VOLUNTARY CLEANUP REPORT
FRANK WEAR CLEANERS
106 South Third Avenue
Yakima, Washington

Project Number 00-163

May 3, 2001

Prepared for: Greg Stoffers
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1.0 INTRODUCTION

Fulcrum Environmental Consulting, Inc. (Fulcrum) was retained by Greg Stoffers to complete an Independent Remedial Action of an area of assumed soil contamination. Suspect contaminants included tetrachloroethylene (PCE) and associated breakdown products including trichloroethylene (TCE), cis 1-2 Dichlorethylene (DCE), and Vinyl Chloride. The subject site is the former Frank Wear Cleaners facility located at 106 South Third Avenue, in Yakima Washington. See Figure 1 for subject site location. Transglobal Environmental Geosciences Northwest, Inc. (TEG) was subcontracted by Fulcrum to provide on-site analytical services. Tri-Valley Construction (Tri-Valley) was contracted independent of Fulcrum to provide excavation and soil transportation services.

2.0 SCOPE OF WORK

Purpose of this independent remedial action was to investigate the site for subsurface USTS, piping, or drywells that could result in PCE impact to site soils or groundwater, to investigate site soils for PCE impact, and to remediate any identified PCE impacted soils through removal and off-site disposal. Towards that end, Ecology indicated that Mr. Stoffers needed to demonstrate that all contaminated soil had been removed from the site, and that the site was no longer adversely impacting groundwater.

The scope of this project was limited to soil investigation and remediation activities associated with assumed PCE impact soils at the former Frank Wear Cleaners facility located at 106 South Third Avenue. Groundwater sampling was not included within project scope of services. Site investigation services included a pre-remediation coordination meeting with representatives of the Washington State Department of Ecology (Ecology); preparation of a Site Sampling Plan and Site Health and Safety Plan (see Appendix A); coordination of on-site activities with Ecology, the analytical laboratory, excavation contractor, and property owner; oversight of excavation activities; characterization of concrete flooring material remaining on-site, limited evaluation of Ecology provided groundwater monitoring results, and completion of a voluntary cleanup report. Scope of services was based in large part upon information provided by the property owner and in previous reports submitted to Ecology.

Fulcrum made no effort to independently verify accuracy or completeness of supplied data. Fulcrum makes no warranties expressed or implied as to the accuracy or completeness of other's work included or referenced herein.

3.0 DISCUSSION OF PERTINENT REGULATIONS AND GUIDANCE

3.1 MTCA Regulations

In March of 1989, the Model Toxics Control Act (MTCA) went into effect in Washington. The MTCA regulations set standards to ensure quality of cleanup and protection of human health and the environment. A major portion of the MTCA regulation (completed in 1991) was the development of numerical cleanup standards and requirements for cleanup actions. MTCA established three options for site-specific cleanup levels: Method A, B, and C. Method A defines cleanup levels for 25 of the most common hazardous substances found at sites. Method B levels are set using a site risk assessment, which enables consideration of site-specific characteristics. Method C is similar to Method B, however, the individual substances' cancer risk portion of the assessment is set at 1 in 100,000 rather than 1 in 1,000,000.

3.2 Proposed Cleanup Standards

Site cleanup criteria and associated cleanup actions will be established consistent with WAC 173-340, the Model Toxics Control Act Cleanup Regulation, and Ecology's recommended Yakima Railroad Area soil cleanup levels. Applicable cleanup levels for PCE and its breakdown products are presented in Table 1.

Table 1: Cleanup Levels

Contaminant	Soil			Groundwater		
	RR Area[1]	Method B[2]	Method A[3]	Method B	Method A	MCL[4]
Tetrachloroethylene (PCE)	19 ppb	85.8 ppb	500 ppb	.858 ppb	5 ppb	5 ppb
Trichloroethylene (TCE)	11 ppb	398 ppb	500 ppb	3.98 ppb	5 ppb	5 ppb
cis 1-2 Dichloroethylene (DCE)	29 ppb	800 ppb	NA	80 ppb	NA	70 ppb
Vinyl Chloride	2 ppb	2.3 ppb	NA	.023 ppb	NA	2 ppb

1 RR = Yakima Railroad Area recommended soil cleanup levels protective of groundwater
2 Method B = Model Toxics Control Act Method B Cleanup Level
3 Method A = Model Toxics Control Act Method A Cleanup Level
4 MCL = Maximum Contaminant Level

Yakima Railroad Area soil cleanup levels protective of groundwater have been selected as the target cleanup levels for this project.

4.0 SITE HISTORY

Information reviewed in Central Washington Regional Ecology's files indicate that the Frank Wear Cleaners site has been occupied by a dry cleaning operation from the early 1940s until the late 1990s. A site investigation conducted by Ecology in 1985 indicated that PCE was being released to a gravel area in the back lot (west of the concrete pad). Ecology performed a second site inspection in 1987, which also indicated that dangerous waste was being discharged into the environment.

Ecology conducted a preliminary assessment of the Frank Wear Cleaners site in 1989. Ecology recommended further investigation including soil borings and groundwater sampling. Two pit samples (unknown depth) collected by Ecology in 1989 were reported to have PCE concentrations of 660 parts per billion (ppb) and 10,000 ppb respectively.

PLSA Engineering and Surveying also conducted a preliminary investigation in 1989. They collected two soil samples (independent of Ecology's activities) with reported PCE concentrations of 3,000 ppb and 63 ppb.

In 1991, Ecology named the Frank Wear Cleaners site as one of the potentially liable parties (PLPs) to the Yakima Railroad Area, which is an area of widespread groundwater contamination located in general proximity to the rail line passing through central Yakima, Washington.

In 1995, Maxim Technologies, Inc., (Maxim) performed site investigation/remediation activities. Maxim oversaw installation of four on-site monitoring wells and 11 test pits. Analytical results confirmed PCE contamination of site groundwater and soils including soils beneath the building footprint. Numerous surface soil samples were also collected. Approximately 610 tons of soil was removed from the gravel area

west of the building footprint. Approximately 310 tons was found to be above applicable cleanup levels (determined to be 80 ppm) and was transported to the Rabanco landfill near Roosevelt Washington for disposal. The remaining 300 tons was determined to be below the cleanup level and placed back in the excavation.

In 1996, Ecology issued an Agreed Owner to Frank Wear Cleaners requiring a remedial investigation, feasibility study, implementation of feasibility study activities, and submittal of groundwater sampling data. As a result, in 1997, Environmental Economic Solutions, Inc., (EES) oversaw installation of one off-site, up-gradient groundwater monitoring well, installation and sparge testing of one gas spargepoint/well, and soil and groundwater sampling of the installed wells. Groundwater samples were also collected from pre-existing monitoring wells. Sampling confirmed presence of PCE in site soils and site groundwater. EES oversaw installation of a KVA Analytical Systems "C-Sparge" and controller system for sparging of site groundwater with ozone. EES estimated groundwater remediation would be achieved in 6 to 12 months. Groundwater sparging was discontinued in 1998.

Groundwater monitoring data provided by Ecology indicates that quarterly sampling from February of 1995 to August of 2000 (including sampling during and after sparging) confirms PCE contamination in site groundwater in excess of regulatory limits. Concentrations appear to be generally decreasing over time.

In 2000, Greg Stoffers arranged for demolition of the on-site building to facilitate investigation and remediation of suspect contaminated soil beneath the building footprint. Demolition was performed by Tri-Valley using 40 hour hazardous materials trained workers.

In November of 2000, a meeting was held between Greg Stoffers, the site owner, Travis Trent of Fulcrum Environmental Consulting, Inc., (Fulcrum) and Rick Roeder with Ecology, to discuss means of final site closure. Mr. Stoffers indicated that the on-site building had been demolished and that he was preparing to have the remaining on-site contaminated soil excavated, characterized, and disposed under Fulcrum's direction. Mr. Roeder indicated that removal of all site soil containing PCE in excess of 20 ppb has been a significant step toward achieving final site closure at other PCE contaminated locations in the Yakima area. Mr. Roeder indicated that as a minimum, a period of post removal groundwater monitoring would be required to confirm absence of groundwater contamination before Ecology would accept final site closure or issue a no further action (NFA) determination for the site.

5.0 ENVIRONMENTAL SETTING

The subject site is located just west of the downtown area of Yakima in a commercialized area. The property is located in the N.E. ¼ of the S.E. ¼ of Section 24, Township 13 North, Range 18 East of the Willamette Meridian, Yakima County, Washington. Site elevation is approximately 1,077 feet above mean sea level. Site geology is characterized by unconsolidated to poorly consolidated sand and gravel deposits. Groundwater depth and flow direction is strongly influenced by seasonal variations and irrigation activities. Groundwater depth is estimated at 13 to 20 feet. Based on Ecology quarterly Railroad Corridor monitoring, groundwater flow direction in the near surface sedimentary aquifer is generally southeast with some seasonal variation in more southerly or easterly directions.

Regionally the subject site is located within the Yakima Folds Geomorphic Province on the western margin of the Columbia River Plateau. The Columbia River Basalt Group is comprised of a number of formations. The three youngest formations of the Columbia River Basalt Group are present in the Yakima region. These basalt formations and the interbedded and overlying sedimentary lithologies of the Ellensburg Formation

comprise the near surface stratigraphy of the Yakima Region. Quaternary alluvial sediments and landslide deposits are present in valley environments. Anticlinal ridge and synclinal valley structures of the Yakima Fold Belt dominate topography.

According to Biggane, 1982, two regional aquifers are known to be present in the Yakima Area. The two regional aquifers are loosely characterized as the sedimentary aquifer and the basalt aquifer. Both regional aquifers consist of a large number of water bearing subunits. The sedimentary aquifer typically overlies the basalt aquifer except in regions where the basalt aquifer is exposed at land surface. Water bearing subunits and the regional aquifers are hydraulically connected.

The sedimentary aquifer is composed of Upper Ellensburg and Quaternary sedimentary units. Water bearing units of the sedimentary aquifer vary lithologically, typically are not laterally extensive, and demonstrate heterogeneous and anisotropic water transmission properties. Groundwater occurs in perched, unconfined and confined conditions. Recharge to the sedimentary aquifer occurs through infiltration from precipitation and irrigation and from influent portions of irrigation canals, local streams, and rivers. Recharge also occurs via flow from the underlying basalt aquifer. The sedimentary aquifer discharges to effluent reaches of local streams and rivers as well as to the underlying basalt aquifer.

The basalt aquifer is composed of basalt flows and sedimentary interbeds. Sedimentary interbeds vary lithologically, typically are not laterally extensive, and demonstrate heterogeneous and anisotropic water transmission properties. Sedimentary interbeds act as regional aquitards in some areas although the extent of interbed members is unknown. Recharge to the basalt aquifer occurs via infiltration from precipitation and irrigation where the basalt aquifer is exposed at land surface. Recharge also occurs via flow from the overlying sedimentary aquifer. The basalt aquifer discharges to effluent reaches of local streams and rivers as well as to the overlying sedimentary aquifer.

6.0 SITE INVESTIGATION

Travis Trent of Fulcrum oversaw on-site investigation and remediation activities on February 13, and 14, 2001. Tri-Valley was on-site to perform soil excavation activities. TEG was on-site with a mobile laboratory to provide analytical services. Site investigation began with a review of the site health and safety plan, discussion of site specific hazards, and review of proposed sampling/investigation activities.

Prior to start of site investigation activities, Fulcrum confirmed areal extent of previous remedial activities conducted under Maxim's direction in 1995. Tri-Valley was the excavation contractor and Greg Stoffers was the property owner during the 1995 work. Greg Huylar of Tri-Valley and Greg Stoffers were both on-site during the 1995 work and were able to confirm the 1995 excavation/remedial area. This area was generally excluded from this investigation, although limited excavation and confirmation sampling was performed to confirm concentration of soil returned to the excavation during the 1995 remediation.

Through discussions with Greg Stoffers (property owner) and Rick Roeder of Ecology, Fulcrum identified a former drywell area, former dry cleaning machine area, former shed area, and piping located along the northern property border as the areas of greatest concern. Mr. Roeder also expressed concern about the possibility of an underground storage tank (UST) being present on the subject site. During excavation, a northeast/southwest trending abandoned 4 inch diameter line was encountered in the central portion of the property at 4 ft bgs. In addition to the areas of concern identified above, the investigation included excavation and sampling determined by Fulcrum to be representative of areas of the subject site not specifically targeted or previously investigated.

6.1 February 13, 2001 Site Investigation

Excavation began on February 13, 2001, with a 1 to 2 ft lift in each area of concern (see figure 3). Exposed soil was evaluated for any changes in texture, odor, or appearance. Samples were collected and submitted to TEG's on-site laboratory for specific Halogenated and Aromatic Hydrocarbon (EPA 8021B) analysis. Areas shown through analysis to contain detectable quantities of PCE were over-excavated and then re-sampled at greater depth until analytical results were non-detect. Soil excavated from any area with analytical results above the detection limit was placed on plastic pending off-site disposal. The only analyte detected under EPA 2081B was PCE. Analytical results for February 13, 2001 soil sampling are presented in Table 2.

Table 2: Analytical Results for 2/13/01 Soil Sampling

Sample Number	Location/Depth	Analytical Results (ppm)
Method Blank		ND
FW0213-01	Former dry cleaning machine area, 2 ft bgs	ND
FW0213-01 D.	Duplicate analysis of sample FW0213-01	0.06
FW0213-02	Former boiler room, 1.5 ft bgs	ND
FW0213-03	Former shed area, 1 ft bgs	0.23
FW0213-04	Former dry cleaning machine area, below sample FW0213-01, 3 ft bgs	ND
FW0213-05	Former drywell area, 2 ft bgs	0.19
FW0213-06	North boundary piping area, 1.5 ft bgs	ND
FW0213-07	North boundary piping area, 1 ft bgs	0.11
FW0213-08	Former shed area, below sample FW0213-03, 3 ft bgs	0.13
FW0213-09	Former shed area, below sample FW0213-03 and 08, 5 ft bgs	ND
FW0213-09 D.	Duplicate analysis of sample FWO213-09	ND
FW0213-10	Former drywell area, below sample FW0213-05, 3 ft bgs	ND
FW0213-11	North boundary piping area, below sample FW0213-07, 3 ft bgs	ND
FW0213-12	Former shed area, 5 ft bgs	ND
FW0213-13	Center of property, 3 ft bgs	0.15
FW0213-14	Center of property, below sample FW0213-13, 7 ft bgs	ND
FW0213-15	Center of property, 4 ft bgs	0.10
FW0213-16	Center of property, below sample FW0213-15, 9 ft bgs	ND

ppm = parts per million
Note: EPA 8021B Method Detection Limit for PCE = 0.05 ppm.
Laboratory QA/QC sample results were within acceptable recover limits.

The February 13, 2001 analytical results document detectable PCE concentrations in 4 localized areas. Excavation and sampling continued in each area until EPA 2081B sample results were non-detect. Based upon analytical results and areal extent of excavation and sampling, Fulcrum and Greg Stoffers determined that a second day of excavation and sampling would be required to characterize and remediate soil at the subject site.

6.2 February 14, 2001 Site Investigation

The second day of site excavation (February 14, 2001) began with additional excavation of the central area to evaluate potential for impact at depth. Excavation and sampling continued in the eastern area of the subject site, below the former building footprint. Concrete was removed as necessary to facilitate sample collection. Exposed soil was evaluated for any changes in texture, odor, or appearance. Samples were

collected and submitted to TEG's on-site laboratory for specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) analysis. Areas shown through analysis to contain detectable quantities of PCE were over-excavated and then re-sampled at greater depth until analytical results were non-detect. Soil excavated from any area with analytical results above the detection limit was placed on plastic pending off-site disposal. The only analyte detected under EPA 2081B was PCE. Analytical results for February 14, 2001 soil sampling are presented in Table 3.

Excavation along the east property boundary was performed as close to the sidewalk as possible to assist in evaluating potential for presence of a UST. No visual indication of UST presence, such as tank sidewalls or piping, was noted.

Table 3: Analytical Results for 2/14/01 Soil Sampling

Sample Number	Location/Depth	Analytical Results (ppm)
Method Blank		ND
FW0214-01	Center of property, 10 ft bgs	ND
FW0214-02	Center of property, 9 ft bgs	ND
FW0214-03	Center of property, 9 ft bgs	ND
FW0214-04	North property boundary, 3.5 ft bgs	ND
FW0214-05	Northeast corner of property, 2 ft bgs	ND
FW0214-06	North property boundary, 2 ft bgs	ND
FW0214-07	East end of north property boundary, 2 ft bgs	ND
FW0214-07 D.	Duplicate analysis of FW0214-07	ND
FW0214-08	East end of property, 1 ft bgs	.290
FW0214-09	South end of property, 1 ft bgs	1.500
FW0214-10	Former shed area, 3 ft bgs	ND
FW0214-11	Center of property, 2.5 bgs	ND
FW0214-11 D.	Duplicate analysis of sample FW0214-11	ND
FW0214-12	South end of property, 3 ft bgs	ND
FW0214-13	East end of property, 3 ft bgs	0.21
FW0214-14	South end of property, below sample FW0214-09	ND
FW0214-15	East end of property, 1.5 ft bgs	ND
FW0214-16	Composite sample of concrete	ND
FW0214-17	East end of property, below sample FW0214-08, 5 ft bgs	ND
FW0214-18	East end of property, 3 ft bgs	ND
FW0214-19	East end of property, below sample FW0214-13	ND

ppm = parts per million
Note: EPA 8021B Method Detection Limit for PCE = 0.05 ppm
Laboratory QA/QC sample results were acceptable recover limits

February 14, 2001 analytical results document PCE concentrations above the method detection limit in 3 localized areas. Excavation and sampling continued in each area until EPA 2081B sample results were non-detect. Based upon analytical results and areal extent of excavation and sampling all PCE impacted soil had been excavated and stockpiled on plastic pending off-site disposal. Fulcrum and Greg Stoffers determined that no further investigation was necessary to characterize or remediate soil at the subject site.

7.0 WASTE DISPOSAL

Approximately 432 tons of excavated soil was determined to contain PCE concentrations below MTCA Method A cleanup levels but potentially above the target Railroad area threshold values. Through discussion with Ecology, it was determined that this soil would could be conservatively characterized as state only problem waste. The 432 tons of PCE impacted soil was transported to Columbia Asphalt & Gravel for recycling with verbal approval from Ecology and The Yakima Regional Health District. See Appendix D for soil disposal receipts.

A composite sample (FW0214-16) of concrete from the former building floor was collected from three locations. Concrete samples were preferentially selected from areas formerly occupied by dry cleaning equipment or areas overlying soil with detectable quantities of PCE. Analytical results from the composite concrete sample were non-detect for PCE concentrations. Concrete was determined to be solid waste and was transported to Andersons Rock and Demolition landfill for disposal.

No other suspect hazardous waste was generated during this investigation.

8.0 EVALUATION OF DATA

8.1 Soil Analytical Results

Analytical results document PCE concentrations above the method detection limit in 7 localized areas of the subject site. Detected concentrations ranged from 0.06 ppm to 1.5 ppm with the average concentration for the 10 samples with detectable levels of PCE being 0.297 ppm. Highest concentrations detected where in the upper 1 ft bgs. Concentrations decreased with depth. Only one sample with detectable concentrations of PCE was found at depths greater than 3 ft bgs. No PCE was detected in any samples collected below 4 ft bgs.

All areas with detectable concentrations of PCE where over-excavated and re-sampled until sample results were non-detect for PCE concentration. Excavation and sampling was performed to 10 ft bgs in the center of the property to evaluate potential for PCE impact at depth. This area was selected based upon presence of PCE in near surface samples and proximity to the drywell area and the area where piping was found. Samples collected at 9 to 10 ft bgs were non-detect for PCE. Sample results for all areas of soil remaining on the subject site were non-detect for PCE. Analytical results confirm that all areas of soil with detectable concentrations of PCE were excavated and removed from the subject site.

8.2 Groundwater Analytical Results

No groundwater sampling was conducted by Fulcrum as part of this investigation. Groundwater monitoring has been conducted on and adjacent to the subject site under Ecology's oversight since 1995. The investigation outlined in this report was specifically scheduled to fall between Ecology's previously scheduled quarterly groundwater sampling events. Groundwater analytical results provided by Ecology are presented in Table 4.

Table 4: Ecology Groundwater Monitoring Results for PCE

Date	FW-MW-1	FW-MW-2	FW-MW-3	FW-MW-4	FW-MW-5	Cleanup level[1]
2/22/95	66	210.02	150	1.7		5
4/20/95	1140	109	5	18		5
9/6/95	23.9	8.8	11.5	6		5
12/26/95	298	605	1080	332		5
12/5/97	400	54	42	1100	83	5
3/4/98	830	72	860	210	390	5
6/4/98	690	110	16	280	120	5
8/31/98	33	39	29	34	17	5
6/3/99	1100	94	13.8	530	90.9	5
9/8/99	150	31.5	12.4	51.7	19.1	5
12/8/99	193	28.1	28.5	139	69.1	5
3/9/00	13.6	71	37.6	700	103	5
6/8/00	320	36	18	52	25	5
8/29/00	20	12	17	11	6.6	5
12/00	150	13	30	23	41	5
3/00	17	9.6	34	876	17	5

1 MTCA Method A cleanup level for PCE
Results are reported in parts per billion
Note: PCE concentration data has been supplied by Ecology and was not independently verified or checked by Fulcrum

Monitoring well 04 is located southwest and downgradient of the subject site. Monitoring well 02 is located on the west-end of the subject site and monitoring well 05 is located northeast and upgradient of the subject site. Data provided is insufficient to determine whether monitoring wells 01 and 03, located to the south and west of the subject site respectively, are cross gradient or downgradient of the subject site. Railroad area data suggests that MW-01 is down gradient part of the year and cross gradient the remainder. Railroad area data suggests that MW-03 is cross gradient. Well locations are shown on Figure 2.

On-site concentrations (MW-02) are generally lower than upgradient concentrations (MW-05). The data provided is insufficient to determine whether PCE concentrations in MW-01, 03, and 04 reflect adverse environmental impact from the subject site.

9.0 CONCLUSIONS AND RECCOMENDATIONS

Purpose of this independent remedial action was to investigate the site for subsurface USTS, piping, or drywells that could result in PCE impact to site soils or groundwater, to investigate site soils for PCE impact, and to remediate any identified PCE impacted soils through removal and off-site disposal. Towards that end, Ecology indicated that Mr. Stoffers needed to demonstrate that all contaminated soil had been removed from the site, and that the site was no longer adversely impacting groundwater. The scope of this project was limited to soil investigation and remediation activities associated with assumed PCE impact soils at the former Frank Wear Cleaners facility located at 106 South Third Avenue.

9.1 Subsurface Investigation

A subsurface investigation was performed across all suspect areas of the subject site not excavated during the 1995 investigation. Purpose was to determine if a subsurface structure, such as UST, utility piping, or drywell, capable of causing PCE impact the site soils or groundwater. A 6" pipe was uncovered in the center of the property, which terminated at gravel at both ends (approximately 15 ft long). Mr. Stoffers indicated that pipe location was consistent with the location of a former sewer line that had been historically replaced. A former drywell area was also excavated during the investigation. The drywell had historically used to address stormwater runoff from the west end of the subject site. A sump area in the former boiler room was also excavated and investigated. No USTs or related piping such as vent line, fill lines, or fuel lines were found on the subject site.

Excavation along the east property boundary was performed as close to the sidewalk as possible to assist in evaluating potential for presence of a UST. No visual indication of UST presence, such as non-native fill, tank sidewalls or piping was noted. No patched areas of concrete were present in the sidewalk area to suggest historic UST presence. The site owner/operator indicated that to the best of his knowledge, no USTs were present under the east sidewalk area. Given the limited distance from the excavation to the street, consistent native fill, and absence of any UST related piping such as fill or vent lines, it is unlikely that USTs are present under the sidewalk at the east end of the subject site.

This investigation finds it extremely unlikely that a UST or other potentially contributing structure, is present on the subject site or under the sidewalk at the east-end of the subject site. Fulcrum recommends no further investigation to determine potential for presence of on or near site PCE impacting structures.

9.2 Soil

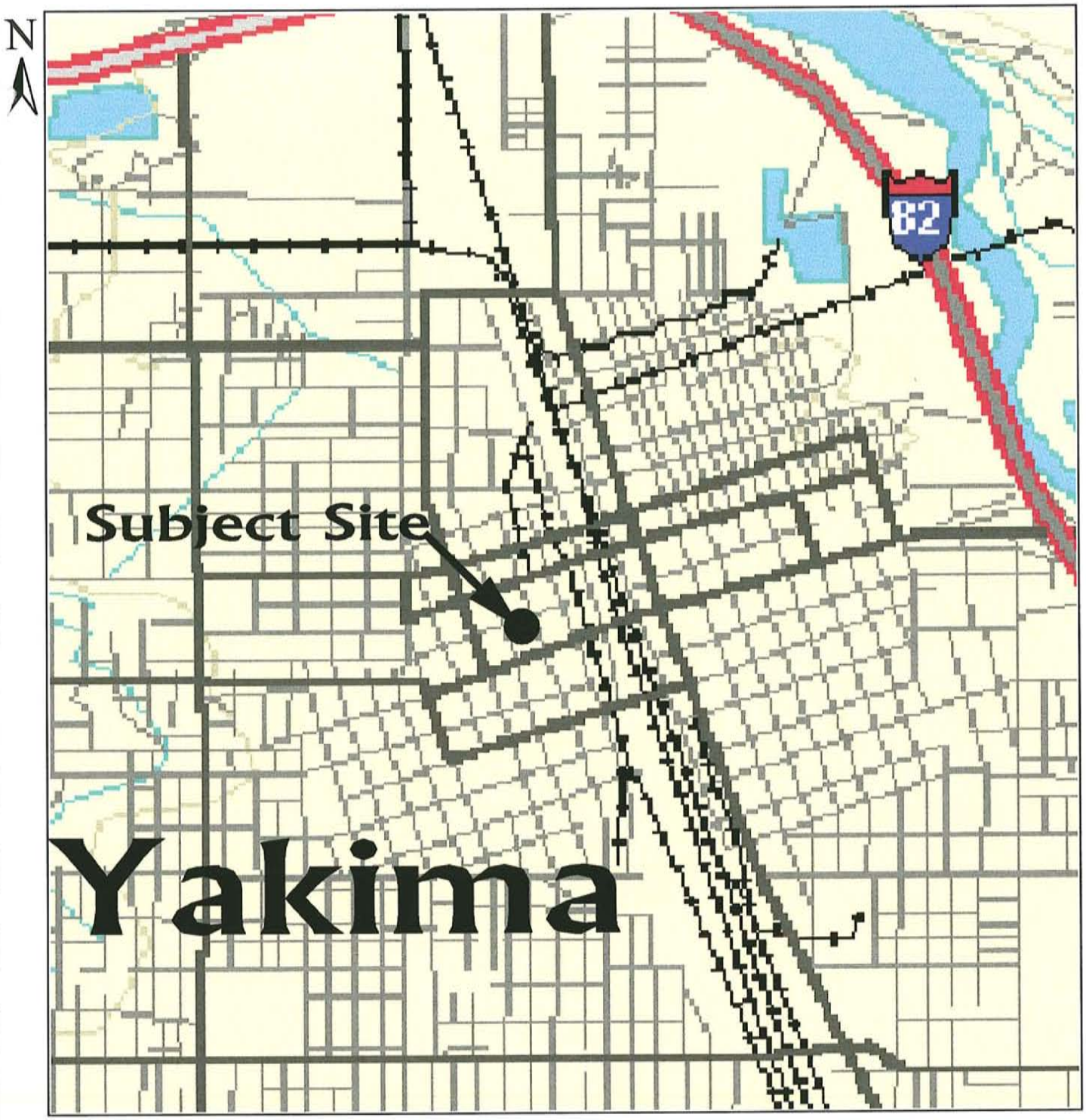
This investigation identified localized areas of low concentration PCE impacted soil from 0 to 4 ft bgs. Only one localized area of less than 1 cubic yard was identified with PCE concentrations above the MTCA Method A cleanup level. No analyte included under EPA 2081B, besides PCE, was detected. All areas of impacted soil were over-excavated and re-sampled until analytical results were non-detect for PCE. Approximately 423 tons of low concentration PCE impacted soil was removed from the subject site in addition to the approximate 310 tons removed during the 1995 investigation. Samples from all remaining areas of the subject site, including sampling at depth (9 to 10 ft bgs) were non-detect for PCE concentrations. Absence of detectable concentrations of PCE in near surface samples suggest a very low probability of connection between site soils and PCE concentrations in adjacent groundwater. Focused sampling in areas most likely to have been impacted, combined with the concentration of sample locations over the limited areal extent of the subject site suggest a very low probability that any areas of impacted soil remain on the subject site. Fulcrum recommends no further investigation to assess potential for PCE impacted soil at the subject site.

9.3 Groundwater

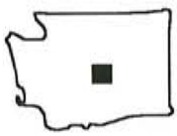
No groundwater sampling was conducted as part of this investigation. Evaluation of the subject site's impact was limited under the scope of services to review of Ecology provided pre and post-investigation data. Review of Ecology provided data to evaluate whether the subject site is the source of PCE impact to adjacent groundwater is inconclusive.

Generally lower concentrations in the on-site monitoring well than the off-site upgradient well do not support the premise that the subject site is the source of groundwater contamination. Information provided by Ecology was insufficient to determine whether MW-01 and MW-03 are hydrologically downgradient of the subject site. However, Railroad area data suggests that for at least part of the year, these wells are located cross gradient to the subject site. Review of pre and post groundwater monitoring results suggest that excavation and removal of low concentration PCE impacted soil resulted in no significant change to PCE concentrations from the same season during the previous year.

Fulcrum recommends that quarterly monitoring results continue to be evaluated for effect of suspect source removal. Fulcrum also recommends further investigation be conducted to evaluate hydrologic gradient and potential for off-site sources of PCE impact to near site groundwater.



LEGEND



Subject Site: ●

Scale: 1 mile

Contour Interval: 50 feet

Source: DeLorme 3-D Topoquad Maps 1:40 625

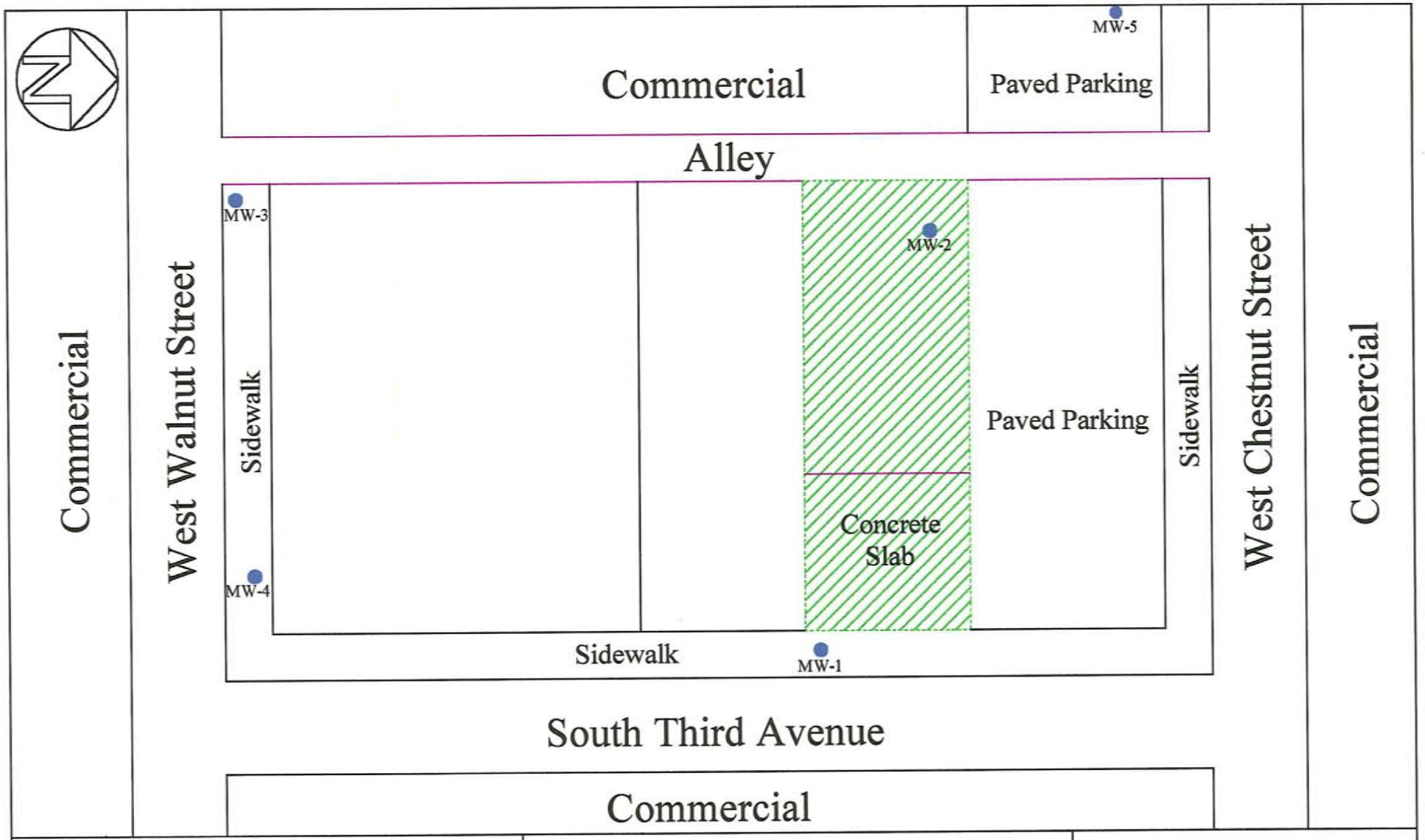
FIGURE 1

Site Location Map

Frank Wear Cleaners
106 South Third Avenue
Yakima, Washington 98902

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
509-574-0839

DRAWN BY: AMP	PROJECT NUMBER: 01-165
DATE: 04/20/2001	FILE NAME: Frank Wear Cleaners



LEGEND

Subject Site:
Property Boundary:
Monitoring Wells:
Scale: NTS

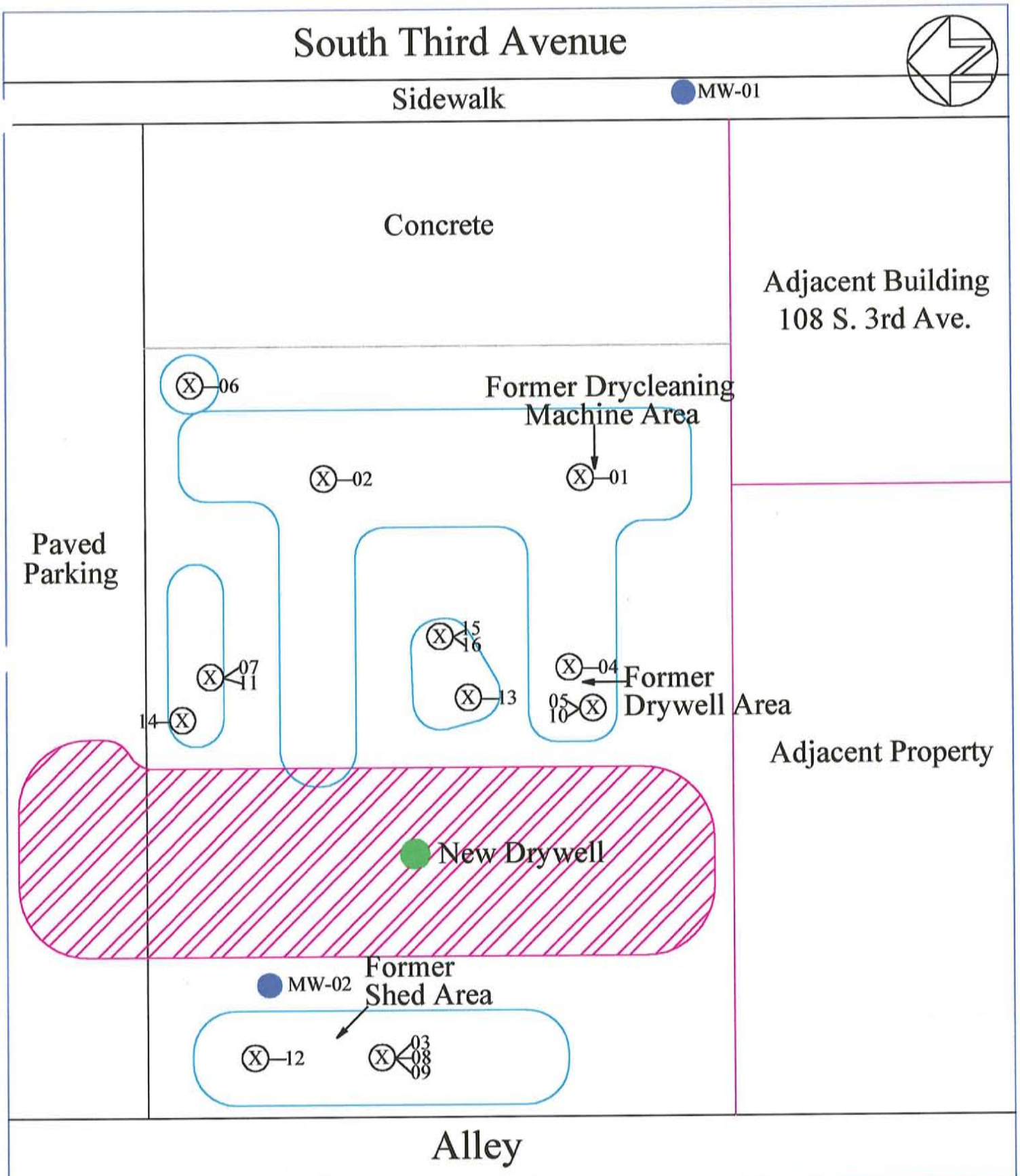
FIGURE 2

Site Layout Map

Frank Wear Cleaners
106 South Third Avenue
Yakima, Washington

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
Phone(509) 574-0839 Fax(509) 575-8453

DRAWN BY: AMP	DATE: 01/17/2001
FILE NAME: Frank Wear Cleaners	
PROJECT NUMBER: 01-165	



Legend

Sample Locations: FW0213 - ##
Excavation Sites:
Prior Excavation:
Scale: NTS

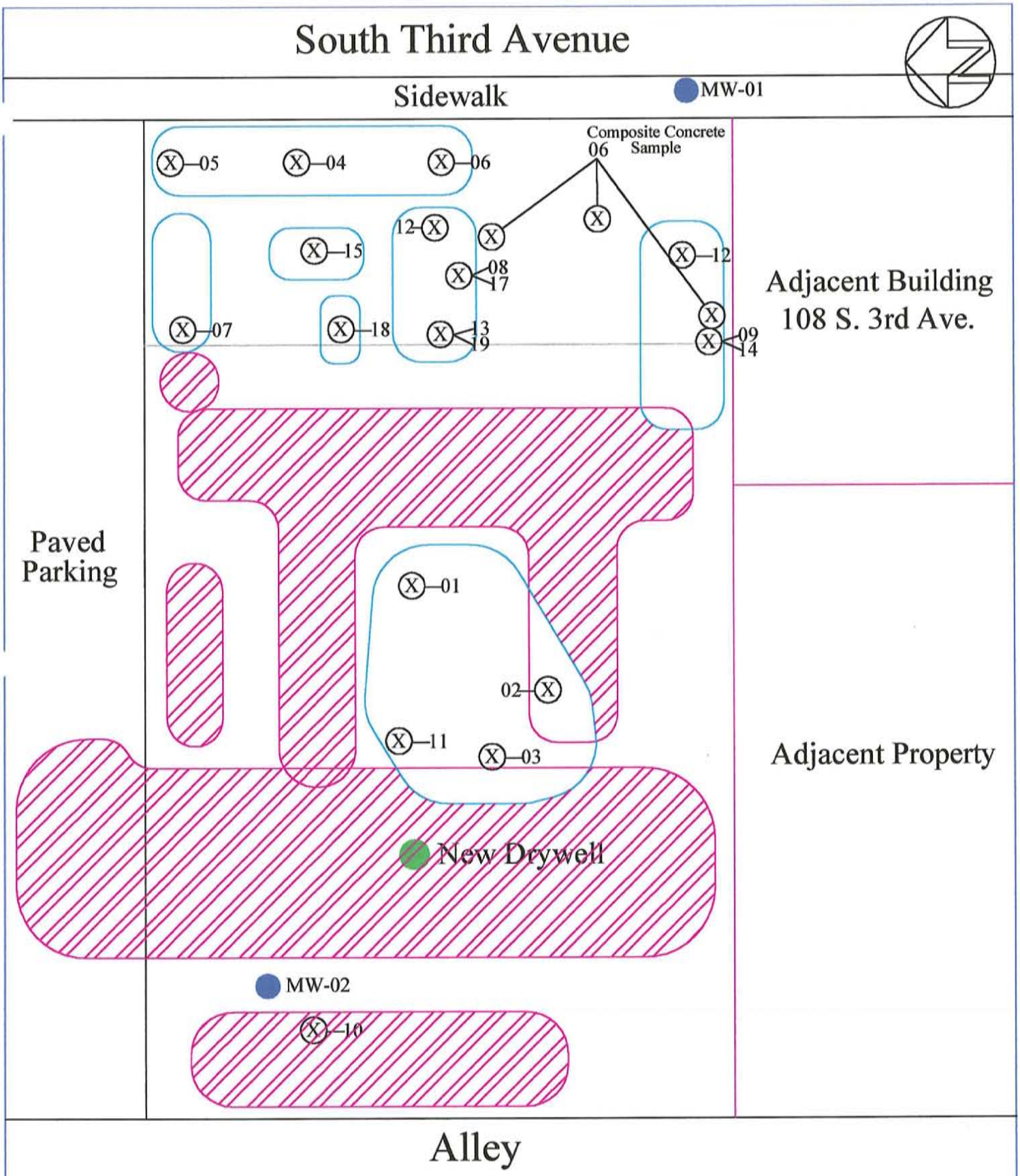
Figure 3

2/13/01 - Sample Location Map

Frank Wear Cleaners Site
106 South Third Avenue
Yakima, Washington

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
Phone (509) 574-0839 Fax (509) 575-8453

Drawn by: AMP	Project Number: 01-165
Date: 04/20/2001	File Name: Frank Wear Cleaners



Legend

Sample Locations: FW0213 - ##
Excavation Sites:
Prior Excavation:
Scale: NTS

Figure 4

2/14/01 - Sample Location Map

Frank Wear Cleaners Site
106 South Third Avenue
Yakima, Washington

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
Phone (509) 574-0839 Fax (509) 575-8453

Drawn by: AMP	Project Number: 01-165
Date: 04/20/2001	File Name: Frank Wear Cleaners

APPENDIX A

SITE DRAFT SAMPLING PLAN

Frank Wear Cleaners Site
Draft Site Sampling Plan
Yakima, Washington

January 30, 2001

Prepared for: Greg Stoffers
Stoffers Martinizing Dry Cleaning
812 Summitview Avenue
Yakima, Washington, 98902
(509) 248-6071

Prepared by: Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
(509) 574-0839

Authored by: _____ Date:_____

Travis Trent, RPG
Environmental Geologist
Fulcrum Environmental Consulting, Inc.

Reviewed by: _____ Date:_____

Peggy Williamson, CHMM
Central Washington Regional Manager
Fulcrum Environmental Consulting, Inc.

TABLE OF CONTENTS

1.0 Introduction

Purpose of this sampling and analysis plan is to facilitate excavation and characterization of an area of soil contaminated by tetrachloroethylene (PCE) and associated breakdown products including trichloroethylene (TCE), cis 1-2 Dichlorethylene (DCE), and Vinyl Chloride. Total investigation area is approximately 2,000 square feet. Subject site is located at 106 South Third Avenue in Yakima, Washington. The subject site encompasses approximately 6,300 square feet (ft^2) and was formerly occupied by a single story building occupied by a dry cleaning business called Frank Wear Cleaners. The building has been demolished and the site currently consists of an approximately 2,800 ft^2 rectangular concrete pad (floor of the demolished building) and an approximately 3,500 ft^2 gravel area. See Figure 1 for location of the Frank Wear Cleaners site.

2.0 Environmental Setting

The subject site is located just west of the downtown area of Yakima in a commercialized area. The property is located in the N.E. ¼ of the S.E. ¼ of Section 24, Township 13 North, Range 18 East of the Willamette Meridian, Yakima County, Washington. Site elevation is approximately 1,077 feet above mean sea level. Site geology is characterized by unconsolidated to poorly consolidated sand and gravel deposits. Groundwater is estimated at 13 to 20 feet and is strongly influenced by seasonal variations and irrigational activities.

Regionally the subject site is located within the Yakima Folds Geomorphic Province on the western margin of the Columbia River Plateau. The Columbia River Basalt Group is comprised of a number of formations. The three youngest formations of the Columbia River Basalt Group are present in the Yakima region. These basalt formations and the interbedded and overlying sedimentary lithologies of the Ellensburg Formation comprise the near surface stratigraphy of the Yakima Region. Quaternary alluvial sediments and landslide deposits are present in valley environments. Anticlinal ridge and synclinal valley structures of the Yakima Fold Belt dominate topography.

According to Biggane, 1982, two regional aquifers are known to be present in the Yakima Area. The two regional aquifers are loosely characterized as the sedimentary aquifer and the basalt aquifer. Both regional aquifers consist of a large number of water bearing subunits. The sedimentary aquifer typically overlies the basalt aquifer except in regions where the basalt aquifer is exposed at land surface. Water bearing subunits and the regional aquifers are hydraulically connected.

The sedimentary aquifer is composed of Upper Ellensburg and Quaternary sedimentary units. Water bearing units of the sedimentary aquifer vary lithologically, typically are not laterally extensive, and demonstrate heterogeneous and anisotropic water transmission properties. Groundwater occurs in perched, unconfined and confined conditions. Recharge to the sedimentary aquifer occurs through infiltration from precipitation and irrigation and from influent portions of irrigation canals, local streams, and rivers. Recharge also occurs via flow from the underlying basalt aquifer. The sedimentary aquifer discharges to effluent reaches of local streams and rivers as well as to the underlying basalt aquifer.

The basalt aquifer is composed of basalt flows and sedimentary interbeds. Sedimentary interbeds vary lithologically, typically are not laterally extensive, and demonstrate heterogeneous and anisotropic water transmission properties. Sedimentary interbeds act as regional aquitards in some areas although the extent of interbed members is unknown. Recharge to the basalt aquifer occurs via infiltration from precipitation and irrigation where the basalt aquifer is exposed at land surface. Recharge also occurs via flow from the overlying sedimentary aquifer. The basalt aquifer discharges to effluent reaches of local streams and rivers as well as to the overlying sedimentary aquifer.

3.0 Site Background

Information reviewed in Central Washington Regional Ecology's (Ecology) files indicates that the Frank Wear Cleaners site has been occupied by a dry cleaning operation from the early 1940s until the late 1990s. A site investigation conducted by Ecology in 1985 found that PCE was being released to a gravel area in the back lot (west of the concrete pad). Ecology performed a second site inspection in 1987 which also indicated that dangerous waste was being discharged to the environment.

Ecology conducted a preliminary assessment of Frank Wear Cleaners in 1989. Ecology recommended further investigation including soil borings and groundwater sampling. Two pit samples (unknown depth) collected by Ecology in 1989 were reported to have PCE concentrations of 660 parts per billion (ppb) and 10,000 ppb respectively.

PLSA Engineering and Surveying also conducted a preliminary investigation in 1989. They collected two soil samples (independent of Ecology's activities) with reported PCE concentrations of 3,000 ppb and 63 ppb.

In 1991, Ecology names the Frank Wear Cleaners site one of the potentially liable parties (PLPs) to the Yakima Railroad Area, which is an area of widespread groundwater contamination located in general proximity to the rail line passing through central Yakima, Washington.

In 1995, Maxim Technologies, Inc., (Maxim) performed site investigation/remediation activities. Maxim oversaw installation of four on-site monitoring wells and 11 test pits. Analytical results confirmed PCE contamination of site groundwater and soils, including soils beneath the building footprint. Numerous surface soil samples were also collected. Approximately 610 tons of PCE contaminated soil was removed from the gravel area west of the building footprint. Approximately 310 tons was found to be above applicable cleanup levels (identified as 80 ppb) and was transported to the Rabanco Landfill near Roosevelt Washington for disposal. The remaining 300 tons was placed back in the excavation.

In 1996, Ecology issued an Agreed Owner to Frank Wear Cleaners requiring a remedial investigation, feasibility study, implementation of feasibility study activities, and submittal of groundwater sampling data. As a result, in 1997, Environmental Economic Solutions, Inc., (EES) oversaw installation of one off-site, up-gradient groundwater monitoring well, installation and sparge testing of one gas sparge point/well, and soil and groundwater sampling of the installed wells. Groundwater samples were also collected from pre-existing monitoring wells. Sampling confirmed presence of PCE in site soils and site groundwater. Environmental Economic Solutions, Inc., oversaw installation of a KVA Analytical Systems "C-Sparge" and controller system for sparging of site groundwater with ozone. EES estimated groundwater remediation would be achieved in 6 to 12 months.

Groundwater sparging was discontinued in 1998. Groundwater monitoring data provided by Ecology indicates that quarterly sampling from February of 1995 to August of 2000 (including sampling during and after sparging) confirms PCE contamination in site groundwater in excess of regulatory limits. Concentrations appear to be generally decreasing over time.

In November of 2000, a meeting was held between Greg Stoffers, the site owner, Travis Trent of Fulcrum Environmental Consulting, Inc., (Fulcrum), and Rick Roeder with Ecology to discuss means of final site closure. Mr. Stoffers indicated that the on-site building had been demolished and that he was preparing to have the remaining on-site contaminated soil excavated, characterized, and disposed under Fulcrum's direction. Mr. Roeder indicated that removal of all site soil containing PCE in excess of 20 ppb has been a significant step toward achieving final site closure at other PCE contaminated locations in the Yakima area. Mr. Roeder indicated that as a minimum, a period of post removal groundwater monitoring would be required to confirm absence of groundwater contamination before Ecology would accept final site closure or issue a "no further action" (NFA) determination for the site.

4.0 Investigation Activities and Schedule

4.1 Summary of Proposed Soil and Groundwater Activities

Materials necessary for site remedial activities will be mobilized to the site. All on-site personnel will review the site Health and Safety Plan. Signs, barrier tape, etc., shall be erected to denote the restricted work area and establish a decontamination area. Remedial activities will start with removal of the concrete pad over the remedial area. Concrete will be staged on-site pending results of waste characterization. Sub-concrete sediments will then be excavated in approximate 2 foot lifts and staged on-site pending results of characterization analysis performed in the on-site analytical laboratory. Once on-site laboratory analysis shows site cleanup levels have been achieved, soil samples will be collected from excavation extents and submitted to Transglobal Environmental Geosciences Northwest, Inc.'s (TEG) laboratory in Lacey, Washington for confirmatory analysis. Following receipt of confirmatory sample analysis showing concentrations below site cleanup levels, the excavation will be refilled and compacted to grade.

Quarterly groundwater samples are being collected from on-site and adjacent monitoring wells under Ecology's direction. Soil investigation activities will be scheduled in early February 2001, approximately mid-point between Ecology's December 2000 and March 2001 quarterly groundwater sampling. Post-remediation groundwater contaminant concentrations will be compared to pre-remediation concentrations as reported by Ecology to evaluate groundwater impact of remedial activities. Subsequent quarterly sampling of monitoring wells will be conducted by Ecology.

4.2 Tentative Schedule

Soil investigation activities will be scheduled in the week of February 12, 2001, pending coordination with TEG, Tri-Valley, and Ecology. Soil excavation activities are expected to be completed within 2 days of the start of site activities. Soil removal and site reclamation activities are expected to be completed within 2 - 3 weeks of the start of site activities.

5.0 Applicable or Relevant and Appropriate Requirements

Washington State:
Washington Water Pollution Control Laws: RCW 90-48, 52, 59
Washington Water Pollution Control Regulations: WAC 173-10, 216, 220, 221
Washington Water Quality Standards: WAC 173-201
Washington Dangerous Waste Regulations: WAC 173-303
Washington State Solid Waste Regulations: WAC 173-304
Washington State Solid Waste Management Law: RCW 70-95
Washington State Air Pollution Control Regulations: WAC 173-400.490
Washington Clean Air Act: RCW 70-94
Washington State Environmental Policy Act: RCW 43.21C
Washington Industrial Health and Safety Act: WAC 296-155, RCW 49.17

Federal:
Clean Air Act
Clean Water Act
Department of Transportation Rules for the Transportation of Hazardous Materials
Occupational Safety and Health Act (OSHA)
Resource Conservation and Recovery Act (RCRA)
Safe Drinking Water Act
Toxic Substances Control Act

5.1 Site Clean-up levels

Site clean-up criteria and associated clean-up actions will be established consistent with WAC 173-340, The Model Toxics Control Act Cleanup Regulation, and Ecology's Yakima Railroad Area soil cleanup levels that are protective of groundwater. Applicable Cleanup levels for PCE and its breakdown products are presented in Table 1.

Table 1 Cleanup Levels

	Soil			Groundwater		
Contaminant	RR Area[1]	Method B[2]	Method A[3]	Method B	Method A	MCL[4]
Tetrachloroethylene (PCE)	19 ppb	85.8 ppb	500 ppb	.858 ppb	5 ppb	5 ppb
Trichloroethylene (TCE)	11 ppb	398 ppb	500 ppb	3.98 ppb	5 ppb	5 ppb
cis 1-2 Dichloroethylene (DCE)	29 ppb	800 ppb	NA	80 ppb	NA	70 ppb
Vinyl Chloride	2 ppb	2.3 ppb	NA	.023 ppb	NA	2 ppb

1 RR = Yakima Railroad Area soil cleanup levels protective of groundwater
2 Method B = Model Toxics Control Act Method B Cleanup Level
3 Method A = Model Toxics Control Act Method A Cleanup Level
4 MCL = Maximum Contaminant Level

Yakima Railroad Area soil cleanup levels protective of groundwater have been selected as the target cleanup levels for this project.

6.0 Organization and Responsibilities for the Sampling and Analysis Activities

Fulcrum personnel will conduct all field sampling activities. Any excavations or soil borings defined under this SAP will be performed by contractors with OSHA/WISHA hazardous waste worker certifications. Samples will be analyzed on-site by TEG or delivered via common carrier under chain-of-custody to TEG's laboratory in Lacey Washington. Fulcrum will review laboratory results and laboratory quality assurance/quality control (QA/QC) documentation.

7.0 Sample Collection and Handling Methodology

Soil:

Site characterization samples will be obtained by grab sampling select points for stockpiled soil and excavation sample locations that are less than 4 ft below grade. Samples will be collected by backhoe bucket for sample locations greater than 4 ft below grade. For samples collected directly from the excavation, a plastic disposable spoon will be used to obtain samples from undisturbed soil, 3 to 6 inches into the soil from the bottom or sidewall of the remedial excavation. When appropriate, samples may be collected by hand using new latex or vinyl gloves.

The following method will be used to collect samples using a backhoe bucket:

1) The equipment operator will place the backhoe bucket at the desired sample location.
2) The bucket will be carefully brought into the soil to obtain a representative sample without disturbing overburden.
3) The sample will be quickly brought to the surface.

4) A new plastic spoon or gloved hand will be used to obtain soil samples directly from the bucket near the teeth of the bucket. Care will be taken to obtain samples that have not been exposed to the atmosphere or been in contact with the backhoe bucket.
5) The sample will be transferred by spoon or glove hand to the sample container.

Samples for laboratory analysis will be deposited into labeled borosilicate glass sample containers, packaged on ice, and hand delivered to TEG's on-site laboratory or delivered via common carrier under chain-of-custody to TEG's laboratory in Lacey, Washington.

8.0 Sampling Decontamination Procedures

Backhoe bucket decontamination will be performed if more than insignificant amounts of residual soil is observed on bucket prior to sampling. Dry decontamination techniques, such as brushing or scraping off residual soil/waste, will be preferable over water washing, to minimize waste generation.

Because only disposable equipment will be used during sampling, no other decontamination is anticipated between sampling events.

9.0 Quality Assurance and Quality Control

Quality Assurance (QA) is a system of activities that assures the user of analytical data at a stated level of confidence. Quality Control (QC) is an overall system of activities that controls the quality of a product or service so that the needs of the user are met. The overall data quality objectives are to develop and implement QA/QC procedures for sampling, chain of custody, laboratory analysis, and reporting as described in this work plan; and to provide results that are technically sound and properly documented that can be used to support decisions regarding soil removal and disposal. An environmental lab with on-site capability to perform the desired analysis will be used for analysis. The methods of analysis will be as follows:

Volatile Organic Analysis: Method 8010, 8260, 8240, or 8021B

Each of the indicated analytical methods mandates specific laboratory QA/QC requirements including duplicates, matrix spikes, and various blanks in accordance with lab QA/QC requirements. One duplicate sample and one field blank sample for every 20-site characterization samples submitted to the on-site lab will be analyzed as part of the QA/QC plan. Site confirmatory samples will be submitted under chain of custody to TEG's Lacey, Washington, laboratory for analysis.

10.0 Sample Labeling, Preservation, and Chain-of-Custody

Each sample will be given a unique sample identification number. The following procedure will be used to label samples at the site:

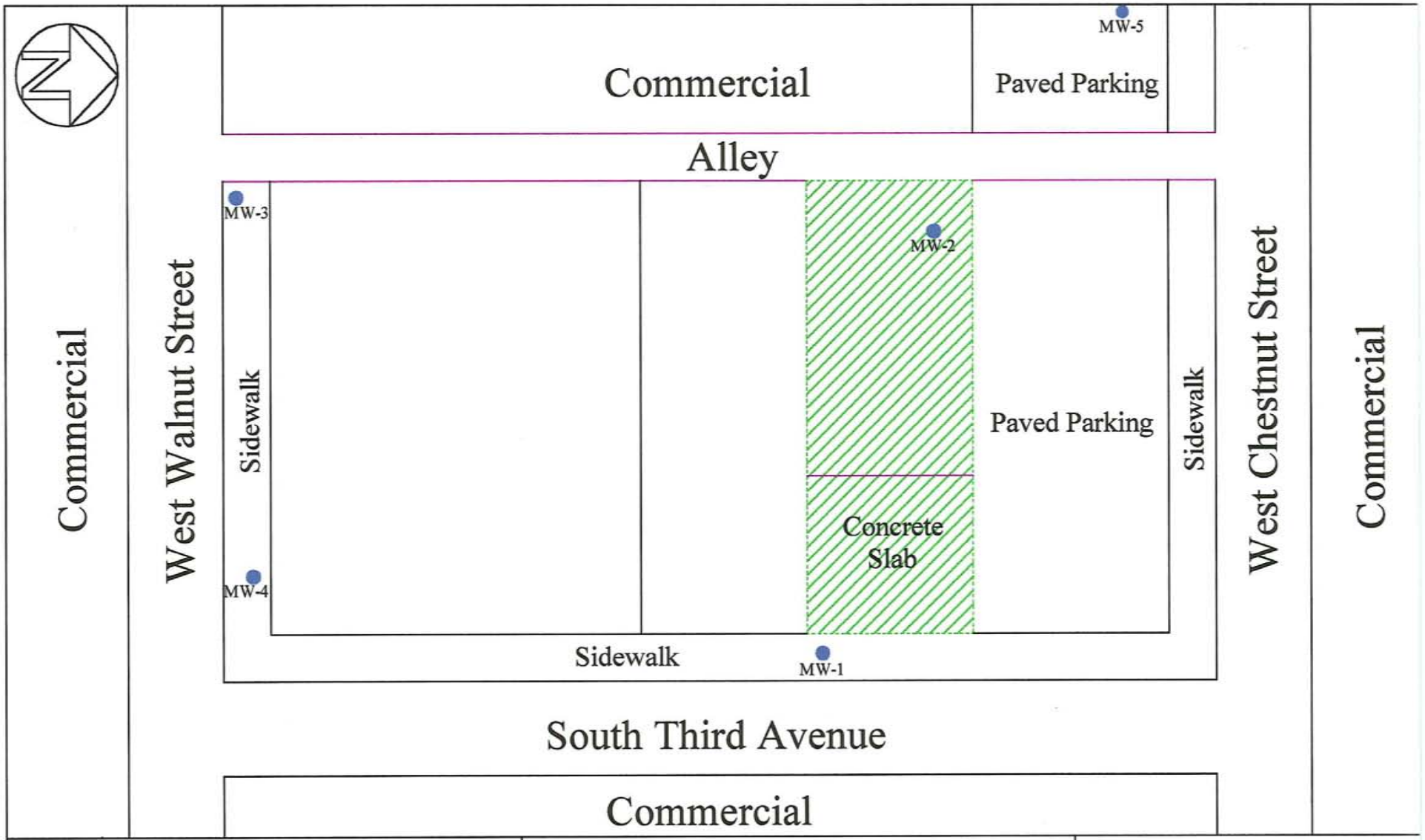
Sample numbers will be designated by the site name abbreviation FW, date, and sample number. For example, the third site characterization soil sample collected on February 12 will be FW0212-03.

Immediately following sampling, each sample will be logged with exact sample location (i.e. map location and depth), analysis, laboratory, and sampler's name. Sample information will also be entered onto the appropriate chain-of-custody form.

Following sampling and labeling of containers, each container will be logged on a chain-of-custody form and delivered directly to the on-site laboratory. Samples that will be shipped will be placed into a sealable quart-sized plastic bag and securely packed in an ice chest. Ice packed in plastic bags or reusable freeze blocks such as "blue ice" will be used to preserve soil samples. The ice chests will be delivered to the laboratory by common carrier under chain-of-custody.

11.0 Reporting

Upon completion of the soil removal activities, a project report shall be prepared. The report will include discussion of field activities, quantities and types of materials removed, ultimate destination of removed materials, analytical results, and accompanying appendices containing relevant documentation generated during the project.



LEGEND

Subject Site:
Property Boundary:
Monitoring Wells:
Scale: NTS

FIGURE 1

Site Location Map

Frank Wear Cleaners
106 South Third Avenue
Yakima, Washington

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
Phone(509) 574-0839 Fax(509) 575-8453

DRAWN BY: AMP	DATE: 01/17/2001
FILE NAME: Frank Wear Cleaners	
PROJECT NUMBER: 01-165	

APPENDIX B

SITE HEALTH & SAFETY PLAN

HEALTH AND SAFETY PLAN
Frank Wear Cleaners Site
Yakima, Washington

Prepared by:
Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901

January 30, 2001

Plan Prepared by: Travis Trent

TABLE OF CONTENTS

1.0 GENERAL PROJECT INFORMATION AND DESCRIPTION OF ACTIVITIES

1.1 Description of Activities

Activities anticipated under this health and safety plan include excavation and characterization of an area of soil contaminated by tetrachloroethylene (PCE) and associated breakdown products including trichloroethylene (TCE), cis 1-2 Dichlorethylene (DCE), and Vinyl Chloride.

1.2 Site Location and Description:

Subject site is located at 106 South Third Avenue in Yakima, Washington. The property is located in the N.E. ¼ of the S.E. ¼ of Section 24, Township 13 North, Range 18 East of the Willamette Meridian, Yakima County, Washington. The subject site is located just west of the downtown area of Yakima in a commercialized area.

Total investigation area is approximately 2,000 square feet (ft^2). The subject site encompasses approximately 6,300 ft^2 and was formerly occupied by a single story building, which contained a dry cleaning business called Frank Wear Cleaners. The building has been demolished and the site currently consists of an approximately 2,800 ft^2 rectangular concrete pad (floor of the demolished building) and an approximately 3,500 ft^2 gravel area. See Figure 1 for location of the Frank Wear Cleaners site.

Site elevation is approximately 1,077 feet above mean sea level. Site geology is characterized by unconsolidated to poorly consolidated sand and gravel deposits. Groundwater is estimated at 13 to 20 feet and is strongly influenced by seasonal variations and irrigational activities.

1.3 Contact List:

Fulcrum Project Manager:	**Travis Trent, Environmental Geologist**	**509-574-0839**
Fulcrum Site Health & Safety Officer:	**Travis Trent, Environmental Geologist**	**509-574-0839**
Fulcrum Field Services Person:	**Travis Trent, Environmental Geologist**	**509-574-0839**
Client Contact:	**Greg Stoffers, Site Owner**	**509-248-6071**
Other Project Personnel:	**Greg Huylar, Excavation Contractor**	**509-457-6341**

1.4 Proposed Start Date: February 12, 2001

1.5 Overall Hazard Ranking: Moderate

2.0 GENERAL SITE SAFETY

All work shall be performed in compliance with Title 29 of the Code of Federal Regulations, Part 1910 (29 CFR, General Industry Standards), 29 CFR 1926 (Construction Industry Standards), WAC 296-155 (Washington Industrial Health and Safety Act), and other applicable federal, state, and local Health and Safety regulations. In addition, no personnel will jeopardize the health and safety of themselves or others, or any property, during the course of this investigation.

3.0 SITE INFORMATION

3.1 Site History

Information reviewed in Department of Ecology's (Ecology) files indicates that the Frank Wear Cleaners site has been occupied by a dry cleaning operation from the early 1940s until the late 1990s. A site investigation conducted by Ecology in 1985 found that PCE was being released to a gravel area in the back lot (west of the concrete pad). Ecology performed a second site inspection in 1987 which also indicated that dangerous waste was being discharged to the environment.

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Groundwater sparging was discontinued in 1998. Groundwater monitoring data provided by Ecology indicates that quarterly sampling from February of 1995 to August of 2000 (including sampling during and after sparging) confirms PCE contamination in site groundwater in excess of regulatory limits. Concentrations appear to be generally decreasing over time.

In November of 2000, a meeting was held between Greg Stoffers, the site owner, Travis Trent of Fulcrum Environmental Consulting, Inc., (Fulcrum), and Rick Roeder with Ecology to discuss means of final site closure. Mr. Stoffers indicated that the on-site building had been demolished and that he was preparing to have the remaining on-site contaminated soil excavated, characterized, and disposed under Fulcrum's direction. Mr. Roeder indicated that removal of all site soil containing PCE in excess of 20 ppb has been a significant step toward achieving final site closure at other PCE contaminated locations in the Yakima area. Mr. Roeder indicated that as a minimum, a period of post removal groundwater monitoring would be required to confirm absence of groundwater contamination before Ecology would accept final site closure or issue a "no further action" (NFA) determination for the site.

3.2 Planned Duration of Activities

It is anticipated that the investigation and characterization and site reclamation will take approximately 2-3 weeks.

3.3 Site Topography and Accessibility

Subject site is a relatively flat lying. The site is accessed from Third Avenue.

4.0 SITE SPECIFIC SAFETY AND HEALTH HAZARDS

4.1 Physical Hazards

Physical hazards are those associated with heavy machinery, vehicular traffic, and climate. Heavy equipment hazards include the possibility of coming in contact with utilities, such as pressurized natural gas lines and overhead. Workers need to be aware of the limits of the machinery and operators need to be aware of the location of other workers. Hard hats and steel-toed boots must be worn by all personnel when in proximity of such equipment.

Personnel working near trenches or excavations need to be aware of the possibility of caving along the sidewalls. Under no circumstance shall personnel enter a trench or excavation deeper than four feet without first appropriately sloping or shoring the trench or excavation. No person shall enter a seepage catch basin or a drywell unless trained and certified for confined space entry.

Machinery and heavy equipment can emit strong sound waves capable of creating permanent hearing damage to those in close proximity. Personnel must wear hearing protection, such as ear plugs or ear muffs while near operating machinery and heavy equipment.

Traffic hazards exist at any site that has vehicular access. Often the operator of a motor vehicle will be to busy watching the heavy equipment to notice personnel on site. In areas of high traffic, personnel will wear red reflective vests to increase personal visibility and one person will be designated as a "flagger" to direct traffic near the working area.

During periods of cold weather, personnel should take measures to prevent hypothermia and frost bite. Layering clothing enables personnel to adjust to changing temperatures. All personnel on-site should be aware of the various symptoms and treatments of frostbite and hypothermia.

4.2 Chemical Hazards

The hazardous chemicals of concern under this Health and Safety Plan are volatile organic compounds including tetrachloroethylene and its breakdown products. See Appendix A for further chemical information.

5.0 ENVIRONMENTAL AND PERSONNEL PROTECTION

5.1 Personnel and Environmental Monitoring

Because of the low level of contamination present, air monitoring is not warranted. Changes to site activities will be based on the presence of visible dust generation. If visible dust is generated by site activities, engineering methods will be utilized to control dust and personnel protective equipment (PPE) will be upgraded to level C.

5.2 Personal Protection

All activities are to be conducted in Level D PPE. Level D PPE will consist of hard-hats, gloves, and steel-toed boots. If visible dust is generated by site activities, PPE will be upgraded to Level C. Inhalation of vapors or particulates during site activities will be minimized by the use of engineering controls. Ingestion of contaminated materials will be minimized by the use of appropriate personal hygiene procedures. Workers will thoroughly wash face and hands with soap and water after leaving the work area and prior to eating, drinking, or using the restroom.

5.3 Environmental Delineation

Environmental delineation will be achieved through the set-up and maintenance of an exclusion zone surrounding the contaminated area. All personnel and equipment that enters the exclusion zone must be decontaminated prior to leaving the exclusion zone. Primary means of decontamination will be dry removal of debris and disposable gloves. Disposable equipment will be deposited in marked containers within the exclusion zone for later disposal. Wet decontamination, if necessary, will be performed such that rinsate water can be collected and placed into barrels pending characterization and disposal.

6.0 EMERGENCY RESPONSE

FIRE: 911
POLICE: 911
HOSPITAL: Providence Medical Center
110 South Ninth Avenue
Yakima, Washington 98902
Phone: 575-5000

POISON CONTROL CENTER: 1-800-572-5842

EXPLOSIVE UNIT: 911

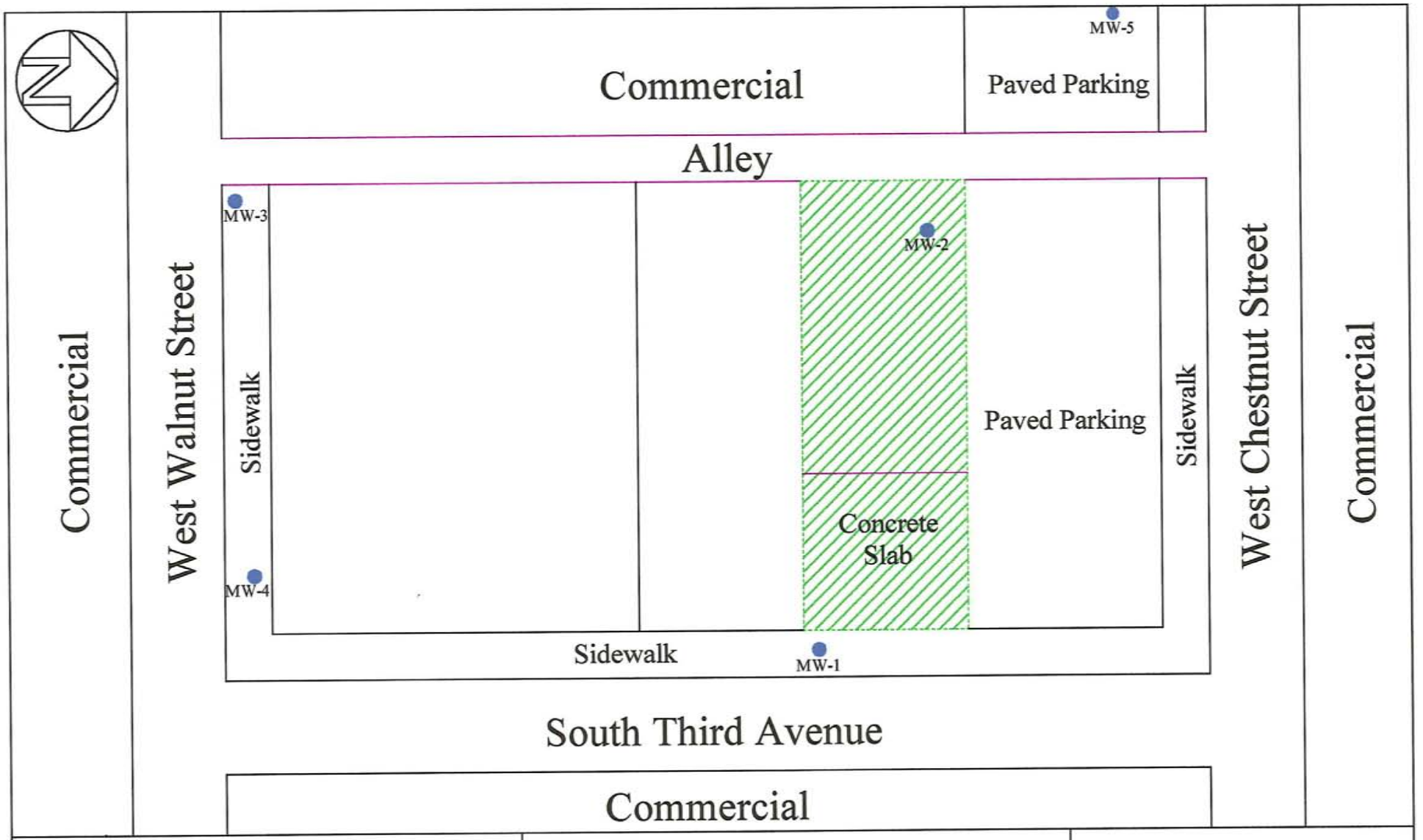
DIRECTIONS TO HOSPITAL: From site, head south (right) on third avenue ¼ block to the intersection with Walnut Avenue. Turn west (right) on Walnut Avenue approximately 5 blocks to Providence Medical Center. See Figure 2 for locations.

6.1 Training Requirements

All Fulcrum personnel involved in activities on-site in which the potential for chemical exposure or physical exertion exists must be enrolled in an active medical monitoring program and have completed their 40-hour Haz-Mat and Safety course.

I have read the above Health and Safety Plan for the Frank Wear Cleaners Investigation, Fulcrum Project Number 01-166. I am aware of the risks associated with this project as discussed both verbally and as stated in the aforementioned Health and Safety Plan, and will perform in a manner to decrease the risk of bodily injury to myself or others; property damage; or negatively impact to the environment.

Name (print)	Signature	Date	Company
Travis Trent		2/12/01	Fulcrum
Chris Purdom		2-12-01	Tri Valley
Donald Abbott		02-13-01	Ecology
Rick Roach		2-13-01	"
Tim McCall		2/13/01	TEG
Greg Huglar		2/13/01	Tri Val/GY
Greg Stothers		2/13/01	F.W Clnr
Peggy Williamson		2/13/01	Fulcrum.



LEGEND

Subject Site:
Property Boundary:
Monitoring Wells:
Scale: NTS

FIGURE 1

Site Location Map

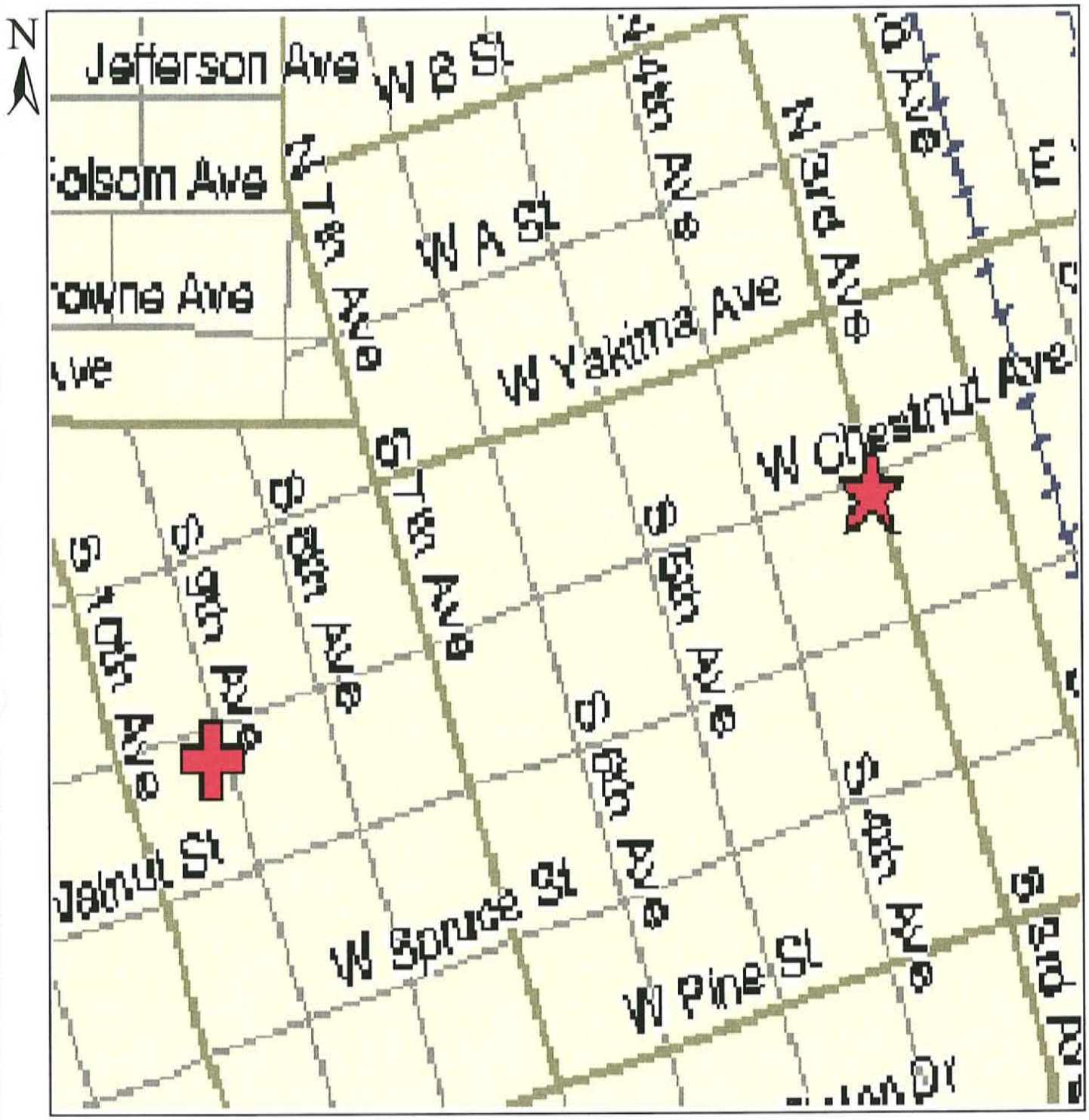
Frank Wear Cleaners
106 South Third Avenue
Yakima, Washington

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
Phone(509) 574-0839 Fax(509) 575-8453

DRAWN BY: AMP | DATE: 01/17/2001

FILE NAME: Frank Wear Cleaners

PROJECT NUMBER: 01-165



LEGEND

Subject Site: ★

Hospital: ✚

Source: Map Quest

FIGURE 2

Health & Safety Plan Map

Frank Wear Cleaners
106 South Third Avenue
Yakima, Washington 98902

Fulcrum Environmental Consulting, Inc.
122 South Third Street
Yakima, Washington 98901
509-574-0839

DRAWN BY: AMP	PROJECT NUMBER: 01-165
DATE: 01/17/2001	FILE NAME: Frank Wear Cleaners

APPENDIX A
CHEMICAL INFORMATION

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Tetrachloroethylene

CASRN 127-18-4

Contents

0106
Tetrachloroethylene; CASRN 127-18-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Tetrachloroethylene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1988
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4
Last Revised -- 03/01/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hepatotoxicity in mice, weight gain in rats	NOAEL: 20 mg/kg/day (converted to 14 mg/kg/day)	1000	1	1E-2 mg/kg/day
6-Week Mouse Gavage Study Buben and O'Flaherty, 1985	LOAEL: 100 mg/kg/day (converted to 71 mg/kg/day)			

*Conversion Factors: Doses have been adjusted for treatment schedule (5 days/week)

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: a dose-effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

Buben and O'Flaherty (1985) exposed Swiss-Cox mice to tetrachloroethylene in corn oil by gavage at doses of 0, 20, 100, 200, 500, 1500, and 2000 mg/kg, 5 days/ week for 6 weeks. Liver toxicity was evaluated by several parameters including liver weight/body weight ratio, hepatic triglyceride concentration, DNA content, histopathological evaluation, and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight/body weight ratios were significantly higher than controls for animals

treated with 100 mg/kg. At higher doses, hepatotoxic effects included decreased DNA content, increased SGPT, decreased levels of G6P and hepatocellular necrosis, degeneration and polyploidy.

A NOEL of 14 mg/kg/day was established in a second study, as well (Hayes et al., 1986). Groups of 20 Sprague-Dawley rats of both sexes were administered doses of 14, 400, or 1400 mg/kg/day in drinking water. Males in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The uncertainty factor of 1000 results from multiplying factors of 10 to account for intraspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Other data support the findings of the principal studies. Exposure of mice and rats to tetrachloroethylene by gavage for 11 days caused hepatotoxicity (centrilobular swelling) at doses as low as 100 mg/kg/day in mice (Schumann et al., 1980). Mice were more sensitive to the effects of tetrachloroethylene exposure than rats. Increased liver weight was observed in mice at 250 mg/kg, while rats did not exhibit these effects until doses of 1000 mg/kg/day were reached. Relative sensitivity to man cannot be readily established but the RfD of 1E-2 mg/kg/day is protective of the most mild effects observed in humans [diminished odor perception/modified Romberg test scores in volunteers exposed to 100 ppm for 7 hours; roughly equivalent to 20 mg/kg/day (Stewart et al., 1961)].

The principal studies are of short duration. Inhalation studies have been performed which indicate that the uncertainty factor of 10 is sufficient for extrapolation of the subchronic effect to its chronic equivalent. Liver enlargement and vacuolation of hepatocytes were found to be reversible lesions for mice exposed to low concentrations of tetrachloroethylene (Kjellstrand et al., 1984). In addition, elevated liver weight/body weight ratios observed in animals exposed to tetrachloroethylene for 30 days were similar to those in animals exposed for 120 days. Several chronic inhalation studies have also been performed (Carpenter, 1937; NTP, 1985; Rowe et al., 1952). None are inconsistent with a NOAEL of 14 mg/kg/day for tetrachloroethylene observed by Buben and O'Flaherty (1985) and Hayes et al. (1986).

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Low
Data Base -- Medium
RfD -- Medium

No one study combines the features desired for deriving an RfD: oral exposure, large number of animals, multiple dose groups, testing in both sexes and chronic exposure. Confidence in the principal studies is low mainly because of the lack of complete histopathological examination at the NOAEL in

the mouse study. The data base is relatively complete but lacks studies of reproductive and teratology endpoints subsequent to oral exposure; thus, it receives a medium confidence rating. Medium confidence in the RfD follows.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-82/005F.

U.S. EPA. 1987. Quantification of Toxicological Effects for Tetrachloroethylene. Prepared from the Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Drinking Water, Washington, DC.

Agency Work Group Review -- 05/20/1985, 08/05/1986, 09/17/1987

Verification Date -- 09/17/1987

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4

Not available at this time.

_VI. BIBLIOGRAPHY

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4
Last Revised -- 07/01/1989

__VI.A. ORAL RfD REFERENCES

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

Carpenter, C.P. 1937. The chronic toxicity of tetrachloroethylene. J. Ind. Hyg. Toxicol. 19(7): 323-336.

Hayes, J.R., L.W. Condie, Jr. and J.F. Borzelleca. 1986. The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats. Fund. Appl. Toxicol. 7: 119-125.

Kjellstrand, P., B. Holmquist, M. Kanje, et al. 1984. Perchloroethylene: Effects on body and organ weights and plasma butyrylcholinesterase activity in mice. Acta Pharmacol. Toxicol. 54(5): 414-424.

NTP (National Toxicology Program). 1985. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Tetrachloroethylene (perchloroethylene). U.S. Dept. Health and Human Services, NIH Publ. No. 85-2567.

Rowe, V.K., D.D. McCollister, H.C. Spencer, E.M. Adams and D.D. Irish. 1952. Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. Arch. Ind. Hyg. Occup. Med. 5: 566-579.

Schumann, A.M., J.F. Quast and P.G. Watanabe. 1980. The pharmacokinetics and macromolecular interaction of perchloroethylene in mice and rats as related to oncogenicity. Toxicol. Appl. Pharmacol. 55: 207-219.

Stewart, R.D., H.H. Gay, D.S. Erley, C.L. Hake and A.W. Schaffer. 1961. Human exposure to tetrachloroethylene vapor. Arch. Environ. Health. 2: 40-46.

U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (perchloroethylene). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-82-005F. Office of Drinking Water, Washington, DC.

U.S. EPA. 1987. Quantification of Toxicological Effects for Tetrachloroethylene. Prepared from the Health Assessment Document for Tetrachloroethylene (perchloroethylene). Office of Drinking Water, Washington, DC.

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

_VII. REVISION HISTORY

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4

Date	Section	Description
12/23/1987	I.A.	RfD withdrawn pending further review
03/01/1988	I.A.	Revised Oral RfD sumary added - RfD changed
03/01/1988	III.A.	Health Advisory added
07/01/1989	VI.	Bibliography on-line
05/01/1990	II.	Carcinogen assessment now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.	Regulatory action section withdrawn
08/01/1995	II.	EPA's RfD/RfC and CRAVE workgroups were discontinued in M 1995. Chemical substance reviews that were not completed September 1995 were taken out of IRIS review. The IRIS Pi Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III.,IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, Supplementary Data were removed from IRIS on or before Ap 1997. IRIS users were directed to the appropriate EPA Pro Offices for this information.

VIII. SYNONYMS

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4
Last Revised -- 01/31/1987

127-18-4
Ankilostin
Antisal 1

Antisol 1
Carbon bichloride
Carbon dichloride
Czterochloroetylen
Dee-Solv
Didakene
Didokene
Dowclene EC
Dow-Per
ENT 1,860
Ethene, tetrachloro-
Ethylene tetrachloride
Ethylene, tetrachloro-
Fedal-Un
NCI-C04580
Nema
PCE
PER
Perawin
PERC
Perchloorethyleen, per
Perchlor
Perchloraethylen, per
Perchlorethylene
Perchlorethylene, per
Perchloroethylene
Perclene
Percloroetilene
Percosolv
Percosolve
PERK
Perklone
Persec
Tetlen
Tetracap
Tetrachlooretheen
Tetrachloraethen
Tetrachlorethylene
Tetrachloroethene
Tetrachloroethylene
1,1,2,2-Tetrachloroethylene.
Tetracloroetene
Tetraguer
Tetraleno
Tetralex
Tetravec
Tetroguer
Tetropil
WLN: GYGUYGG
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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0106.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Trichloroethylene

CASRN 79-01-6

Contents

0199
Trichloroethylene; CASRN 79-01-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Trichloroethylene

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	08/01/1992
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	withdrawn	07/01/1994

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

The carcinogen assessment summary for this substance has been withdrawn following further review. A new carcinogen summary is in preparation by the CRAVE Work Group.

Agency Work Group Review -- 12/04/1986, 04/06/1989, 05/30/1989, 09/22/1993, 06/09

EPA Contacts:

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Not available at this time.

_VII. REVISION HISTORY

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Date	Section	Description
03/01/1988	II.B.3.	Text revised
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.2.	Text added
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	II.D.4.	Documentation corrected
05/01/1989	II.	Carcinogen assessment summary noted as pending change
06/01/1989	II.D.3.	Primary contact changed
07/01/1989	II.	Withdrawn; new assessment verified (in preparation)
12/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
07/01/1992	II.	EPA contact changed; work group review dates added
08/01/1992	I.A.	Oral RfD now under review
11/01/1993	II.	Work group review date added
07/01/1994	II.	Work group review date added
08/01/1995	I.A.,I.B., II.	EPA's RfD/RfC and CRAVE workgroups were discontinued in M 1995. Chemical substance reviews that were not completed September 1995 were taken out of IRIS review. The IRIS Pi Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III.,IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, Supplementary Data were removed from IRIS on or before Ap 1997. IRIS users were directed to the appropriate EPA Pro Offices for this information.

VIII. SYNONYMS

Substance Name -- Trichloroethylene
CASRN -- 79-01-6
Last Revised -- 03/31/1987

79-01-6
ACETYLENE TRICHLORIDE

ALGYLEN
ANAMENTH
BENZINOL
BLACOSOLV
BLANCOSOLV
CECOLENE
CHLORILEN
1-CHLORO-2,2-DICHLOROETHYLENE
CHLORYLEA
CHLORYLEN
CHORYLEN
CIRCOSOLV
CRAWHASPOL
DENSINFLUAT
1,1-DICHLORO-2-CHLOROETHYLENE
DOW-TRI
DUKERON
ETHINYL TRICHLORIDE
ETHYLENE TRICHLORIDE
ETHYLENE, TRICHLORO-
FLECK-FLIP
FLOCK FLIP
FLUATE
GEMALGENE
GERMALGENE
LANADIN
LETHURIN
NARCOGEN
NARKOGEN
NARKOSOID
NCI-C04546
NIALK
PERM-A-CHLOR
PERM-A-CLOR
PETZINOL
PHILEX
RCRA WASTE NUMBER U228
TCE
THRETHYLEN
THRETHYLENE
TRETHYLENE
TRI
TRIAD
TRIAL
TRIASOL
TRICHLOORETHEEN
TRICHLOORETHYLEEN, TRI
TRICHLORAETHEN
TRICHLORAETHYLEN, TRI
TRICHLORAN
TRICHLOREN
TRICHLORETHENE
TRICHLORETHYLENE
TRICHLORETHYLENE, TRI
TRICHLOROETHENE
Trichloroethylene
1,1,2-TRICHLOROETHYLENE
1,2,2-TRICHLOROETHYLENE
TRI-CLENE
TRICLORETENE
TRICLOROETILENE
TRIELENE
TRIELIN
TRIELINA
TRIKLONE
TRILEN
TRILENE
TRILINE

TRIMAR
TRIOL
TRI-PLUS
TRI-PLUS M
UN 1710
VESTROL
VITRAN
WESTROSOL
.

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Vinyl chloride

CASRN 75-01-4
08/07/2000

Contents

Note:	***A TOXICOLOGICAL REVIEW*** is available for this chemical in Adobe* PDF format (197 Pages, 1.2M). Similar documents can be found in the List of Available IRIS Toxicological Reviews

1001

Vinyl chloride; CASRN 75-01-4; 08/07/2000

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices, Regional Offices, and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Vinyl chloride

File First On-Line 08/07/2000

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	On-line	08/07/2000
Inhalation RfC Assessment (I.B.)	On-line	08/07/2000
Carcinogenicity Assessment (II.)	On line	08/07/2000

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Vinyl Chloride
CASRN -- 75-01-4
Last Revised -- 08/07/2000

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver cell polymorphism	NOAEL: 0.13 mg/kg-day NOAEL (HED): 0.09 mg/kg-day	30	1	3E-3 mg/kg-day
Rat chronic feeding study	LOAEL: 1.3 mg/kg-day LOAEL (HED): 0.9 mg/kg-day			

Til et al., 1983, 1991

*Conversion Factors and Assumptions -- The PBPK model of Clewell et al. (1995a,b) was used to convert the administered animal dose to the human equivalent dose (HED). At the HED, the time-integrated liver concentration of reactive metabolites calculated by the model is predicted to be equal to or less than that achieved for the animal NOAEL or LOAEL.

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

The vinyl chloride (VC) PBPK model of Clewell et al. (1995a,b) was used in this RfD assessment as well as in the accompanying RfC and cancer assessments. Use of this model allows improved calculation of the human dose that would be expected to result in the same level of toxicity as that observed in animals. Its use is based on the assumption that equal tissue concentrations of reactive metabolite would result in the same level of toxicity. As indicated in the RfC file, the PBPK model was also used to perform route-to-route extrapolation of the doses used in the oral study of Til et al. (1983, 1991).

Til, HP; Immel, HR; Feron, VJ. (1983) Lifespan oral carcinogenicity study of vinyl chloride in rats. Final report. CIVO Institutes. TNO Report No. V 83.285/291099, TSCATS Document FYI-AX-0184

0353, Fiche No. 0353.

Til, HP; Feron, VJ; Immel, HR. (1991) Lifetime (149-week) oral carcinogenicity study of vinyl chloride in rats. Food Chem Toxicol 29:713-718.

Til et al. (1983, 1991) incorporated VC into the diet of Wistar rats, administering diets containing 1% polyvinyl chloride (PVC) with varying proportions of VC monomer. Diets were available to experimental animals for 4 hours per day. Food consumption and VC concentrations were measured several times during the feeding period to account for loss of VC from the diet through volatilization. This information was used to calculate the ingested dose. Evaporative loss averaged 20% over 4 hours The ingested dose was adjusted downward by the amount of VC measured in the feces to arrive at the bioavailable doses of 0, 0.014, 0.13, or 1.3 mg VC/kg-day, which were fed to Wistar rats (n = 100, 100, 100, and 50/sex/group, respectively) for a lifetime. Rats were weighed at 4-week intervals throughout the study. All males surviving 149 weeks and all females still alive until week 150 were killed in extremis. A variety of lesions were observed histologically at the highest dose level of 1.3 mg/kg-day, including increased incidences of angiosarcomas, neoplastic nodules, hepatocellular carcinoma, cellular foci (clear-cell, basophilic, and eosinophilic), liver-cell polymorphism, and cysts. Of the above lesions, all except cysts and liver cell polymorphism are considered neoplastic or preneoplastic. Cysts, described as proliferating bile duct epithelium, are not considered precursors of hepatocellular tumors because tumors did not develop from this location. Liver cell polymorphism is considered to be a noncarcinogenic cytotoxic effect (Schoental and Magee, 1957, 1959). The incidenc of female rats having "many" hepatic cysts was 3/98 in controls, 4/100 at 0.014 mg/kg, 9/96 at 0.13 mg/kg, and 24/49 at 1.3 mg/kg. The incidence of male rats with liver cell polymorphism characterized as moderate or severe was 5/99 in controls, 5/99 at 0.014 mg/kg, 8/99 at 0.13 mg/kg, and 13/49 at 1.3 mg/kg; the corresponding incidence in females was 16/98, 16/100, 12/96, and 24/49. Benchmark dose analysis was attempted but was not successful with these data. The LOAEL based on these endpoints is clearly at the highest dose of 1.3 mg/kg-day and the NOAEL at the next highest dose of 0.13 mg/kg day.

PBPK MODELING: The PBPK model used was developed by Clewell et al. (1995a,b). The basis of the model and of this assessment is the production of reactive metabolites, most likely chloroethylene oxide, through two saturable pathways: one by cytochrome P450 IIE1 and the other by other isozyme of cytochrome P450. Because VC liver toxicity is related to production of reactive metabolites, the appropriate dose metric for liver toxicity endpoints was the total amount of the metabolite generated, divided by the volume of the tissue in which the metabolite is produced, that is, mg metabolite/L liver (Andersen et al., 1987).

The human dose corresponding to the NOAEL in animals was determined by first calculating the value of the dose metric for the NOAEL in the animals, i.e., the value of the total metabolites per liver volume for rats exposed to 0.13 mg/kg-day under the protocol of the study. This metric was then directly compared with that generated by the PBPK model from the results of a sample scenario of a continuous human exposure of 1 ppm ingestion in water by a 70 kg person, or 0.0286 mg VC/kg-day. PBPK outputs also demonstrated that the relationship between this dose metric and oral intake was linear in the dose range of interest (up to around 25 mg/kg-day). The metric generated from the simulated human scenario was 1.01 mg/L liver. The metric generated from the rat NOAEL was 3.00 mg/L liver (from the average of the male value of 3.03 and the female value of 2.96), which was then converted by a simple proportion to the corresponding human continuous exposure of 0.09 mg/kg-day = NOAEL human equivalent dose (HED). The modeling predicts that an average daily exposure of a human to this NOAEL(HED) would generate the same concentration of metabolites in the liver as wa calculated for the rats at the study NOAEL. Further details of the PBPK model development and results and the conversions between animal and human doses are in the Vinyl Chloride Toxicological Review and Appendices B and D.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 30. An uncertainty factor of 10 was used for protection of sensitive human subpopulations and 3 for animal-to-human extrapolation. The uncertainty factor for intraspecies variability includes the variability in risk estimates that would be predicted by the PBPK model for different individuals through variability in physiology, level of activity, and metabolic capability. A factor of 3 rather than default of 10 was used for interspecies extrapolation because, although PBPK modeling refines the animal-to-human comparison regarding the toxicokinetic portion (delivered dose), it does not address the uncertainty regarding the toxicodynamic portion (differential tissue sensitivity) of interspecies extrapolation. Uncertainty relating to toxicodynamics exists for the basic mode of action for noncance liver effects, that is, whether the epoxide or its rearrangement product (the aldehyde) are causal of the noncancer liver toxicity. The limited evidence of human susceptibility to certain hepatic effects of VC from the problematic study of Ho et al. (1991) also supports retaining the toxicodynamic portion of th interspecies UF (see Toxicological Review Section 4.1.2). No uncertainty factor for database insufficiency is considered necessary, because adequate chronic, developmental, and multigeneration reproductive studies exist. The total uncertainty factor is 30.

MF = 1.

___I.A.4. ADDITIONAL STUDIES/COMMENTS (ORAL RfD)

The Feron et al. (1981) study preceded that of Til et al. (1983, 1991). Because effects were note at the lowest concentration, the study was repeated by Til et al. using lower doses. Compound and die were administered to Wistar rats (n = 80, 60, 60, and 80, respectively) as in Til et al. at bioavailable doses of 0, 1.7, 5.0, or 14.1 mg VC/kg-day for a lifetime. All surviving animals were necropsied at week 135 (males) or week 144 (females). Significant clinical signs of toxicity in the 5.0 and 14.1 mg/kg-day groups included lethargy, humpbacked posture, and emaciation. Significantly increased mortality was seen consistently in males at 14.1 mg/kg-day, and in females at 5.0 and 14.1 mg/kg-day Relative liver weight was significantly increased at 14.1 mg/kg-day, but not reported for the other dos groups. A variety of liver lesions in male and female rats were observed histologically to be dose-related and the incidence was statistically significant. These lesions included cellular foci (clear-cell, basophilic, and eosinophilic), neoplastic nodules, hepatocellular carcinoma, angiosarcoma, necrosis, cysts, and liver cell polymorphism. Several of these endpoints were significantly increased in the group exposed to 1.7 mg/kg-day, including liver-cell polymorphisms and cysts, both of which were observed in the principal study of Til et al. and are not considered preneoplastic. This oral study defines a NOAEL of 1.7 mg/kg-day and a LOAEL of 5.0 mg/kg-day for liver effects that are not thought to be preneoplastic. Using the PBPK model of Clewell et al. (1995b), a NOAEL(HED) and LOAEL(HED) of 1.1 mg/kg-day and 9.2 mg/kg-day, respectively, were calculated. The results of Feron et al. (1981) are consistent with the results reported in the principal study of Til et al. (1983, 1991), in that the same noncancer liver endpoints were observed.

No other studies were located of oral administration of VC to animals or of oral exposure of humans to VC. However, the observation of nonneoplastic effects in the liver following exposure to VC is supported by several inhalation animal studies, as well as by occupational studies. These studie are discussed in the RfC summary and the Toxicological Review and are only briefly summarized here. The oral equivalents, in mg/kg-day, were estimated from the inhalation scenarios by the PBPK model and are presented for comparative purposes with the principal study.

Bi et al. (1985) exposed Wistar rats (apparently 75 per group) to 0, 10, 100, or 3000 ppm VC (99.99% pure), 6 hours/day, 6 days/week for up to 12 months, with interim sacrifices at 3 months (n = 8), 6 months (n = 30), 9 months (n = 6), and 12 months (n = 10), and sacrifice of surviving animals at 18 months (6 months after the end of exposure). This report presented histopathology results for the testes but not the liver. Body weight was significantly decreased in the mid- and high-exposure group Relative liver weight was increased in a concentration-dependent manner after 6 months (LOAEL at 10 ppm), but was affected only in the 3000 ppm group at 12 months, and no significant effect on liver weight was reported at 18 months. There was a concentration-related increase in the incidence of

damage to the testicular seminiferous tubules (incidence at 0, 10, 100, or 3000 ppm was 18.9%, 29.7 36.5%, and 56%, respectively), with significant increases at the two highest levels. This damage consisted of cellular alterations, degeneration, and necrosis. The NOAEL(HED) for testicular damage is estimated at 1.4 mg/kg-day and the LOAEL(HED) for liver alterations at 0.9 mg/kg-day.

Sokal et al. (1980) exposed male Wistar rats (7–34/sex/group) to 0, 50, 500, or 20,000 ppm of VC 5 hours/day, 5 days/week for 10 months. Relative liver weight was increased at 500 and 20,000 ppm, and absolute liver and testes weight were increased at 20,000 ppm. Treatment-related histological changes developed in the liver and testes. After 10 months, significant increases in polymorphism of hepatocytes (2/28, 5/21, 18/34, and 10/17 in 0, 50, 500, and 20,000 ppm groups, respectively) and proliferation of reticulo-endothelial cells lining the sinusoids (3/28, 3/21, 13/34, and 8/17 in 0, 50, 500, and 20,000 ppm groups, respectively) were observed. These effects were also seen at 6 months in the 500 and 20,000 ppm groups (incidences not reported). Damage to the spermatogeni epithelium was significantly higher than in controls following exposure to 500 ppm (3/28, 3/21, 13/34 and 5/17 in the 0, 50, 500, and 20,000 ppm groups, respectively; NOAEL at 50 ppm). The NOAEL (HED) for liver alterations is estimated at 3.1 mg/kg-day and for testicular alterations, 4.8 mg/kg-day.

In a related study, male Wistar rats (7–10/group) were exposed under dynamic conditions to nominal concentrations of 50, 500, or 20,000 ppm VC or to air only, 5 hours/day, 5 days/week for 10 months with interim sacrifices at 1, 3, and 6 months (Wisniewska-Knypl et al., 1980). Tissue examinations were limited to the liver. Relative liver weight was increased at all sacrifice times at 500 and 20,000 ppm. Ultrastructural alterations, including lipid droplet formation and accumulation, were seen at all exposure levels (LOAEL at 50 ppm). The LOAEL(HED) for lipid accumulation is estimated at 2.6 mg/kg-day.

Increased liver weight was also observed in rats exposed to concentrations of 100 ppm or highe for up to 6 months, and rabbits exposed to 200 ppm or higher exhibited histological changes (characterized as granular degeneration and necrosis with some vacuolization and cellular infiltration, NOAEL at 50 ppm, LOAEL at 100 ppm) in the centrilobular area of the liver (Torkelson et al., 1961). Histopathological lesions of the liver (centrilobular granular degeneration) also occurred in rats exposed to 500 ppm. For females, the NOAEL(HED) for increased liver weight was 3.1 mg/kg-day; the LOAEL was 6.6 mg/kg-day.

A two-generation inhalation reproductive study, done in accordance with GLP, was performed in rats (CD, 30/sex/group) exposed by whole-body inhalation for 6 hours/day to concentration levels o 0, 10, 100, and 1100 ppm VC monomer (CMA, 1998). Evaluation for the parental animals included body weights, food consumption, and estrous cycling as well as fertility, reproductive performance, and sperm assessments. Both F1 and F2 pups were examined and weighed at birth and on several day during lactation. At weaning, one pup/sex/litter was randomly selected, sacrificed, and given a macroscopic exam. No adverse effect of the measured parameters was seen in the parental generations and no adverse effect of treatment was indicated in the F1 and F2 pups. Liver effects typical of VC (increased weights, hypertrophy, and occurrence of altered hepatocellular foci) were noted in parental animals at 1100 and 100 ppm, but not at 10 ppm, with increased incidence occurring in the P2 as opposed to the P1 animals. Whether this increased incidence in P2 animals was due to in utero or juvenile susceptibility (the P1 animals were not exposed during these periods whereas the P2 animals were) or to a longer duration (P2 animals were exposed longer than were P1 animals) is not clear. However, tumor incidence has been documented to increase at maturity among laboratory animals treated with VC during the first 6 months of life when compared with those exposed during the secon or third 6-month period of life (Maltoni et al., 1981; Drew et al., 1983). The NOAEL for reproductive effects is >1100 ppm. PBPK analysis (Section 4.3 and Appendix D of the Toxicological Review) indicates that liver effects are seen in Til et al. (1991) at doses to the liver that are much lower than th NOAEL for liver effects (10 ppm; NOAEL(HED)=1 mg/kg-day) in this reproductive study.

Several epidemiology and case studies have associated chronic occupational exposure to VC with impaired liver function and/or biochemical or histological evidence of liver damage, notably subcapsular, portal and perisinusoidal fibrosis, hyperplasia of hepatocytes and sinusoidal cells, and

portal hypertension (Buchancova et al., 1985; Doss et al., 1984; Gedigk et al., 1975; Lilis et al., 1975; Marsteller et al., 1975; Popper and Thomas, 1975; Tamburro et al., 1984). Ho et al. (1991) reported VC-related liver dysfunction in 12 of 271 workers who were exposed to environmental levels of 1–20 ppm, with a geometric mean of 6 ppm; latent periods from first exposure to the first abnormal test ranged from 1 to 13 years. These studies are described in more detail in the Toxicological Review.

Data on potential reproductive or developmental effects of VC following oral exposure of animals or humans are not available. However, because VC is rapidly absorbed and distributed throughout the body following both oral and inhalation exposure, and because a PBPK model with route-to-route extrapolation capabilities is employed, data from inhalation studies can be used to predict potential effects from oral exposure.

Insufficient data exist to evaluate the teratogenicity of VC in humans. Several epidemiology studies have investigated the effects of VC exposure on the incidence of fetal loss and birth defects (Hatch et al., 1981; Infante et al., 1976; Waxweiler et al., 1977); however, no solid association has been found. Studies of communities near VC plants (Edmonds et al., 1978; Theriault et al., 1983) hav found no clear association between parental residence in a region with a VC plant and incidence of birth defects in the exposed community.

Inhalation experiments in animals have associated developmental toxicity only with concentrations at or above those associated with maternal toxicity and above those concentrations extrapolated by the PBPK model to a human equivalent concentration (HEC) that are associated with liver effects in the principal study of Til et al. (1983, 1991) of 2.5 mg/m^3 or a HED of 0.09 mg/kg-day John et al. (1977) exposed pregnant mice to 0, 50, or 500 ppm on gestation days 6 to 15. Exposure to 500 ppm induced maternal effects, including increased mortality, reduced body weight, and reduced absolute (but not relative) liver weight. Fetotoxicity also occurred in mice at 500 ppm, and was manifested as significantly increased fetal resorption, decreased fetal body weight, reduced litter size, and retarded cranial and sternal ossification. There was no evidence of a teratogenic effect in mice at either concentration. Pregnant rats exposed to 500 ppm on gestation days 6 through 15 had reduced body weight, and one rat exposed to 2500 ppm died. Fetal body weight was significantly decreased at 500 ppm, and an increased incidence of dilated ureters was observed at 2500 ppm. No signs of maternal or developmental toxicity were observed in pregnant rabbits exposed to 500 or 2500 ppm. In another study, rats were exposed continuously to 1500 ppm during the first, second, or third trimester of pregnancy (Ungvary et al., 1978). During the first third of pregnancy, maternal toxicity was manifested by increased relative liver weight; increased fetal mortality and embryotoxic effects were also observed. There were no embryotoxic or teratogenic effects following exposure during the secon or last trimester. In a dominant lethal study of VC, reduced fertility was observed at a concentration (250 and 1000 ppm) above the concentration that caused liver effects in rats (Short et al., 1977).

As discussed in the RfC summary, human and animal studies indicate that absorption following inhalation exposure occurs rapidly, with peak retention reached within 15 minutes. No human studies of absorption of ingested VC were located, although the principal study (Til et al., 1983, 1991) reported that absorption of VC monomer in animals following oral exposure is nearly quantitative. Peak blood levels were reached within 10 minutes when VC was administered to male rats by gavage in an aqueous solution at doses up to 92 mg/kg. In the same study, more complex and slightly delayed absorption was observed following VC gavage in oil, although peak blood levels were reached within 40 minutes (Withey, 1976). At 72 hours after a single gavage dose of 100 mg/kg VC in oil, unmetabolized VC was detected in exhaled air, indicating that metabolism was saturated (Watanabe and Gehring, 1976; Watanabe et al., 1976).

The primary route of VC metabolism is by the action of cytochrome P450 IIEI on VC to form a highly reactive epoxide intermediate, chloroethylene oxide (CEO), which spontaneously rearranges to form chloroacetaldehyde (CAA). These intermediates are detoxified mainly through conjugation with glutathione catalyzed by glutathione S-transferase (Hefner et al., 1975; Bolt et al., 1976; Jedrychowsk et al., 1984; Watanabe et al., 1978). The conjugated products are excreted in urine as substituted

cysteine derivatives (Bolt et al., 1980; Hefner et al., 1975). Although VC has often been cited as a chemical for which saturable metabolism should be considered in the risk assessment, saturation appears to become important only at very high exposure levels (greater than 250 ppm by inhalation or 25 mg/kg-day orally) compared with levels associated with the most sensitive noncancer effects or tumorigenic levels, and thus has little impact on the risk estimates.

Several different PBPK models for VC have been described in the literature. These models are described in detail and compared in the accompanying Toxicological Review Appendix A. The PBPK model used in this assessment was developed to support a cancer risk assessment based on the pharmacokinetic and metabolic data available in the literature for VC (Clewell et al., 1995a,b). The initial metabolism of VC was hypothesized to occur via two saturable pathways, one representing low capacity–high affinity oxidation by cytochrome P450 IIE1 and the other representing higher capacity–lower affinity oxidation by other isozymes of P450, producing in both cases CEO as an intermediate product. The parameter values for the two metabolic pathways describing the initial step in VC metabolism were determined by simulation of gas uptake data from mice, rats, hamsters, monkeys, an controlled human inhalation exposures, as well as from data on total metabolism and glutathione depletion in both oral and inhalation exposures. Successful simulation of pharmacokinetic data from a large number of studies over a wide range of concentrations using primarily inhalation exposure and different measures of effect (decreased chamber concentrations of VC, decreased serum levels of GSH) served as evidence that the PBPK model was valid over the exposure range of interest, especially for inhalation exposure scenarios. One limitation of the model is the lack of pharmacokinetic data via the oral route available for simulation and model validation. Model parameters for deriving dose metrics via the oral route have therefore been established such that the dose metrics generated would be "conservative," that is, predictive of higher human risk from animal results. This model, including the parameters and the rationale for their choice, pharmacokinetic data and model fit to these data, the sensitivity analysis of the model, and the actual dose metrics derived, i presented in the appendices of the Toxicological Review.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- High
Database -- Medium to high
RfD -- Medium

The overall confidence in this RfD assessment is medium. Confidence in the study of Til et al. (1983, 1991) is high because it used adequate numbers of animals, was well controlled, and reported i detail on the histological effects on the liver and their absence in other tissues (e.g., testes) at these same exposure levels. The critical effects, liver alterations and histopathology, are corroborated by other long-term studies including oral studies (Feron et al., 1981), inhalation studies (Sokal et al., 1980), and a reproductive study (CMA, 1998).

The confidence in the database is medium to high. The route-to-route capability of the PBPK model allows use of inhalation data, such as the developmental studies, to fill gaps in the oral databas The multigeneration reproductive study (CMA, 1998) and the dominant lethal study of Short et al. (1977) indicate at least in animals that if reproductive effects were to occur from exposure to VC, they would occur at a much higher exposure than that producing liver effects.

Concern for the confidence of dose metrics derived by the PBPK model from the oral study of Til et al. is offset by procedures instituted within the model when calculating oral dose metrics, including assumption of a maximum rate of VC uptake (i.e., designating it a zero-order process) and spreading the applied dose over a 24-hour period, which would minimize the concentration and maximize the likelihood that the parent VC would be metabolized to reactive species (i.e., the basis of this assessment, mg VC metabolized).

The high degree of confidence in the principal study of Til et al. (1983, 1991), combined with

the medium to high assessment of the database and less than high confidence in the qualitative aspects of the PBPK model, is considered to result in an overall medium confidence in the RfD.

MF = 1.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 2000

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Vinyl Chloride in Support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 2000).

Agency Consensus Date – 07/20/2000

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRI in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Vinyl Chloride
CASRN -- 75-01-4
Last Revised -- 08/07/2000

___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfC
Liver cell polymorphism	NOAEL: 0.13 mg/kg-day NOAEL(HEC): 2.5 mg/m^3	30	1	1E-1 mg/m^3
Rat chronic feeding study	LOAEL: 1.3 mg/kg-day LOAEL(HEC): 25.3 mg/m^3			

Til et al., 1983, 1991

*Conversion Factors and Assumptions: MW = 62.5. The NOAEL/LOAEL(HEC) were calculated for gas: extrarespiratory effect based on the PBPK model of Clewell et al. (1995a,b). The continuous human exposure concentration (HEC) that achieved a time-integrated liver concentration of metabolites less than or equal to that achieved for the animal simulation is defined as the HEC. The model parameters, assumptions, and results are explained below and at length in the accompanying Toxicological Review for Vinyl Chloride (U.S. EPA, 2000).

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

The chronic dietary study of Til et al. (1983, 1991) in rats is the principal study for both the inhalation RfC and oral RfD. The rationale for basing an inhalation RfC on an oral study is based on

evidence for a mode of action common to exposures from either route (liver toxicity) and availability of PBPK models to perform route-to-route extrapolations. The critical effect, increases in the incidenc of liver cell polymorphism and cysts, is reported in both oral studies (lifetime feeding studies of Feron et al., 1981; Til et al., 1983, 1991) and inhalation studies (10-month inhalation study of Sokal et al., 1980). In addition, the existing inhalation studies report no direct effects at the portal of entry (i.e., the respiratory tract). The inhalation database for VC, although deficient in chronic inhalation studies fro which an RfC could be derived, has nevertheless allowed for development of PBPK models capable o converting VC exposures not only from animals to human equivalents but also from route to route. Use of this PBPK model is based on the principal assumption that equal tissue concentrations of reactive metabolite would result in the same level of toxicity whether in animals or humans, or from inhalation or oral exposures. Complete documentation of the choice, application, assumptions, and limitations of the PBPK model used in this assessment are in the supporting Toxicological Review an appendices.

Til, HP; Feron, VJ; Immel, HR. (1991) Lifetime (149-week) oral carcinogenicity study of vinyl chloride in rats. Food Chem Toxicol 29:713-718.

Til, HP; Immel, HR; Feron, VJ. (1983) Lifespan oral carcinogenicity study of vinyl chloride in rats. Final report. Civo Institutes. TNO Report No. V 83.285/291099, TSCATS Document FYI-AX-0184-0353, Fiche No. 0353.

The lifetime dietary study of Til et al. (1983, 1991) was performed in order to study a range of oral doses below those delivered in a nearly identical study by Feron et al. (1981), because tumors and other pathological effects were observed at all doses in the Feron et al. study. To incorporate VC into the diet of Wistar rats, Til et al. administered diets containing 1% PVC with varying proportions of V monomer. Diets were available to experimental animals for 4 hours per day. Food consumption and VC concentrations were measured at several times during the feeding period so as to account for the loss of VC from the diet through volatilization. This information was used to calculate the ingested dose. Evaporative loss averaged 20% over 4 hours. The ingested dose was adjusted downward by the amount of VC measured in the feces to arrive at the bioavailable doses of 0, 0.014, 0.13, and 1.3 mg/kg-day VC, which were fed to Wistar rats (n = 100, 100, 100, and 50/sex/group, respectively) for lifetime. Rats were weighed at 4-week intervals throughout the study. All males surviving 149 weeks and all females alive until week 150 were killed in extremis. Mortality was slightly increased in the high-dose group near the end of the study. A variety of lesions were observed histologically at the highest dose level of 1.3 mg/kg-day. These included increased incidences of angiosarcomas, hepatocellular carcinomas, neoplastic nodules, cellular foci, liver-cell polymorphism, and cysts. All these may be considered as neoplastic or preneoplastic save for cysts and liver cell polymorphism. Cysts described as proliferating bile duct epithelium are not considered precursors of hepatocellular tumors because tumors did not develop from this location. Liver cell polymorphism is considered a noncarcinogenic cytotoxic effect (Schoental and Magee, 1957, 1959). The incidence of female rats having "many" hepatic cysts was 3/98 in controls, 4/100 at 0.014 mg/kg, 9/96 at 0.13 mg/kg, and 24/49 at 1.3 mg/kg. The incidence of male rats with liver cell polymorphism characterized as moderat or severe was 5/99 in controls, 5/99 at 0.014 mg/kg, 8/99 at 0.13 mg/kg, and 13/49 at 1.3 mg/kg; the corresponding incidence in females was 16/98, 16/100, 12/96, and 24/49. Benchmark dose analysis was attempted but was not successful with these data. The LOAEL based on these endpoints is clearly at the highest dose of 1.3 mg/kg-day and the NOAEL at the next highest dose of 0.13 mg/kg-day.

PBPK Modeling: The PBPK model used was developed by Clewell et al. (1995a,b). The basis of the model and this assessment is the production of reactive metabolites, most likely chloroethylene oxide, through two saturable pathways: one by cytochrome P450 IIE1 and the other by other isozymes of cytochrome P450. Because VC liver toxicity is related to production of reactive metabolites, the appropriate dose metric for liver toxicity endpoints was the amount of the metabolite generated, divided by the volume of the tissue in which the metabolite is produced, that is, mg/L liver (Andersen et al., 1987) expressed as a daily average.

The NOAEL(HEC) was derived by first calculating the value of the appropriate dose metric for

the NOAEL in the animals, that is, the value of the total metabolites per liver volume for rats exposed to 0.13 mg/kg under the protocol of the study. This metric was calculated to be 3.00 mg/L liver (from the average of the male value of 3.03 and the female value of 2.96) and a factor was then used to convert this metric to a continuous human inhalation exposure. The conversion factor to a human equivalent inhalation concentration (HEC) was generated by exercising the PBPK model to determine this same dose metric for a continuous human inhalation exposure, that is, the continuous exposure concentration that would result in the same dose of metabolites to the human liver. The results from a range of exposure concentrations (1 $\mu g/m^3$ to 10,000 mg/m^3) showed that the relationship was linear up to nearly 100 mg/m^3, with the factor in this range being 1.18 mg/L liver/1 mg/m^3 VC. Conversion of the study NOAEL of 0.13 mg/kg-day was then accomplished by dividing the animal dose metric fo this concentration by the conversion factor (3.00 /1.18) to arrive at NOAEL(HEC) of 2.5 mg/m^3. For the LOAEL(HEC) the figures and calculation are 29.9/1.18, or 25.3 mg/m^3.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF = 30. An uncertainty factor of 10 was used for protection of sensitive human subpopulations and 3 for animal-to-human extrapolation. The uncertainty factor for intraspecies variability includes the variability in risk estimates that would be predicted by the model for different individuals, through variability in physiology, level of activity, and metabolic capability. A factor of 3 rather than a default of 10 was used for interspecies extrapolation because, although PBPK modeling refines the animal-to-human comparison of delivered dose, it does not address the uncertainty regarding the toxicodynamic portion of interspecies extrapolation (relating to tissue sensitivity). Uncertainty relating to toxicodynamics exists for the basic mode of action for noncancer liver effects, that is, whether the epoxide or its rearrangement products (the aldehyde) are causal of the noncancer liver toxicity. The limited evidence of human susceptibility to certain hepatic effects from VC from the problematic stud of Ho et al. (1991) also supports retaining the toxicodynamic portion of the interspecies UF. No uncertainty factor for database insufficiency is considered necessary, because adequate chronic, developmental, and multigenerational reproductive studies exist. The total uncertainty factor is 30 (se Toxicological Review Section 4.1.2).

MF = 1.

___I.B.4. ADDITIONAL STUDIES/COMMENTS (INHALATION RfC)

Bi et al. (1985) exposed Wistar rats (apparently 75 per group) to 0, 10, 100, or 3000 ppm VC (99.99% pure), 6 hours/day, 6 days/week for up to 12 months. Animals were weighed monthly and observed daily for clinical signs. Interim sacrifices were reported at 3 (n = 8), 6 (n = 30), 9 (n = 6), an 12 (n = 10) months, with surviving animals examined after 18 months (6 months after the end of exposure). Organ weights and histopathology were reported to have been assessed on lung, liver, hear kidney, testes, spleen, and brain, but only partial organ weight information was presented, and only testicular histopathology results are discussed in the report. Body weight was significantly decreased in the mid- and high-exposure groups (320, 310, 280, and 240 g in 0, 10, 100, and 3000 ppm groups, respectively). Relative liver weight was increased in a concentration-dependent manner after 6 months At 12 months, increased relative liver weight was observed only in the 3000 ppm group, although the power to detect this effect was limited by the small number of animals examined. No effect on liver weight persisted at 18 months after the start of the exposure. Relative kidney weight in the 3000 ppm group was increased at 3 and 12 months but not at 6 or 18 months, and in the 100 ppm group only at 18 months. Relative testes weight was decreased in the 100 and 3000 ppm groups at 6 months, but the effect was not concentration related, in that the relative testes weight was less at 100 than at 3000 ppm and no other time points showed significant effects. The study did not report absolute organ weights, relative weights for groups with no significant differences, standard deviations, or histopathology results (except in the testes), making the organ weight differences in tissues other than the liver and testes difficult to interpret. The incidence of damage to the testicular seminiferous tubules in rats (n = 74 total) exposed to 0, 10, 100, or 3000 ppm was 18.9%, 29.7%, 36.5%, and 56%, respectively. The incidence was statistically elevated at 100 and 3000 ppm ($p < 0.05$ and $p < 0.001$, respectively)

compared with controls, and appeared to be concentration related. This damage consisted of cellular alterations, degeneration, and necrosis. Thus, 10 ppm is considered a LOAEL for liver weight changes and the NOAEL for biologically significant testicular degeneration.

In determining an HEC for testicular damage by use of the PBPK model, the effects are assume to be caused by metabolites produced in the testes, in that cytochromes P450 are known to be present in this tissue. Because specific information on VC metabolism in testes is not available, the relative amount of metabolism in testes and liver was assumed to be the same across species, so the amount of metabolite produced in the testes would be proportional to the total metabolism. An appropriate dose metric for the testicular effects would then be the total amount of metabolite produced divided by the body weight expressed as a daily average, which was determined by the PBPK model to be 0, 1.3, 12.5, and 43.2 mg metabolites per kg body weight. Using a conversion factor for this dose metric derived for a continuous human inhalation exposure as described above (0.0308 mg/kg body weight), the NOAEL(HEC) would be 1.3/.0308 = 42 mg/m^3. Benchmark analysis of the incidence of testicular degeneration using the Weibull and polynomial models and the HECs calculated using the PBPK model resulted in a BMC10 for extra risk of 182 mg/m^3. The testicular effects noted in this subchroni study are considered to occur at higher HEC concentrations than do the liver effects. In addition, it may be that testicular effects from VC exposure have concentration dependency and a route component (i.e., inhalation only), in that testicular effects were not reported in either of the lifetime oral exposure studies in which liver toxicity was prominent.

A two-generation inhalation reproductive study, done in accordance with GLP, was performed in rats (CD, 30/sex/group) exposed by whole-body inhalation for 6 hours/day to concentration levels o 0, 10, 100, and 1100 ppm (0, 26, 256, and 2816 mg/m^3) VC monomer (CMA, 1998). Evaluation for the parental animals included body weights, food consumption, and estrous cycling as well as fertility, reproductive performance, and sperm assessments. Both F1 and F2 pups were examined and weighed at birth and on several days during lactation. At weaning, one pup/sex/litter was randomly selected, sacrificed, and given a macroscopic exam. No adverse effect of the measured parameters was seen in the parental generations and no adverse effect of treatment was indicated in the F1 and F2 pups. Liver effects typical of VC (increased weights, hypertrophy, and occurrence of altered hepatocellular foci) were noted in parental animals at 1100 and 100 ppm, but not at 10 ppm, with increased incidence occurring in the P2 as opposed to the P1 animals. Whether this increased incidence in P2 animals was due to in utero or juvenile susceptibility (the P1 animals were not exposed during these periods whereas the P2 animals were) or to a longer duration (P2 animals were exposed longer than were P1 animals) is not clear. However, tumor incidence has been documented to increase at maturity among laboratory animals treated with VC during the first 6 months of life when compared with those exposed during the second or third 6-month period of life (Maltoni et al., 1981; Drew et al., 1983). Th NOAEL for reproductive effects is >2816 mg/m^3. PBPK analysis (Section 4.3 and Appendix D, Table D-2, of the Toxicological Review) indicates that liver effects are seen in Til et al. (1991) at doses to the liver that are much lower than the NOAEL for liver effects (mg/m^3) in this reproductive study.

The Feron et al. (1981) study preceded the one reported by Til et al. (1983, 1991). Because effects were noted at the lowest concentration, the study was repeated by Til et al. using lower doses. Compound in the diet was administered to Wistar rats (n = 80, 60, 60, and 80, respectively) as in Til e al. (1983, 1991) at bioavailable doses of 0, 1.7, 5.0, or 14.1 mg/kg-day VC for a lifetime. All survivin animals were necropsied at week 135 (males) or week 144 (females). Significant clinical signs of toxicity in the 5.0 and 14.1 mg/kg-day groups included lethargy, humpbacked posture, and emaciation Significantly increased mortality was seen consistently in males at 14.1 mg/kg-day and in females at 5.0 and 14.1 mg/kg-day. Relative liver weight was significantly increased at 14.1 mg/kg-day, but was not reported for the other dose groups. A variety of liver lesions were observed histologically to be dose-related and statistically significant in male and female rats. These included cellular foci (clear-cell, basophilic, and eosinophilic), neoplastic nodules, hepatocellular carcinoma, angiosarcoma, necrosis, cysts, liver-cell polymorphism, and necrosis. Several of these endpoints were significantly increased in the group exposed to 1.7 mg/kg-day, including liver-cell polymorphism, cysts, and necrosis, all of which were observed in the principal study of Til et al. and are not considered preneoplastic. This oral study defines a NOAEL of 1.7 mg/kg-day and a LOAEL of 5.0 mg/kg-day for

liver effects that are not thought to be preneoplastic. Using the PBPK model of Clewell et al. (1995b), a NOAEL(HEC) and LOAEL(HEC) of 33 and 97 mg/m^3, respectively, were calculated. Application o benchmark analysis and the PBPK model (for extensive liver necrosis because liver-cell polymorphism could not be modeled), using the internal dose metric from the PBPK model to get the BMC at a benchmark response of 10% extra risk, and then using the human PBPK model to get the human equivalent of the BMC as described for the Bi et al. (1985) study, resulted in a BMC(HEC) of 34 mg/m^3 for the effect in females and a BMC(HEC) of 59 mg/m^3 for males. This study corroborates the results reported in the principal study of Til et al. (1983, 1991), in that the same noncancer liver endpoints were observed.

Male Wistar rats (7–34/sex/group) were exposed via inhalation to 0, 50, 500, or 20,000 ppm of VC for 5 hours/day, 5 days/week for 10 months (Sokal et al., 1980). Histopathology was conducted o all major organs, including the lungs, with groups sacrificed at 1.5, 3, 6, and 10 months of exposure. Ultrastructural examination of the liver was carried out at 3, 6, and 10 months. No adverse effects on the lung were reported. There was a statistically significant ($p < 0.05$) and biologically significant (e.g., >10% relative to concurrent controls) decrease in body weight at 10 months in the high-exposur group only. Relative liver weight was increased at 500 and 20,000 ppm and absolute liver and testes weight were increased at 20,000 ppm. Treatment-related histological changes developed in the liver and testes. After 10 months, significant increases in polymorphism of hepatocytes (2/28, 5/21, 18/34, and 10/17 in 0, 50, 500, and 20,000 ppm groups, respectively) and proliferation of reticulo-endothelial cells lining the sinusoids (3/28, 3/21, 13/34, and 8/17 in 0, 50, 500, and 20,000 ppm groups, respectively) were observed. These effects were also seen at 6 months in the 500 and 20,000 ppm groups (incidences not reported). Fatty degeneration was also observed and ultrastructural changes, including proliferation of smooth endoplasmic reticulum and lipid droplets, were reported, but no data were given. The report indicated that more detailed description of the histopathology and ultrastructur would be published separately, but no such record was found. Damage to the spermatogenic epithelium was significantly greater than in controls following exposure to 500 ppm (3/28, 3/21, 13/34, and 5/17 in the 0, 50, 500, and 20,000 ppm groups, respectively). A NOAEL of 50 ppm was identified for hepatocellular and testicular histopathology. Using the PBPK model of Clewell et al. (1995b), the NOAEL of 50 ppm corresponds to a duration-adjusted NOAEL(HEC) of 93 mg/m^3 for liver effects and a NOAEL(HEC) of 145 mg/m^3 for testicular effects. Applying benchmark modeling using the dosimetry provided by the PBPK model in the same manner as described for the principal study, the BMC(HEC) values are 59–169 mg/m^3 for liver effects (59 mg/m^3 for nuclear proliferation o hepatocytes, 92 mg/m^3 for liver cell polymorphism, and 169 mg/m^3 for the continuous endpoint of increased relative liver weight), and 122 mg/m^3 for testicular effects.

In a related study, male Wistar rats (7–10/group) were exposed under dynamic conditions to nominal concentrations of 50, 500, or 20,000 ppm VC or to air only, 5 hours/day, 5 days/week (duration adjusted to 19, 190, or 7607 mg/m^3, respectively) for 10 months with interim sacrifices at 1, 3, and 6 months (Wisniewska-Knypl et al., 1980). This study appears to be a different experiment fro that reported by Sokal et al. (1980) because of different initial animal weights and chemical purity, although this is not entirely clear. Body weight was significantly affected only in the 20,000 ppm group exposed for 10 months. Tissue examinations were limited to the liver. Relative liver weight was increased at all sacrifice times at 500 and 20,000 ppm. Examination of liver tissue from exposed animals showed ultrastructural changes at all exposure levels, with the intensity of the effects increased in a dose-response manner, although no quantitative information was provided. This study identifies a minimal LOAEL of 50 ppm for minor liver histopathology and a NOAEL of 50 ppm for liver weight effects. Based on the PBPK model of Clewell et al. (1995a,b), this corresponds to a LOAEL(HEC) of 80 mg/m^3. Because the exposure conditions and number of animals tested in this study were the same as in the Sokal et al. (1980) study, and the response data were the same as those i the Sokal study, although rounded off, the BMC(HEC) value of 169 mg/m^3 identified in the Sokal study also applies here. The liver ultrastructural data are not amenable to benchmark analysis because only descriptive information was presented.

Several species of animals were exposed to 0, 50, 100, 200, or 500 ppm VC via inhalation for u

to 6 months (Torkelson et al., 1961). Hematologic determinations, urinalysis, clinical biochemistry, organ weight measurement, and histopathology examination were conducted. Rats (24/sex/group), guinea pigs (12/sex/group), rabbits (3/sex/group), and dogs (1/sex/group) exposed to 50 ppm (128 mg/m^3), 7 hours/day, for 130 of 189 days did not exhibit toxicity as judged by appearance, mortality, growth, hematology, liver weight, and pathology. At an exposure concentration of 100 ppm administered 138–144 times in 204 days, a statistically significant increase in the relative liver weight of male and female rats was noted. Exposure to 200 ppm (138-144 times in 204 days) for 6 months resulted in increased relative liver weight in male and female rats, but there was no biochemical or microscopic evidence of liver damage. Rabbits exposed under the same conditions exhibited histological changes (characterized as granular degeneration and necrosis with some vacuolization an cellular infiltration) in the centrilobular area of the liver. There was no effect at this level in guinea pigs or dogs. Histopathological lesions of the liver (centrilobular granular degeneration) and increased organ weight occurred in rats exposed to 500 ppm. Although relative liver weights were slightly elevated in male rats (n = 5) exposed to 100 or 200 ppm for 2–4 hours/day (duration adjusted to 15–3 and 30–60 mg/m^3, respectively), the increases were not statistically significant. A NOAEL for liver effects of 50 ppm (duration adjusted to 25.6 mg/m^3) is identified in this study. Based on the PBPK model of Clewell et al. (1995b), this corresponds to a duration-adjusted NOAEL(HEC) of 93 mg/m^3. These data were not amenable to benchmark analysis because standard deviations on the weight measurements were not reported.

Maltoni et al. (1980, 1981) exposed Sprague-Dawley or Wistar rats to 1–30,000 ppm 4 hours/day, 5 days/week for 52 weeks, and mice and hamsters to 50–30,000 ppm for 30 weeks, followed by an observation period. A statistically significant increase in tumor incidence, including liver angiosarcoma, was observed in all three species at 50 ppm (duration adjusted to 15.2 mg/m^3). This study primarily investigated the development of tumors. However, the incidence of neoplastic an preneoplastic lesions including hepatomas, neoplastic liver nodules, nodular hyperplasia of the liver, and diffuse hyperplasia of the liver was presented. Using the combined results for two experiments in SD rats (one exposing 60 male and 60 female rats to 1–25 ppm and the second using exposure concentrations of 250–10,000 ppm with 120 male and 120 female rats), the incidence for diffuse hyperplasia at 0, 1, 5, 10, 25, 50, 250, 500, 2500, 6000, and 10,000 ppm for combined males and females was 1.9%, 0.8%, 0%, 8.3%, 7.5%, 3.0%, 1.7%, 10%, 1.7%, 3.3%, and 5.0%, respectively. Diffuse hyperplasia was increased significantly in most exposure groups, but did not appear to be concentration related. Likewise, the results for nodular hyperplasia, neoplastic nodules, and hepatoma in SD rats, and for these lesions in Wistar rats, showed significant increases but did not appear to be concentration related.

Several epidemiology and case studies have associated chronic occupational exposure with impaired liver function and/or biochemical or histological evidence of liver damage, notably subcapsular, portal, and perisinusoidal fibrosis; hyperplasia of hepatocytes and sinusoidal cells; and portal hypertension (Buchancova et al., 1985; Doss et al., 1984; Gedigk et al., 1975; Lilis et al., 1975; Marsteller et al., 1975; Popper and Thomas, 1975; Tamburro et al., 1984). Focal hepatocellular hyperplasia and focal mixed (hepatocytes and sinusoidal cells) hyperplasia are early histological alterations indicative of VC exposure (Popper and Thomas, 1975), and are the principal anatomic lesions in VC-associated liver disease (Berk et al., 1976). Doss et al. (1984) reported coproporphyrinuria in 46 males occupationally exposed to VC for 18 months to 21 years. Gedigk et al (1975) correlated liver damage manifested as parenchymal damage, fibrosis, and proliferation of the sinusoidal cells with duration of exposure to VC in 51 patients. The severity of degenerative lesions increased with increasing duration of exposure, and appeared to be reversible upon exposure cessation Another study reported the progressive nature of the liver changes that resulted in "chronic hepatitis" (Lilis et al. 1975). Thresholds for hepatotoxicity cannot be identified because data regarding exposure concentrations and duration were not available. The symptoms and signs of liver disease associated with occupational exposure to VC include pain or discomfort in the right upper quadrant of the abdomen, hepatomegaly, splenomegaly, and thrombocytopenia, in addition to fibrosis, cirrhosis, and portal hypertension; however, these observations are not pathognomonic for VC-induced liver disease (Lilis et al., 1975; Marsteller et al., 1975; Popper and Thomas, 1975). Fibrosis frequently occurs in the elderly and patients with diabetes mellitus (Popper and Thomas, 1975).

Ho et al. (1991) reported VC-related liver dysfunction in 12 of 271 workers who were exposed to environmental levels of 1–20 ppm, with a geometric mean of 6 ppm (15 mg/m^3). The affected workers were identified as a result of a medical surveillance program of biochemical liver function tests. Although results suggested effects at very low levels, the exposure estimates may well be flawe and other problems exist with using this study (see Toxicological Review Section 4.1.2). Prior to 198 concentrations of VC were reported to range from 2000 to 5000 ppm during tank washing and as high as 10,000 ppm near reactors. Du et al. (1995) found that serum levels of gamma-glutamyl transferase (GGT), but not other indicators of liver function, correlated with exposure in a group of 224 VC workers with time-weighted average (TWA) exposure ranging from 0.36 to 74 ppm (0.92–189 mg/m^3 Such tests, however, are not specific for VC. Hepatomegaly, altered liver function as shown by biochemical tests, and Raynaud's phenomenon (cold sensitivity and numbness of fingers) were reported in chemical plant workers exposed to 25–250 ppm VC (64–639 mg/m^3) (Occidental Chemica Corporation, 1975).

An occupational study attempted to correlate the effects of VC on the liver function of exposed workers (77 total), as measured by the plasma clearance of the ^{99m}Tc-N-(2,4-dimethylacetanilido) iminodiacetate (HEPIDA) complex (Studniarek et al., 1989). The duration of exposure varied from 3 to 17 years. Personal air samplers were used to determine the mean VC concentrations in 1982 at various regions of the plant. Polymerization operators (n = 13) had the highest mean exposure to VC, 30 mg/m^3, with a mean duration of employment of 10 years. Autoclave cleaners (n = 9) and auxiliary personnel (n = 12) in polymerization rooms were exposed to mean concentrations of 9 mg/m^3 for a mean duration of 8 and 12 years, respectively, whereas technical supervisors (n = 6) had the lowest mean VC exposure of 6 mg/m^3 for a mean duration of 13 years. The investigators found a significant correlation between degree of exposure to VC and frequency of low clearance values; however, no concentration-response relationship was detected among the groups with respect to plasma clearance of ^{99m}Tc-HEPIDA. This study is of limited value because personal air sampling was conducted for only 1 year. The yearly geometric means of VC atmospheric concentrations in various departments of the plant were provided, but these concentrations fluctuated dramatically between 0.1 and 600 mg/m^3 from 1974 to 1982.

There was no evidence of decrements in pulmonary function over the course of a work shift in a group of 53 chemical, plastics, and rubber workers exposed to higher VC levels (up to 250 ppm, 639 mg/m^3) (Occidental Chemical Corporation, 1975). In an analysis of causes of death in a cohort of 10,173 VC workers for up to 30 years after the onset of exposure, the only noncancer cause for which the SMR was significantly elevated was emphysema (Dow Chemical Company, 1986). There was no correlation with exposure duration or latency. There was also no control for smoking, although there was no excess of lung cancer.

Insufficient data exist to evaluate the teratogenicity of VC in humans. Several epidemiology studies have investigated the effects of VC exposure on incidence of fetal loss and birth defects (Hatc et al., 1981; Infante et al., 1976; Waxweiler et al., 1977); however, no solid association has been found. Studies of communities near VC plants (Edmonds et al., 1978; Theriault et al., 1983) have found no clear association between parental residence in a region with a VC plant and the incidence of birth defects in the exposed community.

VC does not appear to be teratogenic in animals and is embryotoxic only at high levels. Inhalation experiments in animals have associated developmental toxicity only with concentrations at or above those associated with maternal toxicity. John et al. (1977) examined the effects of inhaled V on the fetuses of mice, rats, and rabbits. Pregnant CF1 mice (30–40/group) were exposed to 0, 50, or 500 ppm VC on gestational days 6–15. Sprague-Dawley rats (20–35/group) and New Zealand white rabbits (15–20/group) were administered 0, 500, or 2500 ppm VC, 7 hours/day on gestational days 6–15 for rats and 6–18 for rabbits. Parameters of maternal and developmental toxicity were evaluated; both the fetuses and litters were evaluated. Mice were more sensitive to the toxic effects of VC than either rats or rabbits. In mice, concentrations of 500 ppm induced maternal effects that included

increased mortality, reduced body weight, and reduced absolute, but not relative, liver weight. Fetotoxicity also occurred in mice at 500 ppm, and was manifested as significantly increased fetal resorption, decreased fetal body weight, reduced litter size, and retarded cranial and sternebral ossification. However, there was no evidence of a teratogenic effect in mice at either concentration. In rats exposed to 500 ppm, but not to 2500 ppm, maternal effects were restricted to reduced body weight. Maternal effects in rats at 2500 ppm were death of one rat, elevated absolute and relative liver weights, and reduced food consumption. A significant reduction in fetal body weight and an increase in the incidence of lumbar spurs were observed among rats exposed to 500 ppm but not 2500 ppm, an are not considered signs of VC-induced fetotoxicity. At 2500 ppm, increased incidence of dilated ureters was observed, which may represent a chemical-induced effect. No signs of maternal or developmental toxicity were observed in rabbits at either dose. This study identifies a NOAEL of 50 ppm (130 mg/m^3) for maternal and fetotoxicity in mice and a NOAEL of 2500 ppm (6500 mg/m^3) for rabbits.

Ungvary et al. (1978) exposed groups of pregnant CFY rats continuously to 1500 ppm (4000 mg/m^3) on gestational days 1–9, 8–14, or 14–21 and demonstrated that VC is not teratogenic and has no embryotoxic effects when administered during the second or last third of pregnancy. During the first third of pregnancy, maternal toxicity was manifested by increased relative liver weight; increased fetal mortality and embryo toxic effects were evident. Slightly reduced body weight gain was noted in dams exposed on days 14–21.

VC does not appear to produce germinal mutations as manifested by a dominant lethal effect in male rats. In a dominant lethal study, Short et al. (1977) exposed male CD rats to 0, 50, 250, or 1000 ppm VC 6 hours/day, 5 days/week for 11 weeks. At the end of the exposure period, the exposed males were mated with untreated females, and there was no evidence of either preimplantation or postimplantation loss in pregnant females. However, reduced fertility was observed in male rats exposed to 250 and 1000 ppm (650 and 2600 mg/m^3) VC.

Absorption of VC in humans after inhalation exposure is rapid. A study conducted in five youn adult male volunteers showed that 42% of inhaled VC in the lung was retained, that maximum retention was reached within 15 minutes, and that the percent retention was independent of inspired VC concentration at least to the maximum used in the experiment, 60 mg/m^3. After cessation of exposure, the VC concentration in expired air decreased rapidly within 30 minutes to 4% of the inhaled concentration (Krajewski et al., 1980). Animal inhalation studies also showed that VC is rapidly absorbed. Exposure of male Wistar rats (number/group unspecified) to 1000, 3000, or 7000 ppm VC (99.9% pure) for 5 hours using a head-only apparatus resulted in rapid uptake into the blood, as measured by gas-liquid chromatography (GLC) (Withey, 1976). Equilibrium blood levels were achieved within 30 minutes for all exposures. Upon cessation of exposure, blood levels declined to a barely detectable level after 2 hours. Rat studies show that the distribution of VC is rapid and widespread, but the storage of VC in the body is limited by its rapid metabolism and excretion (Bolt e al., 1977).

The primary route of VC metabolism is by the action of cytochrome P450 isozymes, primarily CYP IIE1, to form a highly reactive epoxide intermediate, CEO, which spontaneously rearranges to form CAA. These intermediates are detoxified mainly through conjugation with glutathione catalyzed by glutathione S-transferase (Hefner et al., 1975; Bolt et al., 1976; Jedrychowski et al., 1984; Watanabe et al., 1978a). The conjugated products are excreted in urine as substituted cysteine derivatives (Bolt et al., 1980; Hefner et al., 1975). Although VC has often been cited as a chemical for which saturable metabolism should be considered in the risk assessment, saturation appears to becom important only at very high exposure levels (greater than 250 ppm by inhalation or 25 mg/kg-day orally) compared with those associated with the most sensitive noncancer effects or tumorigenic levels, and thus has little impact on the risk estimates.

Based on the elimination of VC observed following administration by various routes of exposure, the metabolism of VC appears to be a dose-dependent, saturable process in animals (Green

and Hathway, 1975; Hefner et al., 1975; Gehring et al., 1978) and in humans (Krajewski et al., 1980). Saturation of metabolic pathways occurred at exposure concentrations of 250 ppm VC in male Wistar rats and 200 ppm in Rhesus monkeys; at concentrations below this, a straight, first-order decline in radioactivity was observed (Bolt et al., 1977; Buchter et al., 1980; Filser and Bolt, 1979). Studies hav demonstrated the binding of metabolites of ^{14}C-VC to liver macromolecules in vitro, and in rats exposed by inhalation (Guengerich and Watanabe, 1979; Guengerich et al., 1979, 1981; Kappus et al., 1976; Watanabe et al., 1978a,b). In single-exposure experiments at concentrations ranging from 1 to 5000 ppm ^{14}C-VC, the binding to macromolecules increased proportionately with increasing metabolites of VC, and disproportionately with VC exposure concentration (Watanabe et al., 1978b).

The observation of Watanabe et al. (1978b) of a disproportionate relationship between effects (e.g., binding to macromolecules, liver effects, tumors) and exposure concentrations of unmetabolized VC is a principal reason for using PBPK modeling. The important contribution of PBPK modeling is to provide a more biologically plausible estimate of the effective dose, that is, the total production of reactive metabolites at the target tissue. The ratio of this biologically effective dose to exposure concentration or administered dose is not uniform across routes and species. Therefore, any estimate o administered dose is less adequate for performing route-to-route and interspecies extrapolation of risk.

Several different PBPK models for VC have been described in the literature. These models are described in detail and compared in the accompanying Toxicological Review Appendix A. The PBPK model used in this assessment was developed to support a cancer risk assessment based on the pharmacokinetic and metabolic data available in the literature for VC (Clewell et al., 1995a,b). The initial metabolism of VC was hypothesized to occur via two saturable pathways, one representing low capacity–high affinity oxidation by cytochrome P450 IIE1 and the other representing higher capacity–lower affinity oxidation by other isozymes of P450, producing in both cases CEO as an intermediate product. The parameter values for the two metabolic pathways describing the initial step in VC metabolism were determined by simulation of gas uptake data from mice, rats, hamsters, monkeys, an controlled human inhalation exposures, as well as from data on total metabolism and glutathione depletion in both oral and inhalation exposures. Successful simulation of pharmacokinetic data from a large number of studies over a wide range of concentrations using primarily inhalation exposure and different measures of effect (decreased chamber concentration of VC, decreased serum levels of GSH) served as evidence that the PBPK model was valid over the exposure range of interest, especially for inhalation exposure scenarios. One limitation of the model is the lack of pharmacokinetic data via the oral route available for simulation and model validation. Model parameters for deriving dose metrics via the oral route have therefore been established such that the dose metrics generated would be "conservative," that is, predictive of higher human risk from animal results. This model, including the parameters and the rationale for their choice, pharmacokinetic data and model fit to these data, the sensitivity analysis of the model, and the actual dose metrics derived, is also presented in the appendices of the Toxicological Review.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- High
Database -- Medium to High
RfC -- Medium

The overall confidence in this RfC assessment is medium. Confidence in the study of Til et al. (1983, 1991) is high because it used adequate numbers of animals, was well controlled, and reported i detail on the histological effects on the liver. Bi et al. (1985) and Sokal et al. (1981) both give corroborative information on liver effects following inhalation exposure. Because of the close similarity of the pharmacokinetics via the inhalation and oral routes and the use of a PBPK model, inhalation data can be used to fill gaps in the inhalation database and vice versa.

Confidence in the database is medium to high. The two-generation reproductive study of CMA (1998) showed no indication of reproductive effects while demonstrating liver effects corroborative of

results from other studies, both oral and inhalation. The repeated exposure dominant lethal study of Short et al. (1977) showed reduced fertility, but only at concentrations well above those producing effects in the target organ (liver). Two developmental inhalation studies were located that reported embryotoxic effects only at levels much higher than those causing maternal toxicity in mice, rats, or rabbits (John et al., 1977; Ungvary et al., 1978). Several other inhalation studies report on other endpoints and support the use of the liver effects. Concern for the confidence of dose metrics derived by the PBPK model from the oral study of Til et al. is also offset by procedures instituted within the model when calculating oral dose metrics, including assumption of a maximum rate of VC uptake (i.e designating it a zero-order process) and spreading the applied dose over a 24-hour period, which woul minimize the concentration and maximize the likelihood that the parent VC would be metabolized to reactive species (i.e., the basis of this assessment, mg VC metabolized).

The high degree of confidence in the principal study of Til et al. (1983, 1991), combined with the medium to high assessment of the database and less than high confidence in the qualitative aspects of the PBPK model, is considered to result in an overall medium confidence in the RfC.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA, 2000

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Vinyl Chloride in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 2000).

Agency Consensus Date – 07/20/2000

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRI in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Vinyl Chloride
CASRN -- 75-01-4
Last Revised --08/07/2000

Section II provides information on three aspects of the carcinogenic assessment for the substance in question, the weight-of-evidence judgment of the likelihood that the substance is a huma carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per µg/L drinking water or risk per µg/m^3 air breathed. Th third form in which risk is presented is a concentration of the chemical in drinking water or air providing cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed sinc the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utiliz those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

On the basis of sufficient evidence for carcinogenicity in human epidemiology studies, VC is considered to best fit the weight-of-evidence Category "A," according to current EPA Risk Assessme Guidelines (U.S. EPA, 1986). Agents classified into this category are considered known human carcinogens. This classification is supported by positive evidence for carcinogenicity in animal bioassays including several species and strains, and strong evidence for genotoxicity.

Under the Proposed Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1996), it is concluded that VC *is a known human carcinogen by the inhalation route of exposure*, based on huma epidemiological data, and by analogy *the oral route* because of positive animal bioassay data as well as pharmacokinetic data allowing dose extrapolation across routes. VC *is also considered highly likel to be carcinogenic by the dermal route* because it is well absorbed and acts systemically. The weight of evidence for human carcinogenicity is based on (1) consistent epidemiologic evidence of a causal association between occupational exposure to VC via inhalation and the development of angiosarcoma, an extremely rare tumor; (2) consistent evidence of carcinogenicity in rats, mice, and hamsters by both the oral and inhalation routes; (3) mutagenicity and DNA adduct formation by VC and its metabolites in numerous in vivo and in vitro test systems; and (4) efficient VC absorption via all routes of exposure tested, followed by rapid distribution throughout the body. In light of the very high percentage of angiosarcomas worldwide that are associated with VC exposure, the evidence for VC carcinogenicity is considered strong.

The International Agency for Research on Cancer (IARC) has also concluded that sufficient evidence for carcinogenicity in humans exists and has placed VC in carcinogenicity category 1, that is carcinogenic to humans (IARC, 1979).

VC carcinogenicity occurs via a genotoxic pathway and is understood in some detail. VC is metabolized to a reactive metabolite, probably chloroethylene oxide (CEO), which is believed to be the ultimate carcinogenic metabolite of VC. The reactive metabolite then binds to DNA, forming DN adducts that, if not repaired, ultimately lead to mutations and tumor formation. Therefore, a *linear* extrapolation was used in the dose-response assessment. Because of uncertainty regarding exposure levels in the occupationally exposed cohorts, recommended potency estimates are based on animal bioassay data.

___II.A.2. HUMAN CARCINOGENICITY DATA

Sufficient: Several independent retrospective and prospective cohort studies demonstrate a statisticall significant elevated risk of liver cancer, specifically angiosarcomas, from exposure to VC monomer (Monson et al., 1974; Tabershaw and Gaffey, 1974; Byren et al., 1976; Waxweiler et al., 1976; Fox and Collier, 1977; Cooper, 1981; Weber et al., 1981; Jones et al., 1988; Wu et al., 1989; Pirastu et al., 1990; Simonato et al., 1991; Wong et al., 1991; Du and Wang, 1998; Pirastu et al., 1998; CMA, 1998) The possible association of brain, soft tissue, and nervous system cancer with VC exposure was also reported (Monson et al., 1975; Waxweiler et al., 1976; Cooper, 1981; Wong et al., 1991; CMA et al., 1998). The evidence supporting a causal link between brain cancer and VC exposure is limited by the fact that most of the positive studies utilized the same CMA cohort, or workers from the same plants, and according to Doll (1988) contain certain weaknesses in the data. Some studies have found an association between VC exposure and cancer of the hematopoietic and lymphatic systems (Simonato al., 1991; Weber et al., 1981); observed increases in other studies fell below statistical significance du to the small numbers of these types of cancers (Tabershaw and Gaffey, 1974). VC exposure has also been associated with lung cancer (Buffler et al., 1979; Monson et al., 1975; Waxweiler et al., 1976, 1981), but this response was considered more likely due to PVC particles than to VC. An excess of melanoma was reported in one study (Heldaas et al., 1984), but other studies have not substantiated

this report.

In 1974, Creech and Johnson reported for the first time an association between exposure to VC and cancer in humans: three cases of liver angiosarcoma were reported in men employed in a PVC plant. Angiosarcoma of the liver is considered to be a very rare type of cancer, with only 20–30 cases per year reported in the United States (Gehring et al., 1978; ATSDR, 1995). As described in the following paragraphs, greater than expected incidences of angiosarcoma of the liver have since been reported in a number of other cohorts of workers occupationally exposed to VC.

In a proportionate mortality study analyzing the causes of death of 142 workers exposed to VC monomer or VC/PVC, Monson et al. (1974) found an excess incidence of liver cancer (8 observed versus 0.7 expected). Five of these were angiosarcomas. The study also found an excess of brain cancer (5 observed versus 1.2 expected) and lung cancer (13 observed versus 7.9 expected). No statistical analysis was conducted by tumor target.

Byren et al. (1976) reported a significantly elevated risk of pancreas/liver cancer (4 observed versus 0.97 expected) in a cohort of 750 Swedish workers exposed to VC. They also found a small excess of brain cancer (2 observed versus 0.33 expected).

Waxweiler et al. (1976) found a significantly elevated risk (7 observed versus 0.6 expected) of liver cancer in a cohort of 1294 workers who were exposed to VC monomer for a minimum of 5 years and followed for 10 or more years. In a separate phase of the study, the authors identified 14 cases of liver and biliary cancer, 11 of which were angiosarcomas. Several of the identified subjects were not included in the main study because they were still alive, or because they did not meet the minimum criteria for inclusion in the cohort. Brain cancer incidence was significantly increased in workers observed for 15 years or more after initial exposure (3 observed versus 0.6 expected); a nonsignificant increase was observed for a 10-year latency. The cohort study also found a slight excess risk of lymphatic and hematopoietic system cancer (4 observed versus 2.5 expected). Of the 14 cases of primary lung cancer identified, 5 were large-cell undifferentiated, three were adenocarcinomas, and there were no squamous cell or small cell bronchiogenic carcinomas, suggesting that these cancers were not associated with smoking. In a study of 4806 workers at the same plants, an elevated risk of lung cancer was found in those exposed to PVC and chemicals other than VC. PVC appeared to be th most likely etiologic agent (Waxweiler et al., 1981).

A large number of occupational studies reported an association between VC and liver angiosarcoma or hepatocellular carcinoma, but quantitative exposure information is available for only a few studies. Fox and Collier (1977) reported 4 cases of liver cancer, 2 of which were angiosarcomas in a cohort of 7717 British VC workers. The study authors grouped the subjects by estimated exposur levels and exposure duration. From these data, average exposure levels have been estimated as 12.5, 70, and 300 ppm (Clement Associates, 1987) or 11, 71, and 316 ppm (Chen and Blancato, 1989). Because workers were classified on the basis of the maximum exposure for each worker, cumulative exposure is overestimated, leading to a probable underestimation of risk using these data. Both angiosarcoma cases were considered to have had high exposure to VC monomer, at the level of 200 ppm and above TWA. There was no effect on other cancers, in comparison with cancer rates in England and Wales. In a followup study, Jones et al. (1988) analyzed mortality in 5498 male VC workers. This study found a significant excess of primary liver tumors, with 11 deaths, 7 of which were angiosarcomas. The median latency for angiosarcomas was 25 years.

Weber et al. (1981) examined mortality patterns in 7021 German and Austrian VC monomer/PVC workers and 4007 German PVC processing workers. Comparisons were with West German population death rates. A significantly elevated risk of liver cancer (12 observed versus 0.79 expected) was observed in the VC monomer/PVC cohort, but a significant increase (4 observed versu 1 expected) was also observed in an unexposed reference group. However, the risk in the VC monome cohort increased with exposure duration. The study authors implied that four cases of angiosarcoma were identified in the study cohort, although it was not clear if all of the cases belonged to this cohort.

A significant excess risk of brain cancer (Obs = 5, SMR = 535, $p < 0.05$) was also observed in the PVC processing workers, but not in VC monomer/PVC workers. Risk of lymphatic and hematopoieti cancer (Obs = 15, SMR = 214) was significantly increased in VC monomer/PVC production workers, and there was a tendency for increased risk at longer exposure durations.

In a preliminary mortality followup study of 464 workers at an Italian VC monomer production facility, a significant excess of respiratory cancers was observed (Obs = 5, SMR = 289, $p < 0.03$) (Bel et al., 1987). The excess remained after correction for smoking and was associated with longer exposure durations and higher exposure levels. They also found a significant excess of lung cancer in preliminary report of a cohort of 437 VC monomer/PVC workers.

Smulevich et al. (1988) investigated a cohort of 3232 workers (2195 men, 1037 women) in a Soviet VC/PVC chemical plant. No cases of angiosarcoma or other liver tumors were reported. Workers who were highly exposed to VC (> 300 mg/m^3) had a significantly elevated risk of lymphomas and leukemias (apparently 7 observed versus about 1.1 expected for combined men and women, but there are inconsistencies in the reported numbers). The risk of brain cancer was elevated i women (Obs = 2, SMR = 500), but the effect was not statistically significant and the incidence in men was unaffected. Although mammary tumor incidence was increased in some of the animal studies reported in the following section, no cases of breast cancer were reported in the workers. This is the only study having a significant number of females in the cohort.

Wu et al. (1989) investigated a cohort of 2767 VC monomer workers, most of whom had been employed for fewer than 5 years. There was a significant excess risk of liver cancer (14 observed versus 4.2 expected). The incidence of angiosarcomas was not reported, but 12/18 liver cancers were angiosarcomas in a larger cohort of 3620 workers that included workers exposed to PVC, as well as the VC monomer workers. In a case-control study with the controls taken from a NIOSH database, angiosarcomas were related to higher cumulative exposure to VC monomer, but other liver cancers were not. Brain and lung cancer were not elevated for the VC monomer workers, but were elevated fo the combined cohort.

Pirastu et al. (1990) evaluated clinical, pathological, and death certificate data for 63 deaths in 3 VC monomer/PVC manufacturing or PVC extruding plants in Italy. Fourteen deaths from primary liver cancer were found, of which seven were identified as angiosarcoma and two as hepatocellular carcinoma. No comparison with a control population was conducted. However, the authors stated that this study indicated a relationship between VC exposure and primary liver cancer, as well as with angiosarcoma. Pirastu et al. (1998) updated the cohort through 1996 for one of the plants and 1997 for the other two. The combined SMR for liver cancer at the three plants equaled 364 ($p<0.05$).

Simonato et al. (1991) reported on the results of a large multicentric cohort study of 12,706 European VC/PVC workers. A significant increase in liver cancer deaths was observed (Obs = 24, SMR = 286). Workers were classified on the basis of maximum exposure level into ranges of < 50 ppm, 50–499 ppm, and ≥500 ppm. Estimating an average exposure duration of 9 years, average exposure levels for these groups can be estimated at 25, 158, and 600 ppm. Histopathology was available for 17 of the liver cancers; 16 were confirmed as angiosarcoma and one was a nonangiosarcoma primary liver cancer. Excess risk from liver cancer was related to the time since firs exposure, duration of exposure, and estimated total exposure. An increased risk of lymphosarcoma wa observed (SMR = 661, 95% CI = 136–1931), but there was no relationship to duration of employment Brain cancer had an elevated risk in certain analyses, but there was no clear relationship to exposure duration. There was no excess risk of lung cancer.

Du and Wang (1998) studied 2224 workers employed at 5 factories in Taiwan during the period 1989–1995. A significantly increased risk of hospital admission among VCM workers due to primary liver cancer was reported, resulting in a morbidity odds ratio (MOR) of 4.5–6.5. Of the 12 cases of liver cancer found, 6 were diagnosed as hepatocellular carcinoma. The other 6 appeared to be the same, although angiosarcoma could not be ruled out without pathological confirmation. Ten of 11

cases of liver cancer for which detailed information was available were carriers of hepatitis B virus.

In a preliminary report with only 85% followup completed, Tabershaw and Gaffey (1974) compared mortality in a cohort of 8384 men occupationally exposed to VC with death rates among U.S. males. Each VC plant classified workers as exposed to high, medium, or low levels of VC, but n quantitative estimate of exposure was provided, and no attempt was made to establish consistent gradations of exposure between plants or exposure periods. No significant increases in any general cancer classification were found. However, six cases of angiosarcoma of the liver identified by other investigators occurred in the study population; only two of these were identified as angiosarcomas on the death certificate. The study authors also noted that 6 of 17 (40%) deaths in the category "other malignancies" were due to brain cancer. They stated that only 22% of the deaths in this category woul be expected to be due to this cause, but they did not provide any supporting documentation. This preliminary report also noted a slight excess risk of lymphomas (5 observed versus 2.54 expected) in the group with the higher exposure index.

Cooper (1981) enlarged the Tabershaw and Gaffey (1974) study to include 10,173 VC workers; vital status was ascertained for 9677 men. Cooper noted that, of the nine angiosarcomas identified in the United States workers during the study period (prior to 1/93), eight were included in the study cohort. Statistical analyses were conducted for broad categories of tumors; a significant increase (Obs = 12, SMR = 203, $p < 0.05$) was observed for brain and central nervous system malignancies.

An update on this cohort (Wong et al., 1991) also found an association between VC exposure and angiosarcoma. Fifteen deaths from angiosarcoma were identified, a clear excess over the incidenc in the general population, although no statistical analysis was conducted for this malignancy. This study also attempted to determine whether other cancers are associated with VC exposure. Excluding the 15 angiosarcomas identified from death certificates, a significant increase was observed in liver and biliary tract cancers alone (Obs = 22, SMR = 386, $p < 0.02$). However, the study authors suggeste that these 22 cancers probably included some cases of angiosarcoma that were misdiagnosed. Based o a comparison of death certificates and pathology records in 14 cases, they estimated that the correct number of primary liver/biliary tract cancers (excluding angiosarcomas) is 14, which is still significantly increased over background (SMR = 243, $p < 0.01$). This study also found a significantly increased risk of cancer of the brain and central nervous systems (Obs = 23, SMR = 180, $p < 0.05$). There was no excess in cancer of the respiratory system or the lymphatic and hematopoietic system. Expected deaths were based on U.S. mortality rates, standardized for age, race, and calendar time.

CMA (1998) updated the Wong et al. (1991) study through 1995. This study was also designed to evaluate possible induction of cancer at a larger number of target sites than the Wong et al. study. I this study, all liver and biliary cancers were included in a single category. Mortality rate for these cancers, based on 80 deaths, was again significantly increased (SMR = 359; 95% CI = 284–446). The SMRs increased with duration of exposure from 83 (95% CI = 33–171), to 215 (95% CI = 103–396) t 679 (95% CI = 483–929), to 688 (95% CI = 440–1023) for workers exposed from 1 to 4 years, 5 to 9 years, 10 to 19 years, and 20 years or more, respectively. Mortality from brain and CNS cancer showed an excess based on 36 deaths (SMR = 142; 95% CI = 100–197). The elevation was statisticall significant for those exposed 5– 9 years (SMR = 193; 95% CI = 96–346) and for those exposed 20 years or more (SMR = 290; 95% CI = 132–551). Finally, mortality from connective and other soft tissue cancers, based on 12 deaths, was also increased significantly (SMR = 270; 95% CI = 129–472). The increases were significant for those exposed 10–19 years (SMR = 477; 95% CI = 155–1113) and 20 or more years (SMR = 725; 95% CI = 197–1856). This cause of death category had not been evaluated in the Wong et al. (1991) study. Deaths were based upon regional (State-weighted) mortalit rates for white males.

In conclusion, strong evidence exists for a causal relationship between exposure to VC in humans and a significantly excessive risk of liver angiosarcoma; the highest relative risk is associated with this cancer type. There is also highly suggestive evidence of a causal relationship with other liver cancers. Brain cancer and cancer of the lymphopoietic system, connective tissue, and soft tissue have been associated with VC exposure in some studies, but not others, suggesting a possible relationship.

Lung cancer has also been associated with VC exposure, but, based on the data of Waxweiler et al. (1981), the increased risk of lung cancer observed in some cohorts may be due to exposure to PVC dust, rather than VC monomer. In reviewing the effects of exposure to VC, both Doll (1988) and Storm and Rozman (1997) concluded that evidence for induction of nonliver tumors is weak. Because of the consistent evidence for liver cancer in all the studies, knowledge that VC is metabolized primarily by the liver, and the weaker association for other sites, it is concluded that the liver is the most sensitive site, and protection against liver cancer will protect against possible cancer induction in other tissues.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient: VC is carcinogenic in rodents by both oral and inhalation routes, and some data indicate that it produces tumors when given i.p., s.c., and transplacentally.

Feron et al. (1981) conducted lifespan oral bioassays of VC in Wistar rats. In order to incorporate VC into the diet of Wistar rats, Feron et al. (1981) administered diets containing 10% PV with varying proportions of VC monomer. Diets were available to experimental animals for 4 hours per day and food consumption and VC concentrations were measured several times during the feeding period to account for loss of VC from the diet due to volatilization. This information was used to calculate the ingested dose. Evaporative loss averaged 20% over 4 hours. The ingested dose was adjusted downward by the amount of VC measured in the feces to arrive at the bioavailable doses of 0 1.7, 5.0, or 14.1 mg VC/kg-day, which were fed to Wistar rats (n = 80, 60, 60, and 80, respectively) fo a lifetime. The amount actually absorbed was used in estimating risk because, although VC absorptio is near 100% under most conditions, in the present case a small amount of VC was attached to or encapsulated in the PVC present in the feed and not taken up. Animals were sacrificed at 135 weeks (males) or 144 weeks (females). An additional group of 80/sex were administered 300 mg/kg bw/day by gavage in oil 5 days/week for 83 weeks. Increased mortality was noted in all treated groups, as was increased tumor incidence. Almost exclusively angiosarcomas were observed in the groups administered 300 mg/kg-day by gavage, whereas a mixture of angiosarcomas, hepatocellular carcinomas, and neoplastic nodules was observed at the middle and high dietary doses. Only hepatocellular carcinomas and neoplastic nodules were reported at the low dose. Several other rare tumors were identified as possibly associated with VC exposure. With one exception, animals with pulmonary angiosarcomas (significant at $p < 0.05$) also had liver angiosarcoma, suggesting metastases from the liver. A few Zymbal gland tumors, a rare tumor type, were noted, although the increases wer not statistically significant. These neoplasms occurred at and above doses of 5 mg/kg bw/day. Abdominal mesotheliomas were elevated over controls in all dosed groups, but there was no clear dos response. Significant increases in preneoplastic proliferative lesions (clear-cell foci, basophilic foci, and eosinophilic foci) were observed in all dose groups. These foci are hepatocyte-derived, whereas angiosarcomas are derived from sinusoidal cells, indicating that the foci are precursors of hepatocellular carcinomas, not angiosarcomas.

Til et al. (1983, 1991) extended the study of Feron et al. (1981) to lower doses. The oral doses were delivered in the same way except that the diets contained a final concentration of 1% PVC, rathe than 10%. Groups of 50 or 100 male and female Wistar rats were administered lifetime dietary doses of VC at 0, 0.014, 0.13, or 1.3 mg/kg bw/day (149 weeks for males and 150 weeks for females). An additional control group of 100 males and 100 females was held in a separate room. Mortality differences were not remarkable for males, but were slightly increased for females receiving 1.3 mg/kg-day. Angiosarcomas were observed in one high-dose male and two high-dose females. Although this incidence did not achieve statistical significance, angiosarcomas are rare in rats. Other significant increases in tumors were limited to neoplastic nodules in females and hepatocellular carcinomas in males. No Zymbal gland tumors or abdominal mesotheliomas were observed. In this study, VC at 0.13 mg/kg-day did not induce tumors, whereas at 1.3 mg/kg-day, neoplastic and nonneoplastic lesions in the liver were clearly increased by comparison to controls. Significant increases in foci of cell proliferation were observed in males and females at the high dose, with significant increases in basophilic foci extending down to 0.014 mg/kg-day.

Male and female Sprague-Dawley rats (40/sex/group) were administered 0, 3.33, 16.65, or 60 mg/kg VC in olive oil, by gavage, 5 days/week beginning at 13 weeks of age and continuing for 52 weeks (Maltoni et al., 1981, 1984). Animals were observed for their lifetime (136 weeks). In a separat phase of testing, groups of 75 Sprague-Dawley rats received 0, 0.03, 0.3, or 1.0 mg/kg-day VC using the same dosing protocol. During the second year of the study, all three groups of treated males showed a lower rate of survival than controls. The rate of survival in controls was very low in terms o adequate number surviving for development of neoplasms appearing late in life. Nonetheless, angiosarcomas of the liver appeared with a dose-related incidence, down to a dose of 0.3 mg/kg-day. Nephroblastomas, a rare tumor type in rats, were also reported at 16.65 and 60 mg/kg-day. There was no effect on mammary tumors.

Male and female Sprague-Dawley rats (30/sex/group) were exposed to 0, 1, 5, 10, 25, 50, 100, 150, 200, 250, 500, 2500, 6000, or 10,000 ppm VC by inhalation for 4 hours/day, 5 days/week for 52 weeks (Maltoni et al., 1981, 1984). Animals were observed throughout their lifetime (135 weeks). Tumor incidence (including liver angiosarcomas) and latency were concentration dependent. Additional tumor types seen included liver hepatoma, nephroblastoma, neuroblastoma of the brain, Zymbal gland tumors, and mammary carcinomas. The study authors particularly noted the rarity of angiosarcoma, hepatoma, nephroblastoma, and neuroblastoma in their animal colony.

These results in rats are confirmed in similar experiments in other species. Maltoni et al. (1984) also exposed male and female Swiss mice and male Syrian golden hamsters (approximately 40–80/sex/species/group) to 0, 50, 250, 500, 2500, 6000, or 10,000 ppm VC by inhalation for 4 hours/day 5 days/week for 30 weeks. Animals were observed for life. The following types of tumors were increased in exposed mice: mammary, liver (including angiosarcomas), forestomach, lung, and epithelial. Tumor types in hamsters were liver (including angiosarcomas), forestomach, and epithelial.

Other inhalation experiments support the carcinogenicity of VC. Rats and mice exposed to 0, 5 250, or 1000 ppm for 6 hours per day, 5 days per week for up to 6 months (mice) or 10 months (rats) (Hong et al., 1981) or up to 12 months (mice and rats) (Lee et al., 1978) had a significantly increased incidence of hemangiosarcoma of the liver at $\geq$250 ppm. Animals were sacrificed 12 months after the end of exposure. Mice in this study exposed to $\geq$250 ppm also had an increase in bronchoalveolar adenoma of the lung and mammary gland tumors in females (adenocarcinomas, squamous and anaplastic cell carcinomas). Male rats exposed to concentrations as low as 100 ppm for 6 hours per day, 6 days per week, for 12 months and sacrificed at 18 months (6 months after the end of exposure) had significantly increased incidences of angiosarcoma of the liver (Bi et al., 1985). Rats exposed to 3% VC (30,000 ppm) for 4 hours per day, 6 days per week, for 12 months had significantly increased incidences of epidermoid carcinoma of the skin, adenocarcinoma of the lungs, and osteochondroma in the bones (Viola et al., 1971), and rats exposed to 0–5000 ppm for 52 weeks had primary tumors in th brain, lung, Zymbal gland, and nasal cavity (Feron and Kroes, 1979). Keplinger et al. (1975) provided a preliminary report of a concentration-dependent increase in tumor formation (alveologenic adenoma of the lung, angiosarcomas of the liver, and adenosquamous carcinoma of the mammary gland) in mic exposed to 0, 50, 200, or 2500 ppm VC.

Suzuki (1978, 1983) investigated the effect of VC on lung tumor formation. In a preliminary study conducted with a limited number of animals, alveologenic lung tumors developed in 26 of 27 mice exposed to 2500 or 6000 ppm for 5–6 months (Suzuki, 1978). A concentration-related increase i the incidence of alveologenic tumors was observed in a study in which 30–40 mice/group were exposed to 1–660 ppm or filtered air for 4 weeks and then observed for up to 41 weeks postexposure (Suzuki, 1983). An increase in bronchoalveolar adenoma was observed in a lifetime study of mice exposed to 50 ppm for 100 1-hour exposures, and 5000 or 50,000 ppm for a single 1-hour exposure (Hehir et al., 1981). The statistical significance of these observations was not presented.

The available evidence from either inhalation or oral studies in animals supports the findings in humans that VC is carcinogenic. Additional evidence for cancer induction by intraperitoneal,

subcutaneous, and transplacental administration of VC was presented by Maltoni et al. (1984). IARC reached similar conclusions (IARC, 1979).

Several studies have compared the carcinogenic effects of VC in newborn animals and adults. Newborn rats treated with VC respond with both angiosarcoma and hepatocellular carcinoma, in contrast with adult animals, in which angiosarcomas generally predominate (Maltoni et al., 1981). Consistent with this observation, VC was found to induce preneoplastic foci in newborn rats, but not i adults (Laib et al., 1979). Interestingly, in the same study it was found that VC did induce preneoplastic foci in adult rats after partial hepatectomy, indicating that the appearance of foci, and presumably of hepatocellular carcinoma, in neonatal animals was a consequence of the increased rate of cell proliferation at that age. Similarly, Laib et al. (1989) found that inhaled radiolabeled VC was incorporated into physiological purines of 11-day-old Wistar rats at eightfold higher levels than in similarly treated adult rats (presumably reflecting the DNA replication activity), and roughly fivefold higher levels of the DNA adduct 7-(2-oxoethyl) guanine (OEG) were found in the livers of young animals, reflecting an increased alkylation rate. In a similar study, roughly fourfold greater concentrations of both OEG and N2,3-ethenoguanine (EG) were also seen in preweanling rats expose to VC (Fedtke et al., 1990). The higher cell proliferation rates found in newborn animals suggest that VC, or any other DNA-reactive carcinogen, could be more potent in newborns than in adults. Nevertheless, increased cell proliferation and DNA adduct formation are not in themselves adequate t demonstrate a quantitative potency difference for tumor formation between infants and adults.

Additional evidence indicating that young animals are more sensitive than adults to VC carcinogenicity is provided by Maltoni et al. (1981). Pregnant rats were exposed from gestation day 1 through 18 to 6000 or 10,000 ppm VC for 4 hours/day, and tumors were ascertained at 143 weeks postexposure. Nephroblastomas, forestomach tumors, epithelial tumors, and mammary gland carcinomas were observed only in the offspring, and the incidence of Zymbal gland carcinomas was higher in transplacentally exposed animals than in maternal animals. Because the dams and offspring were followed for the same period, latency is not an issue for this experiment. However, it is importan to note that the offspring were exposed during organogenesis, a period of rapid cell division, and any genotoxic carcinogen would be expected to have a higher potency during this period.

Drew et al. (1983) studied the effects of age and exposure duration on cancer induction by VC i rats, mice, and hamsters. Female golden Syrian hamsters, F344 rats, Swiss CD-1 mice, and B6C3F1 mice were exposed for 6 hours/day, 5 days/week to VC (50, 100, or 200 ppm for mice, rats, and hamsters, respectively) for 6, 12, 18, or 24 months, with the exception of mice, which were exposed only up to 18 months. All animals were sacrificed at month 24 or 18 (mice), and about 50 animals/species/group were tested. Other groups of rodents were held 6–12 months, and then exposed for 6 or 12 months, and also sacrificed at month 24. Unfortunately, time-to-tumor data were not reported in this study, making it impossible to deconvolute the impact of survival on the observation o tumors from later exposure periods. Because both mice and hamsters showed significant survival effects (life-shortening) from the VC exposures, only the data on exposures of rats during the first 12 months of life are appropriate for analysis. In the rats, exposure from 0 to 6 months showed an overall similar potency to exposure from 6 to 12 months of life. In particular, the incidence of hepatocellular carcinoma combined with neoplastic nodules and hemangiosarcoma was 24% and 5%, respectively, i rats exposed from 0 to 6 months, whereas for exposure from 6 to 12 months, the incidence was 31% and 4%, respectively. In this study, however, even the 0- to 6-month animals were 8–9 weeks old at the start of exposure and thus approaching maturity.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Several lines of evidence indicate that VC metabolites are genotoxic, interacting directly with DNA. Occupational exposure to VC has resulted in chromosome aberrations, micronuclei, and sister chromatid exchanges (SCEs); response levels were correlated with exposure levels (Hansteen et al., 1978; Purchase et al., 1978; Sinues et al., 1991). VC is mutagenic in the *Salmonella typhimurium* reverse mutation assay, with the mutagenic activity decreased or eliminated in the absence of

exogenous metabolic activation (Bartsch et al., 1975; Rannug et al., 1974). The VC metabolites CEO and CAA are both mutagenic in the *Salmonella* assay (Bartsch et al., 1975; Rannug et al., 1976). The highly reactive metabolite CEO was much more mutagenic than CAA, suggesting that this is the metabolite responsible for VC carcinogenicity. DNA adducts formed by VC have also been identified (Swenberg et al., 1992, 1999).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

____II.B.1.1. Oral Slope Factor	(a) LMS method (per mg/kg-day)	(b) LED 10/linear method (per mg/kg-day)
Continuous lifetime exposure during adulthood	7.2E-1	7.5E-1
Continuous lifetime exposure from birth	1.4	1.5

____II.B.1.2. Drinking Water Unit Risk	(a) LMS method (per µg/L)	(b) LED 10/linear method (per µg/L)
Continuous lifetime exposure during adulthood	2.1E-5	2.1E-5
Continuous lifetime exposure from birth	4.2E-5	4.2E-5

_____II.B.1.3. Extrapolation Method: (a) Linearized multistage (b) LED 10/linear method

The oral slope factor of 7.2E-1 per mg/kg-day to account for continuous lifetime exposure during adulthood, based on use of the linearized multistage model is recommended. A twofold increase to 1.4 per mg/kg-day to account for continuous lifetime exposure from birth is also recommended. According to the EPA Cancer Risk Assessment Guidelines of 1986 (U.S. EPA, 1987) "in the absence of adequate evidence to the contrary, a linearized multistage procedure will be employed." The 1996 proposed guidelines (U.S. EPA, 1996) recommend employment of the LED 10/linear method in similar situations. This approach is to draw a straight line between the point of departure from observed data, generally as a default the LED_{10}. The LED_{10} is the lower 95% limit on dose that is estimated to cause a 10% response. As can be seen, the derived values using either approach are virtually identical.

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration (µg/L)	
	Adult exposure	Exposure from birth
E-4 (1 in 10,000)	4.8	2.4
E-5 (1 in 100,000)	4.8E-1	2.4E-1
E-6 (1 in 1,000,000)	4.8E-2	2.4E-2

__II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor type: Total of liver angiosarcoma, hepatocellular carcinoma, and neoplastic nodules
Test animals: Female Wistar rats
Route: Oral, diet
Reference: Feron et al., 1981

Admin. Dose (mg/kg/-day)	mg Metabolite/ L liver[a]	HED[b]	Tumor Incidence
0	0	0	2/57
1.7	38.6	1.07	28/58
5.0	113.2	3.13	49/59
14.1	316.6	8.77	56/57

[a]Dose metric (lifetime average delivered dose in rats) calculated from PBPK modeling of administere animal dose.
[b]Lifetime daily human oral dose required to produce an equivalent liver concentration.

__II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The slope factor is the 95% upper confidence limit on risk for female Wistar rats. Human equivalent doses were calculated using the PBPK model of Clewell et al. (1995), based on a dose metric of the daily metabolite generated, divided by the volume of the tissue in which the metabolite i produced, that is, mg metabolites/L liver (Andersen et al., 1987). The initial VC metabolism was hypothesized to occur via two saturable pathways, one representing low-capacity, high-affinity oxidation by cytochrome P450 IIE1, and the other representing higher capacity, lower affinity oxidation by other isozymes of P450, both of which were addressed by the PBPK model in calculatio of the dose metric described above.

Modeling of risk was conducted on the basis of the animal dose metric (mg metabolites/L liver) generated by the PBPK model from input of the administered dose. PBPK analysis showed that when generated with the pathway operative at low concentrations (low-capacity, high-affinity), the dose metric was linear with concentration. At high concentrations used in rodent bioassays, the second pathway becomes more involved, causing the metric-concentration relationship to become nonlinear. The PBPK model addressed both pathways. Then, consistent with the statement that "tissues experiencing equal average concentrations of the carcinogenic moiety over a full lifetime should be presumed to have equal lifetime cancer risk" (U.S. EPA, 1992), the calculated risk values based on th animal dose metric were assumed to correspond to the same risk for the same human dose metric. It was further assumed that the linear relationship between the dose metric and concentrations of interest (i.e., low) demonstrated by PBPK modeling was valid (see Appendix B of the Toxicological Review).

In order to convert the human dose metric to a human dose, the model was run for a sample human continuous oral exposure (1 mg/L in drinking water) to determine the dose of metabolites to th human liver corresponding to a given ingested dose. Because VC metabolism is linear in the human dose range of interest, this equivalence factor could be used to convert the risk based on the dose metric (now in humans) into the human oral dose. Calculation of a slope factor is appropriate in this case, in spite of the use of a pharmacokinetic model, because VC metabolism is linear in the human exposure range. Risk was based on results from the species and sex with the greatest response if differences between sexes were significant. In this case female rats are the most sensitive. This is in accordance with EPA guidelines (U.S. EPA, 1986).

Because statistically significant increases in liver angiosarcomas, neoplastic nodules, and hepatocellular carcinomas were reported in the oral studies of Feron et al. (1981), risk was calculated based on animals exhibiting any of these endpoints. This is in agreement with EPA policy to combine data from animals with any tumor that is statistically significantly increased. Although nodules may not progress to malignancy in every case, a conservative approach avoids possible underestimation of total cancer risk. Lung angiosarcoma incidences were significantly increased, but these animals also had liver tumors, so they were included in the counts.

HECs were derived using a PBPK model, an approach considered to be more accurate than a scaling factor, because it accounts for many more interspecies variables. Although the model does not account for possible pharmacodynamic differences, no adjustment was made for this variable because available evidence suggests that humans are considered unlikely to be more susceptible to cancer induction by VC than laboratory species. A number of published cancer risk estimates based on epidemiologic data provided no evidence for greater carcinogenic sensitivity to VC in humans than in rats or mice, and some evidence for less sensitivity (see Section 5.3.3 of the Toxicological Review of Vinyl Chloride).

Confidence is high that the steady-state concentration of the active metabolite in the liver is accurately modeled, although the possibility of cancer induction at sites other than the liver is of some concern. Increases in nonliver tumors have been detected in some of the animal studies. They were generally sporadic in nature, however, with little evidence of a positive dose response. Although increases in nonliver tumors were noted in some of the epidemiology studies, the increase in relative risk was generally less than for liver tumors, and the evidence is considered to be fairly weak. Becaus VC is activated in the liver and because both human and animal data indicate that the liver is the most sensitive site for cancer induction, it is concluded that adequate protection against liver cancer will be protective against cancers at other sites. For a discussion of possible cancer induction at nonliver sites, see Section 5.3.5 of the Toxicological Review of Vinyl Chloride.

Animal evidence indications of age-dependent sensitivity warrant concern for young children potentially exposed to VC. This is based on several observations in animals regarding early-life studies. Exposure periods in the early-life studies do not overlap those of the chronic studies from which chronic slope factors and unit risk are derived. The angiosarcoma incidence after short-term, early-life exposure is approximately equal to that of long-term exposure starting after maturity. Based on these observations, continuous lifetime exposure from birth would about double cancer risk. Although there is some uncertainty regarding differences in sensitivity during early exposure, and although a portion of the increased responsiveness may be due to greater air or liquid intake per unit body weight, nevertheless the recommendation put forth is considered prudent. For further discussion on the basis for recommending adjustment to account for early-life sensitivity, as well as methodolog to adjust for partial lifetime exposure, see Section 5.3.5.1 of the Toxicological Review of Vinyl Chloride.

In general, the potential for added risk from early-life exposure to VC is accounted for in the quantitative cancer risk estimates by a twofold uncertainty factor. If exposure occurs only during adult life, the twofold factor need not be applied.

Although increases in mammary tumors were reported in several inhalation bioassays, the increases were sporadic, with little evidence of a positive dose response. Because of the uncertainty in the animal data, the lack of reported breast cancer in occupationally exposed males or in one small cohort of females, and the knowledge that VC is primarily activated in the liver, it is concluded that the liver is the most sensitive organ and no adjustment is necessary for possible breast cancer induction. For further discussion see section 5.3.5 of the Toxicological Review of Vinyl Chloride.

In summary, extrapolation of dose was based on equivalent concentration of the active metabolite per unit of liver volume. Because individual animal data, including time-to-tumor data, were available, a time-to-tumor model was used. In accordance with the 1986 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1987), a linearized low-dose extrapolation was conducted fo this genotoxic carcinogen. In accordance with the proposed cancer guidelines (U.S. EPA, 1996) a linear approach was also utilized by drawing a straight line between the LED_{10} and the origin (zero dose). The results are nearly identical to those derived using the linearized multistage model. The values derived are recommended for lifetime exposure beginning at adulthood. For exposures beginning at birth an additional twofold safety factor is recommended.

The unit risk should not be used if the water concentration exceeds 10^5 µg/L, because above thi concentration the slope factor may differ from that stated.

__II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The study was well conducted, used an adequate number of rats, and is supported by results of a followup study by Til et al. (1991) as well as those reported by Maltoni et al. (1981, 1984). Although inclusion of neoplastic nodules may represent a conservative approach, it should be noted that low body weights in the Feron et al. (1981) study, due to restriction of food intake to 4 hours per day, are likely to decrease tumorigenesis.

Use of a pharmacokinetic model reduces the uncertainty in extrapolating from animals to humans. A sensitivity analysis conducted on the parameters for the model found no amplification of error from inputs to outputs (Clewell et al., 1995). This is the desired result in a model used for risk assessment. A Monte Carlo uncertainty/variability analysis (2 realizations, 500 simulations/realization was conducted to evaluate the impact of parameter uncertainty and variability on the risk prediction. The 95th percentile of the distribution of UCL risks was within 50% of the mean UCL risk. It should be noted that the slope factor was not based on the Monte Carlo analysis.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

____II.C.1.1. Inhalation Unit Risk:	(a) LMS method (Risk per µg/m^3)	(b) LED 10/linear method (Risk per µg/m^3)
Continuous lifetime exposure during adulthood	4.4E-6	4.4E-6
Continuous lifetime exposure from birth	8.8E-6	8.8E-6

The unit risk estimate of 4.4 E-6/ (µg/m)^3 to account for continuous, lifetime exposure during adulthood, based on use of the linearized multistage model is recommended. A twofold increase to 8. E-6/ (µg/m)^3 , to account for continuous lifetime exposure from birth, is also recommended (see Toxicological Review Section 5.3.5.1). According to the EPA Cancer Risk Assessment Guidelines of 1986 (U.S. EPA, 1986) "in the absence of adequate evidence to the contrary, a linearized multistage procedure will be employed." The 1996 proposed guidelines (U.S. EPA, 1996) recommend employment of the LED 10/linear method in similar situations. This approach is to draw a straight lin between the point of departure from the observed data, generally as a default the LED_{10}. The LED_{10} is the lower 95% limit on a dose that is estimated to cause a 10% response. As can be seen, the derived values using either approach are virtually identical.

____II.C.1.2. Extrapolation Method: (a) Linearized multistage (b) LED 10/linear method

Air Concentrations at Specified Risk Levels for continuous lifetime exposure during adulthood (based on female rats using the linearized multistage method):

Risk Level	Concentration
E-4 (1 in 10,000)	23 µg/m^3
E-5 (1 in 100,000)	2.3 µg/m^3
E-6 (1 in 1,000,000)	2.3E-1 µg/m^3

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Tumor type: Liver angiosarcomas, angiomas, hepatomas, and neoplastic nodules
Test animals: Female Sprague-Dawley rats
Route: Inhalation
Reference: Maltoni et al. (1981, 1984)

Admin. conc.(ppm)[a]	Metabolite mg/L liver[b]	HEC (ppm)[c]	Tumors[d]
0	0	0	0/141
1	0.59	0.20	0/55
5	2.96	1.0	0/47
10	5.90	2.0	1/46
25	14.61	4.6	5/40
50	31.27	10.1	1/29
100	55.95	19	1/43
150	76.67	26	5/46
200	90.00	31	10/44
250	103.45	35	3/26
500	116.94	40	11/28
2500	134.37	48	10/24
6000	143.72	51	13/25

[a] Animals exposed 4 hours/day, 5 days/week for 52 weeks.
[b]Dose metric (lifetime average delivered dose in female rats) calculated from PBPK modeling of the administered animal concentration.
[c]Continuous human exposure concentration over a lifetime required to produce an equivalent mg metabolite/L of liver.
[d]Based on number of animals alive after detection of first liver tumor.

HEDs were calculated with the aid of the PBPK model of Clewell et al. (1995), using a dose metric of the daily metabolite generated, divided by the volume of the tissue in which the metabolite i produced (Andersen et al., 1987). The initial VC metabolism was hypothesized to occur via two saturable pathways, one representing low-capacity, high-affinity oxidation by cytochrome P450 IIE1, and the other representing higher capacity, lower affinity oxidation by other isozymes of P450. In order to convert the human dose metric to a human dose, the model was run for a sample human continuous inhalation exposure (1 mg/m^3) to determine the dose of metabolites to the human liver corresponding to a given inhalation dose. As described for the oral slope factor, the risk modeling was conducted based on the animal dose metric, and the resulting risk was converted to a human risk value based on an equivalence factor. The equivalence factor for inhalation exposure was calculated by determining the human dose metric for continuous human inhalation exposure to a range of exposure concentrations (1 $\mu g/m^3$ to 10,000 mg/m^3). This calculation showed that the model was linear up to nearly 100 mg/m^3, and the calculated equivalence factor was used to convert the risk from the inhalation experiments conducted in animals (in the units of the dose metric) to human risk values. The slope factor is based on the 95% upper confidence on risk in female rats. Calculation of a slope factor is appropriate in this case, in spite of the use of a pharmacokinetic model, because VC metabolism is linear in humans in this exposure range.

HECs were derived using a PBPK model, an approach considered to be more accurate than a scaling factor, because it accounts for many more interspecies variables. While the model does not account for possible pharmacodynamic differences, no adjustment was made for this variable because available evidence suggests that humans are considered unlikely to be more susceptible to cancer induction by VC than laboratory species (see Section 5.3.3 of the Toxicological Review of Vinyl Chloride as well as the following section for additional details).

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

Although human studies are preferable for deriving human cancer risk estimates, exposure data from most of the epidemiology studies are inadequate to derive risk estimates. For those that do provide exposure information, cumulative exposure (e.g., ppm-years) can be calculated. Because VC metabolism becomes nonlinear at high exposure concentrations, however, cumulative exposure is not sufficient for quantitating risk.

Hepatomas, angiomas, and neoplastic nodules were not statistically significantly increased in th Maltoni et al. (1981, 1984) studies. However, because hepatocellular tumors were significantly increased in the Feron et al. study, it was concluded that all liver tumors in the Maltoni et al. studies are likely the result of exposure to VC as well, and should be included as a conservative approach. Th additional numbers of tumors were quite small and only minimally influenced the quantitative estimates.

A twofold adjustment is recommended to account for greater responsiveness to VC exposure during early life. Although there is some uncertainty regarding differences in sensitivity during early exposure, and although a portion of the increased responsiveness may be due to increased minute-volume ventilation, nevertheless the recommendation put forth is considered prudent. For a more detailed discussion regarding application of this adjustment, see Section 5.3.5.2 of the Toxicological Review.

Although increases in mammary tumors were reported in several inhalation bioassays, the increases were sporadic, with little evidence of a positive dose response. Because of the uncertainty in the animal data, the lack of reported breast cancer in occupationally exposed males or in one small cohort of females, and the knowledge that VC is primarily activated in the liver, it is concluded that the liver is the most sensitive organ and no adjustment is necessary for possible breast cancer induction. For further discussion see section 5.3.5 of the Toxicological Review of Vinyl Chloride.

The unit risk estimate, even with the addition of adjustments for early exposure, is about 10-fol lower than the previous EPA "HEAST" value of 8.4E-5 (U.S. EPA, 1994). There are several reasons for this. First, in the earlier estimate, absorption was assumed to equal 50% in the rat versus 100% in humans. Such an assumption is invalid in that virtually all VC is absorbed in both species until a bloo concentration determined by the inspired concentration and the blood-to-air partition coefficient is reached. Because the partition coefficient is about twice as large in rats as in humans, arterial blood concentration will be greater in rats than humans, rather than less. Metabolic activation of VC (Vmax/Km) is about 10 times faster in rats than humans. Blood flow to the liver is more rapid. After accounting for these and other pharmacokinetic differences, the model predicts that rats will have a considerably greater steady-state concentration of the active metabolite of VC than humans and thereby greater risk. Use of administered dose and standard defaults, on the other hand, would result i a prediction of lower risk in rats than in humans.

The unit risks can be compared with those derived from human epidemiology data. Risk estimates have been derived from four epidemiology studies (Fox and Collier, 1977; Jones et al., 1988 Simonato et al., 1991; Wong et al., 1991). Uncertainties associated with use of these studies are described in greater detail in the Toxicological Review. The primary weakness of the Fox and Collier (1977) study is the relatively small cohort associated with only two liver cancer cases. The Jones et al.

(1978) study is an update of the Fox and Collier study. Workers were categorized according to cumulative exposure, which was considered to vary with duration, but not concentration. The Simonato et al. (1991) study was the largest, but the data were collected from many different workplaces in different countries, resulting in considerable uncertainty regarding exposures.

Chen and Blancato, using one pathway model, derived a unit risk estimate of 1.5E-6 per $\mu g/m^3$ based on the Fox and Collier study. Clewell et al. (1995) developed risk estimates, using the two-pathway model, based on epidemiology studies reported by Fox and Collier (1977), Jones et al. (1988), and Simonato et al. (1991) ranging from 1.6 E-7 to 1.5E-6 per $\mu g/m^3$. Reitz et al. (1996) also assessed risk based on the Simonato et al. (1991) study. Although they did not develop a formal unit risk estimate using this study, they did report that a unit risk estimate of 5.7E-7 per $\mu g/m^3$ derived using the Maltoni et al. (1981, 1984) animal inhalation studies overpredicted tumor counts from the Simonato et al. (1991) study by 10- to 35-fold.

The epidemiology-based estimates thus vary over about an order of magnitude, with the upper end of this range still somewhat lower than the animal inhalation–based estimates. Although each of these estimates contains a considerable degree of uncertainty, collectively they indicate that the anima data–based unit risk estimates are unlikely to underestimate true risk, despite being considerably lowe than an earlier EPA estimate (ATSDR, 1997).

As discussed for the oral slope factor, a linear extrapolation from the 95% lower bound on the ED10 (LED10) was also considered for this genotoxic carcinogen. The maximum likelihood estimate (MLEs) and risk based on the MLEs were also derived. The LED10s were slightly less conservative than those risk estimates derived using the linearized multistage approach.

The unit risk should not be used if the air concentration exceeds 10^4 $\mu g/m^3$, because above this concentration the slope factor may differ from that stated.

__II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

Maltoni et al. (1981, 1984) conducted a series of experiments in which rats were exposed to varying concentrations of VC, resulting in a broad, well-characterized concentration-response curve based on experiments conducted with an adequate number of animals.

Use of a pharmacokinetic model reduces the uncertainty in extrapolating from animals to humans. A sensitivity analysis conducted on the parameters for the model found no amplification of error from inputs to outputs (Clewell et al., 1995). This is the desired result in a model used for risk assessment. A Monte Carlo uncertainty/variability analysis (4 realizations, 500 simulations/realization was conducted to evaluate the impact of parameter uncertainty and variability on the risk prediction. The 95th percentile of the distribution of UCL risks was within approximately a factor of 2 of the mean UCL risk.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Documents --

U.S. Environmental Protection Agency (U.S. EPA). (2000) Toxicological review of vinyl chloride in support of summary information on the Integrated Risk Information System (IRIS). Available online

from National Center for Environmental Assessment, http://www.epa.gov/iris.

U.S. Environmental Protection Agency. (1985) Health and environmental effects profile for chloroethene. EPA/600/85/374.

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This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Vinyl Chloride in Support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 2000).

___II.D.2. EPA REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Consensus Date -- 07/20/2000

___II.D.3. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRI in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address)

___III. [reserved]
___IV. [reserved]
___V. [reserved]

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Vinyl Chloride
CASRN -- 75-01-4
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_VII. REVISION HISTORY

Vinyl Chloride
CASRN -- 75-01-4

Date	Section	Description
08/07/2000	I-VIII	RfD, RfC, Cancer assessment first on-line
09/26/2000	II.B.1.3.	Corrected drinking water concentration at E-4, E-5, and E-6 risk level associated with exposure from birth.
09/26/2000	I.A.2, I.B.2., VI.A, VI.B., VI.C.	Correction to Til...(1983), HP et al reference
09/26/2000	Tox Review	RFD given in Section 6.2.2 should be 3E-3. Exponent of E-6 should be part of the 4.4 per ug/cu.m figure in Section 5.3.5.

_VIII. SYNONYMS

Vinyl Chloride
CASRN -- 75-01-4
Last Revised -- 08/07/2000

vinyl chloride
vinyl chloride monomer
chloroethylene
chloroethene
VC
VCM

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Last updated: 7 November 2000
URL: http://www.epa.gov/iris/subst/1001.htm

EPA United States Environmental Protection Agency

IRIS
Integrated Risk Information System

cis-1,2-Dichloroethylene

CASRN 156-59-2

Contents

0418
cis-1,2-Dichloroethylene; CASRN 156-59-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR cis-1,2-Dichloroethylene

File On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1995

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2
Last Revised -- 02/01/1995

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no data in humans or animals and generally nonpositive results in mutagenicity assays.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

None.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

cis-1,2-Dichloroethylene did not yield positive results for a Salmonella typhimurium spot test assay in the absence of mammalian liver homogenates; however, this compound did cause a dose-dependent increase in mutations in a host-mediated assay (Cerna and Kypenova, 1977). cis-1,2-Dichloroethylene at a medium concentration of 2.9 mM produced no positive results in a mutagenicity assay for Escherichia coli K12 (Greim et al., 1975). Galli et al. (1982a) reported no positive results for cis-1,2-dichloroethylene in point mutation, mitotic gene conversion and mitotic recombination assays (all for Saccharomyces cerevisiae). In addition, it did not yield positive results in an in vivo (intravenous) host-mediated mutagenicity assay (Galli et al., 1982b). cis-1,2-Dichloroethylene did not induce chromosomal aberrations in mouse bone marrow in vivo (Cerna and Kypenova, 1977).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984

The 1984 Health Effects Assessment document has received Agency review and has been approved for publication as an EPA document.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/07/1989

Verification Date -- 09/07/1989

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

None

_VI.B. INHALATION RfC REFERENCES

None

_VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Cerna, M. and H. Kypenova. 1977. Mutagenic activity of chloroethylenes analyzed by screening system tests. Mutat. Res. 46(3): 214-215.

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U.S. EPA. 1984. Health Effects Assessment for cis-1,2-Dichloroethylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

_VII. REVISION HISTORY

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
02/01/1995	II.D.3.	Primary contact changed
04/01/1997	III.,IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, Supplementary Data were removed from IRIS on or before Ap 1997. IRIS users were directed to the appropriate EPA Pro Offices for this information.

VIII. SYNONYMS

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2
Last Revised -- 12/01/1990

156-59-2
Ethene, 1,2-dichloro-, (Z)-
(Z)-1,2-Dichloroethene
(Z)-1,2-DICHLOROETHYLENE
cis-DICHLOROETHYLENE
CIS-1,2-DICHLORETHYLENE
CIS-1,2-DICHLOROETHENE
cis-1,2-DICHLOROETHYLENE
Ethene, 1,2-dichloro-, (Z)-
Ethylene, 1,2-dichloro-, (Z)-
HSDB 5656
NSC 6149
1,2-CIS-DICHLOROETHYLENE
.

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0418.htm

APPENDIX C

ANALYTICAL RESULTS

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

800 Sleater-Kinney SE, PMB #262
Lacey, Washington 98503-1127

Mobile Environmental Laboratories
Environmental Sampling Services

Telephone: 360-459-4670
Fax: 360-459-3432

February 19, 2001

Travis Trent
Fulcrum Environmental
105 S 3rd Street
Yakima, WA 98901

Dear Mr. Trent:

Please find enclosed the analytical data report for the Frank Wear Cleaners Project in Yakima, Washington. Mobile Lab services were conducted on February 13 & 14, 2001. Soil samples were analyzed for Specific Halogenated Hydrocarbons and BTEX by Method 8021B.

The results of these analyses are summarized in the attached table. All soil values are reported on a dry weight basis. Applicable detection limits and QA/QC data are included. An invoice for this analytical work is also enclosed.

TEG Northwest appreciates the opportunity to have provided analytical services to Fulcrum Environmental for this project. If you have any further questions about the data report, please give me a call. It was a pleasure working with you on this project, and we are looking forward to the next opportunity to work together.

Sincerely,

Michael A. Korosec
President

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		Method Blank	FW0213-01	FW0213-01 Dup.	FW0213-02	FW0213-03	FW0213-04
Date Sampled			2/13/01	2/13/01	2/13/01	2/13/01	2/13/01
Date Analyzed		2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01
	MDL (mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd	nd	nd	nd	nd	nd
Benzene	0.05	nd	nd	nd	nd	nd	nd
Toluene	0.05	nd	nd	nd	nd	nd	nd
Ethylbenzene	0.05	nd	nd	nd	nd	nd	nd
Total Xylenes	0.05	nd	nd	nd	nd	nd	nd
1,1-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd
Methylene chloride	0.05	nd	nd	nd	nd	nd	nd
trans-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd
1,1-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd
cis-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd
Chloroform	0.05	nd	nd	nd	nd	nd	nd
1,1,1-Trichloroethane (TCA)	0.05	nd	nd	nd	nd	nd	nd
Carbon tetrachloride	0.05	nd	nd	nd	nd	nd	nd
1,2-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd
Trichloroethene (TCE)	0.05	nd	nd	nd	nd	nd	nd
1,1,2-Trichloroethane	0.05	nd	nd	nd	nd	nd	nd
Tetrachloroethene (PCE)	0.05	nd	nd	0.06	nd	0.23	nd
1,1,1,2-Tetrachloroethane	0.05	nd	nd	nd	nd	nd	nd
Surrogate Recovery (%)		95	94	97	109	97	119

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		FW0213-05	FW0213-06	FW0213-07	FW0213-08	FW0213-09	FW0213-09 Dup.
Date Sampled		2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01
Date Analyzed		2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01
	MDL (mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd	nd	nd	nd	nd	nd
Benzene	0.05	nd	nd	nd	nd	nd	nd
Toluene	0.05	nd	nd	nd	nd	nd	nd
Ethylbenzene	0.05	nd	nd	nd	nd	nd	nd
Total Xylenes	0.05	nd	nd	nd	nd	nd	nd
1,1-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd
Methylene chloride	0.05	nd	nd	nd	nd	nd	nd
trans-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd
1,1-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd
cis-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd
Chloroform	0.05	nd	nd	nd	nd	nd	nd
1,1,1-Trichloroethane (TCA)	0.05	nd	nd	nd	nd	nd	nd
Carbon tetrachloride	0.05	nd	nd	nd	nd	nd	nd
1,2-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd
Trichloroethene (TCE)	0.05	nd	nd	nd	nd	nd	nd
1,1,2-Trichloroethane	0.05	nd	nd	nd	nd	nd	nd
Tetrachloroethene (PCE)	0.05	0.19	nd	0.11	0.13	nd	nd
1,1,1,2-Tetrachloroethane	0.05	nd	nd	nd	nd	nd	nd
Surrogate Recovery (%)		108	112	112	90	68	111

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		FW0213-10	FW0213-11	FW0213-12	FW0213-13	FW0213-14	FW0213-15	FW0213-16
Date Sampled		2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01
Date Analyzed		2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01	2/13/01
	MDL (mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd	nd	nd	nd	nd	nd	nd
Benzene	0.05	nd	nd	nd	nd	nd	nd	nd
Toluene	0.05	nd	nd	nd	nd	nd	nd	nd
Ethylbenzene	0.05	nd	nd	nd	nd	nd	nd	nd
Total Xylenes	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Methylene chloride	0.05	nd	nd	nd	nd	nd	nd	nd
trans-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
cis-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Chloroform	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,1-Trichloroethane (TCA)	0.05	nd	nd	nd	nd	nd	nd	nd
Carbon tetrachloride	0.05	nd	nd	nd	nd	nd	nd	nd
1,2-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Trichloroethene (TCE)	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,2-Trichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Tetrachloroethene (PCE)	0.05	nd	nd	nd	0.15	nd	0.10	nd
1,1,1,2-Tetrachloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Surrogate Recovery (%)		105	106	105	100	107	102	105

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

QA/QC Data - EPA 8021B Analyses

	Sample Description: FW0213-09						
	Matrix Spike			Matrix Spike Duplicate			RPD
	Spiked Conc. (mg/kg)	Measured Conc. (mg/kg)	Spike Recovery (%)	Spiked Conc. (mg/kg)	Measured Conc. (mg/kg)	Spike Recovery (%)	(%)
Benzene	1.00	1.10	110	1.00	1.00	100	9.52
Toluene	1.00	0.97	97	1.00	1.00	100	3.05
1,1-Dichloroethene	1.00	1.13	113	1.00	1.06	106	6.39
Trichloroethene (TCE)	1.00	0.97	97	1.00	0.88	88	9.73
Surrogate Spike			95			109	

	Laboratory Control Sample		
	Spiked Conc. (mg/kg)	Measured Conc. (mg/kg)	Spike Recovery (%)
Benzene	1.00	0.97	97
Toluene	1.00	0.99	99
1,1-Dichloroethene	1.00	1.03	103
Trichloroethene (TCE)	1.00	0.87	87
Surrogate Spike			117

ACCEPTABLE RECOVERY LIMITS FOR MATRIX SPIKES: 80%-120%
ACCEPTABLE RPD IS 20%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

teg

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES

CHAIN-OF-CUSTODY RECORD

CLIENT: Fulcrum Environmental Consulting

ADDRESS: 122 South 3rd Street

PHONE: (509) 574-0839 FAX:

CLIENT PROJECT #: PROJECT MANAGER: Travis Trent

DATE: 2/13/01 PAGE 1 OF 1

PROJECT NAME:

LOCATION: Front Wear Cleaners Yakima

COLLECTOR: T. Trent DATE OF COLLECTION 2/13/01

Sample Number	Depth	Time	Sample Type	Container Type	ANALYSES: VOA 8010/8021B	VOA 8021B BTEX	VOA 8260	SEMI VOL 8270	TPH - HCID	TPH 8015 (gasoline)	TPH 8015 (diesel)	TPH 8015 (d & o)	PAH 8100	PCBs 8082	Pesticides 8081	TOTAL LEAD	pH	NOTES	Total Number of Containers	Laboratory Note Number
1. FW0213-01	2ft	8:10	soil	Glass																
2. FW0213-02	1.5ft	8:11	"	"																
3. FW0213-03	1ft	8:12	"	"																
4. FW0213-04	2ft	9:40																		
5. FW0213-05	2ft	10:10																		
6. FW0213-06	1.5ft	11:12																		
7. FW0213-07	1ft	10:15																		
8. FW0213-08	3ft	10:30																		
9. FW0213-09	5ft	11:30																		
10. FW0213-10	3ft	12:00																		
11. FW0213-11	3ft	12:30																		
12. FW0213-12	5ft	2:05																		
13. FW0213-13	7ft	2:45																		
14. FW0213-14	~~12ft~~ 10ft	3:00																		
15. FW0213-15	~~3ft~~ 8ft	~~3:00~~ 3:10																		
16. FW0213-16	9ft	3:10																		
17.																				
18.																				

RELINQUISHED BY (Signature)	DATE/TIME	RECEIVED BY (Signature)	DATE/TIME
[signature]	2/13/01	[signature]	2/13/01
RELINQUISHED BY (Signature)	DATE/TIME	RECEIVED BY (Signature)	DATE/TIME

SAMPLE DISPOSAL INSTRUCTIONS

☐ TEG DISPOSAL @ $2.00 each ☐ Return ☐ Pickup

SAMPLE RECEIPT	
TOTAL NUMBER OF CONTAINERS	
CHAIN OF CUSTODY SEALS Y/N/NA	
SEALS INTACT? Y/N/NA	
RECEIVED GOOD COND./COLD	
NOTES:	

LABORATORY NOTES:

Turn Around Time:

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		Method Blank	FW0214-01	FW0214-02	FW0214-03	FW0214-04	FW0214-05	FW0214-06
Date Sampled			2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01
Date Analyzed		2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01
	MDL (mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd	nd	nd	nd	nd	nd	nd
Benzene	0.05	nd	nd	nd	nd	nd	nd	nd
Toluene	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Methylene chloride	0.05	nd	nd	nd	nd	nd	nd	nd
trans-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
cis-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Chloroform	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,1-Trichloroethane (TCA)	0.05	nd	nd	nd	nd	nd	nd	nd
Carbon tetrachloride	0.05	nd	nd	nd	nd	nd	nd	nd
1,2-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Trichloroethene (TCE)	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,2-Trichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Tetrachloroethene (PCE)	0.05	nd	nd	nd	nd	nd	nd	nd
Surrogate Recovery (%)		113	84	98	88	128	93	90

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		FW0214-07	FW0214-07 Dup.	FW0214-08	FW0214-09	FW0214-10	FW0214-11	FW0214-11 Dup.
Date Sampled		2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01
Date Analyzed		2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01
	MDL (mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd	nd	nd	nd	nd	nd	nd
Benzene	0.05	nd	nd	nd	nd	nd	nd	nd
Toluene	0.05	nd	nd	nd	nd	nd	nd	nd
Ethylbenzene	0.05	nd	nd	nd	nd	nd	nd	nd
Total Xylenes	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Methylene chloride	0.05	nd	nd	nd	nd	nd	nd	nd
trans-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
cis-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Chloroform	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,1-Trichloroethane (TCA)	0.05	nd	nd	nd	nd	nd	nd	nd
Carbon tetrachloride	0.05	nd	nd	nd	nd	nd	nd	nd
1,2-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Trichloroethene (TCE)	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,2-Trichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Tetrachloroethene (PCE)	0.05	nd	nd	0.29	1.5	nd	nd	nd
Surrogate Recovery (%)		104	93	96	97	99	99	86

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		FW0214-12	FW0214-13	FW0214-14	FW0214-15	FW0214-16	FW0214-17	FW0214-18
Date Sampled		2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01
Date Analyzed		2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01	2/14/01
	MDL (mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd	nd	nd	nd	nd	nd	nd
Benzene	0.05	nd	nd	nd	nd	nd	nd	nd
Toluene	0.05	nd	nd	nd	nd	nd	nd	nd
Ethylbenzene	0.05	nd	nd	nd	nd	nd	nd	nd
Total Xylenes	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Methylene chloride	0.05	nd	nd	nd	nd	nd	nd	nd
trans-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
1,1-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
cis-1,2-Dichloroethene	0.05	nd	nd	nd	nd	nd	nd	nd
Chloroform	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,1-Trichloroethane (TCA)	0.05	nd	nd	nd	nd	nd	nd	nd
Carbon tetrachloride	0.05	nd	nd	nd	nd	nd	nd	nd
1,2-Dichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Trichloroethene (TCE)	0.05	nd	nd	nd	nd	nd	nd	nd
1,1,2-Trichloroethane	0.05	nd	nd	nd	nd	nd	nd	nd
Tetrachloroethene (PCE)	0.05	nd	0.21	nd	nd	nd	nd	nd
Surrogate Recovery (%)		90	101	70	90	109	85	100

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

QA/QC Data - EPA 8021B Analyses

Sample Description: FW0213-16							
	Matrix Spike			Matrix Spike Duplicate			RPD
	Spiked Conc. (mg/kg)	Measured Conc. (mg/kg)	Spike Recovery (%)	Spiked Conc. (mg/kg)	Measured Conc. (mg/kg)	Spike Recovery (%)	(%)
Benzene	1.00	0.99	99	1.00	1.04	104	4.93
Toluene	1.00	0.98	98	1.00	1.04	104	5.94
1,1-Dichloroethene	1.00	1.07	107	1.00	1.03	103	3.81
Trichloroethene (TCE)	1.00	0.87	87	1.00	0.96	96	9.84
Surrogate Spike			93			112	

	Laboratory Control Sample		
	Spiked Conc. (mg/kg)	Measured Conc. (mg/kg)	Spike Recovery (%)
Benzene	1.00	0.98	98
Toluene	1.00	1.00	100
1,1-Dichloroethene	1.00	1.04	104
Trichloroethene (TCE)	1.00	0.87	87
Surrogate Spike			113

ACCEPTABLE RECOVERY LIMITS FOR MATRIX SPIKES: 80%-120%
ACCEPTABLE RPD IS 20%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES NORTHWEST, INC.

FRANK WEAR CLEANERS PROJECT
Yakima, Washington
Fulcrum Environmenal Consulting, Inc.

Specific Halogenated and Aromatic Hydrocarbons (EPA 8021B) in Soil

Sample Description		FW0214-19
Date Sampled		2/14/01
Date Analyzed		2/14/01
	MDL (mg/kg)	(mg/kg)
Vinyl chloride	0.25	nd
Benzene	0.05	nd
Toluene	0.05	nd
Ethylbenzene	0.05	nd
Total Xylenes	0.05	nd
1,1-Dichloroethene	0.05	nd
Methylene chloride	0.05	nd
trans-1,2-Dichloroethene	0.05	nd
1,1-Dichloroethane	0.05	nd
cis-1,2-Dichloroethene	0.05	nd
Chloroform	0.05	nd
1,1,1-Trichloroethane (TCA)	0.05	nd
Carbon tetrachloride	0.05	nd
1,2-Dichloroethane	0.05	nd
Trichloroethene (TCE)	0.05	nd
1,1,2-Trichloroethane	0.05	nd
Tetrachloroethene (PCE)	0.05	nd
Surrogate Recovery (%)		101

"nd" Indicates not detected at listed detection limit.
"int" Indicates that interference prevents determination.

ACCEPTABLE RECOVERY LIMITS FOR SURROGATE (Chlorobenzene): 65%- 135%

ANALYSES PERFORMED BY: Tim McCall
DATA REVIEWED BY: Sherry Chilcutt

teg TRANSGLOBAL ENVIRONMENTAL GEOSCIENCES

CHAIN-OF-CUSTODY RECORD

CLIENT: Fulcrum Environmental

ADDRESS: 122 South 3rd Street

PHONE: (509) 574-0839 FAX:

CLIENT PROJECT #: PROJECT MANAGER: Travis Trent

DATE: 2/14/01 PAGE 1 OF 1

PROJECT NAME: Frank Wear Cleaners

LOCATION: Yakima, WA

COLLECTOR: Travis Trent DATE OF COLLECTION 2/14/01

Sample Number	Depth	Time	Sample Type	Container Type	ANALYSES: VOA 8010/8021B	VOA 8021B BTEX	VOA 8260	SEMI VOL 8270	TPH - HCID	TPH 8015 (gasoline)	TPH 8015 (diesel)	TPH 8015 (d & o)	PAH 8100	PCBs 8082	Pesticides 8081	TOTAL LEAD	pH	NOTES	Total Number of Containers	Laboratory Note Number
1. FW0214-01	1ft	10:00	Soil	glass																
2. 02	7ft	10:00																		
3. 03	7ft	10:00																		
4. 04	35	10:30																		
5. 05	2	10:30																		
6. 06	2	10:30																		
7. 07	35	11:00																		
8. 08	.5	12:30																		
9. 09	.5	12:30																		
10. 10	3ft	1:00																		
11. 11	2.5ft	1:00																		
12. 12	3ft	2:00																		
13. 13	3ft	2:00																		
14. 14	3ft	2:00																		
15. 15	1.5ft	3:30																		
16. Concrete 16																				
17. 17	5ft	3:45																		
18. 18	3ft	3:45																		

RELINQUISHED BY (Signature)	DATE/TIME	RECEIVED BY (Signature)	DATE/TIME
Travis Trent	2/14/01	Jim McCall	2/14/01 0900
RELINQUISHED BY (Signature)	DATE/TIME	RECEIVED BY (Signature)	DATE/TIME

SAMPLE DISPOSAL INSTRUCTIONS

☐ TEG DISPOSAL @ $2.00 each ☐ Return ☐ Pickup

SAMPLE RECEIPT	
TOTAL NUMBER OF CONTAINERS	
CHAIN OF CUSTODY SEALS Y/N/NA	
SEALS INTACT? Y/N/NA	
RECEIVED GOOD COND./COLD	
NOTES:	

LABORATORY NOTES:
Do not include analysis after C1D2. (TM)

Turn Around Time:

APPENDIX D

DISPOSAL RECEIPTS

LOAD TICKET

ANDERSON

Rock & Demolition Pits
Petroleum Contaminated Soils Site
Topsoil - Shale - Crushed Rock

№ 20634

41 Rocky Top Road
Yakima, WA 98908

Bus. (509) 965-3621
Fax (509) 965-8656

DELIVERIES
We make deliveries inside the curb line at customer's risk only and accept no responsibility whatsoever for damages resulting from such deliveries.

Name Russell

Address

Phone Home

Office

P.O #

Job # Frank Wear

Hauled by

Received by

Date 2-20-01 Sold by

WEIGHT TICKET #	TIME	TRUCK NO	QUANTITY	PRODUCT	UNIT PRICE	AMOUNT
	8:12	301	1load	~~Demo~~ Concrete		~~90.00~~ 50.00
	8:35	302	"	" "		~~90.00~~ 50.00
	8:50	384	1Bigload	" "		~~120.00~~ 50.00
	9:20	301	1load	"		50.00
	9:41	302	1load	"		50.00
	10:07	384	1bigload	"		50.00
	10:39	301	1load	"		50.00
	11:08	302	1load	"		50.00
	1:45	384	1bigload	"		50.00
	2:50	301	1load	"		50.00
	1:30	302	1load	"		50.00
	~~2:10~~		~~1bigload~~	"		~~50.00~~
						550.00
		TOTAL				

DATE BILLED

CUSTOMER AGREES TO PAY (a) A LATE CHARGE OF 1.5% PER MONTH IF ACCOUNT IS NOT PAID WITHIN 10 DAYS OF INVOICE, AND (b) ATTORNEY'S FEES INCURRED IN COLLECTION.

Frank Wear.

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

211883

AGGREGATE TICKET

45,660 lb Manual Wt.
IN GROSS 22.83 Tons
20,660 lb Manual Wt. 03/01/2001 DATE
P.T. TARE 10.33 Tons
25,000 lb 11,340 kg 12:26 TIME
NET 12.50 Tons 11.34 Metric Tonnes

WEIGH MASTER TYLER STEWART Pit: P104

P.O. # JOB NO.:

SIGNATURE Bob

SOLD TO: EVANS CRANE RUSSELL CRANE SERVICE # of Loads: 2
Job Total: 40.54

DELIVER TO: TIM CURTIS

Load

ZONE NO.:

PRODUCT: DUMP DUMP CHARGE

TRUCKER: CUST03 RUBIN

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212522 TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457-3654 OR 877-6102

211082

AGGREGATE TICKET

50,360 lb Manual Wt.
IN GROSS 25.18 Tons
20,660 lb Manual Wt. 03/01/2001 DATE
P.T. TARE 10.33 Tons
29,700 lb 13,472 kg 11:01 TIME
NET 14.85 Tons 13.47 Metric Tonnes

WEIGH MASTER TYLER STEWART Pit: P104

P.O. # JOB NO.:

SIGNATURE X Bob

SOLD TO: ~~FOSS~~ CRANE RUSSELL CRANE SERVICE # of Loads: 4
Job Total: 94.64

DELIVER TO: ~~TIM FOSS~~ Frank Sear

ZONE NO.: Contaminated Rock/Sand

PRODUCT: ~~CONC SAND~~ CONCRETE SAND

TRUCKER: ~~CUST03~~ RUBIN

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212515

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

211080

AGGREGATE TICKET

60,420 lb Manual Wt.
TN GROSS 30.21 Tons
20,660 lb Manual Wt. 03/01/2001 DATE
P.T. TARE 10.33 Tons
39,760 lb 18,035 kg 10:16 TIME
NET 19.88 Tons 18.04 Metric Tonnes

WEIGH MASTER TYLER STEWART Pit: P104

P.O. # JOB NO.:

SIGNATURE X

SOLD TO: RUSS CRANE RUSSELL CRANE SERVICE # of Loads: 2
Job Total: 50.46

DELIVER TO: TIM CURTIS

ZONE NO.: ZONE 1 Contaminated

PRODUCT: CONC SAND CONCRETE SAND

TRUCKER: CUSTMR RUBIN

Franklin

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212512

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212525

AGGREGATE TICKET

Manual wt.

49,760 lb
OUT GROSS 24.88 Tons
23,180 lb
P.T. TARE 11.59 Tons
26,580 lb
NET 13.29 Tons

Manual wt.

12,057 kg
12.06 Metric Tonnes

03/01/2001 DATE

12:55 TIME

WEIGH MASTER TYLER STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE X Bell

SOLD TO: CHASS CRANE RUSSELL CRANE SERVICE

# of Loads: 6
Job Total: 144.52

DELIVER TO:

J.K. Hall FRANKS

ZONE NO.: ~~Zone~~ 1

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUSTOM CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212526

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212544

AGGREGATE TICKET

OUT GROSS 48,760 lb Manual Wt.
24.38 Tons

P.T. TARE 20,660 lb Manual Wt. 03/01/2001 DATE
10.33 Tons

NET 28,100 lb 12,746 kg 15:14 TIME
14.05 Tons 12.75 Metric Tonnes

WEIGH MASTER
TYLER STEWART Pit: WILDL

P.O. # JOB NO.:

SIGNATURE

SOLD TO: ROSS CRANE RUSSELL CRANE SERVICE # of Loads: 15
Job Total: 401.45

DELIVER TO: CURTIS

ZONE NO.:

PRODUCT: RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST03
RUBIN

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212544

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

AGGREGATE TICKET 212536

GROSS 71,160 lb
OUT 35.58 Tons
TARE 23,180 lb
T. T. 11.59 Tons
NET 47,980 lb
23.99 Tons

Manual Wt.

Manual Wt.

21,764 kg
21.76 Metric Tonnes

03/01/2001 DATE

14:14 TIME

WEIGH MASTER
TYLER STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE
Butch

SOLD TO: ROSS CRANE RUSSELL CRANE SERVICE

# of Loads: 10
Job Total: 248.71

DELIVER TO:

ZONE NO.:

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST04

CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212537

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212523

AGGREGATE TICKET

Manual Wt.

70,920 lb
OUT GROSS 35.46 Tons
23,180 lb
P.I. TARE 11.59 Tons
47,740 lb 21,655 kg
NET 23.87 Tons 21.65 Metric Tonnes

Manual Wt.

03/01/2001 DATE

12:36 TIME

WEIGH MASTER: TYLER STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE: Butch

SOLD TO: FISS CRANE RUSSELL CRANE SERVICE

# of Loads: 4
Job Total: 104.29

DELIVER TO:

ZONE NO.:

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST04 CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212524

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212548

AGGREGATE TICKET

OUT GROSS 67,000 lb Manual Wt.
33.50 Tons

P.T. TARE 23,180 lb Manual Wt. 03/01/2001 DATE
11.59 Tons

NET 43,820 lb 19,877 kg 15:29 TIME
21.91 Tons 19.88 Metric Tonnes

WEIGH MASTER
TYLER STEWART Pit: WILDL

P.O. # JOB NO.:

SIGNATURE
Butch

SOLD TO: ROSS CRANE RUSSELL CRANE SERVICE # of Loads: 18
Job Total: 461.32

DELIVER TO:

ZONE NO.:

PRODUCT: RUN UNCLASSIFIED PIT RUN

TRUCKER: 1704 CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212548

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212514

AGGREGATE TICKET

79,260 lb Manual Wt.
OUT GROSS 39.63 Tons
23,180 lb Manual Wt. 03/01/2001 DATE
P.T. TARE 11.59 Tons
56,080 lb 25,438 kg 12:10 TIME
NET 28.04 Tons 25.44 Metric Tonnes

WEIGH MASTER STEWART Pit: P104

P.O. # JOB NO.:

SIGNATURE

SOLD TO: BUSS CRANE RUSSELL CRANE SERVICE # of Loads: 2
Job Total: 63.76

DELIVER TO:

ZONE NO.:

PRODUCT: PUMP PUMP CHARGE
CUST04
TRUCKER: CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212518 TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212528

AGGREGATE TICKET

62,500 lb
OUT GROSS 31.29 Tons
Manual Wt.

23,100 lb
P.T. TARE 11.59 Tons
Manual Wt.

03/01/2001 DATE

39,400 lb 17,872 kg
NET 19.70 Tons 17.87 Metric Tonnes

13:32 TIME

WEIGH MASTER: TYLER STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE

Butch

SOLD TO: RUSS CRANE RUSSELL CRANE SERVICE

# of Loads: 5
Job Total: 108.35

DELIVER TO:

ZONE NO.:

PRODUCT: DUMP DUMP CHARGE

TRUCKER: CUST04 CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212529

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212515

AGGREGATE TICKET

120,000 lb
OUT GROSS 60.00 Tons
37,160 lb
P.T. TARE 18.58 Tons
82,840 lb
NET 41.42 Tons

Manual Wt.

Manual Wt.

37,576 kg
37.58 Metric Tonnes

03/01/2001 DATE

12:15 TIME

WEIGHMASTER PETER STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE

SOLD TO: RUSS CRANE RUSSELL CRANE SERVICE

# of Loads: 2
Job Total: 67.92

DELIVER TO:

ZONE NO.:

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST01 CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212520

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212324

AGGREGATE TICKET

99,040 lb Manual Wt.
OUT GROSS 49.52 Tons

37,160 lb Manual Wt. 03/01/2001 DATE
P.T. TARE 18.58 Tons

61,880 lb 28,069 kg 12:44 TIME
NET 30.94 Tons 28.07 Metric Tonnes

WEIGH MASTER TYLER STEWART Pit: P104

P.O. # JOB NO.:

SIGNATURE

SOLD TO: PIGG CRANE RUSSELL CRANE SERVICE # of Loads: 3
Job Total: 135.21

DELIVER TO:

ZONE NO.:

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST01 CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212525

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457-3654 OR 877-6102

211881

AGGREGATE TICKET

Manual Wt.

IN GROSS 92,120 lb 46.06 Tons
T. TARE 33,460 lb 16.73 Tons
NET 58,660 lb 29.33 Tons

Manual Wt.

26,608 kg
26.61 Metric Tonnes

03/01/2001 DATE

10:46 TIME

WEIGH MASTER TYLER STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE

SOLD TO: RUSS CRANE RUSSELL CRANE SERVICE

# of Loads: 3
Job Total: 79.79

DELIVER TO: FOB PLANT FRANKS (clean)

ZONE NO.: Contaminated Rock/Sand.

PRODUCT: CONC SAND CONCRETE SAND

TRUCKER: CUST02 RUBEN

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212514

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212545

AGGREGATE TICKET

	GROSS	82,800 lb 41.40 Tons	Manual Wt.	
OUT				
	TARE	33,460 lb 16.73 Tons	Manual Wt.	03/01/2001 DATE
P.T.				
	NET	49,340 lb 24.67 Tons	22,381 kg 22.38 Metric Tonnes	15:16 TIME

WEIGH MASTER
TYLER STEWART

Pit: WILDL

P.O. # JOB NO.:

SIGNATURE
David W Quarles

SOLD TO: ROSS CRANE RUSSELL CRANE SERVICE

# of Loads: 16
Job Total: 426.12

DELIVER TO: PLANT

ZONE NO.:

PRODUCT RUN UNCLASSIFIED PIT RUN

LV. JOB	ARR. PLANT
ARR. JOB	

TRUCKER: T02
RUBEN

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212545

TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212512

AGGREGATE TICKET

Manual Wt.

90,160 lb
OUT GROSS 45.08 Tons
37,160 lb
P.T. TARE 18.58 Tons
53,000 lb
NET 26.50 Tons

Manual Wt.

24,041 kg
24.04 Metric Tonnes

03/01/2001 DATE

11:59 TIME

WEIGH MASTER STEWART

Pit: P104

P.O. # JOB NO.:

SIGNATURE X David Wallace

SOLD TO: RUSS CRANE RUSSELL CRANE SERVICE

# of Loads: 1
Job Total: 26.50

DELIVER TO:

ZONE NO.:

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST01 CUSTOMER TRUCK

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212516 TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212529

AGGREGATE TICKET

47,060 lb Manual Wt.
OUT GROSS 23.53 Tons

33,460 lb Manual Wt. 03/01/2001 DATE
P.T. TARE 16.73 Tons

53,600 lb 24,313 kg 13:52 TIME
NET 26.80 Tons 24.31 Metric Tonnes

WEIGH MASTER STEWART Pit: P104

P.O. # JOB NO.:

SIGNATURE David W Chaplin

SOLD TO: RUSS CRANE RUSSELL CRANE SERVICE # of Loads: 7
Job Total: 175.32

DELIVER TO: FOB PLANT

ZONE NO.:

PRODUCT: PIT RUN UNCLASSIFIED PIT RUN

TRUCKER: CUST02 RUBEN

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212530 TRUCKER'S PAYROLL

COLUMBIA READY MIX, INC.

3307 RIVER ROAD
YAKIMA, WA 98902
(509) 457- 3654 OR 877-6102

212526

AGGREGATE TICKET

81,940 lb
OUT GROSS 40.97 Tons
33,460 lb
P.T. TARE 16.73 Tons
48,480 lb
NET 24.24 Tons

Manual Wt.

Manual Wt.

21,991 kg
21.99 Metric Tonnes

03/01/2001 DATE

13:11 TIME

WEIGH MASTER PEGGY STEWART

Pit: P104

P.O. #

JOB NO.:

SIGNATURE David W Crane & Co

SOLD TO: SUSS CRANE RUSSELL CRANE SERVICE

# of Loads: 4
Job Total: 86.65

DELIVER TO: END PLANT

ZONE NO.:

PRODUCT: PUMP DUMP CHARGE

TRUCKER: CUSTOC RUBEN

LV. JOB	ARR. PLANT
ARR. JOB	

UNIT PRICE	NET PRICE	HAUL
SUB-TOTAL	TAX	TOTAL

212527

TRUCKER'S PAYROLL