The International Scientific Commission's
Assessment of the

IMPACT of the CYANIDE SPILL at BARSKAUN, KYRGYZ REPUBLIC May 20, 1998

AUGUST 1998

MINING AND MINERAL SCIENCES LABORATORIES

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MINING AND MINERAL SCIENCES LABORATORIES
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CANMET

EXECUTIVE SUMMARY

Over the course of five and a half hours on the afternoon of May 20, 1998 some 1762 kg of NaCN (935 kg CN) entered the Barskaun River. Following the incident, a scientific commission of international experts was assembled at the request of the Deputy Prime Minister, Mr. Boris Silaev to study the impact of the cyanide spill. This commission included:

Thomas Hynes, Environmental Specialist, Natural Resources Canada, Ottawa, CANADA
John Harrison, Health Advisor, Health Canada, Ottawa, CANADA
Evgene Bonitenko, Medical Toxicologist, EMERCOM, RUSSIA
Tamara Doronina, Chemist, Moscow Chemical Institute of Chemical Materials, RUSSIA
Harry Baikowitz, Chemist, Bodycote Technitrol, Pointe-Claire, CANADA
Michael James, Medical Consultant, Medisys, Toronto, CANADA

The Commission investigated the short and long term impact of the cyanide spill on the local air, water and soil, vegetables and plants, fish, and milk. The Commission also evaluated the impact on the future of Lake Issyk-Kul following the spill. In addition, medical and transportation issues arising from the spill were also examined. In its assessment of the impact, the Commission has examined all available relevant written material as well as personal field notes taken during visits to the area, May-June, 1998.

The major conclusions reported herein are:

1. All the cyanide concentrations in air were well below safe industrial hygiene /occupational guidelines.
2. There was no possible exposure to cyanogen chloride as it could not have been generated in significant quantities.
3. No cyanide concentrations in soils reached the level at which remediation action would be warranted for human health (Canadian guideline 29 mg/kg).

4. The cyanide concentration in Barskaun River water was potentially high enough to cause serious health effects at least several hours after the spill to anyone who drank a sufficient quantity of the water.
5. There may have been damage to local (Barskaun and Tamga) crops to irrigation systems being closed up until June 2, 1998, as a result of fear of cyanide contamination.
6. No direct cyanide effects were found on plants in Tosor, Tamga, Barskaun, Chon-Dzargylchak or Al-Terek.
7. In the days following the spill, fish habitats had been repopulated indicative of low cyanide levels in the Barskaun River and the Issyk-Kul lake following the spill. This observation provides evidence to confirm the river/lake environment is safe for public use.
8. There was no reasonable cyanide exposure route to people eating fish.
9. No cyanide exposure occurred by drinking milk or eating dairy products from the region.
10. There has been no damage to Lake Issyk-Kul either in the short or long term.
11. No carcinogenic, mutagenic, teratogenic, reproductive or neurotoxological effects to people are expected.
12. Cyanide concentrations in the ditches of the villages of Barskaun and Tamga do not support cases of cyanide exposure causing human health effects. Nor is there any possible cyanide exposure route for the villages of Tosor, Chon-Dzargylchak and Al-Terek
13. Up to 16 cases of cyanide exposure could have occurred; however these cases have not been confirmed. No medical evidence has yet been supplied to support these cases as being cyanide related. Thus this committee is not able on a scientific basis to confirm that these people suffered adverse human health effects as a direct result of the cyanide spill.
14. None of the 16 potential cases are likely to experience long-term health effects in the future. Nor would cyanide exposure normally have required hospitalization for more than a few days.
15. There were no reported deaths within the first 72 hours; deaths after this time cannot be directly attributed to cyanide exposure in this situation.
16. Widespread use of the cyanide antidotes was inappropriate, and may have itself caused harmful health effects to the patients.

17. The cause of the accident appears to have been human error on the part of the truck driver. The container and packaging met all material and international standards for the transportation of cyanide.

Based on the finding presented in this report the Commission has made the following recommendations to Kumtor and the Kyrgyz Republic.

1. The Kyrgyz Ministry of Health and the Ministry of the Environment should consider adopting their own national guidelines for cyanide concentrations in air, soil, water and food and instituting a national certification program for Kyrgyz laboratories.
2. The use of sodium hypochlorite should be discouraged as a response to the treatment of cyanide spills in the environment due to the potential formation of cyanogen chloride. In practice, sodium hypochlorite usage should be restricted only to industrial applications.
3. In the event of such a spill, a first response priority is the notification of downstream water users to prevent the use of contaminated potable water; this would include monitoring, sampling (air, soil and water), analysis and reporting.
4. Kumtor's emergency response plan should be revised in light of the spill and a copy filed with the Emergency Measures Organization. There should be at least one Emergency Response Drill per annum involving Kumtor and Kyrgyz Emergency Measures personnel.
5. The Kyrgyz Ministry of Health should review its records, and provide information on the number of cases reported in the first 72 hours after the spill.
6. A joint Kumtor - Kyrgyz Ministry of Health follow-up on the patients who reported symptoms be completed and an expert committee be established to review documentation on patient symptomology in conjunction with the Kumtor clinic being instituted.
7. A reassessment of the four fatalities should be completed to determine the exact cause of death.
8. Kumtor and the Kyrgyz Ministry of Health should provide joint training to local health personnel on cyanide diagnosis, hazardous material management, emergency response and treatment.

9. Kumtor should perform risk/cost benefit analyses of generating and recycling cyanide on-site.
10. Kumtor and the Kyrgyz Ministry of Transportation should jointly assess the feasibility of using stronger shipping containers or packaging for cyanide transportation.
11. Kumtor should ensure that all employees involved in transportation activities (truckers, security, procurement) be properly trained on cyanide spill response for hazardous materials.
12. The safety aspect of transporting cyanide and other hazardous materials to the mine site should be reviewed regularly.

ACKNOWLEDGEMENTS

The International Committee would like to thank the following people for their efforts in assisting us with the task we were given by the Deputy Prime Minister, Mr. Boris Silaev:

Kyrgyz Ministry of Environment:

Dr. Kulubek Djoomartovich Bokonbaev (Minister)

Dr. Victor Yakimov

Ms. Anna Golovchenko

Kyrgyz Ministry of Emergency Situations and Civil Defense

Capt. Alik A. Aidakeev

Canadian Department of Natural Resources

Ms. Janice Zinck

Canadian Department of Health

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Canadian Department of External Affairs and International Trade

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GLOSSARY

ACGIH: American Conference of Governmental Industrial Hygienists.

amyl nitrate: [$C_5H_{11}ONO_2$] An ester of amyl alcohol added to diesel fuel to raise the cetane number.

carcinogenicity: The capacity of a chemical or physical agent to cause or induce cancer in living organisms.

cassava: A shrubby perennial plant grown for its starchy, edible tuberous roots.

cyanate: A salt or ester of cyanic acid containing the radical CNO.

cyanide: Any of a group of compounds containing the CN group and derived from hydrogen cyanide, HCN.

cyanogen chloride: [ClCN] A poisonous, colorless gas or liquid, soluble in water; used in organic synthesis.

cyanogenic: Capable of producing cyanide.

DNA: Deoxyribonucleic acid; a very long chain -like molecule made up of thousands of sub-units (bases) whose sequence represents in genetic code the "blueprint" for the normal structure and function of the cell and organism.

EC_{25}: The effective concentration of an agent, such as a chemical, produces a specified effect in 25% of the sample population.

EC_{50}: The effective concentration of an agent, such as a chemical, produces a specified effect in 50% of the sample population.

epidemiology: The study of the distribution and causes of diseases and injuries in human populations.

hydrogen cyanide: [HCN] A highly toxic liquid that has the odor of bitter almonds and boils at 25.6°C; used to manufacture cyanide salts, acrylonitrile, and dyes, and as a fumigant in agriculture. Also known as formonitrile; hydrocyanic acid; prussic acid.

LC_{50}: Lethal Concentration 50. The dose of a substance which is fatal to 50% of the test animals.

LD_{50}: Lethal Dose 50. The dose of a substance which is fatal to 50% of the test animals.

LOEC: The lowest-observed-effect concentration of an agent, such as a chemical, is the lowest concentration at which a statistically significant effect occurred

mg/L: Milligram per Litre

mg/kg: Milligram per Kilogram

mutagenicity: The capacity of a chemical or physical agent to cause permanent alteration of the genetic material within living cells

NOEC: No Observed Effect Concentration of an agent, such as a chemical, is the highest concentration in which the measured effect is not statistically different from that of the control.

NOEL: No Observed Effect Level. The greatest dosage of a chemical, found by experiment or observation, that causes no adverse alteration of morphology, functional capacity, growth, development, or life span of the target organism.

phytopathology: The ability of an organism to cause disease in a plant.

prussic acid: hydrocyanic acid [HCN]. Certain sugar compounds called cyanogenic glycosides contain the cyanide ion (CN-) and only form prussic acid when degraded by certain enzymes.

sodium cyanide: [NaCN] A poisonous, water-soluble powder; decomposes rapidly when standing; used to manufacture pigments, in heat treatment of metals, and as a silver- and gold-ore extractant.

sodium hypochlorite: [NaOCl] Air-unstable, pale-green crystals with sweet aroma; soluble in cold water, decomposes in hot water; used as a bleaching agent for paper pulp and textiles, as a chemical intermediate, and in medicine.

teratogenicity: The capacity of a physical or chemical agent to cause non-hereditary congenital malformations or birth defects in offspring.

tonne: A unit of mass in the metric system, equal to 1000 kg or approximately 2200 pounds.

toxic effect: any change in an organism such as a plant, animal or human which results in impairment of functional capacity of the organism, as determined by anatomical, physiological, biochemical or behavioral parameters; causes a reduction in the organism's ability to maintain normal function; or enhances the susceptibility of the organism to the deleterious effect of other environmental influences.

toxicity: The quality or degree of being poisonous or harmful to plant, animal or human life.

μg/kg: Microgram per kilogram. A unit of concentration equal to one millionth of a gram per one kilogram

μg/mL: Microgram per millilitre. A unit of concentration equal to one millionth of a gram per one thousandth of a litre.

WHO: World Health Organization

Source: Dictionary of Scientific and Technical Terms**,** 4th edition, 1989.

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1.0 BACKGROUND

1.1 Kyrgyzstan

Kyrgyzstan is a central Asian country bounded on the east by China, the north by Kazakstan, the west by Uzbekistan and the south by Tajikistan. Kyrgyzstan covers about 198,394 km^2 (or

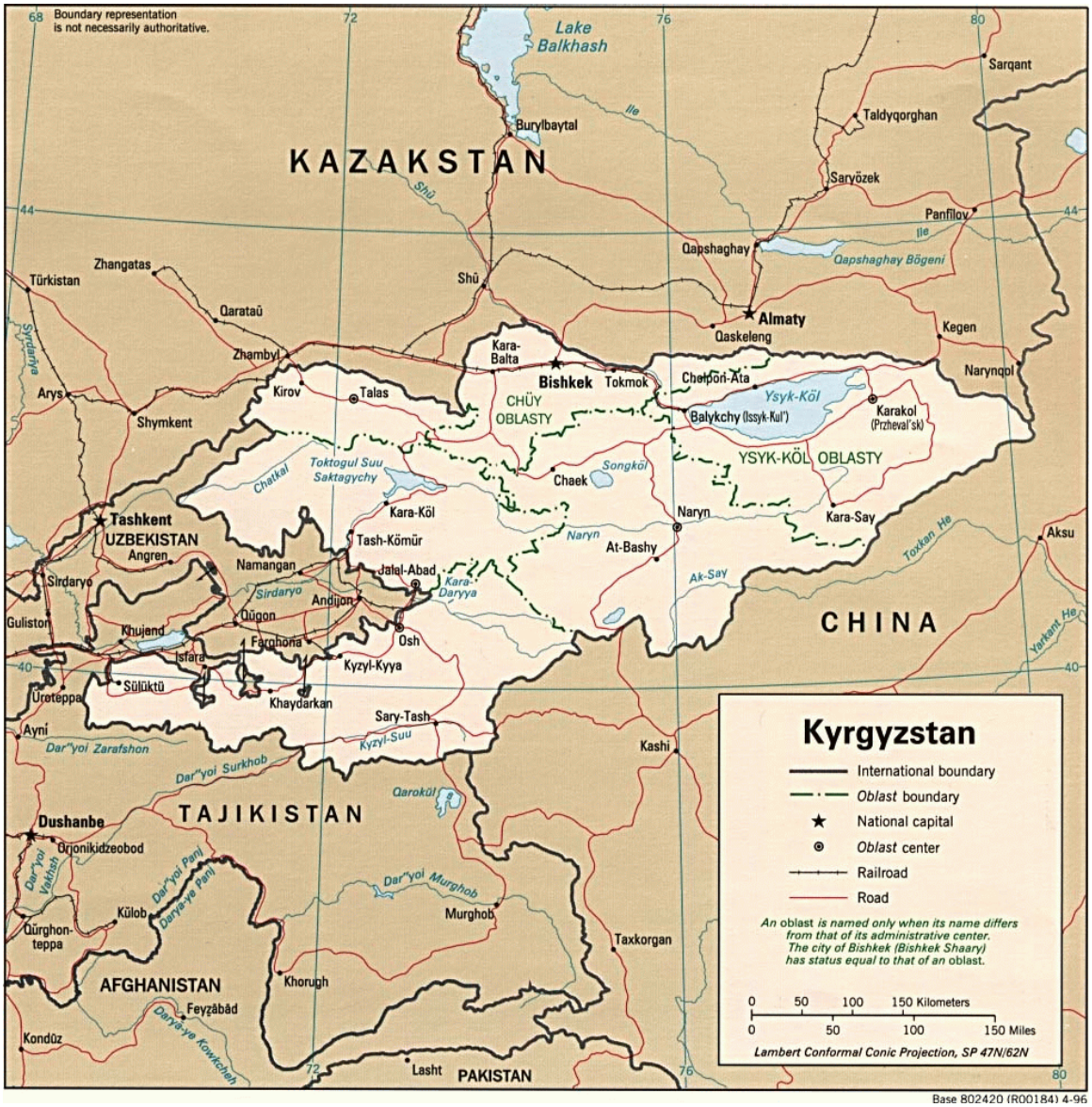

Figure 1: Map of Kyrgyzstan

76,000 sq.mi.) (Figure 1). The Tian Shan, meaning "heavenly mountains" in Chinese, is a towering system covering much of Kyrgyzstan and includes some of the world largest glaciers. Lake Issyk-Kul is one of the most famous mountain lakes in the world, well-known for its magnificent scenery, unique scientific interest and as an unparalleled tourist region. The ethnic composition of Kyrgyzstan is 52% Kyrgyz, 21% Russian, 13% Uzbek and 14% other nationalities.

1.2 The Event

On Wednesday, May 20, 1998 at 12:10 p.m., a transport convoy of five haulage trucks accompanied by two security vehicles was en route from the Balykchy Marshalling Yard to Cameco's Kumtor minesite. Each truck carried a 6-metre sea-container containing 20 tonnes of sodium cyanide (NaCN) briquettes.

On the Barskaun Pass road at about 8 km above the village of Barskaun (population 7,000) the fourth truck in the convoy rolled over on the road adjacent to a bridge over the Barskaun River. The container of sodium cyanide fell into the river, then the truck landed upright on top of the container.

The container was damaged, but despite falling six metres, only seven of the cyanide bulk packages inside were punctured, mainly by crushing as the container distorted. An estimated 1762 kg of sodium cyanide, 935 kg CN^- (Appendix A) was lost into the river. The truck driver suffered a broken arm and leg. The truck was beyond repair.

1.3 The International Scientific Commission

Following the incident, a scientific commission of international experts was assembled at the request of the Deputy Prime Minister, Mr. Boris Silaev to study the impact of the cyanide spill. This commission included:

Thomas Hynes, Environmental Specialist, Natural Resources Canada, Ottawa, CANADA
John Harrison, Health Advisor, Health Canada, Ottawa, CANADA
Evgene Bonitenko, Medical Toxicologist, EMERCOM, RUSSIA
Tamara Doronina, Chemist, Moscow Chemical Institute of Chemical Materials, RUSSIA
Harry Baikowitz, Chemist, Bodycote Technitrol, Pointe-Claire, CANADA
Michael James, Medical Consultant, Medisys, Toronto, CANADA

1.4 Cyanide

Cyanides comprise a distinct group of compounds characterized by the presence of the group, C N . Cyanide compounds take many forms, including free cyanide, simple cyanides, complex cyanides (metallocyanides) and organic cyanides (nitriles or glycosides).

Free cyanide is defined as the sum of the cyanide present either as HCN or CN^-. Hydrogen cyanide is a colourless liquid with an odour characteristic of bitter almonds (threshold value 1-5 mg/m^3 and a vapour pressure of 107.6 kPa at 27.2°C) and it is completely miscible in water. Potassium cyanide, a white granular powder, and sodium cyanide, a white crystalline solid, are both readily soluble in water.

Cyanides in water and soil form hydrogen cyanide that eventually is volatilized into the air and dispenses quickly upwards due to its low density. Micro-organisms in water and soil convert some of the cyanide to less harmful chemicals; some form metal-cyanides.

Most foods contain traces of cyanides, including cassava, sweet potatoes, yams, maize, millet, bamboo, sugarcane, peas and beans, as well as the kernels of almond, lemon, lime, apple, pear, cherry, apricot, prune, and plum. Examples of cyanide levels measured in selected foods include 0.001 to 0.45 mg/kg for cereal grains and their products; 0.07 to 0.3 mg/kg for soy protein products; 1000 mg/kg for cassava; 100 to 300 mg/kg for lima beans. The presence of cyanide in

food of plant origin has been attributed to natural production within the plants and uptake from the surrounding soil. However, soil concentrations are expected to be low as the cyanide ion is strongly adsorbed and retained in soils, and numerous micro-organisms are able to degrade free cyanide to carbon dioxide and ammonia. As well, some cyanide residues have been detected in food treated with cyanide fumigants. Although cyanides can be found in fish from contaminated waters, the cyanides readily decompose upon heating, and cooked foods contain little or no cyanide.

Certain bacteria, fungi, and algae also produce cyanides. Cyanide is an important component of vitamin B-12. Which is an essential vitamin in our diet that prevents vitamin B-12 deficient anemia (iron-poor blood). The cyanide in vitamin B-12 is tightly bound and not harmful.

Cyanide affects the ability of our tissues to use oxygen. Health effects are similar whether cyanide is breathed, ingested, or comes in contact with skin. Rapid damage to the central nervous system and the heart results from breathing high levels of cyanide over a short time. Symptoms include difficulty breathing, irregular heart beat, uncontrolled movement, convulsions, coma, and possibly death. Breathing lesser amounts for a longer time can also be life-threatening. At lower levels of exposure, breathing difficulties, pain in the heart area, vomiting, blood changes, headaches, and enlarged thyroid glands are observed. Skin contact with hydrogen cyanide or cyanide salts can produce skin irritation and sores in some people.

Cyanide exposure can be determined by measuring the amount of cyanide and thiocyanate in urine and blood samples. Small amounts of these compounds are always in the body, so these measurements are only useful when you are exposed to large amounts of cyanide. The exact cyanide exposure levels that result in certain levels of cyanide or thiocyanate in body fluids are not known.

Fatal oral doses of cyanide compounds are reported to range from 0.5 to 3.5 mg CN^-/kg body weight, with most of the values occurring between 0.5 and 1.0 mg CN^-/kg (US EPA, 1992).

In human and mammalian toxicology, we speak of an acute exposure to a chemical as a single dose or exposure for less than 14 days and we speak of chronic exposure as exposure to a chemical for 365 days or longer (normally a daily exposure over this period).

Table 1: Toxic Characteristics of Cyanide Exposure

Concentration of HCN in Air (mg/m^3)	**Toxic Characteristics**
1	Threshold value for smell
5-20	Slight headache from single exposure
20-50	Breathing for hours causes headache, rapid heart beat and vomiting
50-60	Exposure for 30 minutes to 1 hour remains without immediate and long term results
100	Dangerous for life; humans usually die within first hour
120-150	Death within 30 minutes to 1 hour
200	Death within 10 minutes
300	In a quiet situation a human might remain without headache for a 2 minute exposure
400	In a quiet situation a human might remain without headache for a 1.5 minute exposure
550	Exposure for 1 minute remains without serious results
7000-12000	Even using protective equipment, dangerous within a 5 minute exposure due to poisoning via skin.

As the cyanide exposure was over a period of less than a day from the Barskaun River water and most probably less than a few days from any soil source, what had occurred was an acute exposure to the population and not a chronic exposure. Although the health effects from chronic exposure such as mutagenicity will be included in this report for completeness (see Section 9),

they are not pertinent to this situation. Only the acute exposure pattern and its possible health effects are pertinent.

Only acute exposure could have occurred. Any adverse health effects would have resulted from a short or acute exposure and could not be attributed to a lengthy or chronic exposure. It is important to differentiate these two exposure types as the scientific literature is based on whether people or animals are exposed for a short (acute) or long (chronic) period of time to a specific chemical. Detrimental health effects resulting from exposure to cyanide are usually specific to the type of exposure.

2.0 INTERNATIONAL GUIDELINES AND STANDARDS

It is essential to note that the allowable concentration guidelines have been developed only for a chronic exposure whereas we are dealing with an acute exposure in this situation. There is another toxicity indicator, minimum effective concentration, which is 1-2 orders magnitude (10 to 100 times) higher than the chronic allowable concentration limits.

The Russian Maximum Limit Concentration for hydrogen cyanide and its salt are 0.3 mg/m^3 for indoor (working place air) and 0.01 mg/m^3 for atmospheric air. The Russian limit for HCN (CN^-) in spring water is 0.1 mg/L.

Guidelines and criteria for cyanide in soils and groundwater have been developed by several nations, including Canada, the Netherlands, the United Kingdom and Germany. Jurisdictions within Canada with existing guidelines for cyanide in soils and groundwater include Alberta, British Columbia, Ontario and Quebec (Table 2). Cyanide guidelines for soils are usually expressed as criteria for free and total cyanide. The Canadian guideline values for free cyanide vary from 0.9 to 100 mg/kg dry soil, while the values for total cyanide range from 5 to 500 mg/kg dry soil. Guidelines from other agencies vary from 1 to 500 mg/kg dry soil for free cyanide and 5 to 5000 mg/kg dry soil for complex cyanides. The Canadian Water Quality Guideline for protection of aquatic life is 5.0 μg free CN/L (CCREM, 1987) and the Maximum Acceptable Concentration for drinking water is 200 μg free CN/L (HWC, 1992). The Canadian guideline for cyanide in air for an 8 hour occupational exposure limit is 5 mg/m^3 time weighted average (TWA).

The Canadian Environmental Quality Criteria for Contaminated Sites were established for defined land uses by adopting existing criteria for soil and water used by various jurisdictions in Canada. A specific protocol for guidelines derivation was developed to ensure that revised guidelines are scientifically defensible (CCME, 1996).

The protocol considers the effects of contaminated soil exposure on human and ecological receptors for given land uses (e.g. agricultural, residential/parkland, commercial and industrial land).

Deriving human health based soil quality guidelines includes assessing the hazard posed by a chemical, determining estimated daily intake of the chemical unrelated to any specific contaminated site (the background exposure level) and defining generic exposure scenarios appropriate to each land use. The soil guideline must ensure that the total exposure to a contaminant (estimated daily intake plus on-site exposure at the guideline concentration) will present negligible risk.

A soil quality guideline for environmental health is derived using environmental toxicological data to determine the threshold level on key receptors. Exposure from direct soil contact is the primary derivation procedure for environmental guidelines for residential/parkland and commercial/industrial land uses.

Another procedure, exposure from contaminated soil and food ingestion, may be considered for certain land uses if there is adequate data. For agricultural land use, if both derivation procedures are used, the lowest-value result is considered the environmental soil quality guideline.

The final Canadian or recommended guideline for a specific chemical such as cyanide is based on the lowest value generated by the environmental and the human health approaches for each of the four land uses: Agricultural, Residential/Parkland, Commercial and Industrial.

As noted in Table 2, for free cyanide in soil the CCME (1997) guideline for human health (free cyanide) is 29 mg/kg for agricultural land use. This is based on animal and human toxicity data for cyanide compounds.

Also in Table 2 for cyanide in soil, the CCME (1997) guideline for environmental health or potential damage to ecological aspects is 0.9 mg/kg (free cyanide) for agricultural land use. This is based on selected plant and invertebrate toxicity studies for cyanide. These selected organisms include earthworms, bush beans, radishes and lettuce.

The final Canadian recommended guideline for a specific chemical, in this case cyanide, is based on the lowest value of the environmental or the human health guideline. Since the environmental guideline of 0.9 mg/kg is the lowest, it was chosen as the Canadian recommended guideline.

Table.2: Existing Guidelines and Criteria for Free and Total Cyanide in Soil and Water

Jurisdiction	**Medium**	**Description ***	**Maximum concentration ***	**Source**
Russia	Air	Indoor (working place) limit	0.3 mg/m^3	
	Air	Atmospheric limit	0.01 mg/m^3	
	Water	Spring water limit	0.1 mg/L	
Canada	Soil	Recommended guidelines (free cyanide)	AG: 0.9 mg/kg R/P: 0.9 mg/kg C/I: 8.0 mg/kg	CCME 1997
	Soil	Guideline for human health (free cyanide)	AG: 29 mg/kg R/P: 29 mg/kg C: 107 mg/kg I: 420 mg/kg	CCME 1997
	Soil	Guideline for environmental health (free cyanide)	AG: 0.9 mg/kg R/P: 0.9 mg/kg C/I: 8.0 mg/kg	CCME 1997
Alberta	Soil	Clean-up criteria for total cyanide; value will decrease by 50% for soils containing less than 10% clay	5mg/kg	Alberta Environment 1990
British Columbia	Soil	Clean-up criteria for total cyanide	BC-A: 5mg/kg BC-B: 50 mg/kg BC-C: 500mg/kg	BC MOE 1989
	Soil	Clean-up criteria for free cyanide	BC-A: 1mg/kg BC-B: 10 mg/kg BC-C: 100 mg/kg	BC MOE 1989
Quebec	Soil	Guideline for total cyanide	Q-A: 5mg/kg Q-B: 50 mg/kg Q-C: 500mg/kg	MENVIQ 1988
	Soil	Guideline for free cyanide	Q-A: 1 mg/kg Q-B: 10 mg/kg Q-C: 100 mg/kg	MENVIQ 1988
Ontario	Soil	Inert landfill soil placement guideline Urban residential fill soil placement guideline Controlled fill soil placement guideline (cyanide species not identified)	0.02 µg/g 0.05 µg/g 10.0 µg/g	OME 1992

Jurisdiction	Medium	Description *	Maximum concentration *	Source
Ontario	Soil	Proposed clean-up criteria for cyanide (unidentified CN species) for contaminated sites	see below	
	Soil	Surface soil:	AG: 100 µg/g R/P: 100 µg/g C/I: 100 µg/g	
	Soil	Sub-surface soil	R/P: 100 µg/g C/I: 390 µg/g	
Massachusetts	Soil	SG-1 soils, free cyanide SG-2 soils, free cyanide SG-3 soils, free cyanide	100 µg/kg 830 µg/kg 830 µg/kg	DEP 1993
U.K.	Soil	Threshold Trigger: Domestic gardens, allotments, play areas (free cyanide)	25 mg/kg	U.K. DOE 1990
	Soil	Action Trigger: Domestic gardens, allotments, play areas (free cyanide)	500 mg/kg	
	Soil	Threshold trigger: domestic gardens, allotments (complex cyanides)	250 mg/kg	
	Soil	Action trigger: domestic gardens, allotments (complex cyanides)	1000 mg/kg	
	Soil	Threshold trigger: buildings, covered areas (free cyanides)	100 mg/kg	
	Soil	Threshold trigger: buildings, covered areas (complex cyanides)	250 mg/kg	
	Soil	Action trigger: buildings, covered areas (free cyanide)	500 mg/kg	
	Soil	Threshold trigger: Landscapes (complex cyanides)	250 mg/kg	U.K. DOE
	Soil	Action trigger (complex cyanides)	5000 mg/kg	
The Netherlands	Soil	Free cyanide soil/sediment target value Complex cyanide soil/ sediment target value	1 mg/kg 5 mg/kg	MHPPE 1991

Jurisdiction	Medium	Description *	Maximum concentration *	Source
Germany	Soil	Ia) remediation value, total cyanide Ib) remediation value, total cyanide II) remediation value, total cyanide III) remediation value, total cyanide	25 mg/kg dw 25 mg/kg dw 50 mg/kg dw 100 mg/kg dw	Amtsblatt fur Berlin (Berlin Gazett) 1990
Canada	Drinking water	Canadian Drinking Water Guideline MAC (free cyanide) Objective concentration (free cyanide)	 0.2 mg/L 0.002 mg/L	HWC, 1979; 1980; 1989;1991
Ontario	Ground water	Clean-up criterion for ground water restoration (unidentified CD species)	52 µg/L	omee 1994B
Quebec	Ground water	Guidelines for cyanide	Q-A: 40µg/l Q-B: 200 µg/L Q-C: 400 µg/L	MENVIQ 1988 (Quebec)
Canada	Ground water	Groundwater interim assessment criteria (each free and total cyanide)	40 µg/L	CCME 1991
(BC, Alberta, Ontario, Quebec	Air	8 hour exposure limit TWA	5 mg/m^3	CCOHS, 1998
Massachusetts	Ground water	GW-1 groundwater, free CN GW-2 groundwater, free CN GW-3 Groundwater, free CN	200 µg/L – 10 µg/L	DEP 1993
The Netherlands	Ground water	Free cyanide groundwater target value 5mg/L Complex cyanide groundwater target value	5 mg/L 5mg/L	MHPPE 1991

*KEY AG: agricultural land use; R/P: residential land and/or park use; C/I: commercial and/or industrial land use; C: commercial land use; I: industrial land use; BC-A: Investigation criterion for AG and R/P; BC-B: Remediation criterion for AG and R/P; BC-C: Remediation criterion for C/I; Q-A: Natural background level; Q-B: Investigation criterion; Q-C: Remediation criterion.
SG-1: "accessible" soil currently or potentially used for agriculture with "high" frequency of use;
SG-2: "accessible" soil with a lower frequency of use; SG-3: "accessible" soil with a frequency of use or "isolated" soil regardless of use;
GW-2: source of vapour to occupied structures; GW-3: discharges to surface waters.
Ia: water conservation areas; Ib: areas with sensitive use; II: geological river valley; III: areas of higher elevation

3.0 QUALITY OF AIR, SOIL AND WATER

3.1 Air

One of the primary methods of sodium cyanide attenuation is by volatilization. When sodium cyanide comes into contact with water, either in the form of humidity, rain or surface water, hydrogen cyanide (HCN) is formed. The amount of hydrogen cyanide thus formed is essentially dependent on the pH of the solution, with more and more HCN being formed as the pH drops. Since hydrogen cyanide gas is lighter than air, it rises into the atmosphere where it undergoes natural degradation. No gaseous cyanide (HCN) could be detected in the Barskaun Region using a portable cyanide-in-air measurement device. The smell of cyanide in the air, if present, would only last for the day of the accident.

An additional air sampling campaign from May 20 to June 10, 1998 found that in most cases the HCN concentration in air was less than 0.1 mg/m^3 (Appendix B). The highest grab sample concentration of HCN ranged from 0.5-6 mg/m^3 on the day of the accident. These concentrations were consistent with those in the Beak modelling study. Elevated cyanide concentrations of 1.8 and 4.6 mg/m^3 were observed above the north side of mouth of the river and above the lake (bay) on May 22, 1998. These values are inconsistent with cyanide behaviour and may be sample outliers. In the days after the spill the majority of the cyanide levels in the air were below detection, including those in the Lake Issyk-Kul region.

Decontamination of the areas affected by the cyanide spill involved the use of sodium hypochlorite to neutralize the toxicity of the cyanide by converting to cyanate. In the reaction to convert CN^- to CNO a transition compound is formed, cyanogen chloride. Cyanogen chloride is a soluble gas that remains in solution above pH 8. Based on the concentrations of cyanide present and the volume of sodium hypochlorite added the maximum concentration of cyanogen chloride which could have evolved would have been 0.0117 mg/m^3 . The ACGIH Threshold Limit Value for cyanogen chloride is 0.75 mg/m^3. A Threshold Limit Value (TLV-TWA) is a time-weighted average concentration for a conventional 8-hour workday and a 40-hour work week, to which it is believed that nearly all workers may be repeatedly exposed, day after day, without experiencing

adverse health effects. The Russian Industrial limit for cyanogen chloride exposure in the workplace is 0.2 mg/$m^{3.}$ These guidelines are based on indoor air studies. The Kyrgyz case occurred in outdoor air.

Detailed calculations on cyanogen chloride generation can be found in Appendix C. It should be noted that while little or no cyanogen chloride was formed with the application of hypochlorite, there was a possibility that minor amounts of chlorine gas and/or chloramine gas could have been evolved accounting for the odour of the air over the time period in which was used.

3.2 Soil

The major processes affecting the transport and distribution of cyanide in soils are volatilization and biodegradation. Cyanide may also form complexes with heavy metals, particularly iron, and precipitate out of solution. Hydrogen cyanide is not susceptible to photolysis in soils but complex cyanides, such as ferrocyanides and ferricyanides, may rapidly photodissociate and release free cyanide when exposed to sunlight. Cyanides may be adsorbed by several materials, including clays and biological solids. However, existing data indicate that the rate of hydrogen cyanide and metal cyanide adsorption on soils is not significant when compared to the rate of volatilization and biodegradation. Small amounts of cyanide in soil may be oxidized to cyanate (HCNO). The high volatility of cyanide and the action of soil microbes ensure high levels of cyanide do not persist and accumulate in soil under natural conditions.

Nine soil samples were taken and analyzed by WHO representatives from local gardens on May 26-27, 1998. At six of the garden locations, the cyanide concentrations were less than 0.5 mg/kg, whereas in three other sites the cyanide concentrations were 0.7, 0.6 and 0.5 mg/kg. These values were below the desirable allowable environmental cyanide level in soil, as recommended by WHO of 1.0 mg/kg (Appendix D).

Recommended soil criteria for cyanide, from a health perspective, in the Netherlands is 20 mg/kg and 29 mg/kg in Canada. A lower number of 0.9 mg/kg is noted, however this number refers to effects of cyanide on particular biota (e.g. earthworms). The recommended environmental

cyanide level in soil as proposed by WHO is less than 1.0 mg/kg. The analyses above are all below the recommended guidelines in Canada and the Netherlands and below the desirable guidelines set by WHO.

On June 3, 1998, a cooperative sampling program was initiated with the Ministry of the Environment, Academy of Science Moscow, the Sanitation and Epidemiological Department, the Department of Environmental Protection and Kumtor Operating Company. The cyanide concentrations in all the soil samples were less than 0.2 mg/kg. Additional soil samples were collected and analyzed by Canadian experts, T. Hynes and J. Harrison using a portable test kit. Results from these samples indicate cyanide levels below detection levels.

An additional 137 soil and ground samples were collected for cyanide analysis between May 20 and June 10, 1998 (Appendix E). The highest concentration of cyanide was found in soil at the accident site (north bank) on May 20, 1998. Within one day, the concentration had dropped from 20 mg/kg to 6.4 mg/kg. Similarly the concentration of cyanide in soil from the bridge site was 4.6 mg/kg on the day of the accident but the level fell to 0.272 mg/kg by May 26, 1998.

Most of the soil samples contained levels of cyanide less than 1.0 mg/kg, with the exception of the samples collected at the accident site. The average natural background for free cyanide in soil for Northern Kyrgyzstan is 0.059 mg/kg.

A study of 37 soil samples by Bodycote Technitrol Eco found similar total cyanide levels in the soil samples from villages surrounding the Barskaun River (Appendix E). The average total cyanide concentration was 0.62 ± 0.57 mg/kg. The KOC data was consistently higher than other analyses but values were still well below recommended limits. In general, the soil samples do not contain levels of cyanide above what is expected naturally and at these levels there would be no risk to vegetable uptake or ground water contamination.

3.3 Water

Over the course of 5.5 hours some 1762 kg of sodium cyanide entered the Barskaun River on May 20, 1998. Based on flow/volume calculations on June 2, 1998, the cyanide-rich plume would have taken approximately 16 minutes to reach the Barskaun Channel, 25 minutes to reach the Tamga Channel and 4 hours and 10 minutes to reach Lake Issyk-Kul (Appendix G). River modelling completed by Beak International (Appendix H) suggest that more than half of the cyanide present would volatilize.

Environmental cyanide sampling began about 14:15 hours (approximately 2 hours after the spill). The measured concentration of free cyanide in the Barskaun River was between 8.51 mg/L and 16.20 mg/L from 14:15 until 16:15 hours and it is considered likely that it was similar over the period between the accident and the arrival of the response team. The water samples collected were taken 20 metres downstream from the site. The committee recognizes that the samples collected and analyzed are grab samples and may not be representative on the true river concentration.

As the container was being removed from the river at about 16:45 hours, more dissolved cyanide trapped in the container flowed into the river. This caused the cyanide concentration in the river to rise to 78.60 mg/L at a location 20 metres downstream from the accident site. At 17:15 hours the cyanide concentration had dropped to 62.00 mg/L. Thirty minutes later, at 17:45, the container had been successfully removed from the river and the cyanide concentration had dropped to 0.2 mg/L and to less than the detection limit at 18:15 hours.

There is no data on cyanide concentration from the moment the container dropped into the water until 14:15. It can be postulated that filling of the container to the river level took a certain time as the water had to flow into the container through a hole 70 cm x 70 cm (0.49 m^2) (Appendix G). After two hours the cyanide concentration was up to 16.2 mg/dm^3. Using an estimated average concentration of 8.1 mg/dm^3 it is calculated that 163.3 kg of NaCN dissolved. Of the 163.3 kg, it is expected that none entered the Barskaun Channel (as it was closed), 138.8 kg entered Lake Issyl Kul and 24.5 kg entered the Tamga Channel. From 14:00 to 17:00 it is estimated that 348

kg CN dissolved. The corresponding distribution of cyanide was such that 248 kg went to the lake, 67 kg Barskaun Channel and 35 kg into the Tamga Channel. During the time when the container was being lifted from the water an estimated 423 kg entered the river in one hour; 284 kg went to the lake, 77 kg to the Barskaun Channel and 63.7 kg to the Tamga Channel. The weight of the 935 kg of cyanide was partitioned such that the lake received 670 kg, the Barskaun Channel received 144 kg and the Tamga Channel received the remaining 121 kg. These calculations do not account for volatilization of cyanide and are appended (Appendix G).

Beak International completed a detailed modelling study on the Barskaun River and surrounding waterways (Appendix H) and using the river characteristics (flow, pH, temperature) estimated the volatilization rate of the cyanide in the Barskaun River. This study found that a majority of the cyanide was lost to the air by volatilization (535 kg) and only 238 kg CN reached Lake Issyk-Kul, 100 kg entered the Barskaun Channel and 61 kg entered the Tamga Channel.

Another series of analyses (Appendix I) shows the level of cyanide in the river and Lake Issyk-Kul were below 0.5 mg/L on the day afer the accident (May 21, 1998). Additional water samples taken by Dr. R. Cleven, a representative of WHO, on May 26-27, 1998, were observed to have cyanide concentrations less than 0.002 mg/L. Samples collected by the cooperative sampling program found that cyanide concentrations in the water after June 3, 1998 were all less than the detection limit of 0.002 mg/L.

3.4 TLV's for Soil and Water

Recommended industrial levels for workers, referred to as TLV's (Am. Conf. of Gov't Ind. Hyg., 1997) for cyanide will be compared to the soil and water exposure levels which resulted from the cyanide spill.

A Threshold Limit Value - Ceiling (TLV-C) is the indoor air quality concentration that should not be exceeded during any part of the working exposure for people. In the case of exposure of hydrogen cyanide, sodium cyanide, potassium cyanide, or calcium cyanide, the ceiling level for

each of these is 5 mg/m^3 for exposure to skin. This level is much higher than any of the cyanide concentrations in soil or water. There are no published values for a cyanide guideline for ambient air.

3.5 Conclusions

All the cyanide concentrations in air were well below the safe industrial hygiene /occupational guidelines. In outside air, HCN would readily disperse.

There was no possible exposure to cyanogen chloride as it could not have been generated in significant quantities. The estimated maximum concentration of cyanogen chloride was calculated to be 0.01 mg/m^3, well below the ACGIH Threshold Limit Value of 0.75 mg/m^3 and the Russian limit of 0.2 mg/m^3 for indoor air quality.

The potential for cyanogen chloride generation did exist due to the operating pH (pH 8.5). However, the sodium or calcium hypochlorite was added to areas with low cyanide concentrations so insignificant concentrations of cyanogen chloride were formed.

No cyanide concentrations in soils reached the level at which remediation action would be warranted for human health (Canadian guideline 29 mg/kg). However, in a few cases, low cyanide concentrations in the soil may have adverse impact on earthworms and other soil invertebrates and on the growth of radishes, lettuce and bush beans. However, the impact of eating these vegetables would not have caused harm to humans or animals.

The cyanide concentration in Barskaun River water was potentially high enough to cause serious health effects at least several hours after the spill to anyone who drank a sufficient quantity of the water. River modelling indicates the travel time from the accident site to the lake was 4 hours. The total time required for the

cyanide to clear from the Barskaun River would have been approximately 10 hours.

4.0 VEGETABLES

Little data is available in the scientific literature on the uptake of cyanide from soil by plants. Cyanide levels in cyanogenic plants are partially determined by nutrient availability, physical sensors and the growth stage of the plant. Consequently, cyanide concentrations in plants are difficult to correlate with levels in surrounding soils. A bioconcentration factor (ratio of cyanide in plant to cyanide in soil) of 0.8 can be calculated from the data on bush beans. Some species of plants actually produce cyanide, in particular cruciferous vegetables like broccoli and cauliflower, as do most fruit (Appendix J).

The effects of cyanide on seedling emergence of radish and lettuce grown in artificial soil were studied by Environment Canada. The 3-day NOEC, LOEC, EC_{25}, EC_{50} values for radish seedlings were 0.9, 1.9, 1.3, and 2.9 mg CN^-/kg soil. The average 5-day NOEC, LOEC, EC_{25}, EC_{50} values for lettuce seedlings were 5, 10, 7, and 13 mg CN^-/kg soil, respectively.

The Canadian Food and Drugs Regulations have a limit of 25 ppm or 25 mg/kg for hydrogen cyanide in apricots. Indigenous cyanide is concentrated in the pit of the fruit, not the flesh. The levels listed above for radishes are well under this limit for safety to humans. Other foods listed with a 25 ppm maximum residue for hydrogen cyanide in the Canadian Food and Drug Regulations (Section 15, Table 2) are; almonds, barley, cashew nuts, coco-beans, corn, kidney beans, lima beans, navy beans, peanuts, pecans, rice, rye, walnuts and wheat.

Cyanogenic glycosides occur in many food plants like cassava, lima beans, and the seeds of some fruits, such as peaches and apricots. Because of their cyanide content, ingestion of large amounts of cassava and, to a lesser extent, lima beans can be fatal if these foods are eaten raw or are not prepared correctly. Toxicity is much reduced by peeling, washing in running water to remove the cyanogen, and then cooking and/or fermenting to inactivate the enzymes and to volatilize the cyanide. In regions like Africa where cassava is a staple food, care is taken in its preparation for human consumption.

Analysis of cyanide in vegetables and fruit collected from the region after the accident showed levels ranging from 0.57 mg/kg to less than detection (0.002 mg/kg) (Appendix K). The higher concentrations were for apricots which are known to naturally contain cyanide (Appendix J). The majority of the samples examined showed cyanide levels less than 0.02 mg/kg. The Food and Drug Act and Regulations in Canada recommends cyanide levels in apricots not exceed 25 mg/kg. The samples of vegetables and fruit collected from the region were several orders of magnitude below this level indicating no effect from the cyanide spill on May 20, 1998.

The ingestion of a large number of apricot pits by children resulted in a variety of neurological effects ranging from headaches to coma (Lasch and El Shawa, 1981). The amount of the ingested pits could be related to the severity of effects. Comatose patients were admitted to a hospital after ingesting 15 mg CN^-/kg (Liebowitz and Schwartz, 1948), 7.6 mg CN^-/kg (Goodhart, 1994), 114-229 mg CN^-/kg (Kasamo *et al*. 1993), and 5.7 mg CN^-/kg (Valenzuela *et al.*, 1992), all in the form of potassium cyanide.

4.1 Conclusions

There may have been damage to local (Barskaun and Tamga) crops due to irrigation systems being closed up until June 2, 1998, as a result of fear of cyanide contamination.

5.0 PLANT DAMAGE

Sodium cyanide is very toxic to animals and humans, but not to plants. The U.S. Environmental Protection Agency (1997) stopped testing of plants, because results obtained up to that time indicated that no meaningful and measurable uptake of cyanide had taken place. According to Dupont (1994), cyanide tightly binds to soil rendering it quite immobile and thus not available to plants. Any damage to plant life would thus not be expected. According to the U.S. Environmental Protection Agency, the threat to plants from sodium cyanide usage is not significant when compared to the potential environmental impact posed by naturally occurring cyanogenic compounds (Appendix J) and processes.

On June 27-29, 1998 a phytopathological examination of crops along Barskaun River and on 5 farm territories of the Dzety-Oguz region: Tosor, Tamga, Barskaun, Chon-Dzargylchak and Al-Terek was completed. Further details of the study can be found in Appendix L. A summary of the study follows.

5.1 Tosor

Seven lots in Komsomolskaya and Kenesh streets were examined. Apricot and apple trees, potatoes and other vegetables were analyzed. These crops were not irrigated with Barskaun water. The general conditions of the crop were quite satisfactory. Some (single cases) cases of dried-out fruit trees cannot be considered impacted by cyanide, even if to assume the cyanide spread to the air. On some lots the effects of powdery mildew and plant lice were observed on the crops, and were not seen as significant. The fall of some small apricots and apples is connected with physiological peculiarities of the fruit and specific features of the 1998 spring-summer period, and are typical of the whole area of the republic.

5.2 Tamga

The examination of 400 ha of perennial grass crops (sainfoin), winter and spring cereal crops was carried out. The fields had not been irrigated since the time of the Barskaun accident and as a result the crops were dried and shrivelled. Nearby where the fields were irrigated the crops were in good condition. No evidence of pest infestation or disease was apparent.

5.3 Barskaun

In total, there is 1470 ha of plough-land in the farm, 15 ha is boghara (unirrigated land). There are winter and spring cereal crops, perennial grass (sainfoin), potatoes, and vegetables. The following were examined:

1. 40 ha fields irrigated with water from the accident site (10 ha sainfoin, 30 ha wheat). No cyanide contamination was revealed. No pest infestation or disease affect on the crops was observed as well. General claims: everything was dried out due to the accident, water was not let out for a considerable period of time.

2. 2.5 ha of orchards were irrigated by the Barskaun River water (1.2 ha apricot trees, 0.5 ha other fruits, 0.5 ha potatoes). No signs of chemical contamination were seen. A young garden was in good condition. There were no signs of pest infestation or disease in plants.

3. A. Mamytov's lot, on Kenesh Street, was watered on the first day of the Barskaun River accident. Some drying of young apricot trees, evidently, have no relation to chemical contamination, as the rest of the garden is in good condition. There are single cases of powdery mildew or plant lice effect.

4. 74 ha trees (apricot, wild apricot, poplars, karagach of 1-2 years) in TOLUK farm, located at the Barskaun River. All the trees show drying and withering effects. The farmer's statement about cyanide impact on the trees' drying out is unsupported. On some trees

there were signs of vascular bactericide drying. However, it has nothing to do with general drying of crops.

5.4 Chon-Dzargylchak

Fruit trees on two lots were examined. No evidence or indirect signs of contamination by cyanide were seen. There were some signs of plant lice and powdery mildew on the apple trees, and maculas (blotches) was noted on the apricots. There were also some small fruit which had fallen, which is not a sign of contamination.

5.5 Ak-Terek

Two fruit gardens were examined there. Earlier, from these gardens samples of apricots with maculas were delivered to Bishkek. In total there were 100 trees: apricots, apples and pears. As in the above-mentioned places, there was no sign of cyanide contamination. The owners' allegation of cyanide contamination (some leaves yellowing, small fruit falling off, etc.) have no serious grounds. There were some effect of powdery mildew and plant lice on the fruit trees.

5.6 Conclusions

An additional study of plants from these regions by the Kyrgyz Republic Ministry of Agriculture and Waste Management showed no evidence of cyanide contamination (Appendix L).

No direct cyanide effects were found on plants in five farm territories of the Dzety-Oguz region: Tosor, Tamga, Barskaun, Chon-Dzargylchak and Al-Terek.

6.0 FISH

Cyanide is one of the most toxic chemicals to which fish are likely to be exposed. Fish are approximately one thousand times more sensitive to cyanide than are humans. Dose levels as low as 0.03 mg/L HCN can be ultimately fatal to sensitive species, while 0.2 mg/L is lethal to most species. In each case, levels less than lethal do provoke physiological and pathological responses that reduce swimming ability, interfere with reproductive capacity and can lead to seriously deformed offspring, and also leave fish more vulnerable to predators (Table 3).

Cyanide is not a persistent toxic chemical in the environment and under normal conditions will not permanently destroy a fish habitat. Due to this acute sensitivity, fish make excellent biological markers for the presence of cyanide in water. If fish are living after exposure, then no other form of life will be harmed.

Table 3: Acute, Chronic and Sublethal toxicity of cyanides to fish (Ingles, 1982).

LETHAL EFFECTS		SUBLETHAL EFFECTS		
ACUTE (Dynamic LC_{50} - 96 h) mg/L CN	CHRONIC (Juniors/Adults) mg/L CN	Activity or Organ Affected	Nature of Effect	at mg/L CN
0.05 - 0.2	0.0019 - 0.07	Spawning	completely inhibited	0.005
		Egg Production	reduced by 42%	0.01
		Egg Viability	eggs infertile	0.065
		Spermatogenesis	permanent reduction	0.02
		Abnormal embryonic development	severe deformities	0.07
		Hatching	up to 40% failure	0.01-0.1
		Swimming	reduced 90% at 6°C	0.015

In addition to the total cyanide level in the water, a number of other factors associated with water chemistry exert a modifying effect on acute toxicity; these include: dissolved oxygen concentration, temperature, pH, salinity, and other dissolved constituents (e.g., zinc, ammonia). Of particular relevance to the Barskaun River are temperature and salinity.

Cyanide toxicity in fish increases 3-fold with a 12ºC decrease in temperature. Furthermore, seventeen parts per thousand chloride ion (full strength sea water) decreases the survival time.

Table 4: Sensitivity of different fish species to cyanide (Ingles, 1982)

Fish Species	CN Concentration (mg/L)	Temperature (ºC)	Effect (LC_{50})
rainbow trout	0.028 0.042 0.068	6.3 12.3 18.0	96 hours 96 hours 96 hours
brown trout	0.08	15.5	24 hours
small mouth bass	0.13	21	
bluegill	0.28		96 hours
fat head minnows	0.23		96 hours
mosquito fish	0.64		96 hours

The majority of the fish kills were found either at the mouth of the Barskaun River or on the shore of the Issyk-Kul lake. Analysis of cyanide in fish collected between May 22 and May 23, 1998 indicated cyanide levels ranging from 1.5 mg/kg in small fish collected in Barskaun Bay to 0.45 mg/kg in some trout also collected from Barskaun Bay (Appendix L).

6.1 Conclusions

In the days following the spill, fish habitats had been repopulated indicative of low cyanide levels in the Barskaun River and Lake Issyk-Kul. This observation provides evidence to confirm the river/lake environment is safe for public use.

There was no reasonable exposure route to people eating fish because any seriously affected fish would have died and would not be considered for human consumption. Furthermore, cyanide present in the fish would be bound to the

haemoglobin and would not be harmful if eaten, and cooking would also have removed the cyanide.

7.0 MILK

Free cyanide is readily adsorbed by terrestrial animals through inhalation, ingestion and contact with skin and mucous membranes. The most frequent cause of cyanide poisoning in terrestrial animals, particularly livestock, comes from ingestion of plants containing cyanogenic glycosides. Animals that eat rapidly are at greatest risk. Ruminants (e.g., cattle and sheep) tend to be more vulnerable to cyanogenic plants than non-ruminants (e.g., horses and pigs), presumably as a result of greater degradation of plant cells by bacterial enzymes. It is doubtful that any cyanide uptake by plants from soils can cause a greater hazard to animals than those that already exist in the natural environment through cyanogenic plants (Dupont 1994).

Veterinarians conducted pathological and chemico-toxicological examinations of the dead animals bodies (cows and hens). Cyanide was not detected in any of the cases (RMD,1998). Therefore the cyanide from the spill could not have been responsible for any of these animal deaths. An additional veterinarian report by the University of Saskatchewan confirms the findings of the Russian Ministry of Defence and is appended in Appendix M

Cyanide poisoning through ingestion of cyanogenic plants is more prevalent under drought conditions since animals are less selective in choice of forage and plant production of cyanogenic glycosides is enhanced under stressful conditions. Detoxification is quite rapid and there is no evidence of bioaccumulation in any organisms. Low doses of cyanide are rapidly degraded to nontoxic products by most species while large dosages cause death. Milk produced from animals which may have ingested cyanogenic plants would have been detoxified within the days falling the spill. No cyanide would enter the milk produced. Analysis of milk samples collected on May 28, 1998 showed the milk was indeed free of cyanide (Appendix N). Therefore cyanide from the spill could not have possibly poisoned anyone drinking the milk.

7.1 Conclusions

No cyanide exposure occurred by drinking milk or eating dairy products from the region.

8.0 LAKE ISSYK-KUL

Lake Issyk-Kul, an oval-shaped basin lake in the northern Tien Shan (mountains) of Kyrgyzstan, is one of the largest mountain lakes in the world, and is famous for its magnificent scenery and unique scientific interest. It has length of 182 kilometres (113 miles), a width of up to 61 kilometres (38 miles), and a surface area of 6,332 square kilometres (2,445 square miles). It reaches 702 metres (2,303 feet)in depth and has a volume of 1733 cubic kilometres (416 cubic miles). The lake's name, which derives from a word for "hot lake", alludes to the fact that it does not freeze over during the winter, even though the lake is situated at an altitude of 1,609 kilometres (5,278 feet).

Due the large size of Lake Issyk-Kul the maximum concentration of cyanide is expected to be extremely low through the dilution effect. On May 21, 1998 cyanide concentration in the lake was between 0.11 and 0.36 mg/L near the mouth of the Barskaun River. No cyanide was detected (< 0.001 mg/L) in water samples taken in the bay near the outlet of the Barskaun River on May 28, 1998. Further analyses support the conclusion that there is no longer an environmental or human health threat to Lake Issyk-Kul either in the short or long term.

8.1 Conclusions

There has been no damage to Lake Issyk-Kul either in the short or long term. The immense volume of water in the lake diluted the cyanide concentration to less than detection limits (<0.001 mg/L) and below acceptable international drinking water standards. Lake Issyk-Kul remains a pristine, national treasure.

9.0 CARCINOGENICITY, MUTAGENICITY, TERATOGENICITY, REPRODUCTIVE EFFECTS AND NEUROTOXICITY

9.1 Carcinogenicity

No carcinogenicity studies in animals and no epidemiological studies with carcinogenicity in humans have been reported. The lack of mutagenic potential of cyanide does, however, suggest that cyanide may not be a carcinogen in animals or humans.

There are no data in humans or experimental animals upon which to base a conclusion regarding carcinogenic potential of free cyanide. It is therefore "unclassifiable with respect to carcinogenicity in humans" (Group VI.B) according to the classification scheme employed by the Bureau of Hazardous Chemicals of Health Canada (1994). Free cyanide is thus treated as a substance for which the critical effect is believed to have a threshold of exposure for the setting of human health soil quality guidelines.

There are also no acute exposure studies for cyanide in the literature to indicate that carcinogenicity is a health problem.

9.2 Mutagenicity

Cyanides have tested negative for mutagenicity and effects on DNA synthesis *in vivo* (DeFlora, 1981; DeFlora *et al.*, 1984; Owais *et al.*, 1985; Sofuni *et al.*, 1985), except in a study by Kushi *et al.* (1983), who reported a marginally mutagenic response for HCN in *S. typhimurium* strain TA100 without metabolic activation.

An *in vivo* study in mice showed no testicular DNA-synthesis inhibition after a single oral dose of 1 mg/kg cyanide as potassium cyanide (Friedman and Staub, 1976). These results indicate that cyanide, especially in the form of potassium cyanide, is not mutagenic.

There are no acute exposure studies for cyanide in the literature to indicate that mutagenicity constitutes a health problem**.**

9.3 Teratogenicity

There are no reports in the literature which indicate that cyanide can directly cause birth defects. Epidemiological surveys of areas in Zaire where cassava is a staple food indicate that congenital hypothyroidism is present in 15% of newborns, an incidence rate 500 times higher than that observed in industrialized countries (Ermans et al.,1980). High chronic thiocyanate levels as a result of cassava consumption and iodine deficiency probably contribute to this condition.

Birth defects were seen in rats fed cassava root diets. Pregnant rats were fed milled cassava powder as 50 or 80% of their diet on gestation days 1 to 15 with controls receiving commercial rat food (Singh, 1981). A dose-related decrease in maternal weight gain was observed over the course of the study. On gestation day 20, fetuses were collected. At 80% cassava in the diet, rats showed a high incidence of embryo-lethality (19%), and 28% of the fetuses were abnormal. Abnormalities were described as limb defects, open eyes, microcephaly, and retarded growth. At 50% cassava, only low fetal body weight was observed. No resorptions or malformations were found in the control group. As no specific dose or level of cyanide are provided, an actual exposure level and/or NOEL cannot be calculated.

There are no acute exposure studies for cyanide in the literature to indicate that teratogenicity is a health problem**.**

9.4 Reproductive Effects

There are no reports that cyanide can directly cause reproductive problems in people. However, adverse effects on the reproductive system were seen in rats and mice that drank water containing

sodium cyanide (US DHHS, 1997). In a reproduction study female Wistar rats were fed a basal cassava diet containing 12 mg/kg HCN or the basal diet with 500 ppm cyanide added (as 1.25 KCN/kg diet) from 20 days before mating through lactation (Tewe and Maner, 1981a). At weaning, two female pup/litter were randomly allotted to the experimental diets for 28 days. Significant decreases in food consumption and growth rates were observed in weanlings fed KCN-enhanced diets regardless of previous cyanide exposure. Those weanlings exposed to higher levels of cyanide *in utero* and during the 28-day postweaning period had significantly reduced protein efficiency ratios. Serum thiocyanate was significantly increased in lactating rats and offspring during lactation and in the postweaning phase in rats fed KCN-enhanced diets. Interestingly, kidney and liver rhodanese activities were not affected.

Tewe and Maner (1981b) fed groups of six pregnant Yorkshire pigs from the day after breeding until parturition. A low-cyanide (30.3 mg CN^-/kg diet or 0.88 mg CN^-/kg body weight/day) cassava (control) diet or the cassava diet with added cyanide for a total cyanide level of 276 mg CN^-/kg or 520.7 mg CN^-/kg diet were used. On gestation day 110 two sows/group were sacrificed, the fetuses were weighed and thiocyanate levels were determined. The number and the weight of 110-day-old fetuses were comparable. Serum thiocyanate levels were slightly increased in the sows and fetuses of the 520.7 mg CN^-/kg added cyanide group. The serum-bound iodine decreased during gestation in all groups. Histopathological examination showed proliferation of glomerular cells of the kidney in sows of all groups and reduced activity of the thyroid gland in dams fed at 520.7 mg CN^-/kg. As was previously described in the study in rats, the doses administered in the cyanide-added diets would result in values of approximately 10 and 17 mg CN^-/kg body weight/day respectively for the 276 and 520 mg/kg dose groups (using body weight values of 170.8 and 183.5 kg and food consumption values of 5.67 and 6.17 kg/day)

All remaining sows were fed their respective diets unto parturition when their diets were changed to corn and soybeans. After 21 days of lactation, the sows and piglets were bled for thiocyanate, protein, and protein-bound iodine determinations. The results showed significantly ($p<0.05$)

higher levels of serum thiocyanate in sows but not in piglets from sows fed diets containing 500 ppm added cyanide. Protein and protein-bound iodine levels were not significantly different.

The authors concluded that cyanide at a level of 520.7 mg/kg in the diet did not interfere with the production of the first litter of sows. However, metabolic and pathological differences did occur. Histopathological changes were observed in the thyroid glands of sows fed a cassava diet with KCN added at 500 mg CN^-/kg of food through the first 110 days of gestation. No histopathological effects on the thyroid were observed in pigs fed diets containing an additional 250 mg CN^-/kg of food. Again, because of the limited number of animals/group (only six pigs/dose group), this study was not used to determine a NOEL (HC, 1995) for the Canadian Soil Guideline for human health effects (cyanide).

There are no acute exposure studies for cyanide in the literature to show that reproductive effects constitute a health problem**.**

9.5 Neurotoxicity

The central nervous system appears to be the most sensitive end point of cyanide toxicity, partly because of its high metabolic demands. Although no true neurotoxicity studies were identified, a number of studies did look at the central nervous system as a target organ.

High doses of cyanide in animals can result in death via central nervous system effects, which can cause respiratory arrest (ATSDR, 1993). Inhalation and oral studies in animals have shown that cyanide exposure leads to encephalopathy (Levine and Stypulkowski, 1959a; Levine, 1967; Hirano, 1967). Damage has been observed in regions such as the deep cerebral white matter, the corpus callosum, hippocampus, corpora striata, palladium, and the substantia nigrar. The higher sensitivity of the white matter may be related to its relatively low cytochrome oxidase content (ATSDR, 1993). Interestingly, CNS effects have been observed following acute exposures (Levine and Stypulkowski, 1959a,b) as well as longer-term exposures (Hertting *et al*., 1960).

The central nervous system in humans has also been involved with the toxicity of cyanides. Neurologic effects of cyanide poisoning in people may correlate with the amount ingested. The exact doses consumed by the people are usually not known. A patient who accidentally ingested an unknown amount of fluid containing 6.9% sodium cyanide and 2.3% silver cyanide experienced tremors (Chen and Rose, 1952). The ingestion of a large number of apricot pits by children resulted in a variety of neurological effects ranging from headaches to coma (Lasch and El Shawa, 1981). The amount of the ingested pits could be related to the severity of effects. Comatose patients were admitted to a hospital after ingesting 15 mg CN^-/kg (Liebowitz and Schwartz, 1948), 7.6 mg CN^-/kg (Goodhart, 1994), 114-229 mg CN^-/kg (Kasamo *et al*. 1993), and 5.7 mg CN^-/kg (Valenzuela *et al.*, 1992), all in the form of potassium cyanide.

Chronic low-level cyanide exposure through cassava consumption (and possibly through tobacco smoke inhalation) has been associated with tropical neuropathy, tobacco amblyopia, and Leber's hereditary optic atrophy (Freeman, 1988). It has been suggested that defects in the metabolic conversion of cyanide to thiocyanate, as well as nutritional deficiencies of protein and vitamin B_{12}, may play a role in the development of these disorders (Money, 1958; Makene and Wilson, 1972; Conn, 1973; Grant, 1980; Osuntokun *et al.*, 1969; Osuntokun, 1972, 1980; and Monekosso and Wilson, 1966). Acutely, delayed effects on the nervous system have been reported (Parkinsonian-like symptoms) which were correlated with CAT scans and EEG (Grandas *et al.*, 1989; Uittl *et al.*, 1985; Feldman and Feldman 1990; Kadashin *et al.*, 1988).

Some authors have suggested that selectivity of the regions affected by cyanide-induced encephalopathy may be related to the depth of acute intoxication and the distribution of the cerebral vascular system (Levine, 1967). According to some authors, the different sensitivity of those parts towards hypoxia and peculiarities of the blood supply determine the selective character of a lesion of certain parts of central nervous system by cyanide. Other authors suggested that low levels of CO_2 may lead to vasoconstriction and reduction in brain blood flow which could cause brain damage possibly from both cytotoxic and anoxic anoxia (resulting from interference with the source of oxygen) (ATSDR, 1993). Several recent studies have suggested

that a disruption in neuronal calcium regulation may be an important factor in the manifestation of cyanide-induced neurotoxic events following acute exposure (ATSDR, 1993). In any case, there is evidence that cyanides may act directly on the CNS as well as indirectly from the inhibition of oxidative processes.

There are no acute exposure studies for cyanide in the literature to indicate that neurotoxicity is a a health problem**.**

9.6 Conclusions

No carcinogenic, mutagenic, teratogenic, reproductive or neurotoxological effects are expected to people as the event was an acute exposure rather than a chronic or long term exposure.

10.0 MEDICAL ISSUES

Mild or early cyanide poisoning symptoms can include;

(i) General weakness, heaviness of the arms and legs
(ii) Difficulty breathing
(iii) Headaches, giddiness, nausea, vomiting
(iv) Breath may have smell of bitter almonds
(v) Irritation of the nose, mouth and throat

The symptoms of severe cyanide poisoning can include;

(i) Nausea and vomiting
(ii) Gasping of breath
(iii) Loss of consciousness
(iv) Convulsions

10.1.1 Range of Toxicity - Ingestion

Without treatment, ingestion of 200 to 300 mg of cyanide salts or 50 mg of hydrocyanic acid may be fatal in adults. Serious poisoning resulted from ingestion of as little as 50 mg of potassium cyanide in one case. Persons have survived ingestion of 1 or more grams of potassium cyanide. Significant poisoning usually occurs when whole blood cyanide levels are 1.0 μg/mL or greater, and death may occur in untreated patients with blood levels of 3.0 μg/mL or greater.

The lowest published toxic human dose of sodium cyanide (caused hallucinations, distorted perceptions, and weakness) was 714 μg/kg, or about 50 mg for a 70 kg adult. Lowest published human fatal doses have ranged from 2800 - 6557 μg/kg or 196 to about 459 mg for a 70 kg adult.

During the 1982 Chicago acetaminophen-cyanide tampering incident, remaining capsules in bottles involved in fatalities contained from 117-858 mg of potassium cyanide.

10.1.2 Range of Toxicity - Dermal Exposure

Cyanide can be absorbed through the skin in amounts sufficient to cause serious poisoning and death, but actual cases are rare. The cases reported have involved whole-body immersion in vats of concentrated cyanide salt solutions (most likely with inhalation and ingestion exposure as well); drenching with concentrated cyanide solutions in enclosed spaces and when contaminated clothing was not removed; total-body exposure to solid cyanide salt preparations (buried in calcium cyanide during a spill); or large body surface area burns with molten sodium cyanide. Skin exposure to dilute solutions (such as the 0.1-0.2% solutions used in clinical laboratories) generally does not cause poisoning.

10.2 Adverse Effects of Cyanide Antidotes

A solid diagnosis of cyanide poisoning is required. Misdiagnosis and improper treatment with pharmaceutical drugs (e.g., amyl nitrate) could negatively impact on the health of the patient.

Among the antidotes used in many countries of the world amyl nitrite, sodium nitrite, sodium thiosulphate, glucose solutions, oxycabalamin, methylene blue, and chromoomona. The latter two are seldom used now. The new antidotes became available in recent years, like anticyanogen and others.

10.2.1 Amyl Nitrite:

Amyl nitrite pearls are broken in gauze and held close to the nose and mouth of spontaneously breathing patients, or can be placed into the face mask lip or inside the resuscitation bag. Amyl

nitrite should be inhaled for 30 seconds of each minute, with a fresh pearl used every 3 to 4 minutes until intravenous access is established. In some cases, amyl nitrite and mechanical ventilation with supplemental oxygen have revived workers rendered unconscious from hydrogen cyanide inhalation. One such worker was able to immediately return to work and complete his shift.

Adverse effects of amyl nitrite include hypotension (low blood pressure) from its vasodilating effects and cardiac dysrhythmias. Amyl nitrite converts a portion of hemoglobin (the blood pigment which transports oxygen from the lungs to the tissues) to methemoglobin (a form of hemoglobin which cannot bind or transport oxygen). When deliberately abused in excessive amounts, significant methemoglobinemia has occurred, causing signs and symptoms of hypoxia (insufficient oxygen in the tissues) and death in some cases. Other potential adverse effects are headache, nausea, vomiting, heart palpitations, breathing difficulties, cyanosis (blue discoloration of the skin and mucous membranes from induced methemoglobinemia), and dizziness.

10.2.2 Sodium Nitrite

When intravenous access is established, amyl nitrite inhalation is stopped and sodium nitrite is infused intravenously in an adult dose of 300 mg (one 10-ml ampule of 3% solution). The pediatric dose is 0.12 to 0.33 ml/kg. Children with anemia may require dosage adjustments as sodium nitrite is also a methemoglobin-inducing substance. Further sodium nitrite dosing is titrated to the clinical response (awakening from coma, normalization of pulse and blood pressure, cessation of seizures, normalization of the blood acid-base balance from the metabolic acidosis caused by cyanide poisoning).

Sodium nitrite is a potent vasodilator and rapid administration can cause significant hypotension. It should be given either by slow intravenous push over no less than 5 minutes or diluted in 50 to 100 ml of D5W and infused at a rate which does not cause or worsen hypotension.

Rare excessive methemoglobin induction is most often seen only with excessive nitrite doses, but has caused fatalities in children mistakenly administered adult doses. Methemoglobin levels should be maintained at less than 30 percent of total hemoglobin.

Additional adverse effects include all of those listed above for amyl nitrite plus visual disturbances, fainting, and seizures.

10.2.3 Sodium Thiosulphate

Following sodium nitrite administration, sodium thiosulphate is given intravenously over several minutes in an adult dose of 12.5 gm (one 50-ml ampule of 25% solution). Administer 1.65 ml/kg to children. An alternate dosing regimen used in only one reported case was a continuous intravenous infusion a 1 gram/hour for 24 hours.

No significant adverse effects from sodium thiosulphate administration have been reported during decades of clinical use in recommended doses. In volunteer studies, sodium thiosulphate caused nausea, vomiting, light-headedness, and injection site discomfort.

Theoretically, larger than recommended doses might cause hypernatremia (too high a concentration of sodium in the blood) and hyperosmolality (too great a concentration of solutes in the blood), which could result in confusion, sleepiness, coma, seizures, cardiac conduction abnormalities, metabolic acidosis, respiratory paralysis, and death.

10.2.4 Anticyanogen

Anticyanogen (4-aminofen) is an active agent forming met-hemoglobin and is used instead of amyl nitrite. In case of cyanide poisoning the first injection of anticyanogen in the form of 20% solution should be in the amount of 1.0 mL intramuscular or 0.75 intravenous. In case of intravenous injections anticyanogen is dissolved in 10.0 mL of 25 - 40% glucose solution of 0.9%

sodium chloride. Injection speed - 3 mL/minute. If necessary antidote can be injected again 30 minutes later in the dosage of 1.0 mL, but only intramuscular. Thirty minutes later the third injection cm be made in the same dosage if there is a necessity. This injection pattern safely ensures met-hemoglobin content on the level of 25-30%. In case of anticyanogen over-dosage there are the same complications as with excessive injections of amyl nitrite (Military toxicology, 1987).

10.2.5 Oxycobalamin

The mechanism of hydroxycobalamin antidote action is connected with oxycobalamin ability to interact with free cyanide and form cyancobalamin (vitamin B-12). It should be noted specifically that vitamin B-12, which was widely used as "antidote" for the residents of Barskaun and Tamga does not have the antidote qualities. But it can cause allergic reactions. Infusion solution of oxycobolamin is prepared by dissolving 0.1 g in 100 mL of 5% glucose solution. Depending on the disease severity the total dosage of antidote can amount to 5-10 g. There is a possibility of allergic reactions when oxycobalamin is used. **(**Mashkovski, 1996).

Acute lethal poisoning by cyanides can take fulminant and delayed fonns. In the first case, death comes within 3-5 min. The delayed form of poisoning lasts 20-30 min. The development of the fulminant form is described below.

The following excerpt on cyanide symptoms and treatment of cyanide poisoning are taken from: Clinical Toxicology of Commercial Products, Gosselin *et al.* (1984)

Symptoms:

1. *Massive doses may produce, without warning, sudden loss of consciousness and prompt death from respiratory arrest. With smaller but still lethal doses, the illness may be prolonged for 1 or more hours.*

2. *Upon ingestion, a bitter, acrid, burning taste is sometimes noted, followed by a feeling of constriction or numbness in the throat. Salivation, nausea and vomiting are not unusual. Solus of sodium and potassium cyanide are corrosive because of their high alkalinity. Other symptoms follow in rapid progression.*

3. *Anxiety, confusion, vertigo, giddiness, and often a sensation of stiffness in the lower jaw.*

4. *Hypernea and dyspnea. Respirations become very rapid and then slow and irregular. Inspiration is characteristically short while expiration is greatly prolonged.*

5. *The odor of bitter almonds may be noted on the breath or vomitus. This characteristic is sometimes a diagnostic help, but as many as 20 to 40% of all persons are said to be congenitally insensitive to the odor of HCN (e.g., De Busk and Seidl, 1969).*

6. *In the early phases of poisoning, an increase in vasoconstrictor tone causes a rise in blood pressure and reflex slowing of the rapid, weak, and sometimes irregular. The victim notes palpitations and a sensation of constriction in the chest. A bright pink coloration of the skin due to high concentrations of oxyhemoglobin in the venous return may be confused with that of carbon monoxide poisoning.*

7. *Unconsciousness, followed promptly by violent convulsions, epileptiform or tonic, sometimes localized but usually generalized. Opisthotonus and trismus may develop. Involuntary micturition and defecation occur.*

8. *Paralysis follows the convulsive stage. The skin is covered with sweat. The eyeballs protrude, and the pupils are dilated and unreactive. The mouth is covered with foam, which is sometimes blood-stained, indicative of pulmonary edema (Graham et al., 1977; Stewart, 1974). The skin color may be brick red. Cyanosis is not prominent in spite of weak and irregular gasping. In the unconscious patient, bradycardia (above) and the absence of cyanosis may be key diagnostic signs.*

9. *Death from respiratory arrest. As long as the heart beat continues, prompt and vigorous treatment offers some promise of survival.*

Treatment (must be prompt):

1. *If the patient is asymptomatic administer syrup of ipecac and/or perform gastric lavage. Do not use activated charcoal because it is said to be ineffective in trapping cyanide (Andersen, 1946). See also 9, below.*

2. *If the patient is apneic, start artificial respiration immediately. Keep the airway clear.*

3. *Administer amyl nitrite (amyl nitrite perles) by inhalation for 15 to 30 seconds of every minute, while a sodium nitrite solution is being prepared. Check perles for date of expiration and use the freshest product available. Perles in storage should be replaced on an annual basis.*

4. *Discontinue amyl nitrite and immediately inject 10 ml. of a 3% solution of sodium nitrite intravenously over a period of 2 to 4 minutes. If necessary, inject a nonsterile solution. Do not remove the needle. Caution: this dose of nitrite may be lethal to young children and appropriate adjustments in dose should be made on a body weight basis (Berlin, 1970).*

5. *Through the same needle infuse intravenously 50 ml. of a 25% aqueous solution of sodium thiosulphate. The injection should take about 10 minutes. Other concentrations (5 to 50%) are permissible if the total dose is held at approximately 12 gm. Injectable forms of nitrite and thiosulphate have expiration datings of 5 years.*
6. *Oxygen therapy may be of value in combination with nitrite and sodium thiosulphate therapy.*

7. *In the event of cardiac asystole, external massage and artificial pacemakers are indicated.*

8. *If symptoms recur, the injections of nitrite and thiosulphate may be repeated at half the above doses. In very severe poisonings it is safer and perhaps more efficient to keep repeating the thiosulphate injections instead of the nitrite.*

9. *Because of the speed of absorption and the rapidity with which symptoms appear, gastric lavage is seldom a practical procedure and should be postponed at least until after procedures 2 to 5. Perhaps the best lavage fluid is a dilute solution of potassium permanganate (1:5000), but tap water or dilute sodium bicardonate solution are likely to be more quickly available.*

10. *Oxygen therapy and a whole blood transfusion may become necessary if nitrite-induced methemeglobinemia becomes too severe. In laboratory animals, exchange transfusions alone increased survival (Tauberger et al., 1974).*

10.3 Human Exposure to Cyanide

At present we have been informed of approximately 2600 cases of cyanide exposure from the Barskaun spill, with four resulting fatalities. We have attempted to verify the diagnosis of this large number of people by requesting copies of their medical information from the Kyrgyz Ministry of Health, through the Canadian Embassy and later through Kumtor Operating Co. To date, we have not been given access to these records, so we are unable to assess the diagnoses made in individual cases.

However, collectively, it is possible to state that the overwhelming majority of these cases have been misdiagnosed. The vast majority of these cases are not the result of cyanide exposure. This conclusion is based upon the routes available for cyanide exposure from the Barskaun accident.

10.3.1 Air Exposure

When the spill occurred there may have been some atmospheric exposure potential during the first hours of the spill. However, the Kumtor personnel at the accident scene for those hours, during the

recovery of the truck and container from the river, experienced no such exposure. Predicted peak air concentrations would last for less than one hour. The highest level of 7.3 mg/m^3 would be expected immediately downstream of the bridge where the accident occurred assuming that air movement was up the river valley. So, although the possibility did briefly exist for atmospheric exposure, little or no exposure did occur along the river because it would have affected the Kumtor spill response personnel. At a distance of 100 m away from the river, and assuming that the air flow was across the river and not along it, the peak air concentration predicted near the mouth would be approximately 0.1 mg/m^3 and last for on the order of one hour (Appendix H).

10.3.2 Water Exposure

The possibility existed during the spill for significant human exposure through water, both in the river and in the irrigation canals. However, with a few possible exceptions, this did not occur. The main potential route of exposure would have been the drainage canal into Barskaun. However, relatively little cyanide actually went into this canal. We can determine this from the soil concentration in the irrigated fields. Multiple analyses from various agencies do not show elevated levels for cyanide in soil. Cyanide binds to the soil and soil analysis can be used to show where high exposures have occurred (Section 3.2-3, Appendix E). Furthermore, there were no high soil cyanide concentrations in the irrigated land in Barskaun or Tamga, so it means that there was no high cyanide concentration in the water that irrigated the soil. Therefore, only a small amount of cyanide came through the canals, and the bulk of the cyanide continued down the river channel to Lake Issyk-Kul.

This means that the concentration of cyanide in the water going to Barskaun and Tamga was insufficient to cause human health concerns. Nevertheless, the possibility exists for human exposure along the river itself, and particularly among the few families living at the mouth of the river, near the lake.

In any event, since the transit time for the river from the spill site to the lake is only about four hours, the cyanide concentration of concern would only have been present for the first day after the spill (and

probably much less). Since cyanide is a fast-acting, strong , toxic agent, if people were not affected during this time, they were not exposed to significant cyanide concentrations. Only cases reported within 72 hours of the spill can be considered to have any potential health effects. Cases reported later have no credible exposure pathway and cannot be directly related to the spill.

10.3.3 Soil Exposure

Despite the collection of many soil samples, no soil concentrations have been found which indicate any human health risk. With the exception of the accident site, every value found is below 1.0 mg/kg. Even the elevated values at the accident site were less than the Canadian guideline. Furthermore, no cyanide concentrations in soils reached the level at which remediation action would be warranted for human health (Canadian guideline 29 mg/kg).

There were several samples that exceeded 0.9 mg/kg, which is the Canadian recommended guideline for environmental concern. This guideline is based on effects on earthworms, radishes, lettuce and bush beans. For the plots where the soil cyanide concentrations are slightly elevated, there may be some effect on the growth rate of vegetables, but not their safety for consumption (section 3.5).

Therefore, no one would have been affected by soil cyanide concentrations during normal activities.

10.3.4 Food Exposure

Cyanide occurs naturally in many foods, especially fruits such as apricots. The cyanide is broken down easily and quickly by our bodies and so the safe concentration of cyanide in vegetables is quite high, 25 mg/kg. Samples of vegetables analyzed from the Barskaun area had cyanide concentrations well below this value. Therefore, no significant cyanide exposure occurred from eating local fruit or vegetables (see section 4.0).

In addition, cattle feeding on vegetation can easily break down any plant cyanide in their bodies. This means that cyanide transfer to milk does not occur at the levels found in the region. This has also been confirmed by milk analyses from the region. Again, no cyanide exposure occurred from drinking milk (or eating dairy products) in the region.

Some river fish were killed by the spill. However, fish are 1000 times more sensitive to cyanide than are people. There was no reasonable exposure route to people from eating fish, because any seriously affected fish would die, and would not be considered for human consumption and any cyanide would be bound to hemoglobin or protein molecules and would not be harmful if eaten. Again, lower cyanide levels in fish would be broken down by our own bodies.

10.3.5 Exposures in Barskaun & Tamga

Based on the data present the cyanide concentration in the villages of Barskaun and Tamga were found to be too low to support any cases of cyanide exposure. There was no valid exposure route for the cyanide to reach levels for human health concerns.

10.3.6 Exposures Other than in Barskaun & Tamga

The only possible cyanide exposures are those listed above. There was no potential for cyanide to affect people, livestock, plants or soils outside the Barskaun or Tamga area. This is because:

- no contaminated water flowed from the Barskaun River to any other village
- cyanide in the Barskaun river water entering Lake Issyk-Kul was immediately diluted by the massive volume of the lake, bringing cyanide concentrations below any level for human health concern.
- any volatilized cyanide in air over the Barskaun River or Lake Issyk-Kul would have been quickly diluted to levels below any human health concern

Consequently, no cases of cyanide exposure were possible in the villages of Tosor, Chon-Dzargylchak and Al-Terek.

10.3.7 Misdiagnosed Exposures

Based on the foregoing exposure pathways, it is clear that the vast majority of cases of presumed cyanide exposure are misdiagnosed. We cannot quantify the number of credible exposures because we have not been given access to health records by the Kyrgyz Ministry of Health. Credible exposures would be those reported within 72 hours after the spill, where people used river water; or possibly, but not very likely, were affected by air concentrations at the river mouth, or associated lake area. Without access to Ministry of Health records it is not possible to assess the number of credible exposures. Based on the information provided to us no more than 16 cases of cyanide exposure could likely have occurred and number of cases in reality may be a considerable less.

What is the cause of these misdiagnoses? It is clear that panic played a role here. There is no doubt that villagers were genuinely fearful when given news of the spill. Stress resulting from the spill and a host of different pre-existing conditions were probably involved in most misdiagnoses. This was coupled with the non-specific nature of cyanide symptoms, which are easily misinterpreted. A further possible scenario postulated was viral infection from domestic or wild animals or people (see Appendix O). The issue of compensation may have caused a further increase in the registration rate at local hospitals.

Whatever the source of the misdiagnoses, it may have had an impact on the health of the patients treated. The pharmaceutical agents used as cyanide antidotes have their own possible toxic effects, and inappropriate use of these pharmaceutical agents may have adverse health effects on patient health (Cummings, 1998).

These agents should be used only when:

a patient is in acute distress, and cyanide exposure has been absolutely confirmed (not speculated), or

a patient is seriously ill, and blood or urine samples have confirmed cyanide exposure.

The widespread use of these antidotes, if used without proper confirmation of exposure, is inappropriate, and potentially dangerous to the health of such patients.

In addition, the evacuation of 7000 people from the area was unnecessary and further intensified the panic in the community.

10.4 Conclusions

cyanide concentrations in the ditches of the villages of Barskaun and Tamga do not support cases of cyanide exposure causing human health effects. Nor is there any possible cyanide exposure route for the villages of Tosor, Chon-Dzargylchak and Al-Terek.

along the Barskaun River between the hours of 12:10 pm and 22:30 pm, the possibility existed for exposure to elevated cyanide concentrations which could have caused adverse human health effects. Based on the data supplied to the Ministry of Health on the number of people hospitalized in the first 72 hours after the spill, the committee accepts potentially up to 16 cases of CN exposure could have occurred. However, no medical evidence has been supplied to support these cases as being cyanide related. Thus this committee is not able on a scientific basis to confirm that these people suffered adverse human health effects as a direct result of the cyanide spill on May 20, 1998.

if some or all of these 16 cases were cyanide related and since we know there were no reported deaths within the first 72 hours then none of the 16 potential cases are

likely to experience long-term health effects in the future. Furthermore, none of these cases would normally have required treatment for more than a few days for cyanide related symptoms to be resolved.

widespread use of the cyanide antidotes was inappropriate, and may have itself caused harmful health effects to the patients.

11.0 TRANSPORTATION ISSUES

11.1 Cyanide Usage

To minimize or eliminate the shipment of cyanide to Kumtor operations, while still maintaining the economic benefits to the Kyrgyz economy, we have taken a preliminary look at three scenarios. They are

1) replace cyanide with another reagent in the operation
2) generate cyanide on-site from reagents trucked in from Balykchy Marshalling Yard
3) recycle cyanide on-site to minimize the amount transported.

After assessing the data available, we have determined that scenario 1, replacing cyanide with another reagents, is not economically viable at his time. There are several reagents and processes which are under research but none are commercially feasible (Dauphin, 1998). We have therefore discarded this option from future consideration.

11.2 On-site Cyanide Generation

Option 2, the generation of cyanide on-site is possible using methods such as the Andrussow process. Methane, ammonia and air are reacted by passing the mixture over a catalyst in a reaction vessel to form converter gas. Due to the operating temperature of between 1000 and 1100 C, the usual catalyst is 10% rhodium-platinum. This gas is then passed through a column containing a caustic solution to produce a sodium cyanide solution. Due to the numerous side reactions the conversion efficiency is typically between 60 and 70%.

$$2CH_4 + 2NH_3 + 3O_2 \Rightarrow 2HCN + 6H_2O \ (-\ 962.8kJ)$$

The stoichiometric equation for the production of sodium cyanide is given below with the molecular weights for each reactant and products.

Ammonia		Methane		Oxygen		Cyanide		Water
NH_3	+	CH_4	+	$1.5O_2$	=	HCN	+	$3H_2O$
17		16		48		27		54
0.63		0.59		1.78		1.00		
0.35		0.33		0.98		0.55		

		HCN		NaOH		NaCN		H_2O
		27		40		49		18
		0.55		0.82		1.00		

Even if you assume a 100% conversion from the above equations, 1 tonne of sodium cyanide will require the shipping of 0.35 tonnes of ammonia, 0.33 tonnes of methane and 0.82 tonnes caustic soda. Decreased conversion efficiency will mean in practice the amounts are higher. The reaction also requires a catalyst and significant energy input.

Should the company decide to manufacture sodium cyanide at Kumtor, they would have to ship ammonia, natural gas and caustic soda to the mine site. There would also be an increase in the fuel shipments required to accommodate the increased electrical demand.

A cost and risk analysis for building a plant on site versus buying and shipping sodium cyanide to the site would be necessary. There would be a significant increase in capital and operating costs. There would be a risk reduction from the elimination of cyanide shipments, but a risk increase from the shipments of ammonia (toxic), natural gas (flammable, explosive), caustic soda (irritant, corrosive) and increased fuel usage for electrical generation.

11.3 Cyanide Recovery and Recycling

Cyanide is widely used in the mining industry to extract gold from ores. Typically, the ore is ground and agitated with a dilute cyanide solution for several hours in the presence of air or oxygen. During this process, the cyanide reacts with the finely disseminated gold particles to form a gold-cyanide complex, $Au(CN)_2^-$, which goes into solution (Riveros, 1997).

Option 3, the recycling of cyanide, is possible using several processes, such as AVR or Cyanisorb. The processes are generally used for minimizing the amount of cyanide to be discharged from effluent plants to the environment. An over-lying consideration in this case is the minimization of the amount of new cyanide to be shipped to the site, by recycling the cyanide already present.

A cyanide recovery process could be used to recover all free cyanide and about half the complexed cyanide without generation of toxic compounds. One such process to recover cyanide is the Acidification-Volatilization-Reneutralization (AVR) process, which is described below as an example of this type of process.

Typically, the AVR process consists of:

- acidifying the cyanide solution to about pH 2-3.5 with sulphuric acid;
- sparging air through the acidified solution to volatilize and remove the hydrocyanic acid, HCN, gas formed as a result of acidification;
- reneutralization of the aerated solution to precipitate the excess sulphate and the last traces of metals (Riveros, 1997).

The benefits of cyanide recovery are

- reduced risks relating to cyanide transportation
- reduced costs associated with cyanide procurement, transportation and destruction
- reduction in environmental liability, in transportation and effluent discharges.

As with option 2, a cyanide recycling effort would require a cost and risk analysis. Again, there would be increased capital and operating costs. There would be a reduction (but not elimination) of cyanide transportation risks, but there would be no increase in risk from the transportation of ammonia, natural gas and caustic. There would be increased risk from greater fuel usage (as in scenario 2) for electrical generation.

11.4 Guidelines for the Safe Transport of Sodium Cyanide

For discussion purposes, sodium cyanide is regulated as a dangerous good and classified as UN1689, Class 6.1 (9.2). In Canada, shipments of this material in excess of 1000 kg per consignment require that an Emergency Response Plan be in placed and a summary of the plan be provided to Transport Canada for approval.

Summary of Canadian requirements for sodium cyanide UN 1689, PGI, transported in Intermediate Bulk Containers (IBC):

must be UN IBC's [7.1.9];
max 1500 L max capacity [3.1];
flexible IBC's shall not be used for solid materials that release liquid [7.3.3b];
Code 13L IBC's (uncoated or unlined flexible IBC's) may not be used [7.3.4b]

Details of the regulations governing the transport of sodium cyanide in Intermediate Bulk Containers can be found in Appendix N.

Although the shipping container met all applicable material and international standards for cyanide shipments, it should be determined whether or not stronger or more resilient packing for the individual cyanide packages is appropriate.

However, the primary cause of the accident appears to have been human error on the part of the truck driver.

11.5 Transportation Conclusions

The cause of the accident appears to have been human error on the part of the truck driver. The container and packaging met all material and international standards for transportation of cyanide.

12.0 CONCLUSIONS

All the cyanide concentrations in air were well below the safe industrial hygiene /occupational guidelines. In outside air, HCN would readily disperse.

There was no possible exposure to cyanogen chloride as it could not have been generated in significant quantities. The estimated maximum concentration of cyanogen chloride was calculated to be 0.01 mg/m^3, well below the ACGIH Threshold Limit Value of 0.75 mg/m^3 and the Russian limit of 0.2 mg/m^3 for indoor air quality.

The potential for cyanogen chloride generation did exist due to the operating pH (pH 8.5). However, the sodium or calcium hypochlorite was added to areas with low cyanide concentrations so insignificant concentrations of cyanogen chloride were formed.

No cyanide concentrations in soils reached the level at which remediation action would be warranted for human health (Canadian guideline 29 mg/kg). However, in a few cases, low cyanide concentrations in the soil may have adverse impact on earthworms and other soil invertebrates and on the growth of radishes, lettuce and bush beans. However, the impact of eating these vegetables would not have caused harm to humans or animals.

The cyanide concentration in Barskaun River water was potentially high enough to cause serious health effects at least several hours after the spill to anyone who drank a sufficient quantity of the water. River modelling indicates the travel time from the accident site to the lake was 4 hours. The total time required for the cyanide to clear from the Barskaun River would have been approximately 10 hours.

There may have been damage to local (Barskaun and Tamga) crops to irrigation systems being closed up until June 2, 1998, as a result of fear of cyanide contamination.

No direct cyanide effects were found on plants in five farm territories of the Dzety-Oguz region: Tosor, Tamga, Barskaun, Chon-Dzargylchak and Al-Terek.

In the days following the spill, fish habitats had been repopulated indicative of low cyanide levels in the Barskaun River and Lake Issyk-Kul. This observation provides evidence to confirm the river/lake environment is safe for public use.

There was no reasonable exposure route to people eating fish because any seriously affected fish would have died and would not be considered for human consumption. Furthermore, cyanide present in the fish would be bound to the hemoglobin and would not be harmful if eaten.

No cyanide exposure occurred by drinking milk or eating dairy products from the region.

There has been no damage to Lake Issyk-Kul either in the short or long term. The immense volume of water in the lake diluted the cyanide concentration to less than detection limits (<0.001 mg/L) and below acceptable international drinking water standards. Lake Issyk-Kul remains a pristine, national treasure.

No carcinogenic, mutagenic, teratogenic, reproductive or neurotoxological effects are expected to people as the event was an acute exposure rather than a chronic or long term exposure.

Cyanide concentrations in the ditches of the villages of Barskaun and Tamga do not support cases of cyanide exposure causing human health effects. Nor is there any possible cyanide exposure route for the villages of Tosor, Chon-Dzargylchak and Al-Terek.

Along the Barskaun River between the hours of 12:10 pm and 22:30 pm, the possibility existed for exposure to elevated cyanide concentrations which could have caused adverse human health effects. Based on the data supplied to the Ministry of Health on the number of people hospitalized in the first 72 hours after the spill, the committee accepts potentially up to 16 cases of CN exposure could have occurred. However, no medical evidence has been supplied to support these cases as being cyanide related. Thus this committee is not able on a scientific basis to confirm that these people suffered adverse human health effects as a direct result of the cyanide spill on May 20, 1998.

If some or all of these 16 cases were cyanide related and since we know there were no reported deaths within the first 72 hours then none of the 16 potential cases are likely to experience long-term health effects in the future. Furthermore, none of these cases would normally have required treatment for more than a few days for cyanide related symptoms to be resolved.

Widespread use of the antidotes was inappropriate, and may have itself caused harmful health effects to the patients.

The cause of the accident appears to have been human error on the part of the truck driver. The container and packaging met all material and international standards for transportation of cyanide.

13.0 RECOMMENDATIONS

Guidelines and Regulations

The Kyrgyz Ministry of Health and the Ministry of the Environment should consider adopting their own national guidelines for cyanide concentrations in air, soil, water and food and instituting a national certification program for Kyrgyz laboratories.

Air, Soil and Water

The use of sodium hypochlorite should be discouraged as a response to the treatment of cyanide spills in the environment due to the potential formation of cyanogen chloride. Only in cases where the pH is above 10 would its application be safe. Natural waters above pH 10 are unlikely. In practice, sodium hypochlorite usage should be restricted only to industrial applications.

Spill Response

In the event of such a spill, a first response priority is the notification of downstream water users to prevent the use of contaminated potable water; this would include monitoring, sampling (air, soil and water) analysis and reporting.

Kumtor's emergency response plan should be revised in light of the spill and a copy filed with the Emergency Measures Organization. There should be at least one Emergency Response Drill per annum involving Kumtor and Kyrgyz Emergency Measures personnel.

Medical

The Kyrgyz Ministry of Health should review its records, and provide information on the number of cases reported in the first 72 hours after the spill.

A joint Kumtor - Kyrgyz Ministry of Health follow-up on the patients who reported symptoms be completed and an expert committee be established to review documentation on patient symptomology in conjunction with the Kumtor clinic being instituted.

A reassessment of the four fatalities should be completed to determine the exact cause of death.

Kumtor and the Krygyz Ministry of Health should provide joint training to local health personnel on cyanide diagnosis, hazardous material management, emergency response and treatment.

Transportation

Kumtor should perform risk/cost benefit analyses of generating and recycling cyanide on-site.

Kumtor and the Kyrgyz Ministry of Transportation should jointly assess the feasibility of using stronger shipping containers or packaging for cyanide transportation.

Kumtor should ensure that all employees involved in transportation activities (truckers, security, procurement) be properly trained on cyanide spill response for hazardous materials.

The safety aspect of transporting cyanide and other hazardous materials to the mine site should be reviewed regularly.

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Appendix A:

Calculation of the Total Amount of NaCN Released Into the Environment on May 20, 1998

Appendix B:

Analysis of Free Cyanide Ion in Air

Appendix C:

Sodium Hypochlorite Usage

Appendix D:

Report from the World Heath Organization

Appendix E:

Analysis of Free and Total Cyanide in Soil and Ground

Appendix F:

Water Chemistry

Appendix G:

Report on the Research of Consequences of the Cyanide Spill into the Barskaun River - *T.M. Doronina*

Appendix H:

Executive Summary from the Preliminary Assessment of Cyanide Dispersion Following the Cyanide Incident

- BEAK International Inc.

Appendix I:

Analysis of Cyanide in Water

Appendix J:

Plant Species Naturally Containing Cyanide

Appendix K:

Analyses of Cyanide in Vegetables and Fruit

Appendix L:

Phytopathological Examination of Crops

Appendix M:

Veterinarian Report - University of Saskatchewan

Appendix N:

Analysis of Free Cyanide in Biosubstrates

Appendix O:

Medical Reports - *E. Bonitenko, M. James, A. Hall*

Appendix P

Safety Requirements for the Handling or Offering For Transport of Dangerous Goods in Canada

SAFETY REQUIREMENTS FOR THE HANDLING OR OFFERING FOR TRANSPORT OF DANGEROUS GOODS IN CANADA

General

7.21 (1) No person shall handle or offer for transport dangerous goods referred to in section 7.33 that are contained in a package or small container that is not in compliance with CGSB Provisional Standard 43-GP-152MP, Packing for Transportation of Dangerous Goods in Prescribed Packagings, dated September 1985, unless the package or small container is designed, constructed, filled and closed so that, under normal conditions of handling and transport, there will be no discharge, emission or escape of the dangerous goods from the package or small container that could constitute a danger to public safety.

(2) No person shall handle or offer for transport any dangerous goods that are contained in a means of containment that has not been prescribed by these Regulations or in a means of containment the continued use of which is permitted by these Regulations, unless the means of containment is designed, constructed, filled and closed so that, under normal conditions of handling and transport, there is no discharge, emission or escape of the dangerous goods that could cause a danger to public safety and the
applicable requirements of this Part are complied with.

(3) A packaging that is marked with the letters "NRC" (non-reusable container) shall not be reused to transport dangerous goods.

7.22 No person shall handle or offer for transport dangerous goods that are contained in a means of containment if the volume or density of the dangerous goods exceeds the maximum mass, volume or density for the means of containment.

7.23 (1) Where a package or small container that is manufactured in Canada is used or is intended

to be used to transport dangerous goods and is marked in accordance with Chapter 9 of the UN Recommendations, the package or small container shall comply with the requirements of National Standard of Canada CAN/CGSB-43.150-95, Performance Packagings for Transportation of Dangerous Goods, dated February 1995.

(2) Paragraph 8.1(i) and section 11 of the Standard referred to in subsection (l) do not apply to a package or small container manufactured before January 1, 1992.

(3) Section 10 of the Standard referred to in subsection (l) does not apply to a package or small container manufactured before January 1, 1994.

(4) The permitted use period requirements in paragraph 7.6.2 of the Standard referred to in subsection (1) do not apply until July 1, 1997.

Classes 3, 4, 5, 6.1, 8 and 9

Packages or Small Containers-By Road, Rail or Ship

7.33 Subject to section 7.34, no person shall handle or offer for transport by road or by rail, or in a domestic consignment by ship, dangerous goods that are included in Class 3, 4, 5, 6.1, 8 or 9 and that are contained in a package or a small container, unless the requirements of section 7.21 or the requirements of CGSB Provisional Standard 43-GP-152MP, Packing for Transportation of Dangerous Goods in Prescribed Packagings, dated September 1985, are complied with.

Tanks-By Road or Ship

7.33.1 (1) After June 30, 1995, no person shall handle or offer for transport by road or in a domestic consignment by ship dangerous goods that are included in Class 3, 4, 5, 6.1 or 8 and that are contained in a large container, unless the following requirements are complied with:

(a) the requirements of CSA Preliminary Standard B621-1987, Selection and Use of Highway Tanks, Portable Tanks, Cargo Compartments and Containers for the Transportation of Dangerous Goods, Classes 3, 4, 5, 6, and 8, in Bulk by Road, dated March 1987 and clause 8 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992; or

(b) the requirements of section 7.33.5.

(2) Subsection (1) does not apply to

(a) IBCs; and

(b) dangerous goods that are included in Class 3 and Packing Group III, that have no subsidiary classification and that have a flash point exceeding 37. 8°C.

(3) Notwithstanding subsection (1), until January 1, 2005, the requirements of clauses 4.1 and 4.4.2, paragraph 5.1(d), clauses 6.2 to 6.4 and Table 6.1 of CSA Preliminary Standard B621-1987, Selection and Use of Highway Tanks, Portable Tanks, Cargo Compartments and Containers for the Transportation of Dangerous Goods, Classes 3, 4, 5, 6, and 8, in Bulk by Road, dated March 1987, do not apply to a tank used to transport Petroleum Crude Oil (UN1267), if the following conditions are met:

(a) the inhalation toxicity of the petroleum crude oil is less severe than the inhalation toxicity for Packing Group II;

(b) the tank is inspected, tested, retested and marked in accordance with the applicable requirements for TC 306 tanks set out in clause 8 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992;

(c) the tank is inspected, tested, retested and marked at a facility registered for those purpose in accordance with clause 9 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992;

(d) no more than 20 years have elapsed since the date of manufacture of the tank or, if that date is unknown, January 1, 1980;

(e) in the case of a tank that was manufactured in accordance with the requirements for TC 306 tanks set out in CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992, except for specification shortages indicated on the manufacturer's certificate by the tank manufacturer in accordance with the provision entitled "Specification shortages" in subsection 178.340-10 of clause 5.5 of the Standard, or the
equivalent requirements for MC 306 tanks set out in CFR 49 applicable at the time of manufacture,

(i) the vehicle to which the tank is attached was registered in any province, and

(ii) the tank is currently used to transport petroleum crude oil; and

(f) in the case of a tank that was not manufactured in accordance with the requirements for TC 306 tanks set out in CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992, or the equivalent requirements for MC 306 tanks set out in CFR 49, the vehicle to which the tank is attached was registered in any province on or before January 1, 1994, the tank is currently used to transport
petroleum crude oil and the registered facility has permanently attached to the tank a metal plate that is marked with at least the following information:

(i) the words "Non-spec Crude Oil Tank" or "Citerne pour pétrole brut, hors spécifications,"

(ii) the date of the first inspection or test done in accordance with paragraph (b),

(iii) the name of the registered facility, and

(iv) the date of manufacture of the tank or, if that date is unknown, January 1, 1980.

(4) Notwithstanding subsection (1), until July 1, 1999, the requirements of clauses 6.2.3 to 6.4 and Table 6.1 of CSA Preliminary Standard B621-1987, Selection and Use of Highway Tanks, Portable Tanks, Cargo Compartments and Containers for the Transportation of Dangerous Goods, Classes 3, 4, 5, 6, and 8, in Bulk by Road, dated March 1987, do not apply to a vacuum tank used to transport waste, if the following conditions are met:

(a) on January 1, 1994, the tank was being used to transport wastes and the vehicle to which the tank is attached was registered in any province;

(b) the tank is inspected, tested, retested and marked in accordance with the applicable requirements for TC 350 tanks set out in clause 8 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992, and for the purpose of clause 8, the design gauge pressure of the tank, if unknown, is considered to be 100 kPa (14.5 p.s.i.);

(c) the tank is inspected, tested, retested and marked at a facility registered for those purposes in accordance with clause 9 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992; and

(d) the registered facility has permanently attached to the tank a metal plate that is marked with at

least the following information:

(i) the words "Non-spec Vacuum Tank" and "Not for Dangerous Goods Use after July 1, 1999" or "Citerne sous vide, hors spécifications" and "Inutilisable pour les marchandises dangereuses après le 1er juillet 1999",

(ii) the date of the first inspection or test done in accordance with paragraph (b),

(iii) the name of the registered facility, and

(iv) the design gauge pressure of the tank or, if unknown, 100 kPa (14.5 p.s.i.)

(5) Notwithstanding subsection (1), until January 1, 2005, the requirements of clauses 4.1 and 4.4.2, paragraph 5.1(d), clauses 6.2 to 6.4 and Table 6.1 of CSA Preliminary Standard B621-1987, Selection and Use of Highway Tanks, Portable Tanks, Cargo Compartments and Containers for the Transportation of Dangerous Goods, Classes 3, 4, 5, 6, and 8, in Bulk by Road, dated March 1987, do not apply to a tank used to transport dangerous goods included in Class 3 and Packing Group II or III,
other than Petroleum Crude Oil (UN1267), if the following conditions are met:

(a) the tank was manufactured before July 1, 1995;

(b) the tank is inspected, tested, retested and marked in accordance with the applicable requirements for TC 306 tanks set out in subsection 178.341-7 of clause 5.6 and clause 8 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992;

(c) the tank is inspected, tested, retested and marked at a facility registered for those purposes in accordance with clause 9 of CSA Preliminary Standard B620-1987, Highway Tanks and Portable

Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992; and

(d) the registered facility has permanently attached to the tank a metal plate that is marked with at least the following information:

(i) the words, "Non-spec Flammable Liquids Tank" and "Not for Dangerous Goods Use after January 1, 2005" or "Citerne hors spécifications pour liquides inflammables" and "Inutilisable pour les marchandises dangereuses après le 1er janvier 2005",

(ii) the date of the first inspection or test done in accordance with paragraph (b), and

(iii) the name of the registered facility.

(6) A tank referred to in subsection (4) shall be considered to have been tested in accordance with subsection 178.341-7 of clause 5.6 CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992 for the purposes of paragraph (4)(b), where the tank was tested at the time of manufacture

(a) in accordance with the applicable requirements for TC 306 tanks set out in subsection 178.341-7 of clause 5.6 of that Standard; and

(b) by a facility registered for that purpose at the time of manufacture or subsequently in accordance with clause 9 of that Standard.

(7) For the purposes of clause 4.1 of CSA Preliminary Standard B621-1987, Selection and Use of Highway Tanks, Portable Tanks, Cargo Compartments and Containers for the Transportation of Dangerous Goods, Classes 3, 4, 5, 6, and 8, in Bulk by Road, dated March 1987, Specification

DOT 406, DOT 407 and DOT 412 tanks manufactured in accordance with sections 178.345 to 178.348 of CFR 49 are considered to be "MC-Equivalent" to TC 306, TC 307 and TC 312 tanks respectively.

Tanks - By Rail

7.33.2 (1) Subject to subsection (3), no person shall handle or offer for transport by rail dangerous goods that are included in Class 3, 4, 5, 6.1, 8 or 9, unless the following requirements are complied with:

(a) the requirements of National Standard of Canada CAN/CGSB-43.147-94, Construction and Maintenance of Tank Car Tanks and Selection and Use of Tank Car Tanks, Portable Tanks and Rail Cars for the Transportation of Dangerous Goods by Rail, dated November 1994; or

(b) the requirements of sections 7.33.5 and 7.49.

(2) Subsection (1) does not apply to a package, a small container or an IBC.

(3) Until October 1, 1996, a person may comply with the requirements for outage for railway vehicles in clause 73.116, 73.241 or 73.344 of CGSB Provisional Standard 43-GP-147P, Construction of Tank Car Tanks and Selection and Use of Tank Car Tanks, Portable Tanks and Rail Cars for the Transportation of Dangerous Goods by Rail, dated December 1990 instead of the requirements for outage for railway vehicles in sub-clause 4.3.3(1) of National Standard of Canada CAN/CGSB-43.147-94,
Construction and Maintenance of Tank Car Tanks and Selection and Use of Tank Car Tanks, Portable Tanks and Rail Cars for the Transportation of Dangerous Goods by Rail, dated November 1994.

IBCs-By Road, Rail or Ship

7.33.3 (1) No person shall handle or offer for transport, by road or rail or in a domestic consignment by ship, dangerous goods that are included in Class 3, 4, 5, 6.1, 8 or 9, are in liquid form and are contained in an IBC referred to in subsection (4), unless the requirements of that subsection are complied with.

(2) After June 30, 1996, no person shall handle or offer for transport, by road or rail or in a domestic consignment by ship, dangerous goods that are included in Class 3, 4, 5, 6.1, 8 or 9 and are contained in an IBC referred to in subsection (5), unless the requirements of that subsection are complied with.

(3) Subsections (1) and (2) do not apply to dangerous goods that are included in Class 3 and Packing Group III, that have no subsidiary classification and that have a flash point exceeding 37.8° C.

(4) When the dangerous goods are contained in an IBC that meets the definition of a rigid polyethylene intermediate bulk container (RIBC) in clause 3 of CSA Preliminary Standard B616-M1989, Rigid Polyethylene Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated May 1989, or in a code 31HZ IBC referred to in National Standard of Canada CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated December 1994, or in an IBC similar to one of those types of IBC, the following requirements shall be complied with:

(a) the selection and use requirements for a code 31HZ IBC in section 7 of National Standard of Canada
CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated December 1994, and for the purposes of this paragraph, the date of manufacture, if not displayed on the IBC, is considered to be June 30, 1992;

(b) for an IBC manufactured after December 31, 1991 and before July 1, 1996,

(i) the design and manufacturing requirements for TC-225 IBCs in section 4 of CSA Preliminary Standard B616-M1989, Rigid Polyethylene Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated May 1989, or

(ii) the design and manufacturing requirements for code 31HZ IBCs in paragraph 5.4 and section 6 of National Standard of Canada CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated December 1994; and

(c) for an IBC manufactured after June 30, 1996, the design and manufacturing requirements for code 31HZ IBCs in sections 4 to 6 of National Standard of Canada CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated December 1994.

(5) When the dangerous goods are contained in an IBC other than those described in subsection (4), the following requirements shall be complied with:

(a) the selection and use requirements in section 7 of National Standard of Canada CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated December 1994, for the code that most appropriately describes the IBC, and for the purposes of this paragraph, the date of manufacture, if not displayed on the IBC, is considered to be January 1, 1994.

(b) for an IBC manufactured after June 30, 1996, the design and manufacturing requirements in sections 4 to 6 of National Standard of Canada CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous Goods, dated December 1994.

(6) The permitted use period requirements in clause 7.3.4 (g) of National Standard of Canada CAN/CGSB-43.146-94, Intermediate Bulk Containers for the Transportation of Dangerous

Goods, dated December 1994, do not apply until July 1, 1997.

Portable Tanks-By Road, Rail or Ship

7.33.5 (1) Subject to subsection (2), dangerous goods that are included in Class 3, 4, 5, 6.1, 8 or 9 may be handled or offered for transport by road or by rail, or in a domestic consignment by ship, if the dangerous goods are contained in

(a) a Type 1 portable tank or Type 2 portable tank as defined in the IMDG Code and the requirements of section 13 of the IMDG Code are complied with; or

(b) an IM 101 portable tank or IM 102 portable tank as defined in 49 CFR and the requirements of Subpart B of Part 172 and sections 173.32a to 173.32c of 49 CFR are complied with.

(2) Dangerous goods may be contained in a TC Type 1 or 3 portable tank instead of a Type 1 or IM 101 portable tank, or in a TC Type 2 or 3 portable tank instead of a Type 2 or IM 102 portable tank, referred to in subsection (1), where

(a) the selection and use requirements of the IMDG Code or 49 CFR, as the case may be, with respect to portable tanks are complied with; and

(b) the TC Type 1, 2 or 3 portable tank complies with CSA Preliminary Standard B620-1987, Highway Tanks and Portable Tanks for the Transportation of Dangerous Goods, dated October 1987 and amended February 1992.

Loading of Transport Units

7.37 No person shall load dangerous goods into or onto a transport unit unless the transport unit is, under normal conditions of handling and transportation, capable of safely retaining the dangerous goods loaded into or onto it and is free of any projections capable of damaging any package or small container containing the dangerous goods.

7.38 (1) Subject to subsection (2), no person shall load dangerous goods contained in any means of containment into or onto a transport unit unless the person ensures that the means of containment and any other goods loaded into or onto the transport unit are loaded or secured so as to prevent damage to the means of containment, the other goods or the transport unit under normal conditions of transportation.

(2) Where a person loads dangerous goods contained in any means of containment into or onto a transport unit for transport by rail in accordance with methods approved by the AAR, the loading of the dangerous goods shall be deemed to have been done in accordance with the requirements of subsection (1).

(3) No person shall load a transport unit into or onto a road vehicle or railway vehicle unless the person ensures that the transport unit is secured so as to prevent, under normal conditions of transportation, damage to the transport unit, road vehicle or railway vehicle.

7.39 No person shall load dangerous goods into or onto a transport unit together with foodstuffs unless the dangerous goods are separated from the foodstuffs in a manner that would prevent the contamination of the foodstuffs in the event of a discharge, emission or escape of the dangerous goods.