DEVELOPMENT OF RECLAIMED POTABLE
WATER QUALITY CRITERIA

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SPECTRIX
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PART I: PROJECT DEFINITION

1.0 INTRODUCTION/BACKGROUND

In order to minimize launch requirements necessary to meet the demands of future long-term spaceflight, NASA will reuse water reclaimed from various on-board sources including urine, feces, wash water and humidity condensate. Development of reclamation systems through actual flight qualification requires the promulgation of water quality standards for potable reuse of the reclaimed water.

Existing standards for domestic U.S. potable water consumption have been developed by the United States Public Health Service (USPHS), and more recently the Environmental Protection Agency (EPA). Earlier standards evolved over many years of real use experience in which it was determined that waters containing less than the specified amounts of a particular constituent were acceptable for human consumption. In more recent years the EPA has conducted extensive analytical studies of potable water supplies and performed corollary epidemiological and toxicological investigations. However, neither the old USPHS standards or newly proposed EPA standards consider the peculiar problems associated with the potable reuse of recycled water. The need for a study effort to develop a protocol (or management plan) leading to the establishment of potable reuse water standards has been recognized by NASA. This report gives the results of this study effort.

The most mature technique for recovery of potable water from urine and humidity condensate aboard spacecraft is vapor compression distillation or VCD. The process employs motor-driven evacuated rotating concentric cylinders to provide artificial gravity for boiling and condensing at low pressure. Energy efficiency is obtained by compressing the vapor and allowing it to condense at a higher temperature on the opposite side of the boiling surface. Latent heat is thus recycled and the only input energy required is that needed to overcome dynamic friction and provide for thermodynamic work.

The NASA recognizes that any water quality standards program for spacecraft must be based to a great extent on knowledge of the potential contaminants associated with reclamation systems and their associated health hazards. Previously funded study efforts have developed and applied qualitative and quantitative analytical methods for characterization of organic contaminants in reclaimed water (Contract NAS9-14548). These methods have been applied to the analysis of product water from operation of a prototype VCD unit. The results of these analyses and their implications in the light of potential health hazards are discussed in this report.

2.0 SCOPE

The scope of this effort was two-fold: (1) to define a protocol by which comprehensive reclaimed water potability/palatability criteria can be established and updated; and

(2) to continue the effort initiated under Contract NAS9-14548 to characterize the organic content of reclaimed water in the Regenerative Life Support Evaluation (RLSE).

3.0 OBJECTIVES

3.1 The general objective of the study effort was the development of a protocol leading to the formulation of Reuse Water Quality Standards for aerospace applications that will qualify the water for human consumption, with particular emphasis on trace organic contaminants and their potential toxicological significance.

3.2 The objective of the organic constituent characterization was to identify and quantify potentially toxic and adverse taste producing organic components in reclaimed water for the RLSE program to include the development and demonstration of analytical procedures

4.0 SPECIFIC TASKS

4.1 Protocol Study

The following tasks were identified in order to accomplish the first stated objective:

4.1.1 Perform a comprehensive literature search.

4.1.2 Identify regulatory roles, if any, of other government entities.

4.1.3 Examine the total concept of reclaimed water in general, including identification of other potential users of reuse water outside of NASA and consider possible cooperative programs.

4.1.4 Evaluate current information on the identification and toxicology of contaminants in spacecraft water supplies.

4.1.5 Formulate a plan to obtain the necessary toxicological support information leading to the qualification of any potential recycle system.

4.1.6 Define the specific approach for generation of water quality criteria to serve as the basis for water quality standards.

4.1.7 Define an approach for developing Reuse Water Quality Standards based on adopted water quality criteria that will allow qualification of any potential spacecraft water source.

4.1.8 Define the appropriate steps which lead from initial recommended Reuse Water Quality Standards to official acceptance and/or approval.

4.1.9 Formulate a schedule and provide a cost estimate for the total program effort recommended as a result of this program plan development.

4.2 Organic Constituent Identification/Quantification

The following tasks were identified in order to accomplish the second objective:

4.2.1 Develop a sampling plan and procedures consistent with other objectives and the overall objective of the RLSE testing program.

4.2.2 Perform qualitative analyses of organics in product water samples at the ppb level with a gas chromatogram-mass-spectrometer-data system (GC-MS-DS).

4.2.3 Perform quantitative analyses of the carbonaceous matter in the product water samples (total organic carbon, dissolved organic carbon, and particulate organic carbon).

4.2.4 Perform quantitative organic analyses of specific chemical constituents which present potential toxicity/potability/palatability problems.

4.2.5 Assist in evaluation of RLSE performance.

4.2.6 Identify potential toxic hazards and a program of toxicological studies if necessary.

4.2.7 Define spacecraft monitoring requirements for potability/palatability.

4.2.8 Revise as appropriate preliminary spacecraft potable water criteria and specification developed under NAS9-14548.

4.2.9 Investigate the potential formation of iodomethanes following the addition of iodine to VCD product water. Iodine is to be used as a biocide during spacecraft storage of product water.

PART II: PROTOCOL STUDY EFFORT

1.0 LITERATURE SEARCH (Task 4.1.1)

Both manual and computer searching was used to obtain listings of potentially useful reference material. A particularly significant set of abstracts entitled Selected Water Resource Abstracts (Office of Water Research and Technology, Water Resources Scientific Information Center, U.S. Dept. of the Interior) found in the NASA-JSC library proved to be the best reference source. These abstracts were manually examined back to 1969 and more than 250 abstracts evaluated. Of these, approximately 50 were deemed to be of sufficient significance to warrant obtaining and evaluating the complete article. Chemical Abstracts was also examined manually but contained no references not previously found in the above.

Computer searching of STAR abstracts was accomplished using the RECON terminal in the NASA-JSC library. Instruction on use of the terminal was provided by the library staff and then the author was left free to try any and all combinations of descriptor terms desired. Approximately 16 hours was spent at the RECON terminal including instruction time. Potentially significant abstracts were called up on the screen and evaluated. Articles deemed potentially significant were obtained and evaluated. About ten (10) new references not found in the Selected Water Resource Abstracts or Chemical Abstracts were found using RECON. These references were all government contract reports.

All pertinent articles obtained in the literature search were read and evaluated in detail. The results of the search are of limited value in that there are very few articles dealing directly with potable reuse of water. A summary of the findings in the areas of specific interest to this project are given below. References are listed in Appendix I.

1.1 Regulatory role identification (Task 4.1.2)

Federal laws have recently established that the U.S. Environmental Protection Agency along with their state level counterparts are responsible for quality criteria of public potable water supplies. Discussions during personal visits and telephone conversations with Dr. Joseph Cotruvo and Dr. Craig Vogt of the EPA Office of Drinking Water in Washington, D.C. indicate that the EPA will want to assume more than an advisory role in the generation of spacecraft reclaimed potable water quality criteria. This advisory role can best be accommodated by including one or more prominent EPA scientists on an Advisory Committee to review the criteria. The EPA Office of Drinking Water has agreed to provide one of its prominent scientists to serve on such a committee upon request of the NASA.

1.2 Other users and possible cooperative programs (Task 4.1.3)

The following have been identified as intended users of reclaimed water for human consumption in the near future:

1.2.1 The U.S. Army: U.S. Army Medical Bioengineering Research & Development Laboratory (USAMBRDL)
Ft. Dietrich, MD

Contact: James C. Eaton, Jr., Phone (301) 663-8000

1.2.2 The U.S. Navy: Naval Civil Engineering Laboratory (USNCEL)
Port Hueneme, CA

Contact: Dr. D.B. Chan, Phone (805) 982-4173

1.2.3 U.S. Air Force: U.S. Air Force Civil Engineering Center
Panama City, FL

Contact: Major Chester Pauls, U.S.A.F. Headquarters, Forrestal Bldg., Washington, DC

Other groups with ongoing research in the area of reuse water include:

1.2.4 The U.S. EPA Office of Water Planning and Standards

1.2.5 The U.S. EPA Health Effects Research Laboratory

1.2.6 The Water Pollution Control Federation

1.2.7 The American Water Works Association

1.2.8 The Safe Drinking Water Committee of the National Academy of Sciences

Only the former group (the military) have schedules and requirements much in common with NASA's interests.

In December, 1977, the USAMBRDL issued an RFP to provide a project management plan for development of direct reuse water quality criteria for Army and Navy (USNCEL) applications. The evaluation/award was completed on March 16, 1978, and went to Culp, Wesner and Culp, Inc. (CWC). We attended the Technical Transfer meeting given by CWC at USAMBRDL at the conclusion of their study in September, 1978, to determine if coordination of the NASA and Army efforts was possible. At this time it was indicated that (1) potable reuse of reclaimed wastewater by troops in the field was not contemplated, (2) potable reuse is a possibility at advanced/remote bases to support military operations in the field over relatively short periods of time (a few weeks), and (3) potable reuse may be used instead of desalination and to supplement inadequate supplies at permanent Navy bases, and (4) that present plans do not anticipate reclaiming urine and/or fecal wastes. These requirements are so different from those required for future NASA needs that any cooperatively funded effort was not felt to be feasible. We do recommend, however, that NASA remain in contact with USAMBRDL and keep apprised of their developments.

1.3 Reuse water contaminants and associated toxicity (Task 4.1.4)

There is no data available on contaminants in product water from recycled human wastes other than that previously obtained in NASA studies. Many references obtained on the health effects of various chemicals found in drinking water supplies proved useful in evaluating the toxicity of the RLSE product water contaminants. These references are given in the toxicity evaluations.

2.0 ORGANIC CONSTITUENT IDENTIFICATION AND QUANTITATION (Tasks 4.2.1 - 4.2.5)

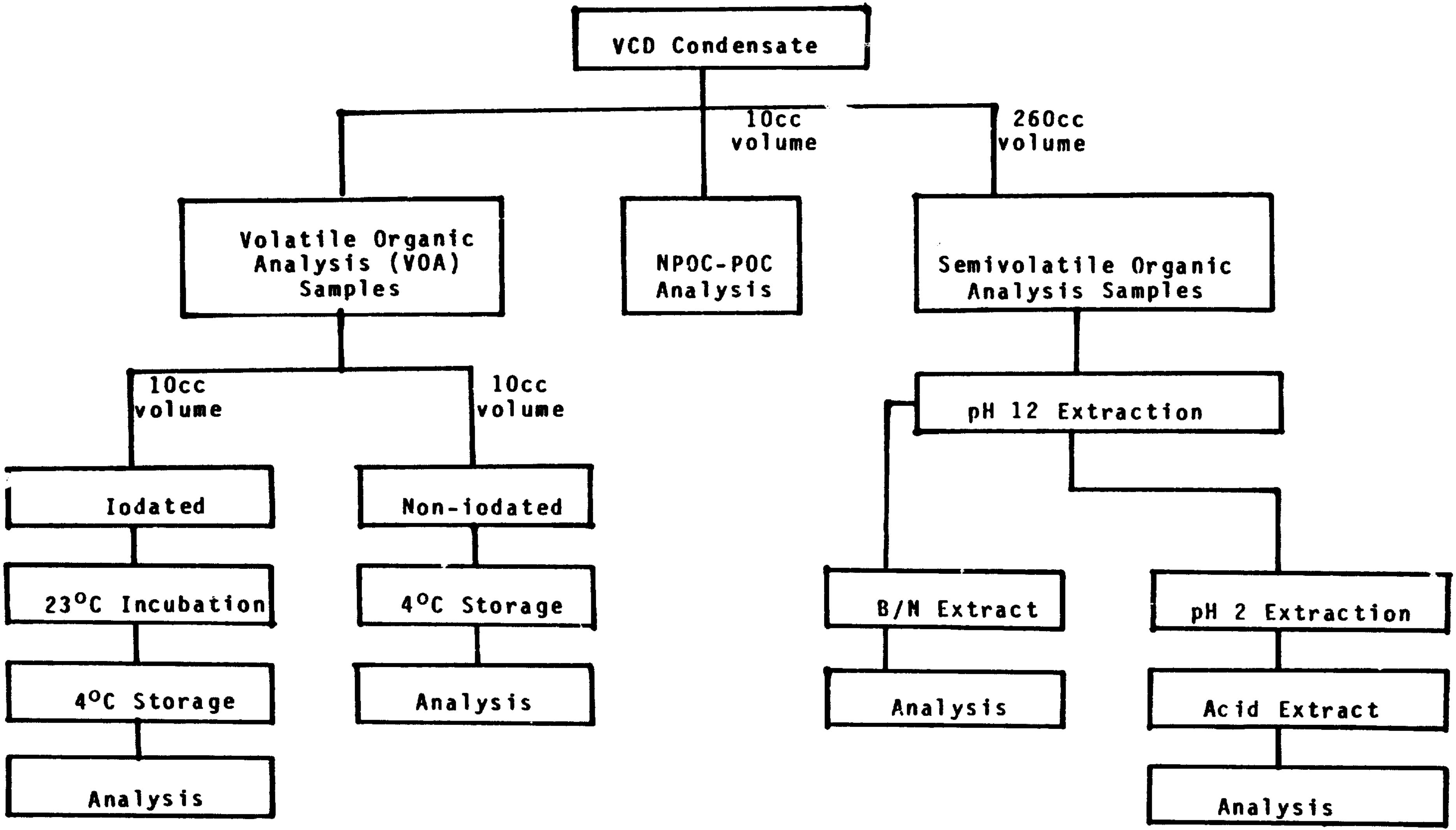
2.1 Sampling of the VCD

The originally planned November, 1977 operation of the VCD unit actually occurred in March, 1978. The unit was operated from March 8, 1978 to March 20, 1978. Eight 160 cc volume samples were collected for semi-volatile analysis and fifteen 10 cc volume samples for volatile organic analysis (VOA). The collection times, analysis times and handling history of the samples are summarized in Tables I and II. All samples were collected in No-Chromix acid cleaned glass septum-seal vials. Care was taken to eliminate any headspace losses by ensuring all bottles were completely full. The vials were sealed with teflon lined silicone rubber septa. A sample of laboratory distilled water from NASA-JSC was provided to use as a blank. All samples collected from the VCD unit were condensates.

2.2 Sample Preparation and Analysis

The samples were maintained at 4° C during storage at Sunnyvale, shipment to Houston and storage at our laboratory. The samples were received at Spectrix on March 31, 1978. Iodine solution (supplied by NASA-JSC) was added to one of each of the five 10 cc samples received in duplicate and to one 130 cc sample to provide a free iodine concentration of 5 ppm. Addition was accomplished by microliter syringe through the septum seal to minimize loss of volatiles. The isolated samples were incubated at room temperature for 10 days to simulate spacecraft storage.

All samples were analyzed for volatiles and semivolatiles according to the scheme which follows:


VCD Condensate
10cc volume
260cc volume
Volatile Organic Analysis (VOA) Samples
NPOC-POC Analysis
Semivolatile Organic Analysis Samples
10cc volume
10cc volume
Iodated
Non-iodated
pH 12 Extraction
23°C Incubation
4°C Storage
B/N Extract
pH 2 Extraction
4°C Storage
Analysis
Analysis
Acid Extract
Analysis
Analysis

The volatiles were placed onto a Tenax trap using a purge and trap technique similar to that developed by Zlatkis, Lichtenstein and Tishbee (Chromatographia, 6:67-70, 1973) later modified by T.A. Bellar and J.J. Lichtenberg (Journal of AWWA, 739-744, 1974) of the EPA. However, rather than using a commercially available liquid sample concentrator, the water was purged in an all glass apparatus developed in this laboratory (Figure 1). (Comparative reproducibilities of the two concentrators are shown in Table III at three concentrations for 13 compounds.) The tenax trap was then heat desorbed in a modified injector port. The effluent gas was cryogenically trapped on a precolumn then rapidly headed to achieve a plug injection for GC-MS analysis. The analysis was accomplished on a 0.2% Carbowax 1500 Carbopack-C.

Three pairs of the 130 cc samples were composited to provide sufficient sample for pH dependent analysis for semi-volatile compounds to give detection limits of 1-10 ppb. The pH was adjusted with 6N NaOH to 11 and a liquid/liquid base/neutral extraction made with methylene chloride. The extract was dried and concentrated prior to analysis. This fraction was then analyzed on a glass column packed with 1% SP-2250 on Supelcoport.

The pH of the base/neutral extracted water was then adjusted with 6N HCl to 2, extracted with methylene chloride, and the extract dried, concentrated and analyzed on a glass column packed with Tenax GC. Prior to analysis of both fractions, an internal standard of d10-anthracene was added to each extract.

All analyses were performed on a Finnigan 3200-INCOS 2300 gas chromatograph-mass spectrometer computer system (GC-MS-COM). The scan range of the mass spectrometer was 40-275 amu for the volatiles and 40-425 amu for the extracts. The scan cycle was 3.0 seconds in both cases. Detection limits were in the range 1-10 ppb (micrograms per liter) depending on the specific component. The GC-MS interface is a glass jet separator resulting in a completely glass or glass-lined sample entry system.

The remaining two 160 cc samples were used for carbon balance studies. Two 10 cc aliquants were removed from each bottle for VOA and ultra low level total organic carbon analysis. The remaining 140 cc was extracted for semivolatile analysis as described above.

The total organic carbon (TOC) analysis was performed on a Dohrmann Model DC-54 Ultra Low Level Total Organic Carbon Analyzer System. The analyzer first purges a 10-50 ml acidified water sample with helium removing inorganic CO_2 produced by the acidification and purgeable (volatile) organics (POC). The CO_2 is removed by a scrubber. The POC is converted to CH_4 by reductive pyrolysis which is then measured by a flame ionization detector (FID). The purged water is then transferred to a UV irradiation cell which oxidizes the nonpurgeable organic carbon (NPOC) to carbon dioxide. This carbon dioxide is then purged and passed to the reductive pyrolysis unit for conversion to methane and measured by the FID to give a value for NPOC. The POC and NPOC are then added to give a value for TOC.

2.3 Results

2.3.1 Volatile Results

Table IV summarizes the results of the volatile analyses. Figure 2 is a typical VOA chromatogram (see Table III: 3-17-78 0900 for results). Compounds appearing on the EPA list of priority pollutants (Appendix II) were identified by a reverse search procedure. The reverse search technique is employed to answer the question, "Is this specific compound present?" A positive identification of a priority pollutant is made when the reverse search technique used in this method meets the following criteria:

(1) The appropriate masses maximize simultaneously;

(2) The retention time is within specified limits; and

(3) The ratio of the masses is correct.

Peaks in the chromatogram not identified by reverse search were then identified by forward search using the NBS library and manual interpretation. Response factors were obtained by analyzing standard mixtures of the seven most prevalent compounds (1,4-dioxane, dichloromethane, methyl isopropenoate, benzene, chloroform, hexane, and toluene) at concentrations of 8, 80 and 240 μg/L. These seven compounds were quantitated using the authentic response factors which were essentially linear over the concentration range found here. The remaining compounds were quantitated using the response of chloroform. A blank space in the table indicates the compounds were not detected. Detection limits for the compounds are indicated at the bottom of Table IV.

2.3.2 Discussion of Volatile Results

These results for methyl iodide are very inconsistent and have no correlation to the laboratory iodation of the samples. After completing these analyses it was learned that pretreated urine feedstock containing iodide was used in the VCD tests. The randomness of the appearance of the methyl iodide is apparently related to the operation of the VCD unit. It would appear that iodation is not a significant factor upon product water since all laboratory iodated samples did not contain methyl iodide. We cannot see any definite trends in the results of the other volatiles that correlate with our knowledge of the operation of the unit or with sample handling and storage. There seems to be no significant difference in samples stored for long periods as compared to shorter storage. One notices rather large differences for quantitative results of dioxane, methyl acrylate and carbon disulfide for two samples collected only a few hours apart. Reproducibilities of samples collected at identical times (3-14-78, 3-17-78 and 3-20-78) are good indicating the above noted differences are real and are probably indicative of operational characteristics of the VCD.

2.3.3 Semivolatile Extract Results

No detectable semivolatile compounds could be found in any of the extracts with a detection limit of 10-100 μg/L (ppb). Typical chromatograms of an acid and base/neutral extract are given in Figures 3 and 4. The detection limit for plasticizers was higher (100 ppb) due to contamination of the extracts by the lid liners used in the extract storage vials. These lid liners are EPA approved, but have recently been shown in our laboratory to badly contaminate the samples. It is also possible that these plasticizers were leached from the plastic tubing known to be used in the VCD unit, but the lid contamination makes it impossible to pinpoint the source. The extract data was reversed searched for all the compounds on the EPA priority pollutant list (see Appendix II) with the only positive results being those for the phthalate plasticizers. Table V gives the results of these searches. All peaks were identified by the reverse search so no forward searches were conducted.

2.3.4 Carbon Balance

Carbon balance results for the two samples checked are given below:

Sample No.	POC	NPOC	Total Volatiles	Total Semivolatiles
1	61 μg/L	20 mg/L	350 μg/L	Indeterminate
2	<50 μg/L	18 mg/L	34 μg/L	Indeterminate

Unfortunately the cap contamination problem makes it impossible to complete the carbon balance.

The intent of a carbon balance study is to account for all measurable TOC when totaling quantities of individual components quantified. Precision of the analyses of volatiles and semivolatiles as described herein is such that one should be able to account for 70 - 80% of the TOC if the analyses performed will "see" all the organic material. Organic material not seen by these analyses could include nonvolatile, non-gas chromatographable high molecular weight organic compounds and particulate carbon (charcoal or graphite, for example). Additional work should be done to achieve this carbon balance on subsequent VCD samples. Failure to account for the TOC by volatile and semivolatile analyses after correction for any particulate organic carbon would indicate the need for high pressure liquid chromatographic (HPLC) analyses.

3.0 TOXICITY OF CONTAMINANTS FOUND IN VCD PRODUCT WATER SAMPLES (Task 4.2.6)

The purpose of this task was to assess the adequacy of the data base regarding the toxicity and hazard potential of the contaminants found in the prototype VCD water reclamation system product water. An intensive search of the available published literature was conducted in regard to each of the 15 compounds identified by chemical analyses of the submitted samples.

Both computer-assisted and direct search of chemical abstracts and Index Medicus were conducted within the library system of the U.T. Health Science Center. Data referral systems such as RTELS, TOXLINE, TOXBACK and CANCERLIT were also searched using appropriate proper names of the compounds and their synonyms. Further, standard texts and current periodicals were screened for relevant documentation.

Primary attention was addressed to physiological and biochemical studies that utilized mammalian species, including man. Included in the review were references dealing with the metabolic fate, acute and chronic toxicity, and any confirmed evidence of carcinogenicity, teratogenicity or embryo toxicity. Available material for each compound was compiled and summarized. This information as well as a recommendation for an interim drinking water standard, where data permit, is presented in Appendix III.

4.0 REVISION OF PRELIMINARY SPACECRAFT POTABILITY/PALATABILITY CRITERIA (Task 4.2.8)

For 12 of the compounds there were adequate toxicity and hazard assessment data available to make a judgment regarding an interim drinking water standard. These are incorporated into Table IV which is an updated suggested reclaimed potable water quality specification which includes all potability/ palatability factors. There was sufficient information to assure little or no hazard to the proposed population at concentrations at or below the levels recommended.

It should be noted that the recommended concentrations are not intended to provide a uniform safety factor. In each case the recommendation was a subjective judgment based on all the available knowledge of and the nature of the health risk. The metabolic fate, the acute and chronic toxicity and the potential for irreversible damage were all considered in making a judgment. It was also assumed that water consumption would not exceed 2 liters per person per day. We recognize this figure may be low since it does not include water for food reconstitution which should be included when future missions are better defined. No consideration was given to acceptability of the recycled water in regard to either smell or taste. Health considerations alone dictated the judgment.

In several cases, the standard might have been relaxed if financial considerations were an important factor. We considered, however, that consistent with the available technology, a conservative interim standard would best protect the population to be exposed.

For the remaining three compounds:

1,2-Dithiol ethane
2-methyl furan
methyl acrylate

there were essentially no data upon which to base any judgment regarding a drinking water standard. In regard to these compounds, there appear to be two courses of action:

(1) Eliminate the source of these materials from the system; or

(2) Develop an adequate data base upon which to make an appropriate judgment.

5.0 <u>HEALTH EFFECTS RESEARCH REQUIREMENTS</u> (Task 4.1.5)

Many health considerations should be included in a management plan for establishing Reuse Potable Water Quality Criteria. However, the U.S. domestic drinking water criteria are of little value in regard to this program since:

(1) this program is primarily concerned with organic compounds whereas the U.S. domestic program has only limited activity in this area; and

(2) there is no assurance that the carbon chloroform extract (CCE) method is adequate for monitoring all organic compounds of toxicological interest. The CCE standard was developed as a test for undesirable tastes and odors in drinking water, not for identifying or quantifying toxic trace organics.

Considerations that have not been adequately addressed include the following:

(1) No one knows, nor is there any satisfactory method of predicting the type or quantity of contaminants produced from multiple re-cycling of physiological system by-products;

(2) There is little or nothing known regarding the potential for harmful effects from mixtures of either the compounds already identified or those potentially present in a re-cycle system;

(3) There is no way of predicting the impact of prophylactic or therapeutic drugs or their biochemical transformation products in any re-cycle water system;

(4) In systems where both sexes would contribute by-products, it is impossible to predict the health consequences of various excreted endocrine materials or their biotransformation products in addition to any or all of the above.

The only satisfactory way to address the above considerations is by way of an appropriate bioassay procedure.

We propose the following investigation to address the above considerations.

(1) Isolate separate groups of male and female rats in metabolic units that are capable of collecting all water from physiological processes, including the expired air.

(2) Maintain these groups of animals on their own recycled water for a one-half life period (1 year). This would permit 50 cycles of water through the animals. The recycling system can be the actual prototype hardware or a laboratory simulator using the same purification principles.

(3) Identical, but separate groups of animals would receive, in addition to the above, various drugs that would find use in a spacecraft mission.

(4) All animals would be maintained for a second year under normal laboratory conditions, then sacrificed to assess any potential for either chronic toxicity or cancer as compared to concurrent control animals.

(5) Evaluate present analytical methods to verify that the detection limits for drugs and endocrine materials (e.g., hormones) are sufficient to meet toxicity requirements.

(6) Chemical analyses would be conducted on each cycle of re-cycled water to evaluate any change in composition of the system contaminants.

(7) An Ames test assay would be conducted on a sample at each re-cycle to predict the development of any mutagenic potential in each system.

PART III: RECOMMENDED PROTOCOL FOR ESTABLISHING POTABLE REUSE WATER QUALITY CRITERIA FOR MANNED SPACEFLIGHT (Tasks 4.1.6 thru 4.1.9)

1.0 REVIEW COMMITTEE

We recommend that NASA establish a five person review committee of nationally recognized experts to consider, review and approve water quality standards for potable reuse of reclaimed water in manned spaceflight. Experts in the following scientific and/or engineering disciplines should be included:

(1) Carcinology;

(2) General toxicology;

(3) Aerospace medicine;

(4) Mutagenicity or virology; and

(5) Water quality.

Members of the committee should be chosen so that they may also serve as official representatives of the National Academy of Sciences, the Environmental Protection Agency and the National Association of American Toxicologists. Although these groups have no mandate requiring their involvement in establishing NASA water standards, their acceptance or approval will be very beneficial. We estimate that this committee will meet quarterly to hear briefings on progress of standards development and to provide feed back to serve as guidelines to future action.

2.0 TASK GROUP

We recommend that a th[illegible]e person Task Group be established to support the Review Committee. This Task Group should be composed of experts in the fields of general toxicology, analytical water chemistry, and spacecraft engineering. We estimate that up to twenty-five percent of full-time will be required for the Task Group members to properly support this effort. Supporting Task Group staff services including secretarial work, technical editing, library services, graphic services, and the mechanical production of Task Group reports will be required.

The assignment of the Task Group will be to develop water quality criteria for potable reuse of reclaimed wastewaters in manned spaceflight for presentation to the Review Committee. The water quality criteria should take the form of maximum allowable levels of contaminants based on health aspects and minimum amounts of any chemicals necessary to maintain suitable palatability. Specific assignments should include, but not be limited to, the following tasks.

2.1 Identification of Systems to be Qualified

At the present time NASA is considering only the VCD reclamation system for future spaceflight. Development of water quality criteria will necessarily be directed more to the peculiar characteristics of this type of system, but care should be taken to insure that the criteria can be applied to any new systems developed in the future.

2.2 Development of a Plan for any Research Effort Required

Previous NASA efforts (Contracts NAS9-14548, NAS9-12969 and NAS9-15368) have shown that additional research is needed to develop analytical methods to completely characterize the organic contamination present in prototype VCD unit product water and to determine the toxic potential of these contaminants under exposure conditions in long term spaceflight. In particular, only 10-25 percent of the total organic carbon (TOC) found in the water has been accounted for in individual component analyses. It shall be the duty of the Task Group to evaluate the existing data and recommend further research in analytical toxicology, microbiology, and health effects which may be required to provide an aedquate basis for setting water quality criteria regarding health aspects. NASA must then fund the required research.

2.3 Palatability Requirements

The removal of essentially all inorganic cations and anions accomplished by the VCD reclamation system results in an essentially tasteless water. This tastelessness could adversely affect astronaut water consumption. NASA experience has shown that it is important that astronauts consume sufficient water to prevent partial dehydration. Previous efforts (NAS9-12969) have identified ingredients which can be added to the VCD product water to enhance the taste and encourage normal consumption. The Task Group shall evaluate this data for inclusion in the water quality criteria.

2.4 Flight Qualification Test Plan

Flight qualification is required for all systems to be flown in spacecraft. The qualification test plan usually requires submitting the equipment to environmental testing simulating extremes of expected flight environments such as gravitational, thermal, mechanical, etc., for time periods that will insure it does not fail during expected flight durations of these environments. The questions of how long one should test a piece of equipment that is supposed to produce a consummable product within rigid specifications for extended periods of time has not normally been addressed in flight hardware test plans. It shall be the duty of the Task Group to develop a suitable flight qualification test plan to insure production of potable water for established flight durations. This plan should be based on operational history of prototype flight equipment insofar as available and shall specify what parameters must be monitored to assure adequate water quality during the flight testing. The fact that no alternate water supply will be available during flight may be a key factor here.

2.5 Flight Monitoring Requirements

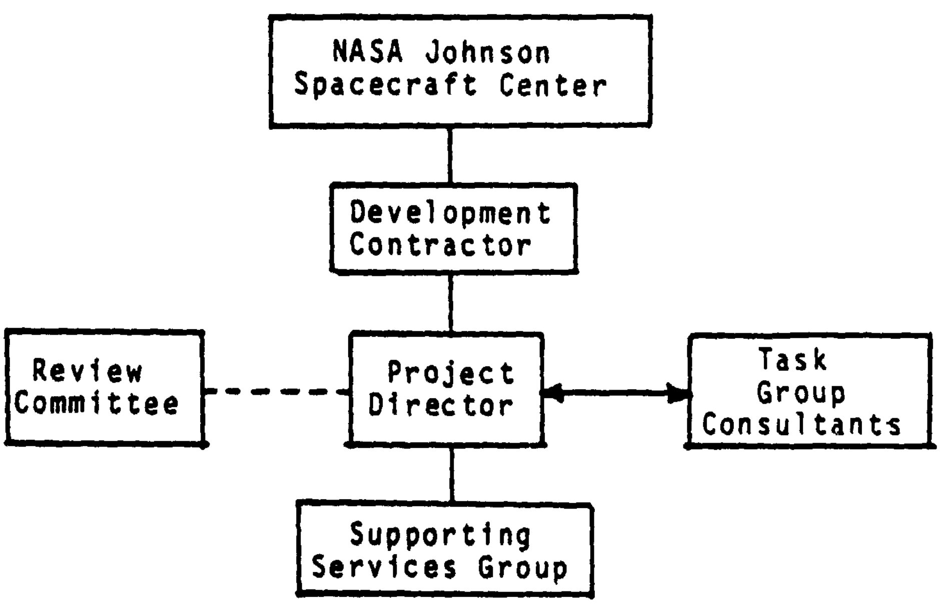
The question of in-flight monitoring requirements must be considered in developing the water quality criteria. Health effects research on expected contaminants may indicate the need for non-conventional monitoring which is not presently available. It shall be the duty of the Task Group to identify these requirements as soon as possible so that any necessary development of monitoring devices may be accomplished in time to meet projected flight schedules.

2.6 Interim Standards

Interim water quality standards may be required to meet design and testing dates prior to development of final water quality criteria. It shall be the duty of the Task Group to identify the need for and recommend Interim Standards to the Review Committee for their approval.

3.0 ORGANIZATION OF THE WATER QUALITY CRITERIA DEVELOPMENT EFFORT

We recommend that the development effort be organized as indicated below:



NASA-JSC should select a Contractor to carry out the effort required to establish approved water quality criteria. This effort can then be carried out under a Project Director. The Project Director shall organize the Review Committee and the

Task Group and arrange consulting contracts between the members and the Development Contractor to cover their time and travel expenses. Review Committee members employed by other government agencies may be covered for their consulting time through inter-agency agreements. Final approval of Review Committee members should be subject to NASA Headquarters approval.

The Project Director shall define, direct, and coordinate the efforts of the Task Group and serve as the interface between the Task Group and the Review Committee. The Project Director shall be responsible for calling Review Committee meetings, establishing the agenda, and insuring Review Committee recommendations are reflected in subsequent Task Group efforts.

The Supporting Services group shall provide secretarial, reproduction, graphics and technical services as requested by the Project Director to support the Review Committee and Task Group Functions.

4.0 SCHEDULE FOR ESTABLISHING APPROVED CRITERIA

An estimated schedule which lists the major tasks and milestones is shown in the enclosed chart.

5.0 COST ESTIMATES

Estimated costs for the proposed four year program are as follows:

5.1 Professional Labor and Overhead $318,400

(1) Project Director (1 man year) $115,000

(2) Secretarial (1 man year) $ 27,600

(3) Other nonprofessional labor (2 man years)	$ 55,800	
(4) Review Committee Consulting Fees 60 days @ $500/day	$ 30,000	
(5) Task Group Consultants 360 days @ $250/day*	90,000	
*Assumes Spacecraft Engineer is a NASA employee		
5.2 Contract Research Effort for Toxicity Studies		$500,000
Total Estimated Cost		$818,400

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APPENDIX II

EPA PRIORITY POLLUTANTS

NAS9-15368

EPA PRIORITY POLLUTANTS

COMPOUND	NO.[1]	COMPOUND
acenaphthene	31A	2,4-dichlorophenol
acrolein	32V	1,2-dichloropropane
acrylonitrile	33V	1,3-dichloropropylene
benzene	34A	2,4-dimethylphenol
benzidine	35B	2,4-dinitrotoluene
carbon tetrachloride	36B	2,6-dinitrotoluene
chlorobenzene	37B	1,2-diphenylhydrazine
1,2,4-trichlorobenzene	38V	ethylbenzene
hexachlorobenzene	39B	fluoranthene
1,2-dichloroethane	40B	4-chlorophenyl phenyl ether
1,1,1-trichloroethane	41B	4-bromophenyl phenyl ether
hexachloroethane	42B	bis(2-chloroisopropyl)ether
1,1-dichloroethane	43B	bis(2-chloroethoxy)methane
1,1,2-trichloroethane	44V	methylene chloride
1,1,2,2-tetrachloroethane	45V	methyl chloride
chloroethane	46V	methyl bromide
bis(chloromethyl)ether	47V	bromoform
bis(2-chloroethyl)ether	48V	dichlorobromomethane
2-chloroethylvinyl ether	49V	trichlorofluoromethane
2-chloronaphthalene	50V	dichlorodifluoromethane
2,4,6-trichlorophenol	51V	chlorodibromomethane
parachlorometa cresol	52B	hexachlorobutadiene
chloroform	53B	hexachlorocyclopentadiene
2-chlorophenol	54B	isophorone
1,2-dichlorobenzene	55B	naphthalene
1,3-dichlorobenzene	56B	nitrobenzene
1,4-dichlorobenzene	57A	2-nitrophenol
3,3'-dichlorobenzidine	58A	4-nitrophenol
1,1-dichloroethylene	59A	2,4-dinitrophenol
1,2-trans-dichloroethylene	60A	4,6-dinitro-o-cresol

As it appears in "Sampling and Analysis Procedures for Screening of Industrial Effluents for Priority Pollutants," EPA, Revised April, 1977.

NAS9-15368

EPA PRIORITY POLLUTANTS
(Continued)

COMPOUND	NO.[1]	COMPOUND
N-nitrosodimethylamine	88V	vinyl chloride
N-nitrosodiphenylamine	89P	aldrin
N-nitrosodi-n-propylamine	90P	dieldrin
pentachlorophenol	91P	chlordane
phenol	92P	4,4'-DDT
bis(2-ethylhexyl)phthalate	93P	4,4'-DDE
butyl benzyl phthalate	94P	4,4' DDD
di-n-butyl phthalate	95P	a-endosulfan-Alpha
di-n-octyl phthalate	96P	b-endosulfan-Beta
diethyl phthalate	97P	endosulfan sulfate
dimethyl phthalate	98P	endrin
benzo(a)anthracene	99P	endrin aldehyde
benzo(a)pyrene	100P	heptachlor
3,4-benzofluorathene	101P	heptachlor epoxide
benzo(k)fluoranthane	102P	a-BHC-Alpha
chrysene	103P	b-BHC-Beta
acenaphthylene	104P	r-BHC-Gamma
anthracene	105P	g-BHC-Delta
benzo(ghi)perylene	106P	PCB-1242
fluorene	107P	PCB-1254
phenanthrene	108P	PCB-1221
dibenzo(a,h)anthracene	109P	PCB-1232
ideno(1,2,3-cd)pyrene	110P	PCB-1248
pyrene	111P	PCB-1260
tetrachloroethylene	112P	PCB-1016
toluene	113P	toxaphene
trichloroethylene	129B	2,3,7,8-tetrachlorodibenzo-p-dioxin

it appears in "Sampling and Analysis Procedures for Screening of
ustrial Effluents for Priority Pollutants," EPA, Revised April,1977.

NAS9-15368

ACETONE

Introduction

MOLFM: C_3H_6O MW: 58.08 d_{25}^{25} 0.788

Water Solubility: infinite

SYN: dimethylketone, DMK, 2-propanone, B-ketopropane

Used as a solvent

Product of normal metabolism resulting in blood levels of 0.3-2.0 mg per 100 ml.

Literature: relatively sparse; most current listings of toxicity refer to older studies.

Metabolism

Acetone is fairly rapidly absorbed and distributed among the various tissues relative to their water content. Acetone is eliminated unchanged in the expired air and urine as well as metabolized. From equivalent blood levels (72 mg/l) in the rat and man, it was found that the rate of disappearance of acetone from the blood was 5.6 mg and 2.2 mg/ kg/hr. respectively.[1] This same study found that at very high blood levels of acetone, elimination as unchanged acetone was of more importance in rate of disappearance but a lower blood levels metabolism may account for more than half of the acetone lost. The metabolic pathways have been characterized primarily as a direct oxidation to a 3-carbon intermediate of glycolysis ("pyruvate" formation) and secondarily as acetoacetate and "formate" production.[2]

Acute Effects

Acetone appears to be relatively safe at fairly high concentrations. Oral doses of 15-20 g/day for several days by human subjects gave no sign of ill effects other than a slight drowsiness.[3] For rats, the median concentration of acetone in the jugular vein at loss of righting reflex was 3000 mg/l, at loss of corneal reflex, 5170 mg/l, and at respiratory failure 9190 mg/l.[1] An eight hour inhalation exposure to slightly over 2000 ppm daily would result in an accumulation of under 350 mg/l of acetone in the blood, which in turn would give no significant symptoms of exposure.[1,3] The single oral lethal dose for rabbits and dogs has been estimated as 8 g/kg.[4] For man the estimated minimum lethal oral dose in 50 ml[5] while 10-20 ml orally may be taken without ill effect.[6]

Chronic Effects

None reported in available literature.

Carcinogenicity/Teratogenicity/Embryotoxicity

None reported in available literature.

Current Standards

The current U.S. TLV is 1000 ppm.[7] No water standard is currently set for the U.S. One report from Russia recommends a standard of 39.6 mg/l for 3 months and 14.0 mg.l for 1 year in recycled drinking water.[8]

Recommendations

Acetone appears to be a relatively safe chemical even at very high levels. However, the lack of recent literature regarding chronic effects coupled with the conservative Russian standard suggests caution.

We recommend an upper limit of 50 mg/l acetone in drinking water.

BENZENE

Introduction

MOLFM: C_6H_6 MW: 78.12 d_4^{15} 0.8787

Water solubility: 0.8ppm

SYN: Benzol, coal naphtha, phenyl hydride

Used as a solvent and as a starting compound for other chemical compounds.

Of ten drinking water supplies surveyed by the EPA, four were found to have concentrations of 0.1-0.3 ug/l.[9a]

Literature: a large number of studies are available, several excellent reviews are available 10-16 which basically refer to the same body of literature and contain many similar discussions.

Metabolism

Benzene is readily absorbed by the lungs and once in the bloodstream is easily concentrated by various organs and tissues especially those that are lipophilic in character. The concentration in bone marrow many become 16-fold to 20-fold over that found in the blood. Benzene is metabolized in the liver primarily to phenol, catechol and quinol and secondarily to hydorxy quinol. Benzene may be excreted in the expired air as unchanged benzene or in the urine as unchanged benzene, free metabolites or metabolites conjungated with glucuronic or sulfuric acids. With the exception of the cat and the pig, 17 species follow this same general pattern of metabolism.

Benzene

Acute Effects

These effects are well documented and range from euphoria to loss of consciousness to death. These effects occur at concentrations much greater than will ever be found in recycled water and will not be discussed in detail at this time.

Chronic Effects

Chronic effects are harder to document. Most exposures are by the inhalation route and are thus hard to extrapolate to the oral route. In one study, mice exposed to 100 ppm benzene for 5 days showed a decrease in wheelturning activity as well as a definite degenerative change in the bone marrow whereas mice exposed to 10 ppm showed no change in wheelturning activity and only a slight degenerative change in bone marrow. Rats exposed to 20 ppm for 5.5 months showed a decreased response time to a conditioned reflex whereas rats exposed to 4 ppm showed no change. Rats exposed to 10 mg/kg orally for 132 days showed signs of leukopenia. An *in vitro* using cultured human leukocytes and 1.1×10^{-3} M benzene resulted in chromosomal gaps and breaks in a 72 hr. culture.

Chronic effects in humans are harder to relate to a definite exposure. Many studies include industrial exposure and concentrations are hard to document completely.

Mechanism of Action

Chronic benzene toxicity seems to be related to the levels and amounts of benzene metabolites in the test subject. It has been observed, however,

Benzene

that inducers of metabolism will reduce toxicity perhaps by resulting in earlier secretion before the benzene starts accumulating in the bone marrow. Inhibitors of metabolism at times will reduce toxicity. Three general hypotheses regarding chronic benzene toxicity are:

1. The phenolic intermediates may produce chromosomal aberrations or reduce mitotic rates.
2. Depletion of glutathione in the bone marrow may subsequently interfere with redox reactions leading to bone marrow depression.
3. The immune surveillance system may be affected allowing the proliferation of cell types normally destroyed.

Problems of Prior Studies

Some of the important points to consider in the evaluation of most of the studes are:

1. Liver metabolism is the primary site of study but the toxic action of interest occurs in the bone marrow.
2. Despite deplication of some of the human blood disorders in animals, there is at present no good animal model for the major toxic affect of interest.
3. There may be a strong interplay of unique genetic sensitivity and other chemical, physicial or viral agents.
4. Lack of mutagenicity in the Ames test.
5. Lack of embryotoxic and teratogenic data.

Benzene

Current Standards

The NIOSH (1974) recommended 8 hr. TWA is 10 ppm [17] No water standard has been recommend for the U.S.[9a]

Recommendations

Current fears that benzene may be a human carcinogen dictates that exposures be maintained at a relatively low level. We recommend an interum standard of 0.1 mg/l benzene in drinking water.

CARBON DISULFIDE

Introduction

MOLFM: CS_2 MW: 76.14 d_4^0 1.293

Water Solubility: 0.294%

Syn: carbon bisulfide, dithiocarbonic anhydride.

Used in various manufacturing processes including the rayon, rubber, solvent and pesticide industries.

Detected in 5 out of 10 drinking water supplies.[9b]

Literature: a large number of studies are available but these deal primarily with occupational studies in the viscous rayon industry or inhalation studies with test animals.

Metabolism

Following an 8 hour expusure, rat blood levels of CS_2 rapidly declined within 7 hours primarily by pulmonary excretion. Some of the inhaled CS_2 was metabolized to nonvolatile, S-containing compounds which showed prolonged retention with respect to free CS_2. CS_2 is postulated to react with endogenous amines or thiols to form dithiocarbamates or trichio-carbonates which may be responsible for subsequent tissue damage. One third of acid-labile CS_2 in brain tissue was still present 16 hours after exposure was stopped.[18] Other investigators have shown metabolism and excretion to CO_2.[19] and trace amounts of thiourea and thiazolidine.[20] A single oral dose of CS_2 inhibited MFO enzymes in rat liver microsomes. Other studies have confirmed depression of MFO activity and cytochrome P-450 levels. Cytochrome P-450 levels can be increased to similar levels both by phenobarbitone pretreatment and starvation, however, the CS_2 -

induced damage occurs only in the phenobarbitone-pretreated group suggesting that a MFO - Metabolite may not be responsible for the damage seen.[2]

Acute Effects

Acute exposure to CS_2 can result in euphoria, restlessness, nausea, vomiting, unconsciousness and terminal convulsions.

Chronic Effects

These effects are primarily the result of worker exposure in the viscous rayon industry.[28] This exposure may tend to stay stable at certain concentrations with intermittent peaks. However, various areas within a plant are often characterized by different levels allowing for relative comparisons, Many of the effects of concern deal with nervous system parameters including polyneuritis, difficulty in walking, vertigo, tremors, various psychoses, dimentia and decrease in nerve conduction velocity. There are also strong indications of increases in coronary heart disease and dysfunction of male and female reproductive systems. Animal data primarily supports or adds to observations made in humans. Other studies have indicated an increased tendency to hemolysis after exposure to CS_2 resulting from disruption of key enzymes in the glycolytic cycle and decrease in ATP levels,[22] continued diene conjugation of liver phospholipids after other parameters returned to normal values during subacute exposure,[23] potentiation by aminotriazole[24] and phenobarbitone with preferential binding of sulfur rather than carbon in the brain[25] and a "dying back" neuropathy similar to that induced by acrylamide.[26]

Carbon Disulfide

Carcinogenicity/Mutagenicity/Teratogenicity

Teratologic/embryotoxic effects have been observed in a least two studies.[96] A recent study[27] exposing pregnant Wistar rats to 50,100 and 200 mg/m^3 throughout gestation showed dose-related, detrimental effects on F_1 progeny. Mating within the F_1 progeny without further exposure to CS_2 resulted in similar structural abnormalities in the F_2 generation for the groups exposed to 100 and 200 mg/m^3.

Current Standards

The U.S. occupational standard is set at 3 mg/M^3,[28] and the Russian occupational standard is 10 mg/M^3.[96] However, it should be noted that another study of CS_2 exposure of 0.1 mg/M^3 for 6 months with rats showed a 16% increase in blood cholinesterase activity[29] and that workers exposed to 10 mg CS_2/M^3, the current Soviet maximum permissible concentration, developed a number of pathological conditions.[30] The National Research Council (1977)[96] has taken the position that not enough is known about CS_2 to set a water standard at this time.

Recommendations

Carbon disulfide is a difficult compound to evaluate. Most of the literature deals with inhalation studies and a large part deals with epidemiological data that is difficult to quantify accurately. The effects of CS_2 appear to be cumulative especially in the case of neuropathy indicating a need for conservative action. We recommend an interim standard of 0.05 mg/l carbon disulfide in drinking water, but suggest further investigations be started to resolve the problem of commulative effects.

CHLOROFORM

Introduction

MOLFM: $CHCl_3$ MW 119.37 d_{20}^{20} 1.484

Water solubility: 0.5%

Syn: trichloromethane

Used as a refrigerant, aerosol propellant, various manufacturing processes and in extraction procedures. Found in almost all finished drinking water supplies (mean concentration 20 μg/l) as a result of the chlorination process.[9c]

Literature: a large number of publications are available.

Metabolism

Chloroform is apparently rapidly absorbed, distributed and excreted in mice, rats and humans. A single oral dosing in man resulted in 40% expired unchanged and 50% expired as CO_2 within 8 hours.[31] Early literature showed that an unknown metabolite was responsible for observed affects.[32,33] This metabolite was later shown to depend upon a C-450 dependent oxidative pathway,[34,35] and to be phosgene.[36, 37]

Acute Effects

Acute effects are characterized by liver enlargement, hepatic and renal lesions and necrosis. These all occur at much higher concentrations than those observed in drinking water.

Chronic Effects

One clinical trial[38] for 1-5 years with an estimated daily oral dose of 0.3-0.96 mg/kg showed no signs of hepatotoxicity whereas another trial[39] with estimated daily oral doses of 23-37 mg/kg resulted in some reversible hepatoxicity. Several animal studies have shown chloroform to be a carcinogen.[40,41] Using dose-response techniques of extrapolation, the 95% upper confidence estimate of lifetime cancer risk per mg/l/day has been calculated as 1.7×10^{-6} and has been "remarkably consistent" from the individual data sets used.[9c] The NCI data sets have been confirmed using the same strains and similar doses.[42]

Carcinogenicity/Mutagenicity/Teratogenicity

A teratology study in rabbits showed fetotoxicity in terms of reduced birth weights at the higher doses but no evidence of teratogenicity looking at external, skeletal and/or soft tissue abnormalities.[43] However, this study does not evaluate the possibility of potential decreased learning ability in the offspring.

Interactions

Pretreatment with metabolic inducers such as phenobarbital and chloropromazine has been shown to potentiate glutathione decrease and liver necrosis following chloroform challenge[23] whereas metabolic inhibitors such as SKF 525A, piperonyl butoxide or CS_2 have been shown to antagonize subsequent chloroform challenge.[44] Chloroform induced hepatotoxicity in mice has been shown to have a strong genetic basis and to be potentiated by testosterone levels in both males and females.[45]

Chloroform

In Vitro Studies

Lack of mutagenicity was shown when cultured Chinese hamster lung fibroblast cells were exposed to 1-3% chloroform for one hour and the bottle sealed for a total of 24 hours.[46] However, it is questionable whether or not the same microsomal oxidation occurred in these lung cells that occurs in liver cells and is responsible for the production of the toxic metabolite.

Current Standards

A total haloform concentration of 70 μg/l has been referred to as "reasonable" in a report referencing both animal studies and epidemiological information.[47] The interim primary drinking water standard for total trihalomethanes has been set at 100 μg/l.[48] Due to its carcinogenic properties, chloroform was banned from human drugs or cosmetics in 1976.[49] A ceiling limit of 2ppm for 1 hr has been recommended for occupational exposures.[50]

Recommendations

The number of studies confirming the potential for chloroform-induced carcinogenesis, the "remarkably consistent" extrapolation of animal data to low doses, and the potential for potentiation by metabolic inducers, supports the need for a strict chloroform standard in drinking water. The Interim Primary Drinking Water Standard for total halomethanes of 0.1 mg/l seems reasonable at this time. Since chloroform represents the major portion of total halomethanes, we recommend a standard of 0.1 mg/l chloroform in drinking water.

DIOXANE

Introduction

MOLFM: $C_4\ H_8\ O_2$ MW;88.10 d^{20}_{4} 1.0329

Winter solubility: 00

Syn: 1,4 - dioxane, p-dioxane, diethylene dioxide

Used as a solvent in many applications

Literature: many published papers available

Metabolism

In an inhalation study of dioxane in human subjects, it was found that a concentration of 50 ppm for a six hour exposure period resulted in a total absored dose of 5.4 $\pm$ 1.1 mg/kg.[51] This study also revealed that dioxane has a half-life of 59 $\pm$ 7min. at 50 ppm exposure is excreted primarily as betahydroxyethxyacetic acid in the urine. This metabolite may also be referred to as p-dioxane -2-one as the two are interchangeable forms depending forms depending upon the pH of the solution.[52] Young, et. al.[51] concluded that 50 ppm in the air would produce no accumulation. The metabolic disposition of dioxane does show saturation[53] (thus no alternate pathway) and induction by metabolic inducers[52,54,55] or high doses of itself. There are indications that the toxic compound may actually be a further metabolite of p-dioxane-2-one.[55]

Acute Effects

Studies indicate that acute toxicity of dioxane is associated with necrosis of the kidney renal cortex and liver centrilobular area as well as edematous lungs and brain.[56]

Dioxane

Chronic Effects/Carcinogenicity

Many chronic studies have been done exposing different test species to different dioxane concentrations for different time periods.[57-60] Most of the observed effects occur at 1% or higher concentrations in drinking water and include increased number of tumors in nasal cavities, liver and kidney. In the liver and kidney, the tumors seem to have a strong association with signs of necrosis and tissue hyperplasia whereas a concentration of 0.01% (9.6 and 19.0 mg/kg/day for male and female rats respectively) for 2 years showed no effect. In general, the data support a dose-response relationship. Another study using detailed electron microscopy documents progressive tissue changes with total oral dose received over time.[61]

Mutagencity/Teratogenicity

None reported in available literature.

Current Standards

The current NIOSH air standard is 1 ppm.[56] There is no drinking water standard.

Recommendations

Although dioxane has carcinogenic potential, it seems to be strongly associated with prior tissue necrosis. This observation from several reports along with differences in pharmacodynamic parameters from high to low doses has lead Watanabe et. al.[62] to argue against extrapolation of dioxane data to very low doses.

We consider that 1 mg/l dioxane in drinking water will provide adequate protection from recognized hazard.

ETHANE, 1,2-DITHIOL

Introduction

MOLFM: $C_2H_6S_2$ MW 94.2 $d^{23.5}$ 1.123

Solubility: relatively insoluble in water

Syn: Dithioethyleneglycol, ethylenedimercaptan, dimethyl sulfide dimethyl disulfide

Literature: there is little available literature on the toxic effects of 1,2-dithiolethane.

Metabolism

No studies noted in available literature.

Acute Effects

The Merck Index[63] warns that vapors may cause severe headaches and nausea. The Registry of Toxic Effects[64] lists a single reference giving the subcutaneous LD50 in mice as 100 mg/kg.

Chronic Effects

No studies noted in available literature

Current Standards

None noted.

Recommendations

No data exist on which to establish a standard.

ETHANE, 1,1,1-TRICHLORO-

Introduction

MOLFM: $C_2 H_3 Cl_3$ MW: 133.40 d_4^{20} 1.3376

Solubility: relatively insoluble in water

Syn: Methyl Chloroform, TCEA

Used as a solvent.

Metabolism

An inhalation exposure to rats of 204 ppm for an 8 hr. day, 5-day week for 14 weeks showed that most of the 1,1,1-trichloroethane (TCEA) is excreted via the lungs as unchanged TCEA.[65] an ip. dose of 700 mg/kg resulted in 98.7% of the TCEA excreted unchanged via the lungs in 25 hours.[66] Another 0.5% was excreted as CO_2 while most of the rest was excreted as the glucuronide of 2,2,2-trichloroethane in the urine. The exact rate of elimination is in apparent dispute with one author arguing for first order uptake and elimination[67] and another for exponential decay with time.[68] Both seem to agree however, that there is a strong relationship between concentration of exposure and length of time of exposure with organ concentrations ten times higher for a total exposure administrated at a high dose and short time period compared to a low dose and a lengthy exposure. In general, there appears to be no accumulation in tissue. TCEA does appear to induce the mixed-function oxidase system.[69]

Acute Effects

TCEA acts as an anesthetic with only a slight capacity for liver damage although some liver stress is produced.[68] TCEA does not appear to enhance

Ethane, 1,1,1-Trichloro

triglyceride levels, increase diene conjugates or decrease glucose -6-phosphatase activity.[67] TCEA seems to act on the electron transport chain[70] and results in peripheral vasodilation followed by depression of myocardial function. Acute exposure also potentiates epinephrine-induced cardiac arrythmia.[71]

Chronic Effects

Exposure of dogs, mice, rats and monkeys continuously for 90 days at 250 ppm produced no significant deleterious findings.[72] Workers exposed to an estimated TWA of 115 ppm TCEA 8 hrs/day, 5 days/week for up to five years showed no adverse effects.[73]

Mutagenicity/Carcinogenicity

No mutagenic effects have been noted using the Ames test system with and without induced rat liver microsomes.[75] However, these same authors failed to show effects with carbon tetrachloride and methylene dichloride. A recent carcinogenesis assay[76] failed to show carcinogenic activity in both mice and rats as the controls had similar neoplasms and the treated animals had abbreviated life spans. In contrast, TCEA wa shown to affect transforming acitvity of Fisher rat embryo cells as measured by relative plating efficiency at a concentration as low as 9.9x10-1 μm thus showing more activity than trichloroethylene.[77]

Current Standards

The recommended NIOSH standard has a TWA of 350 ppm.[74]

Ethane, 1,1,1-Trichloro

Recommendations

Most studies of 1,1,1-trichloroethane show it to be a relatively inoccuous compound even when inhaled at very high levels. However, at this time there do not appear to be any good ingestion studies upon which to base a drinking water standard. We recommend an interim standard of 1 mg/kg TCEA in drinking water.

FURAN, 2-METHYL-

Introduction

MOLFM: $C_5 H_6 O$ MW: 82.11 d_4^{20} 0.9132

Solubility: relatively insoluble in water

Syn: Silvan

3-methyl furan has been found in rainwater samples during an atmospheric smog alert.[78]

Literature: very little literature was noted on 2-methyl furan but there are many articles on furan analogues that suggest that the furan ring is of primary importance in their toxicity. These generalities are discussed below.

Metabolism

Most studies suggest metabolic activation of the furan ring possibly by epoxidation followed by covalent binding to a target site. This activity requires molecular oxygen and a NADPH generating system.[79-84] The damage has been prevented by piperonyl butoxide administration.[82]

Effects of Furan-Containing Analogues

The oral LD 50 and inhalation LD low for 2-methyl furan in the rat have been reported as 167 mg/kg and 377 ppm/4H respectively.[64] The double bond in the terminal furan ring of aflatoxins and analogs was found to be important to the potency of these compounds in both acute and chronic effects.[21,83] It has been found that 0.23 mmol/liter air for 1 hr. or 100-200 mg/kg ip. dosages of 3-methyl furan could result in damage to lung bronchiolar cells[82] or more specifically, the Clara cells.[80] Studies with 2-(N-ethyl carbamoylhydroxymethyl) furan injected ip. showed damage primarily to the lung and secondarily to the liver.[34]

Fu:an, 2-Methyl-

This damage was associated with covalent binding via the furan ring. Studies of furosemide metabolism, distribution and reversible plasma protein binding following toxic and nontoxic doses suggests the reasonableness of a threshold due to required saturation of anion binding sites on plasma proteins following toxic doses thus making more free furosemide available for metabolic conversion.[79] These general effects were found with several simple furan compounds and furan itself. The study by Guengerich[84] also noted threshold effects but at the same time found that doses nontoxic to rats could produce mutations in _S. typhinurium_. Several authors have pointed out the carcinogenic/mutagenic potential of furan compounds due to their alkylating abilities.[79,82-84]

Current Standards

None found.

Recommendations

No data exist on which to establish a standard.

HEXANE

Introduction

MOLFM: C_6H_{14} MW: 86.17 d_4^{20} 0.660

Solubility: relatively insoluble in water

Syn: N-hexane

used as a solvent

Metabolism

In guinea pigs injected ip with 132 mg/kg of hexane, the peak blood concentration of 20 μg/l was reached in 30 min.[85] There appeared to be a phase of rapid elimination with a half-life of 36 min. followed by a phase of slower elimination with a half-life of 4 hours. The metabolite 2-hexanol was found in groups treated with either hexane or MBK whereas the metabolite ,25-hexanedione was found only in the MBK-treated group. However, in vitro studies yielded both metabolites from both solvents.

Acute Effects

No acute effects were seen after exposure to 2000 ppm for 10 min. whereas a dizziness resulted from exposure to 5000 ppm.[5] The LDLo for ip injection in rats is reported to be 9100 mg/kg and the LCLo for inhalation exposure in mice is reported as 120 g/m3.[64]

Chronic Effects

Hexane is apparently capable of producing a neuropathy characterized as an axonal disease in which distal portions of sensory and motor axons undergo a progressive retrograde degeneration with abnormally large accumulations of 10 mm neurofilaments and focal axonal swelling with thinning of the myelin.[86] It has been suggested that MBK and n-hexane are biotransformed to the same metabolite(s) which may provide a common basis for their neurotoxic action.[87] As noted in the section on metabolism, Couri, et. al.[25] showed that there are two common metabolites for both MBK and hexane.

Carcinogenicity/mutagenicity/teratogenecity

No reference in the available literature regarding these effects.

Current Standards

The OSHA standard set in 1974 gave a TWA of 500 ppm[88] but a later criteria document for occupational exposure to alkanes recommends a TWA of 350 mg/m^3.[3] No water quality standards exist for hexane.

Recommendations

The neuropathic effects of hexane suggest a conservative standard. We consider that 1 mg/l hexane in water would provide adequate protection.

METHANE, DICHLORO

Introduction

MOLFM: CH_2 Cl_2 MW 84.94 d_4^{20} 1.3255

Solubility: relatively soluble in water

Syn: methylene chloride, methylene dichloride (-bichloride)

Found in finished water supplies as a result of the chlorination process[9d]

Literature: Many reports are available but most deal with vapor exposure.

Metabolism

Methylene chloride has been found to be eliminated from the body primarily unchanged in expired air within about two hours.[89, 90] Approximately 2% was considered to be left in the liver, kidney and adrenal glands after 24 hrs. by DiVincenzo and Hamilton[89] whereas Carlsson and Hukten[90] characterized the residual as being primarily in white adipose tissue. Both papers report minor CO production. DiVincenzo and Hamilton report that after 24 hours, 91.5% of methylene chloride was expired unchanged, 2% as CO, 3% as CO_2, 1.5% uncharacterized and 1% in the urine. This same report mentions increases in serum formaldehyde related to a decrease in liver formaldehyde but states that it must be a physiological induction and not a metabolite but this was later disputed.[91] Apparently methylene chloride is not inducible by itself or with phenobarbital[91] or 3-MAC[92] but does require NADPH and molecular O_2.[93]

Methane, Dichloro

Acute Effects

An acute oral (low) for humans has been reported to be 500 mg/kg,[5] for rats (LD50, oral) 1.6-2.3 ml/kg.[94] The intraperitoneal LD50 for mice and dogs are given as 1.50 ml/kg and 0.95 ml/kg respectively.[95]

Chronic Effects

Rats given drinking water containing 125 μg/l for 91 days showed no adverse effects.[96] Inhalation exposures for 6 months at a concentration of 5,000 ppm produced no effects in dogs and rabbits.[9d] CNS effects have been seen in humans at exposures over 300 ppm[97] and seen to be one of the more sensitive parameters of exposure. Most of the CNS effects appear to be readily reversible upon removal from exposure. These CNS effects may be strongly related to CO production from metabolism of methylene chloride. There is some concern that methylene chloride may prolong the half-life of CO[98] but this has been recently disputed.[99] However, the potential for additive effects between dichloromethane and CO is sufficiently strong that NIOSH has set a TWA of 75 ppm which must be lowered if exposure to CO also occurs.[97]

Carcinogenicity

Using strain A mice and the pulmonary tumor induction technique, dichloromethane gave elevated though nonsignificant tumor numbers.[100] The authors felt that the nonsignificance may be due to low numbers of test animals and that further testing should be done.

Methane, Dichloro

Teratogenic/Embryotoxic Action

Mice and rats exposed to 1225 ppm dichloromethane for 7 hours daily; days 6-15 of gestation gave no signs of maternal, embryonal of fetal toxicity as well as no signs of teratogenicity.[101]

Mutagenicity

Negative results were obtained in a *Drosophila* test[102] but transforming activity has been shown in a system using Fisher rat embryo cells at concentrations as low as 1.6×10^2 μm [103]

Current Standards

The current NIOSH standard is a TWA of 75 ppm.

No drinking water standards exist.

Recommendations

We consider that a standard 1.0 mg/l methylene chloride in drinking water should provide adequate protection to exposed populations.

METHANE, DICHLORODIFLUORO-

Introduction

MOLFM: $CCl_2 F_2$ MU 120.91 $d_{liq.}^{-29.8}$ 1.486

Solubility: very soluble in water

Syn: Fluorocarbon 12, Freon-12, Propellant-12, Refrigerant 12, Arcton 6, Halon

Used as a refrigerant or aerosol propellant

Literature: a large number of reports are available nut most deal with inhalation exposures

Metabolism

A study using a fixed volume of inhaled, labeled dichlorodifluoromethane over a 7-17 min. period found less than 0.2% of the administered dose in the urine or exhaled as CO_2.[104] Similar results were noted in dogs.[105] Dichlorodifluoromethane has a blood-gas partition coefficient of 0.2 which helps to explain its rapid elimination via expired air. Another study found F12 in the cerebrospinal fluid one minute after start of inhalation but all was essentially gone 20-25 min post exposure.[106] Another study found less than 1% of the administrated dose in tissues 24 hours. after exposure.[107] The addition of fluorine atoms to the dichloromethane molecule greatly stabilizes the molecule and accounts for its slight metabolism.

Acute Effects

There has been some concern with the sudden death that occurs in youths breathing dichlorodifluoromethane. At a tissue bath concentration of 1.06 mg/100 ml, F12 produced a 20% depression in amount and rate of isometric force development in heart Atria.[108] Another report states that cardiac output was decreased by 10-20% FC 12 after 12-30 min.[109] Some studies have reported ECG changes while others have not.[110] The possibility of interaction with other drugs in FC 12 sniffing youths should be considered more thoroughly.

Chronic Studies

An inhalation study exposing test species to 3997 mg/m^3 continuously for 90 days resulted in the death of 2/15 guinea pigs, 0/3 rabbits, 0/2 dogs and 0/2 monkeys.[111] Varying degrees of lung congestion and fatty infiltration of the liver were noted. Both male and female rats exposed *in utero* were also exposed for two years by intragastic intubation at doses of 11-25 mg/kg and 130-256 mg/kg.[112] There were essentially no clinical signs of toxicity or teratogenicity, nor was reproduction or lactation interfered with. The dominant lethal mutation index was not affected. Dogs fed for 2 years on diets resulting in exposures of 10 mg/kg and 100 mg/kg showed no signs of toxicity. Rats, mice and dogs exposed to 164-2240 mg/kg/day for 2 weeks to 23 months showed signs of sedation, ataxia and mild depression only at the highest doses which would disappear rapidly after ending exposure. Gross and microscopic examination gave no signs of toxicity.

Current Standards

Dichlorodifluoromethane has an aquatic toxicity rating of >1000 ppm and a NIOSH standard TWA of 1000 ppm.[64]

Recommendations:

We consider that a standard of 1 mg/l dichlorodifluoromethane in drinking water will provide adequate protection from recognized hazards.

METHANE, IODO-

Introduction

MOLFM: CH_3I MW: 141.94 d_4^{20} 2.28

Solubility: slightly soluble in water

Syn: Methyl iodide

Used as a methylating reagent in the chemical industry.

Literature: Small number of articles available but oral administration is covered.

Metabolism

An inhalation study in humans has shown that very little iodomethane is excreted via the lungs once it has entered the blood-stream.[113] A study of oral dosing in rats found that of a 50-75 mg/kg dose, 40-50% has apparently reacted with GSH within 1.5 hrs. and 22-28% was excreted in the bile within 6 hrs.[114] Smaller amounts of methyl mercapturic acid, methylthioacetic, N-(methylthioacetyl) glycine and S-methyl-L-cysteine have been found in the urine and may be the products of kidney metabolism of any S-methyl glutathione that reaches them.[115]

Acute Effects

Acute effects are associated with congestive changes in lungs, oliguric renal failure, prominent cerebrellar and Parkinsonian neurologia symptoms as well as seizures and coma.[116] Psychiatric disturbances may occur for months to years. Although there has been a fair number of poisoning cases by other monohalide methanes, the low usage of iodomethane has resulted in very few cases of acute poisoning in humans. An oral dose of 75 mg/kg is sufficient to produce an LD50 in rats[114] whereas an oral dose of 0.35 mmoles/kg was found sufficient to reduce liver glutathione

Methane, Iodo-

levels to 58% of control values.[117] Experimental reduction of liver glutathione levels can reduce the excretion of several compounds,[118,119] and potentiate the toxicity of dimethyl phosphorothionates.[120]

Chronic Effects

As reported earlier, an oral dose of 75 mg/kg is sufficient to produce an LD50 in rats,[114] yet in the same study, it was reported that an oral dosing of 30-50 mg/kg, 5 days/week for a month produced no signs of toxicity. This was attributed to the rapid turnover of rat glutathione ($t_{1/2}$ = 3-4 hrs.) thus the animals had sufficient time to overcome accumulative effects.

Carcinogenicity

A 24 week study involving 24 ip injections for a total of 0.06 mmoles iodomethane/kg resulted in a survival rate of 19/20 mice with a nonsignificant increase in lung tumors whereas a total does of 0.31 mmoles resulted in a $p<0.05$ significant increase in mortality.[121] Another study dosing with 50 mg/kg in a single subcutaneous injection or in 10 mg/kg weekly doses resulted in massive local sarcomas.[122] Another report states that subcutaneous injections will produce local sarcomas but that none were seen by intravenous or oral routes of administration in rats.[123]

Current Standards

The current OSHA air standard is a TWA OF 5 ppm.[124]

Recommendations

We consider that a standard of 1 mg/l iodo-methane in drinking water will provide adequare protection from recognized hazards.

PROPIONIC ACID, METHYL ESTER

Introduction

MOLFM: $C_4 H_8 O_2$ MW 88.12 d_4^{20} 0.9151

Water solubility: relatively soluble

Syn: (Propanoic acid, methyl ester), methyl propionate

Literature: essentially no literature appears to be available regarding physiological actions.

Metabolism

No studies reported in the available literature

Acute Effects

The LDLo for an oral dose in rabbits is given as 2550 mg/kg.[64]

Chronic Effects

No studies reported in the available literature.

Current Standards

None noted.

Recommendations

It is impossible to recommend a standard on this substances without further documentation.

TOLUENE

Introduction

MOLFM: C_2 H_8 MW;92.13 D_4^{20} 0.8669

Water solubility: relatively insoluble

Syn: Methylbenzene, phenylmethane

Used as solvent.

Found at concentrations up to 11 µg/L in New Orleans are a drinking water.[9e]

Literature: a large volume of literature is available.

Metabolism

It is estimated that from 75-90% of the absorbed toluene is excreted via the urine as metabolized products and the rest excreted via the lungs as unchanged toluene.[125-127] The general sequence of metabolism is toluene → benzyl alcohol → benzaldehyde → benzoic acid followed by conjugation primarily with glycine giving hippuric acid and a smaller amount with glucuronic acid giving benzoylglucuronic acid.[130] After oral administration to rats, maximum levels were reached in most tissues within 2-3 hours with the exception of white adipose tissue where maximum levels were reached 5 hrs. following dosing.[139] This same study found that maximum levels were reached in most tissues 15-30 min. following a brief inhalation exposure. During the first 9 hrs. following exposure, toluene was more rapidly eliminated following inhalation exposure but tissue levels were essentially the same by 12 hrs. post exposure. Elimination was much slower from white adipose tissue and bone marrow concentrations only showed a slight decrease in 24 hrs. These general trends have been found in other studies[132, 133]

Toluene

Acute Effects

Acute effects can range from irritability and deminished psychomoter performance to disorientation and unconsciousness.[134] There have also been reports of "sudden sniffing death" perhaps associated with an increased sensitivity of the mammalian heart to epinephrine.[135] The calculated LC50 for inhalation exposure using rats is 8800 ppm.[136] References are cited for a LDLo in a human oral exposure of 50 mg/kg and an oral LD50 in rats of 5000 mg/kg in the Registry of Toxic Effects of Chemical Substances.[64]

Chronic Effects

Mice exposed for 3 hr/day, 5 days/week, for 8 weeks at 4000 ppm showed signs of intoxication but no mortality nor was there evidence of damage to lungs, liver or kidneys.[139] No significant effect was seen in rats, guinea pigs, dogs or monkeys exposed to 1085 ppm toluene for 30 sessions.[138] Rats and beagles exposed from 240 to 980 ppm for 6 hrs./day for 13 weeks showed no significant changes in hematology, clinical chemistry or micropathology.[136] In workers exposed to an average of 200 ppm for 3-15 years, a slightly higher but nonsignificant occurrence of unstable chromosomal abberations was noted.[139]

Carcinogenicity

Toluene was reported to be an enhancing agent for skin carcinogenesis but this may be the result of a general irritancy effect with hyperplasia following. No references to systemic carcinogenicity were noted by several recent review authors.[9e, 140, 141] No references to mutagenicity, teratogenicity or embryotoxicity are noted at this time.

Current Standards

NIOSH currently recommends an 8-hrs. TWA of 100 ppm with a 200 ppm C.[134] No recommendation for a water standard was made in a recent review.[9e]

Recommendations

We consider that a standard of 1 mg/l toluene in drinking water will provide adequate protection from recognized hazards.

XYLENE

Introduction

MOLFM: $C_8 H_{10}$ MW: 106.16 d^{20}- 0.86

Water solubility: relatively insoluble

Syn: dimethyl benzene, xylol

Exists in three isomeric forms (ortho, meta, para); most products are composed primarily of the meta isomer.

Used as a solvent.

Found as high as 4.1/μg/liter in finished water of the New Orleans area.[9f]

Metabolism

Most of the aromatic solvents become absorbed onto red blood cell membranes and plasma lipoproteins and are then transported to other tissues where they accumulate in proportion to the fat content of the tissue. Unchanged aromatic solvents are expired in proportion to their vapour pressure and blood concentration. Xylene's low vapor pressure results in relatively little of the absorbed dosage being exhaled as unchanged xylene. In man, it has been estimated that 72% of the absorbed m-xylene was excreted as m-methyl hippuric acid within 18 hrs.[143] Another report gives 90.1% of the absorbed xylene as being metabolized.[142] This latter report studies the kinetics of excretion of ingested m-methyl hippuric acid and found the same rate of excretion, followed by conjugation with glycine, thus concluding that the metabolism of xylene occurred very rapidly. A study in rats showed that phenolic metabolites accounted for $\leq$ 1% of a 100 mg/kg dose.[144]

Acute Effects

Acute effects include a narcotic-type action and muscular weakness. The oral LD50 in rats is reported as 4.3 g/kg;[145] the ip. LD50 in mice as 1.5 g/kg;[144] the inhalation LC50 in rats for a 4 hr. exposure as 6700 ppm.[146] One study giving 1 g/kg by either stomach intubation or ip. injection found a 65-70% reduction in -xylene hydroxylase microsomal activity in the lungs.[147] It was speculated that an unidentified, NADPH-requiring interaction between p-tolualdehyde and the endoplasmic reticulum was taking place. It has been estimated that the LDLO for the oral route in humans is 50 mg/kg.[15]

Chronic Effects

In beagles and rats exposed 6 hr/day, 5 days/week for 13 weeks to 180-810 ppm xylene levels, no significant changes were found when blood, urine, body weight or microscopic tissue parameters were examined.[146] No significant effects were found in the hematopoietic system of experimental animals doses up to 700 mg/kg/day for 9 weeks.[148] In rats and rabbits dosed with 690 ppm for 8 hr./day, 6 days/week for 130 days there were no significant deviations in peripheral blood parameters.[149] Another study found no significant changes in hematologic parameters in rats, dogs, guinea pigs or monkeys.[150]

Carcinogenicity

Xylene application to the skin can increase the tumour yield of subsequent urethane application,[151] but these effects may be related to the general irritancy and subsequent skin hyperplasia seen with several solvents.[152] This latter review found no mention of systemic carcinogenicity by xylene.

Mutagenicity

No reports of mutagenicity were noted in two recent reviews.[9f, 152]

Teratogenicity

When tested as a mixture with an emulsifier, xylene showed no embryotoxic or fetotoxic effects. However, there is a report that xylene may produce developmental defects in chicken embryos.[153]

Present Standards

The current NIOSH standard for an 8 hour TWA is 100 ppm with a ceiling of 200 ppm.[154] However, the authors of this report felt that sufficient epidemiological studies had not been performed to adequately support this limit but there were several experimental studies that suggested that it may be adequate. A recent study committee did not recommend a water quality standard without further research into chronic effects of low level exposure.[9f]

Recommendations:

We consider that a standard of 1 mg/l xylene in drinking water will provide adequate protection from recognized hazards.

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NAS9-15368

APPENDIX IV

TABLES I THRU VI

FIGURES 1 THRU 4

SCHEDULE FOR ESTABLISHING POTABLE
REUSE WATER QUALITY CRITERIA FOR
MANNED SPACEFLIGHT

TABLE I

VCD CONDENSATE

VOLATILE ORGANIC ANALYSIS SAMPLES

Date ollected	Time Collected	Date Analyzed	Sample History
) Mar 78	0900	21 Jun 78	4°C(22d);Iodated;23°C(10d);4°C(82d)
) Mar 78	1500	22 Jun 78	4°C(105d)
l Mar 78	1000	1 Apr 78	4°C(18d)
3 Mar 78	1400	22 Jun 78	4°C(19d); Iodated;23°C(83d)
ł Mar 78	1400	1 Apr 78	4°C(18d)
ł Mar 78	1400	10 Apr 78	4°C(18d); Iodated;23°C(10d)
; Mar 78*	0900	21 Jun 78	4°C(99d)
; Mar 78	1054	1 Apr 78	4°C(16d)
; Mar 78*	1100	21 Jun 78	4°C(98d)
; Mar 78	1500	1 Apr 78	4°C(16d)
' Mar 78	0900	1 Apr 78	4°C(15d)
' Mar 78	0900	10 Apr 78	4°C(15d);Iodated; 23°C(10d)
ı Mar 78	1100	1 Apr 78	4°C(12d)
ı Mar 78	1100	10 Apr 78	4°C(12d);Iodated; 23°C(10d)
ı Mar 78	1400	21 Jun 78	4°C(12d);Iodated; 23°C(10d); 4°C(72d)

ıTE: Distilled water from NASA-JSC was used as an analytical blank for the non-iodated VCD samples. This water was iodated to serve as a blank for the iodated VCD samples.

:arbon balance samples
xxd) = days at indicated temperature

TABLE II

VCD CONDENSATE SEMI-VOLATILE

ANALYSIS SAMPLES

Date Collected	Time Collected		Date Extracted	Sample History
8 Mar 78 9 Mar 78	1500 1100	Composite #1	1 Apr 78	4^{o}C(24d)
13 Mar 78 14 Mar 78	1400 1100	Composite #2	1 Apr 78	4^{o}C(19d)
15 Mar 78*	0900		20 Jun 78	4^{o}C(99d)
16 Mar 78*	1100		20 Jun 78	4^{o}C(98d)
15 Mar 78 16 Mar 78	0900 1000	Composite #3	1 Apr 78 1 Apr 78	4^{o}C(17d)

TABLE III

PRECISION COMPARISON FOR TWO CONCENTRATORS

	% Relative Standard Deviation (1σ) *					
	Spectrix Unit (1)			Tekmar Unit (2)		
	2 μg/l	8 μg/l	80 μg/l	2 μg/l	10 μg/l	20 μg/l
trans-1-2,dichloroethylene	12	9.7	5.3	N/A	N/A	N/A
carbon tetrachloride	2.5	13	3.0	4.3	18	2.5
cis-1,2-dichloroethylene	7.7	11	4.4	N/A	N/A	N/A
chloroform	2.3	4.1	4.4	27	8.2	0.1
1,1,1-trichloroethane	1.4	4.3	4.2	N/A	N/A	N/A
bromodichloromethane	1.4	5.0	5.2	8.8	9.2	2.2
tetrachloroethylene	9.0	30	4.1	3.2	21	9.3
1,2-dichloroethane	1.3	5.1	2.4	3.9	6.3	2.1
chlorodibromomethane	14	14	4.6	10	8.9	2.2
trichloroethylene	N/A	N/A	N/A	22	23	1.5
1,1,1-trichloroethane	1.0	7.4	1.2	9.1	17	4.8
bromoform	20	32	5.7	17	9.4	5.7
p-dichlorobenzene	29	23	N/A	N/A	N/A	N/A

(1) Data obtained with 25 ml samples using a GC-MS detector.

(2) Data obtained with 10 ml samples using GC-MCD.

*All values obtained from five replicate analyses

N/A - No data available

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TABLE IV
Volatile Organic Analysis Results

DATE	TIME	REMARKS See Tables I & II	Dichlorodi-fluoromethane	Dichloromethane *	Acetone	Methyl Iodide	Chloroform *	Carbon disulfide	1,4-dioxane*	1,1,1-trichloroethane	Methyl acrylate *
			Concentration in µg/L (PPB)								
3-10-78	0900	4°C after iodation	5.9	38		1.9			T		86
3-10-78	1500	long storage	1.5	22	2.4			3.3	8.8		25
3-11-78	1000	short storage		15			1.1	1	6	T	T
3-13-78	1400	4°C after iodation		6.8	2.3			T	14		
3-14-78	1400	short storage		2.1			0.9			1.6	T
3-14-78	1400	iodated		2.4			0.5		T	1.4	T
3-15-78	0900	long storage	6.3	29		5.1			T		250
3-16-78	1100	long storage	6.2	17		T	T		7.2		
3-16-78	1500	short storage							28		
3-17-78	0900	short storage		20	10		1.6		16	1.6	42
3-17-78	0900	iodated		8	11		1.5		15	1.5	40
3-20-78	1100	short storage							16		
3-20-78	1100	iodated		T					14		T
3-20-78	1400	4°C after iodation	3.5	15		T	T		7.0		
3-31-78		blank									
4-1-78		iodated blank									
		Detection Limits	0.4	0.4	TBD	TBD	0.1	TBD	TBD	0.4	TBD

*Quantitated from relative response factors / T = 0.1 - 1.0 µg/L TBD = to be determined but <10

Blank indicates not detected at the indicated detection limits

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TABLE IV (continued)

DATE	TIME	REMARKS See Tables I & II	Concentration in µg/L (PPB) Benzene *	Hexane *	Toluene *	Xylene	Dimethyl-disulfide	2-methyl furan			
3-10-78	0900	4° after iodation	1.0		3.8	7.0	T				
3-10-78	1500	long storage	1.3	4.4	1.1	5.5	T				
3-11-78	1000	short storage		T			170	T			
3-13-78	1400	$4^{\circ}C$ after iodation			T		T				
3-14-78	1400	short storage					70				
3-14-78	1400	iodated					T				
3-15-78	0900	long storage		11	4.4		31				
3-16-78	1100	long storage	1.2		2.4						
3-16-78	1500	short storage					9				
3-17-78	0900	short storage					176	T			
3-17-78	0900	iodated					T				
3-20-78	1100	short storage					19				
3-20-78	1100	iodated					17				
3-20-78	1400	$4^{\circ}C$ after iodation	1.3	3.1	5.2	10					
3-31-78		blank									
4-1-78		iodated blank									
		Detection Limits	0.4	TBD	0.1	TBD	TBD	TBD			

* Quantitated from relative response factors / T = 0.1 - 1.0 µg/L TBD = To be determined but <10
Blank indicates not detected at indicated detection limits

TABLE V

RESULTS OF SEMIVOLATILE EXTRACT ANALYSES

Sample I.D.	Component Identified	Quantity (µg/L = PPB)
3/8 - 3/9 Composite	dimethyl phthalate	0.9
	diethyl phthalate	3.7
	di-n-butyl phthalate	2.0
	butyl benzyl phthalate	3.4
	bis(2-ethylhexyl) phthalate	14
3/13 - 3/14 Composite	dimethyl phthalate	1.4
	diethyl phthalate	3.1
	di-n-butyl phthalate	17
	butyl benzyl phthalate	2.0
	bis(2-ethylhexyl) phthalate	15
3/15 - 3/16 Composite	diethyl phthalate	4.7
	di-n-butyl phthalate	26
	butyl benzyl phthalate	2.0
	bis(2-ethylhexyl) phthalate	28

TABLE VI

SUGGESTED RECLAIMED POTABLE WATER QUALITY SPECIFICATION

APPEARANCE

Color - 5 on cobalt scale

Total solids - less than 500 ppm

PALATABILITY

Dissolved gases

CO_2: 1-5 ppm

O_2: 1-5 ppm

Chemical Constituents - Inorganic

Ca^{++}:	20±5 ppm	Cl^-:	30±10 ppm
Mg^{++}, Na^+:	10±2 ppm	$SO_4^=$, NO_3^-:	40±10 ppm
K^+:	2±1 ppm	HCO_3^-, $CO_3^=$:	10±5 ppm

Chemical Constituents - Organic

Nonvolatile

Total Organic Carbon (TOC):	<1 ppm
Dissolved Organic Carbon (DOC):	<1 ppm
Particulate Organic Carbon (POC):	<1 ppm

Volatile Compounds

Component	Upper Limit (mg/L or ppm)
Acetone	50
Benzene	0.1
Carbon disulfide	0.05
Chloroform	0.1
Dioxane	1.0
Ethane, 1,1,1-trichloro	1.0
Hexane	1.0
Dichloromethane	0.1
Dichlorodifluoromethane	1.0
Methyl iodide	1.0
Toluene	1.0
Xylene	1.0
1,2-Dithiolethane	TBD
2-Methyl furan	TBD
Methyl acrylate	TBD

C-2

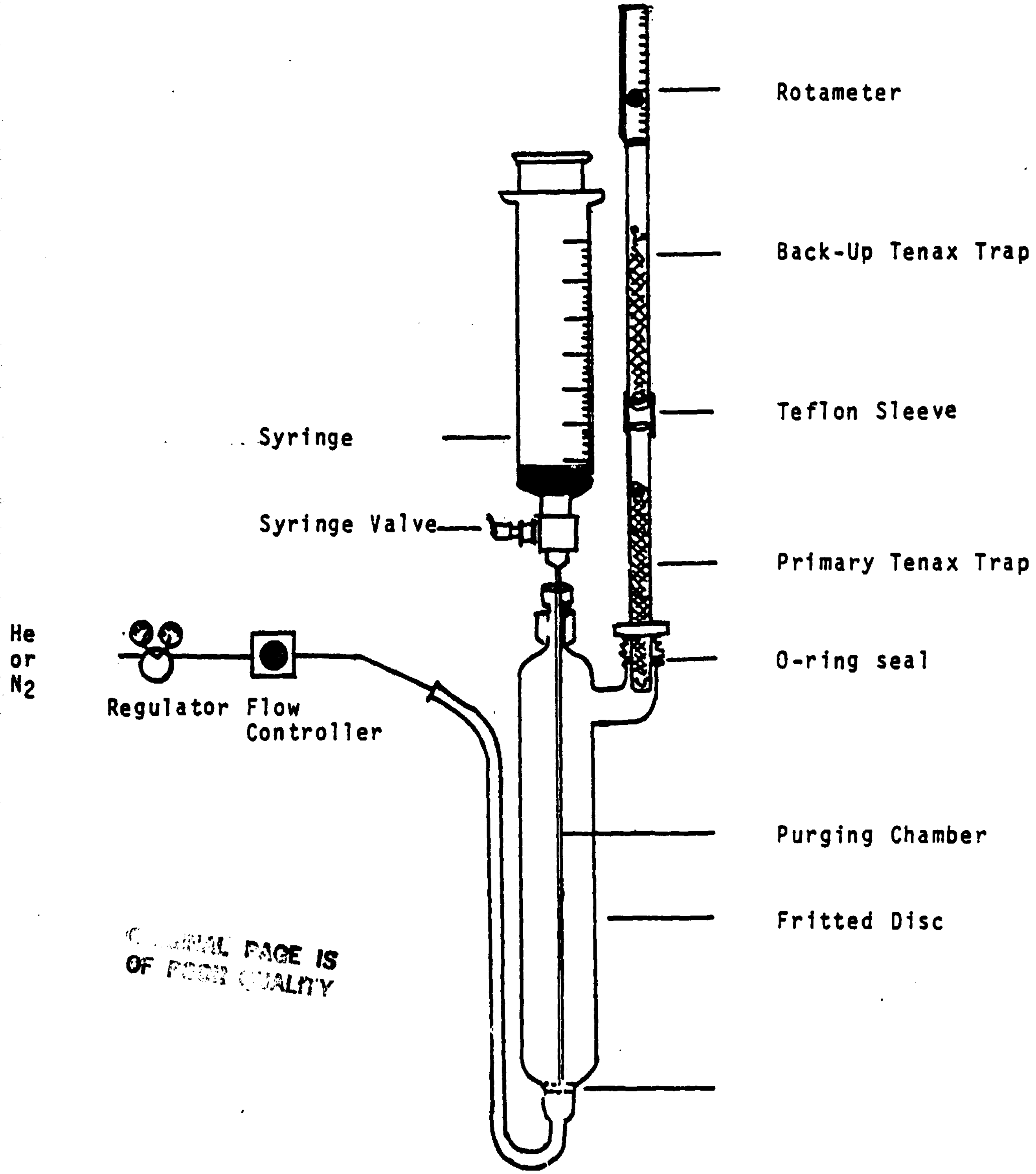

FIGURE 1

SCHEMATIC OF PURGING APPARATUS

SPECTRIX CORPORATION

7408 FANNIN HOUSTON, TEXAS 77054 (713) 790-1600

04/01/78 14:21:00
SAMPLE: 3/17/78 @ 0900 VCD COND
RANGE: G 1, 800 LABEL: N 0, 4.0 QUAN: A 0, 1.0 BASE: U 20, 3

CALI: C060878 #3

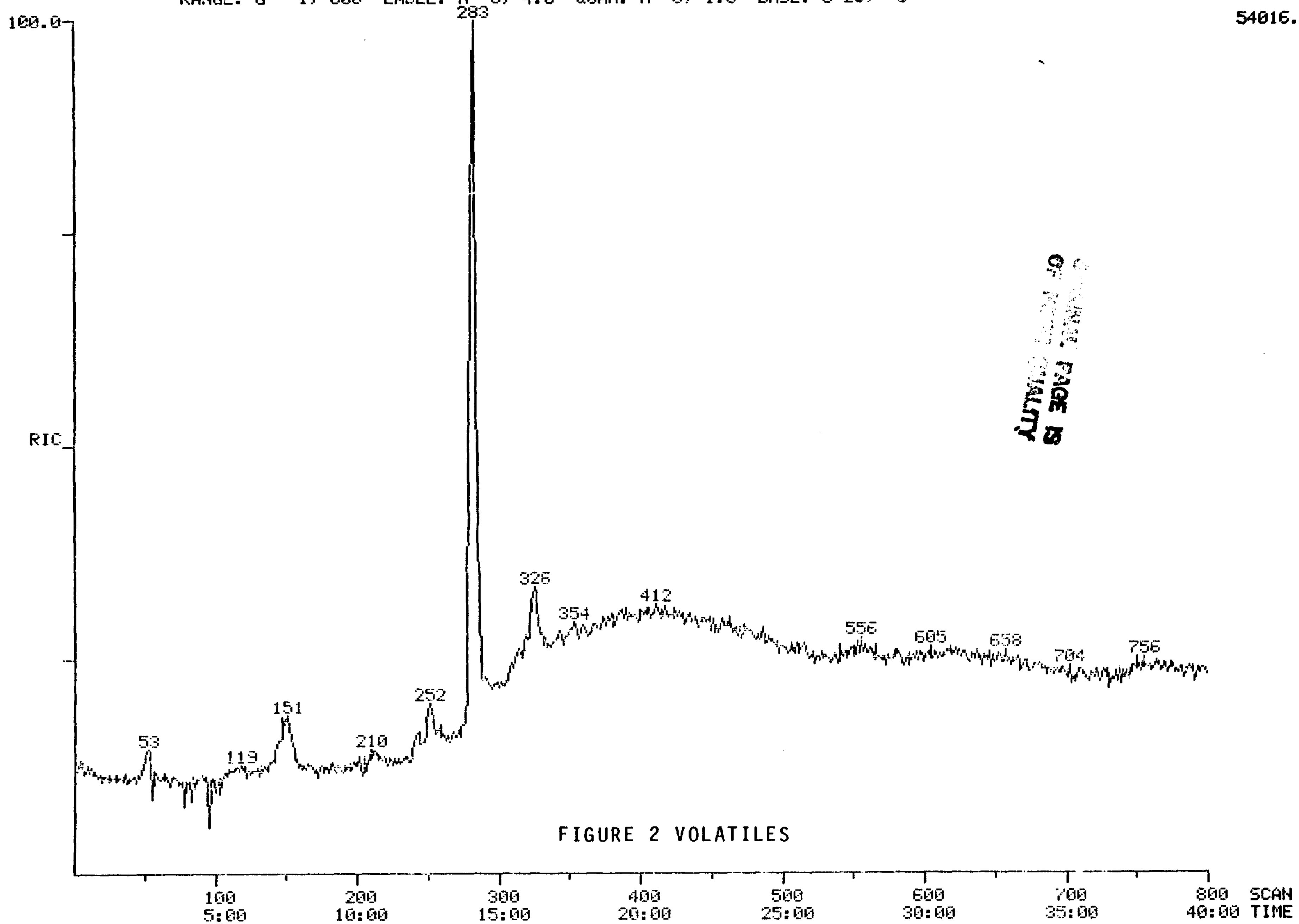

FIGURE 2 VOLATILES

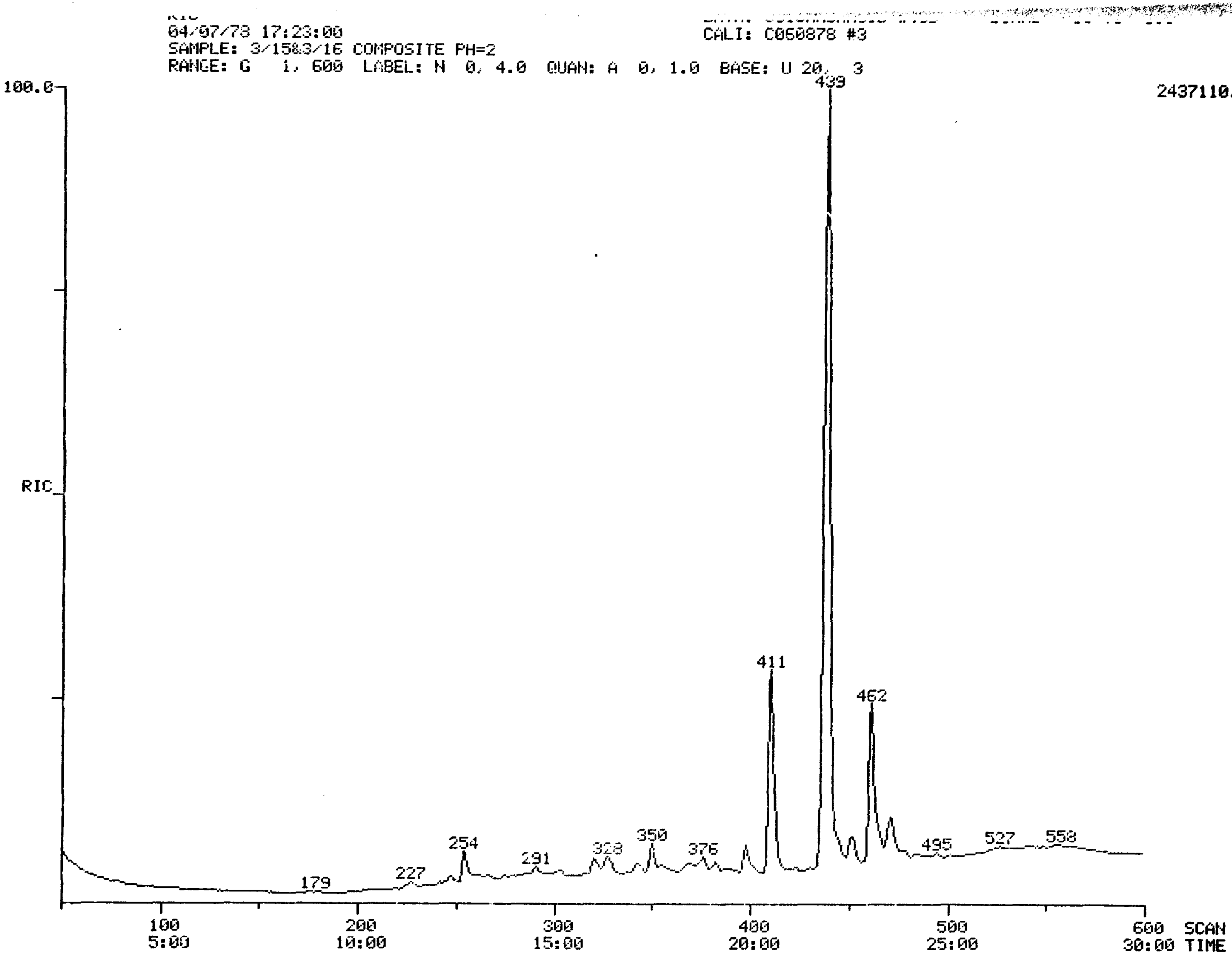

FIGURE 3 ACID EXTRACT

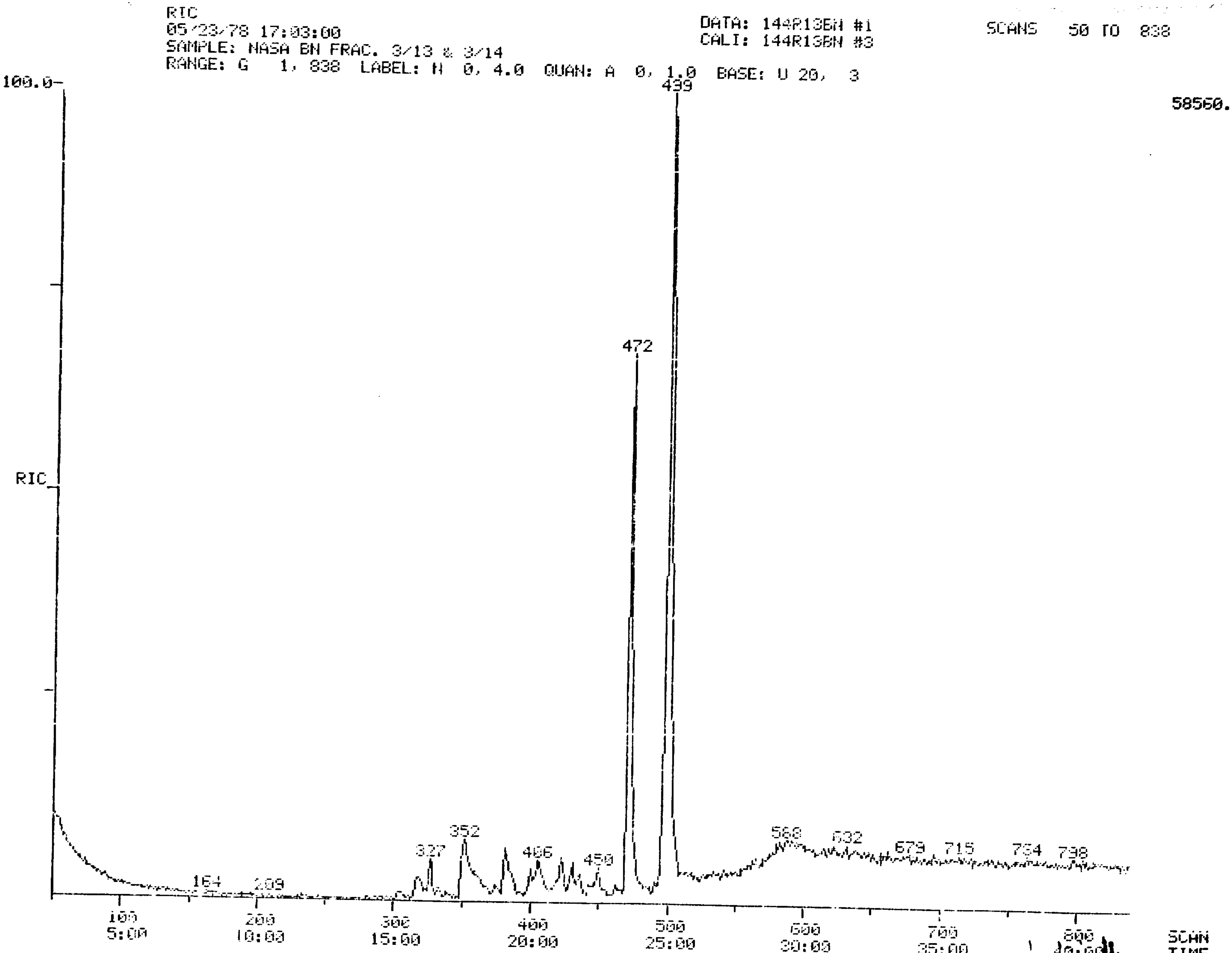

FIGURE 4 BASE/NEUTRAL EXTRACT

Schedule for Establishing Potable Reuse Water Quality Criteria for Manned Spaceflight

Task or Milestone No.	Description	1979				1980				1981				1982				1983			
		1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
T1	Development Contractor Procurement	■	■																		
M1	Award Development Contract		△																		
T2	Organize Review Committee			■	■																
T3	Task Group Preparation for Initial Review Committee Meeting			■	■																
M2	Initial Review Committee Meeting					△															
T4	Development of Health Effects Research Plan					■	■														
M3	Second Review Committee Meeting						△														
T4	Health Effects Research							■	■	■	■	■	■	■	■						
T5	Develop Flight Qualification Test Plan											■	■	■	■						
M4,5	Review Committee Annual Meetings										△				△						
T6	Prepare Water Quality Criteria															■	■				
M6	Submit Water Quality Criteria to Review Committee																△				
T7	Revise Water Quality Criteria																	■			
M7	Final Approval of Water Quality Criteria																	△			

1 = 1st Quarter   3 = 3rd Quarter
2 = 2nd Quarter   4 = 4th Quarter