the exposure to HCB.

#### Research Question / Hypothesis

The previous section explained the possible adverse effects of exposure to HCB and dioxins on human health, and there are two main questions:

1. What are the routes of exposure?

2. What are the factors that will influence the susceptibility of different subsets of the population?

#### Methodology

Previous follow-up studies on the adverse health effects of accidental exposure to HCB and dioxins and the investigation of the health status of residents in communities where ambient HCB concentration is high are particularly useful. In addition, animal studies provide some insight to the action mechanisms of these compounds.

#### Analysis and Discussion

**Routes of Exposure** 

#### Occupational Exposure:

The two main routes of occupational exposure are believed to be inhalation and dermal contact. Nevertheless, other factors and routes of exposure merit attention and further investigation because a study by Currier et al. (1980) found that the HCB level in blood of workers in chlorinated solvent production plant was strongly associated with the number of years worked in the plant, but was poorly correlated with environmental measurements such as work category or activities. The lack of correlation between HCB exposure and HCB blood concentration made the researchers suspect that the exposure was interrelated with other factors such as improper handling of foodstuff and poor personal hygiene. Queiroz et al. (1998) also noticed that the serum HCB level did not correlate with the length of exposure to the substance, which also supports the above explanation for the lack of dose-response relationship for workers who have been exposed to HCB.

### Routes of Exposure for the General Population:

For the general population, the major source of HCB and dioxins exposure is as a contaminant in the diet (Michielsen et al., 1999; Mukerjee 1998). Since both accumulate in fat tissues, it is not surprising that they are detected in food items rich in fat, especially those of animal origin (van Birgelen, 1998). However, HCB can also be found in produces such as vegetables and legumes.

The mean HCB levels in US food during 1990-1991 were less than 1 ppb. An interesting note here is that HCB concentration in UK potato peel was found to be as high as 6 ppb (Wang & Jones 1994). World Health Organization estimated in 1997 that the daily intake of HCB by adults ranges from 0.0004 to 0.003 mg HCB/kg body weight. The total daily intake of PCDD/F in terms of total toxic equivalents (TEQ) is 115pg/person in the Netherlands. This figure should be higher in the United States since their daily intake of TCDD alone (34.8pg/person) is nearly twice that of an average Dutch citizen (20pg/person). The general population exposure will also be from ambient air, contaminated drinking water and also through contact with contaminated soil.

Ballester et al. (2000) also found that although having a spouse who works in an electrochemical factory was associated with an elevated HCB concentration in serum, relatives other than spouses did not show any increase in HCB concentration in serum.

#### Populations in the Vicinity of Magnola

There are three towns in the vicinity of Magnola magnesium extraction plant: Asbestos (population 6700), Danville (1900) and Shipton (3000). Since the Asbestos mines reduced its operation in the 1980s, the surrounding communities have not been growing and they have the characteristics of an aging population. Magnola will be the only major industrial facility in that area, the other nearest industrial establishments are in Sherbrooke.

#### Susceptibility of the General Population

According to Sala et al., (1999) the exposure to HCB did not affect the general health status of a population over 14 years of age in Flix, Spain where the level of HCB in the air is 35ng/m3. This is approximately 100 to 1000 times higher than the readings obtained from other locations. The unusually high HCB level in the air attracts our attention and the possibility of a reading from a contaminated instrument, similar to the observation made by Basu et al. (2000), could not be ruled out. However, we cannot verify the validity of the air samples taken in Flix, Spain.

According to Magnola's estimates, the highest ambient air (i.e. air samples taken at the boundary of the facility) HCB level under all weather conditions will be approximately 1.4ng/m3, which is higher than the air HCB level from other locations. Again, since the sample reading from Flix, Spain is yet to be verified, the elevated air HCB level due to Magnola's emissions justifies the concerns of Comité de Citoyens.

The EPA estimates that, if a person were to inhale air containing HCB at 0.002g/m3 (2ng/m3) over their entire lifetime, that individual will theoretically have no more than a one millionth increased chance of developing cancer as a direct result of the chemical (EPA, 1993). Authors of the Environmental Report by EPA chose 2.2ng/m<sup>3</sup> as the limit. If the air HCB level is in fact 35ng/m3 in Flix, Spain, where the general population of the community has not shown observable adverse health effects, the HCB emission from Magnola at 1.4ng/m3 appears acceptable. The study in Flix, Spain by Sala et al. (1999) is particularly useful because the study site is in a rural environment and the only industrial activity present is the electrochemical factory, which has been producing volatile chlorinated solvents in the last four decades. Furthermore, the concentrations of other airborne organochlorine compounds in the air of Flix, Spain are similar to that of the reference community. This lowers the possibility of the effect of other sources of organochlorine compounds. In addition, 1800 residents (43% of the population over 14 years of age) of that community were included in that study. This large sample size also allows us to use it as a model to predict the effect of HCB in the air for the residents near Magnola project.

## Susceptibility of Individuals with High HCB and Dioxins Exposure

However, Sala et al. (1999) also pointed out that the most highly exposed subjects, males who worked at the electrochemical factory, have a significant increase of pathology related to HCB including goiter, hypothyroidism and cancer. It is important to note that according to Okamura et al. (1987), overt hypothyroidism is more common in women in a normal population. A higher than expected incidence of overt hypothyroidism in male workers would be an implication of adverse health effects due to HCB exposure. Exposure to dioxins was reported to have no effect on thyroid stimulating hormone level in recycling plant workers (Triebig et al. 1998). Grimalt et al. (1994) also found that thyroid and brain neoplasm and soft-tissue sarcomas in males in Flix, Spain are higher than expected. All males who had cancer in that study had worked in the factory, while none of the female subjects in that study worked in the electrochemical factory. This conclusion is supported by another more recent study by Herrero et al. (1999), who found the mean HCB level in serum is 72.8ng/ml for males, and 17.7ng/ml for females of Flix, Spain. This is much higher than the serum HCB level obtained from studies conducted at other locations.

#### Gender Differences

Grimalt et al. (1994) also mentioned the possibility of gender differences in the excretion of HCB derivatives. Burns and Miller (1975), found a higher HCB blood residues in males than in females. In that study there were 22 husband-wife pairs living in exposed households and males had higher residues (5.10ppb) than the females (1.70ppb). However, To-Figueras et al. (2000) believe that the most probable explanation for higher HCB concentration in blood samples from males is their employment in the electrochemical factory, which is the main determinant of the variation in HCB body burden. It is also important to point out that Ballester et al. (2000) also found that among non-factory workers, the HCB concentrations in blood were higher in women (14.3ng/ml) than in men (8.1ng/ml).

In fact, a study conducted by To-Figueras et al. (1997) indicated that pentachlorobenzenethiol (PCBT) is a good urinary marker of HCB internal dose, and by measuring the PCBT in urine samples, the researchers suggest that the HCB metabolism is probably more efficient in men than in women. This means that although males have a more efficient metabolism of HCB and eliminate more PCBT than females, due to the high exposure to the substance at work place, their HCB level in the serum is still much higher than women. Regarding the effects on reproduction, it appears that female subjects are more susceptible than males. HCB is known to be a potent oocyte toxicant in primates. The primordial germ cell appears to be sensitive to HCB levels before the onset of porphyria. Initial analysis of serum HCB and risk for spontaneous abortion by Jarrell et al. (1998) showed that they were significantly associated. However, they concluded that risk of spontaneous abortion was not restricted to patients who experienced a clearly identified high exposure event. The induction of porphyria by HCB was not associated with serum HCB levels or adverse reproductive outcome when assessed at approximately 40 years after exposure of the contamination incident. It is also important to note that currently the women in the control group living in Ankara have a higher blood HCB level than the women who lived in Diyarbakir, where the contamination incident occurred in the 1950s. Moreover, the threshold for risk of spontaneous abortion was possibly in the range of HCB levels of control groups toward the end of their reproductive lifetime. Factors such as number of pregnancies, babies and abortions may play a role in determining body burden of lipophilic chemicals such as HCB (Jarrell et al. 1998).

Another complication in reproduction noted in female porphyria patients is the observation that hormonal changes during pregnancy precipitate the disorder (De Mola et al. 1996).

#### Infants

The most vulnerable individuals in the general population are the infants, particularly breast-fed newborns. There was a high mortality (>95%) among young children, especially those under the age of 1 year, who were exposed to HCB via placenta or maternal milk in the Turkish contamination incident (Michielsen et al., 1999). In fact virtually all children between the ages of 2 months and 5 years were eliminated in the affected communities (Courtney, 1979). One of the reasons why infants are particularly vulnerable is that they have not yet fully developed detoxification mechanisms and their organs are in the process of rapid growth. Moreover the developing immune system seems to be particularly vulnerable to the immunotoxic effects of HCB and dioxins (Michielsen et al. 1999; McGregor et al. 1998). Nakashima et al. (1997) found that a great portion of the HCB accumulated in dams during pregnancy was postnatally transferred to suckling pups through milk after birth. They concluded that prenatal transfer of HCB to rat fetuses was small, and a great portion of the HCB that accumulated in dams during pregnancy was postnatally transferred to suckling pups. Cross-fostering in animal studies also showed that normal pups that were fed by dams that had been exposed to HCB died within days after suckling from the HCB-exposed female (Courtney, 1979). Intakes of dioxins by breast-fed human babies can exceed the Tolerable Daily Intake (TDI) of 10pg/kg body wt/day by almost 20-fold. (Pollitt, 1999).

The above findings show that breast-fed babies are particularly vulnerable. Czaja et al. (1997) also indicated that lipophilic compounds such as HCB accumulate in human fat tissue and are excreted in human milk through lactation. Breast milk is rich in fat and the HCB concentration in the adipose tissue is about 50 times higher than maternal blood HCB concentration (Ando et al. 1985). Similarly, dioxins are lipophilic and are highly concentrated in the milk.

Mothers who consume more fat-rich items such as seal blubber and beluga skin have a higher amount of HCB in their body. This is observed in Inuit communities in the Arctic region (Dewailly et al., 1993). Traditional Inuit diet consists of fat-rich items, and Inuit women breast milk samples were found to have higher concentrations of HCB than breast milk samples from Caucasian women in southern Quebec. To eliminate the discrepancy caused by possible genetic differences in the two populations, populations of two Inuit communities were compared. While 89% Inuit from Hudson Straight region consumed fermented seal or beluga blubber once a year or more, only 28% of women from the Ungava reported the same habit. HCB concentrations in the Hudson Straight group and in the Ungava group were 188ng/g breast milk fat and 115ng/g breast milk fat respectively.

Variations in milk fat dioxin and HCB concentrations in samples from different countries may be in part explained by the differences in diet. Milk samples from developed countries have a higher dioxin TEQ than samples from undeveloped countries (Mukerjee 1998), which may be attributed to differences in meat consumption. Adverse Effect Threshold Level There is little information on the threshold levels and dose-response relationship of HCB and on HCB-related adverse effects in humans (To-Figueras et al., 2000). Animal tests indicate that the LD50 in mice and rats is 10 000mg/kg body weight, and this means that HCB has low to moderate acute toxicity from oral exposure (Courtney 1979). With regard to dioxins, a similar lack of dose-response relationships has been reported. In fact, with regard to quantitative cancer risk assessment for dioxins, the data collected by Becher et al. (1998), do not indicate the presence of a threshold level. The EPA has determined that there is not enough information to establish a Reference Concentration (RfC) for HCB but the Reference Dose (RfD) for HCB is 0.0008 mg/kg/d. (80mg/kg/d) The RfD is a reference point to gauge the potential effects. It is the estimated daily exposure level at which there will not be appreciable risk of deleterious effects even for the most sensitive individuals in their life-time. Exceeding the RfD does not imply that an adverse health effect would necessarily occur. As the amount and frequency of exposure exceeding the RfD increase, the probability of adverse health effects also increases. A comparable measure for dioxin related health effects is the TDI, 10pg/kg body weight/day, which is derived from the No Observed Adverse Effect Level (NOAEL) in animal studies with an added safety factor. Another difficulty is that the half-life of HCB in humans is still unknown (van Birgelen, 1998; WHO 1997), although Currier et al. (1980) suggested that the half-life of HCB in chemical plant workers appeared to be about 2 years. This seems reasonable since the half-life of HCB is reported to be 2.5-3 years in rhesus monkeys (Rozman et al. 1981). As mentioned previously, the World Health Organization estimated (1997) that the daily intake of HCB by adults ranges from 0.0004 to 0.003 mg HCB/kg body weight, while the RfD is 80mg HCB/kg/day. It seems that the daily HCB intake by residents of communities surrounding Magnola will not be likely to exceed this RfD. The TDI of TCDD is 10pg/kg body wt/day. Hence, a person who weighs 50kg can tolerate a maximum of 500pg/person/day. The aforementioned TCDD intake of 20pg/person/day in the Netherlands is below the maximum tolerable daily intake. This suggests that dioxin intakes by the general population are well below the TDI.

# **Conclusions and Recommendations**

Magnola magnesium extraction plant is not at its full operation capacity yet, and it only intends to measure HCB and dioxin levels in the air three times a year. However, this may not truly reflect the distribution of these substances in the air under all weather conditions. Moreover, because the projected air HCB levels in the vicinity of Magnola is more than double that of air HCB levels from other locations around the world, the air quality monitoring program should attempt to provide data on HCB levels in the air at shorter time intervals to allow proper assessment. In addition, the monitoring program should start immediately in order to provide a frame of reference in the future. In other words, this will allow future researchers determine the changes in air quality, particularly air HCB concentration, attributable to Magnola.

It appears that the workers at the magnesium extraction plant will have a higher level of body HCB burden. It is therefore imperative for the company to create occupational hygiene education programs so that workers can avoid excessive exposure.

It is in the general population's best interest to measure the blood HCB level before the plant reaches its full capacity. This will allow future studies to have a frame of reference, and a more accurate estimate of increase in HCB concentration attributable to Magnola will be possible. In addition to blood samples, another good indicator of HCB burden in the human body is the amount of pentachlorobenzenethiol (PTBC) in the urine samples (To-Figueras et al. 1997). Such data should also be collected now for future comparisons.

Moreover the use of oral contraceptives and alcohol consumption by residents in communities around Magnola should be examined. The reasons are that increased reported cases of porphyria in women of childbearing may be related to the use of oral contraceptives (De Mola et al. 1996), and middle-aged males who indulge in alcohol consumption are the most common porphyria patients (Baxi et al. 1983). The potential adverse health effects due to exposure to HCB and dioxins are not limited to the five effects discussed in this section. Currently researchers are investigating the action mechanism of HCB on skin, lung and immune system (Michielsen et al. 1999), and as mentioned above the threshold level of adverse health effects by chronic HCB exposure is yet to be determined (To-Figueras et al. 2000). Therefore, this report should be updated later.

## 5. Ecological Effects

## Introduction

The Comité de Citoyens du Project Magnola have also asked our research group to investigate any potential impacts of Magnola's emissions of hexachlorobenzene (HCB) on ecosystems and wildlife at both local and regional scales.

To answer this question we combined the results from the company's environmental impact assessment with a chemical fate model in order to estimate long term concentrations in the air, water, soil and sediment 'compartments' of the surrounding environment. The environmental impact assessment determined how the HCB emissions disperse from the factory and onto the surrounding landscape, calculating ground air concentration values. The chemical fate model determines how this concentration of HCB will partition itself into the 'compartments' of the environment. Then using information gathered from an extensive literature review, we estimated what the resulting impact will be on the plants, invertebrates, fish, birds and mammals of both aquatic and terrestrial ecosystems.

The limitations of our research include the accuracy and the choice of the chemical fate model and the model used in Magnola's Environmental Impact Assessment. The accuracy of Magnola's reported emissions is a concern. We are also limited by the shortcomings of the literature. There are no studies of impacts on wildlife populations, only impacts on individuals. The data we used come from different ecosystems, as there is insufficient data for southern Quebec. We will also have to generalize from one species to another, and even if closely related, their responses to HCB can differ greatly.

#### Project Issue in a Broader Context / Literature Review

HCB is considered to be a "non-threshold toxicant", a substance for which there is believed to be some chance of adverse health effects at any level of exposure (CEPA, 1993). Because of this, the ministers of the Environment and of National Health and Welfare have concluded that the concentrations of HCB present in the Canadian Environment may be a danger to the environment and to human life and

health. Therefore, HCB is classified as "toxic" as interpreted under section 11 of the Canadian Environmental Protection Act, which states:

"...a substance is toxic if it is entering or may enter the environment in a quantity of concentration or under conditions

a) having or that may have an immediate or long-term harmful effect on the environment;

b) constituting or that may constitute a danger to the environment on which human life depends; or

c) constituting or that may constitute a danger in Canada to human life or health."

HCB has not been used commercially in Canada since 1972 due to concerns about adverse effects on the environment and human health (CEPA, 1993). Sedimentary core studies in the St. Lawrence River have shown a decline in HCB contamination (by a factor of 5-10) since its production was discontinued in the 70's (Carignan et al., 1994). However, it is still released into the environment as a by-product of industrial processes such as Magnola's.

## The Fate of HCB in the Environment

The atmosphere plays an important role in the transport, distribution and cycling of chemicals that are volatile and semi-volatile in nature, such as HCB (Hart et al., 1993). Long-range atmosphereic transport has been shown to be a significant source and means of redistribution of HCB throughout the world (van Pul et al., 1998). In the atmosphere, chemicals attach to aerosols and either settle back down on the soil (dry deposition) or are dissolved by atmospheric moisture and returned to the ground with precipitation (wet deposition) (Ahrens, 1994). HCB is removed from the atmosphere primarily through wet deposition to aquatic and terrestrial systems (Eisenreich and Strachan, 1992). Wet deposition, combined with the low levels of HCB currently in the atmosphere, indicates that HCB is not likely to trap significant quantities of thermal radiation from the Earth's surface, nor is it expected to reach upper layers of the atmosphere. Therefore, HCB is not likely to be associated with global warming (CEPA, 1993).

Because of its mobility in the environment and its resistance to degradation, HCB is widely distributed throughout the Canadian environment. HCB is removed from the air phase via atmospheric deposition to water and soil, or undergoes very slow photolytic degradation (half-life: t  $\frac{1}{2}$  = 80 days) (Mill and Haag, 1986). Volatilization is the major removal process for soil at the surface while slow aerobic (t  $\frac{1}{2}$  = 2.7 - 5.7 years) and anaerobic biodegradation (t  $\frac{1}{2}$  = 10.6 - 22.9 years) are the major removal processes at lower depths (Howard et al., 1991). Due to its low solubility, HCB volatilizes easily from water to air and adsorbs to suspended particles eventually collecting in bottom sediments. Once in sediment, HCB will tend to accumulate and be trapped by overlying sediments (Oliver and Nicol, 1982). Chemical and biological degradation are not considered to be important removal processes of HCB from water or sediments (Oliver and Carey, 1986).

Organisms accumulate HCB from contaminated air, water and food, although benthic organisms may accumulate HCB directly from contaminated sediment (Oliver, 1984b). As with most organochlorides, HCB has a tendency to biomagnify up the food chain. Studies have shown that high trophic level organisms in natural aquatic ecosystems accumulate HCB to levels much greater than those at lower trophic levels (Oliver, 1987; Oliver and Niimi, 1988). For example, Allen-Gil et al. (1997) have shown for four Alaskan Lakes, that HCB concentrations increase from sediment (mean = 0.17 ng/g dry weight) to

snails (mean = 0.15 ng/g wet weight) to greylings (mean = 0.64 ng/g wet weight of liver) and finally to trout (mean = 1.15 ng/g wet weight of liver). Allen-Gil et al. (1997) provide an integrated assessment of HCB distribution within freshwater systems by combining results from sediment, snails and freshwater fish. When viewed as a system, the largest contaminant sink was lake trout, the top predatory fish species. This indicates that, through bioaccumulation, the biological component of ecosystems may function as an important sink for HCB.

# HCB concentrations in various media

Due to global long-rang transport of HCB, measurements of ambient air concentrations taken in the mid 1980's in southern and central Ontario (both downtown and rural regions) and in the Canadian high arctic were all similar (0.15 ng/m3) (CEPA, 1993). The Magnola standard is set at a maximum air concentration of 2.2 ng/m3 (Commité de Citoyens, pers. comm.). Sedimentary concentrations of HCB have been found to range from below the limit of detection (1.0 ng/g dry weight) to 351 ng/g (dry weight) in the St. Lawrence River (Kaiser et al., 1990); and from below the limit of detection (0.2 ng/g dry weight) to 10 ng/g (dry weight) in the Atlantic Provinces (Leger, 1991 in CEPA, 1993). HCB has been detected in invertebrates, fish, reptiles, birds and mammals across Canada since the 1960's, when monitoring of organochlorines began. Appendix C1 summarizes many of the results of these studies.

# Toxicity

Studies have been done on both the acute and chronic toxicity of HCB for various organisms. We concentrated on chronic toxicity studies because exposure to HCB emissions from the Magnola facility is continuous, not short-term. Data on the chronic toxicity of HCB are available for species from a number of trophic levels, including freshwater alga, protozoa, invertebrates and fish. For the terrestrial environment, toxicity data are available only for birds and mammals. The literature on chronic toxicity to various biota is summarized in Appendix C2. The significance of individual toxic responses to effects at the population level is unknown (CEPA, 1993).

Fish eating mammals and predatorial birds tend to be the most susceptible to HCB in the environment as they are at the end of the food chain and get relatively larger doses of HCB than lower trophic levels (because of HCBs tendency to bioaccumulate).

# The local environment

Understanding the composition of the environment surrounding the factory is important when predicting potential impacts of HCB emissions. Magnola is situated at the tail end of the Appalachian range, characterized by mountains of low elevation (150-300 meters). The plant is located in part of a long train of serpentine outcrops beginning in Alabama and ending at the tip of the Ungava Peninsula in Northwestern Quebec. The outcrop Magnola is situated on is about 100 km long, 25 km wide and stretches from Richmond, to East Broughton in the Eastern Townships of Quebec. Historically, this area has yielded 40% of the world's asbestos production (Brooks, 1987).

The serpentine belt is characterized by highly infertile soils, which are largely composed of the minerals antigorite and chrysotile. Thus, the plants that evolved here are adapted to be tolerant of low nutrient levels and heavy metals in the soils. The principal types of forests in the region are the ash maple grove and the fir forest. The two most common species of conifer are white cedar (Thuja occidentalis) and

balsam fir (Abies Balsamea). The terrestrial fauna associated with this vegetation is primarily deer (Odocoileus virginianus), however, black bear (Ursus americanus) and moose (Alces alces) have also been observed. 1.5 km north of Magnola lies Burbank Pond. Typical mammals found around the pond include muskrats (Ondatra zibethicus), mink (Mustela vison), stoats (Mustela erminea), otters (Lontra Canadensis) and raccoons (Procyon lotor). Fifty species of common birds use the pond and the surrounding areas for nesting, summering and as a stop off during migrations. None of these birds are considered rare, vulnerable or in danger; however, only a few species of ducks, two species of heron, and the Canadian goose (Branta Canadensis) compose the majority of the bird population. We can also find a few species of amphibians and turtles in Burbank Pond. The two rivers near the plant contain a few species of fish. The main one's being trout (Salmoninae subfamily), bass (Micropterus salmoides), northern pike (Esox lucius) and yellow pike (Stizostedion vitreum).

# Research Question / Hypothesis

What will be the impacts (if any) of Magnola's Hexachlorobenzene emissions on ecosystems and wildlife at both regional and local scales?

## Methodology

To assess the potential environmental impacts of Magnola's HCB emissions, we first estimated the ambient concentrations of HCB in the soil, water and sediment of both the local and regional environments as a consequence of the factory's emissions into the air. For the regional assessment, we input Magnola's HCB emissions into a chemical fate model or Environmental Equilibrium Partitioning Model, which calculates how a chemical will partition itself between environmental 'compartments' such as air, water, water sediment and soil and the exchange fluxes between these compartments (Robson et al., 1999). For the local assessment, we obtained the results of a short-term industrial source dispersion model done for Magnola's Environmental Impact Assessment (Magnola, pers. Comm.). This model calculated ground air concentration levels of HCB in an area of 2000m2 surrounding the plant. They reported the maximum ground level air concentration of HCB over a period of one-year (1.37 ng/m3 (yr)). We input this value into the chemical fate model to find out what soil, water and water sediment concentrations will be with air concentrations at 1.37 ng/m3.

Next, we used biomagnification values for various organisms obtained from field measurements from previous studies on HCB in order to estimate HCB concentrations in the biota surrounding the factory. Finally, we referred to toxicity studies to see what effects, if any, these concentrations in the biota would have.

#### Analysis and Discussion

#### **Regional Analysis**

To assess any impacts of Magnola's emissions on a regional scale (on the order of 100,000's km2) we ran the chemical fate model, inputting HCB air emissions from the factory (21.4 kg/yr; BAPE report) added to our calculations of HCB emissions from the tailings pond (53.8 kg/yr), to get a total of 75.2 kg/yr. (See Appendix C7 for results). We didn't include the maximum ground concentration values calculated in Magnola's Environmental Impact Assessment because that concentration applies only on a local scale (100`s km2), not on a regional scale of several hundred thousand kilometers. The results show Magnola's contribution to the air, soil, water and sediment HCB concentrations in the Canadian environment as a consequence of long-range atmospheric transport. Ambient air concentrations of HCB have been shown to be similar (0.15 ng/m3) between urban Ontario, rural Ontario and the Canadian Arctic, indicating that concentrations are fairly even across Canada (except near point sources)(CEPA, 1993). The fate model's simulation indicates that Magnola's will increase this regional ambient concentration by about 2%.

As all equations in the fate model are linear, all the results are scaleable (Robson et al., 1999). Therefore, a 2% increase in ambient air HCB concentrations will cause a 2% increase in biota HCB concentrations. Most observations of animals in the wild with potentially dangerous levels of HCB in their tissues are the result of local contamination by a nearby point source. Animals that are only subject to long-range atmospheric transport of HCB tend to have undetectable or extremely low levels of HCB in their tissues. Biota concentrations that are not the result of a nearby point source, but only from long-range transport in the atmosphere and bioaccumulation in the food chain, have been obtained for remote regions of the Canadian North. One such study on mink in the Northwest Territories (Mustela vison) indicated an average HCB concentration of 0.41 ng/g wet weight (Poole et al., 1997). This is very low when compared to the tolerable daily intake of mink, which is 16,000 ng/kg body weight (Moore et al., 1996). A 2% increase in ambient concentrations will cause, through biomagnification, a 2% increase in tissue concentrations in mink. This will not lead anywhere near a daily dose of 16,000 ng/kg body weight, so the impact of Magnola's HCB emissions on a regional level appears minimal for mink.

A study on Beluga whales showed that they averaged 220 ng/g wet weight in eastern Hudson Bay and 930 ng/g wet weight in the St. Lawrence Estuary (Muir et al., 1990). A tolerable intake of beluga whales hasn't been established; however, there have been no obvious impacts on the population level. The St. Lawrence Estuary is considered rather highly contaminated with HCB as much is transported down the St. Lawrence River from the very industrial area of the Great Lakes. The level in Hudson Bay would largely be the result of long-range atmospheric transport, and if this level is increased by 2% it would lead to an average of 224.4, an increase that seems minimal when compared to the exposure beluga whales get in the St. Lawrence Estuary.

Due to HCBs high volatility, it tends to be widely and evenly dispersed when released into the air, except for the immediate vicinity of the plant. Because of this feature, wildlife on a regional level are not likely to be adversely affected by a 2% increase. Obviously, wildlife already highly exposed to nearby point sources will not benefit from any increases in ambient concentrations; however, those cases can most effectively be remedied by mitigating the most accountable point sources. Therefore, Magnola's impact on a regional scale will be minimal.

# Local Analysis

Wildlife in areas affected by point sources of HCB emissions, often have much higher concentrations of HCB in their tissues (CEPA, 1993). Such is the case for the local wildlife surrounding the Magnola facility. To investigate the effects of emissions on the environment at this local level, up to 100 km2 surrounding the factory, we again used the chemical fate model. This time, however, instead of using emissions values, we input the maximum ground level air concentration values calculated in Magnola's Environmental Impact Assessment. The results of the simulations can be found in Appendix C8, and Table 8 summarizes the resulting water, sediment, and soil concentrations from the given air concentrations. The sediment concentration used comes from the results of the fate model (see table 11 in Appendix C8), not the diagram of the results as the diagram only includes the wet weight concentration.

To characterize risks to local wildlife, we used the quotient method used by Moore et al. (1996). This method uses very conservative point estimates to determine if HCB is potentially hazardous to mink (Mustela vison) at selected locations. We will use the same method for mink around the Magnola facility, particularly any living around Burbank Pond (about 1.5 km north of Magnola). Mink are top trophic level carnivores that eat small mammals and fish; thus, they are exposed to contaminants derived from both terrestrial and aquatic food webs. They are an opportunistic species, with aquatic organisms sometimes comprising up to 100% of their diet. Mink readily bioaccumulate environmental pollutants and are extremely sensitive to organochlorides such as HCB (Bleavins et al, 1986). Due to their sensitivity to organochloride contaminants, such as HCB, they make good test subjects. If there are no effects on mink, there are likely to be no effects on other organisms.

Offspring survivability of mink exposed to HCB in their diet was affected at a dose of 160,000 ng/kg body weight/day (Moore et al., 1996). This lowest effects dose was divided by a factor of 10 to derive a no-effects dose and to account for differences between laboratory and field conditions (Moore et al., 1996). This results in a tolerable daily intake (TDI) of 16,000 ng/kg body weight/day for mink.

The quotient method simply multiplies the concentrations of HCB in air, water and fish with the intake amount of each of these mediums by mink. The amounts of HCB accumulated from each medium are added together to get a total daily intake of HCB. This calculation is summarized with the equation:

TDI = CairNIRair + CwaterNIRwater + CfishNIRfish

Where TDI is total daily intake (ng/kg body weight/day), C is concentration, and NIR is intake rate normalized to a 1kg adult female mink.

For the air concentration, we used the maximum ground level concentration calculated in Magnola's Environmental Impact Assessment. The water concentration came from the results of the fate model. The fish concentration comes from multiplying the sediment concentration from the fate model with a sediment to fish biomagnification factor. Allen-Gil et al. (1997) have shown for Lakes in Alaska, that HCB concentrations increase from sediment (mean = 0.17 ng/g dry weight) to lake trout (Salvelinus namaycush) (mean = 1.15 ng/g wet weight of liver), a biomagnification factor of 6.76. If this biomagnification factor is used for the fish in Burbank Pond then with sediment concentrations of HCB at 0.72 ng/g dry weight, the HCB concentrations in fish would be 4.8 ng/g wet weight. We used the same intake rates as used by Moore et al. (1996). From Moore et al. (1996), the tolerable daily intake (TDI) for mink is 16,000 ng/kg body weight/day, 6.65 times higher than our calculated total daily intake. Given that the values used were hyperconservative (maximum exposure concentrations and diets consisting of maximally contaminated fish), it would appear unlikely that mink living near the Magnola facility will be adversely affected by exposure to HCB.

The risk characterization calculated above is deliberately hyper-conservative, as its intent is to create an upper-bound estimate of risk. It is highly unlikely that the entire lifetime diet of an individual mink would consist of the most contaminated individuals in a fish population from the most contaminated site known in the area. This is because the highest air HCB concentration was found in a 100m2 area right next to the plant. We also assume that Lake Trout are representative of other near-shore fish species and that the biomagnification factor measured in 1997 from four Alaskan arctic lakes is representative of current conditions in Quebec (since biomagnification factors are believed to be independent of concentration levels) (Norstrom et al., 1978).

Like mink, predatorial birds are organisms of a high trophic level and are therefore likely to be exposed to high concentrations of HCB (because of it's tendency to biomagnify). Boersma et al. (1986) showed that herring gull eggs (Larus argentatus) with tissue levels of 1,500 ng/g HCB wet weight had significantly reduced embryo weights. For many bird species, reduced embryo weights are associated with lower survival of chicks (CEPA, 1993). As for mink, this effects level was divided by a factor of 10 in order to derive a no-effects level and to account for potential differences in laboratory versus field conditions. Therefore, Environment Canada estimates the no-effect level for HCB in tissues of sensitive bird species to be 150 ng/g wet weight (CEPA, 1993).

Braune et al. (1989), investigated biomagnification factors in Lake Ontario herring gulls. The biomagnification factor between fish and tissue concentration in herring gull eggs is 20 (Braune et al., 1989). As biomagnification factors are not concentration dependent, we can apply this factor to gulls eating fish in the local Asbestos area around Magnola. If we take the HCB concentration in fish (calculated above in the analysis for mink) of 4.8 ng/g wet weight, multiply this by the biomagnification factor of 20, we get a potential tissue concentration of HCB in herring gull eggs of 96 ng/g wet weight. Although this represents the upper bound value of possible tissue concentrations for herring gull eggs (using maximum concentrations), it does come fairly close to the tolerable tissue concentration of 150 ng/g. This is close enough to be of concern, not so much for herring gulls, but for other species of predatorial birds that may be more susceptible than herring gulls to HCB in the environment. For instance, HCB concentrations in peregrine falcon eggs (Falco peregrinus anatum) collected between 1980 and 1987 across Canada had a mean concentration of 279 ng/g wet weight (Peakall et al., 1990). There is no information on HCB bioconcentration factors for peregrine falcons so we cannot conclude if they will be affected by Magnola's emissions. However, if average tissue concentrations of HCB in peregrin falcons across Canada are greater than for other predatorial birds, it's likely that they tend to accumulate HCB more easily and are more susceptible to it. Furthermore, the potential for effects to peregrine falcons from exposure to HCB is considered to be a serious threat to the long-term survival of this species, given its current status as an endangered species in Canada (CEPA, 1993). Ground level air concentrations of HCB would have to be about 2.25 ng/m, or about 65% higher for sediment concentrations to be high enough to get fish so contaminated that herring gulls could have the potential for tissue concentrations of 150 ng/g wet weight.

# Limitations of analysis

The limitations of our research include the accuracy and the choice of the chemical fate model and the model used in Magnola's Environmental Impact Assessment. The accuracy of environmental models tends to be good for making general conclusions, not for specific outcomes. We chose the Fugacity-Based Environmental Equilibrium Partitioning model as our fate model as it was the most recent version available for download from Canada's Environmental Modeling Center. We believe that the choice of the Short Term Industrial Source Complex model used in Magnola's Environmental Impact Assessment to (estimate local ground level air HCB concentrations) is questionable. An article by Eschenroeder (1986) suggests using the Long Term version of the Industrial Source Complex model for modeling dispersion of HCB, not the Short Term version. This is because HCB is a persistent chemical and can last years in the environment.

The accuracy of Magnola's reported emissions is a concern, since this is only a projection of the emissions calculated in the experimental plant they operated in 1997 at Salabery-de-Valleyfied. The experimental plant was 250 times smaller than the actual plant, it only operated for seven full days and

all predicted emissions are extrapolated from this test run. Also, due to Magnola's rights of confidentiality over their industrial process and the complexity of the process, there is no way for outside officials to calculate how much HCB will be emitted. Consequently there is a large potential for error in the published emissions.

We are also limited by the shortcomings of the literature, as there are no studies of impacts on wildlife populations, only impacts on individuals. Furthermore, the data we used comes from different ecosystems, as there is insufficient data for southern Quebec. We also had to generalize from one species to another, and even if closely related, their responses to HCB can differ greatly.

#### Recommendations

Magnola's contribution to a 2% increase in ambient HCB concentrations on a regional level is likely to be minimal, since long-range atmospheric transport tends to disperse HCB fairly evenly and at low concentrations. The results of our local analysis, indicates little risk to piscivorous mammals, despite our choosing of a receptor known to by highly sensitive to HCB. However, for predatorial birds, HCB concentration in the local area may pose a risk to the most susceptable species, especially those that are already endangered.

We recommend to the Comité de Citoyens du Project Magnola that, when up-dates on Magnola's emission levels are available, they encourage Magnola to run a long-term dispersion model (instead of the short-term one used in their Environmental Impact Assessment) including hexachlorobenzene emissions from the tailings pond. Once obtaining the new concentration values, the Comité should re-run our analysis on piscivorous mammals and predatorial birds. This will provide a more accurate picture of any potential impacts on wildlife at both local and regional scales.

# 6. Glossary and Appendix<sup>1</sup>

# Glossary

2,3,7,8-TCDD - Tetrachlorinated dibenzo-p-dioxin, a dioxin congener consisting of four chlorine atoms at the 2, 3, 7, and 8 respective positions on the benzene rings.

Acute exposure - Exposure over a relatively short time period.

Aerobic - Occurring only in he presence of free uncombined molecular oxygen, either as a gas in the atmosphere or dissolved in water.

Aerosol - The suspension of very fine, generally micrometer-size, solid and liquid particles in the atmosphere.

Ambient - Referring to surrounding, external, or unconfined conditions.

Anaerobic - Occurring in environmental conditions devoid of oxygen.

Angina - Sense of suffocation or suffocating pain

<sup>&</sup>lt;sup>1</sup> The *Appendix* for this document is no longer on the website but is available as a separate document.

Aryl hydrocarbon receptor (Ah) - Cytoplasmic protein that TCDD has been shown to bind with and induce changes in the expression of specific genes and translation of their products and hence alter the biological effects.

Atherothrombosis - Formation of blood clots in arteries.

Atmosphere - The gaseous envelope surrounding Earth; a reservoir in Earth's surface system.

Autoimmune - autoimmune disorders are suspected of being caused by inflammation and destruction of tissues by the body's own antibodies

Bioaccumulate - The storage of chemicals in an organism in higher concentrations then are normally found in the environment.

Bioconcentrate - Build-up of chemical elements or substances in organisms in successively higher trophic levels.

Biomagnification factor - Used to describe the accumulation of chemicals in organisms, primarily aquatic, that live in contaminated environments.

Biota - Living organisms, such as animals, plants, fungi, etc.

B-lymphocytes - These lymphocytes produce circulating antibodies, and therefore are responsible for humoral immunity. B cells work chiefly by secreting soluble substances called antibodies into the body's fluids, or humors. (This is known as humoral immunity.)

Cell mediated immunity - See T-lymphocytes.

Chemotaxis - Movement of a cell in response to the stimulus of a gradient of chemical concentration

Chronic exposure - Exposure over a relatively long time period contaminant sink.

Cirrhosis - A condition in which liver responds to injury or death of some of its cells by producing interlacing fibrous tissues.

Degradation - The process by which a chemical is reduced to a less complex form.

Dry deposition - The deposition of material from the atmosphere onto Earth's surface in the form of solid particles. Such particles also may be "washed out" of the atmosphere by rain.

Ecosystem - The biotic community and its abiotic environment functioning as a system.

Environmental impact assessment - Identifies and predicts the impacts from development proposals on both the biophysical environment and on human health and well-being.

Exchange flux - The rate of flow from a source to a sink or receiver.

Fate model - Calculates how a chemical will partition itself between environmental `compartments`such as air, water, water sediment and soil and the exchange fluxes between these compartments.

Geosynthetics - The generic term for all synthetic materials used in geotechnical engirneering applications.

Geotextile - A permeable geosynthetic comprised solely of textiles. Current manufacturing techniques produce non-woven fabrics, knitted (nontubular) fabrics and woven fabrics.

Geomembrane - An essentially impermeable geosynthetic composed of one or more synthetic sheets Cirrhosis: A condition in which liver responds to injury or death of some of its cells by producing interlacing fibrous tissues.

Glutathione - Glutathione (GSH) is a naturally occurring protein of three amino acids - glycine, glutamate (glutamic acid), and cysteine.

Half-life - The time it takes one half of a given material (such as a radioactive isotope or a pesticide) to decay or disappear.

Henry's Law constant - A measure of the tendency for a chemical to volatilize.

Hepatic - Related to the liver

Humoral - Circulating in the bloodstream. Humoral mediated immunity requires circulating antibodies. See also B-lymphocytes.

Immunoglobulins - A group of structurally related proteins that act as antibodies. There are five classes of immunoglobulins: IgA, IgD, IgE, IgG, and IgM. The most important classes are IgG, IgA and IgM.

Intracellular - Within the cell

LD 50 - The dose of a toxic compound that causes in 50% of a group of experimental animals to which the compound is administered.

LOD - Level Of Detection.

Myocardial infarction - Heart attack caused by lack of blood flow in a coronary artery due to a blood clot.

Natural killer - A type of lymphocyte that is able to kill certain types of cancer cells.

Neoplastic - New and abnormal; a new and abnormal growth is called neoplasm

Neutrophil - White blood cells with a lifespan around twelve hours; these cells arrive at the site of an infection or injury to engulf bacterial bodies.

Organochlorides - An organic compound composed of mostly carbon, hydrogen and chlorine.

Pathogen - A microorganism (e.g. bacterium, viruse) that parasitizes a host and produces a disease

PCDD/Fs - Polychlorinated dibenzo-p-dioxins and dibenzofurans, often referred to as dioxins. These compounds consist of two benzene rings enclosing a dioxin or a furan ring. Chlorination refers to the position of chlorine atoms on the benzene rings. Poly means many and prefixes such as di (2), tri (3), tetra (4), penta (5), hexa (6), hepta (7), octa (8) indicate the numbers of chlorine atoms present. Thus Tetrachlorinated dibenzo-p-dioxin (TCDD) is an example of a PCDD congener.

Pentachlorobenzenethiol (PCBT) - A substance derived from the metabolism of HCB

Photolytic degradation - Pertaining to chemical reactions triggered by radiant energy that convert a complex compound to more simple products.

Point source (of pollution) - Easily discernible source of pollution, such as a factory.

Pollution - A substance that adversely alters he physical, chemical, or biological quality of the earth's living systems or that accumulates in the cells or tissues of living organisms in amounts that threaten their health or survival.

Population - A group of individuals of the same species living in a given area at a given time.

Porphyria - A metabolic disorder in which the patient has an excessive amount of porphyrins. This disorder can be due to hereditary factors or can be caused by exposure to exogenous chemical substances such as HCB.

Porphyrin - A pigment that forms a constituent in various proteins

ppb - Parts per billion

Sediment - fragments of rock, soil and organic material transported and deposited in bodies of water by wind, water or other natural phenomena. The term can refer to any size of particles but is often used to indicate only fragments smaller than 6 mm.

Sedimentary core - Technique used to determine past levels of environmental contamination by examining soils layed down through time.

T <sup>1</sup>/<sub>2</sub> - See Half-life.

TDI - Tolerable Daily Intake. Refers to a daily intake of TCDD that is not likely to lead to any adverse effects. Its derivation was based on rat studies at the no-observed-adverse-effects-level (NOAEL) of 1ng/kg body wt/day. A safety factor of 10 was applied to allow for differences in susceptibility between rats and humans.

TEF - Toxic Equivalency Factor. TEFs are consensus values based on available data on relative potency values on a specific compound and are protective in nature. Relative potency values express the potency of a specific compound in comparison to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) the most potent dioxin congener, with a relative potency of 1.

TEQ - A certain amount of toxic equivalents calculated from multiplying the TEF value of a compound by the concentration of a specific matrix used to determine the total dioxin activity.

T-lymphocytes - They interact directly with their targets, attacking body cells that have been affected by viruses or warped malignancy. (This is cellular immunity.)

Trophic level - Functional classification of organisms in an ecosystem according to feeding relationships from first-level autotrophs through succeeding levels of herbivores and carnivores.

Volatilization - The conversion of a substance into the gas or vapor state and its emission into the environment.

Wet deposition - The deposition on Earth's surface of solid particles and dissolved chemical compound in rain.

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