2015 Consumer Confidence Report

Water System Name: **Stockton Pick-n-Pull** Report Date: 06/23/2016

We test the drinking water quality for many constituents as required by state and federal regulations. This report shows the results of our monitoring for the period of January 1 - December 31, 2014 and may include earlier monitoring data.

Este informe contiene información muy importante sobre su agua potable. Tradúzcalo ó hable con alguien que lo entienda bien.

Type of water source(s) in use: groundwater

Name & general location of source(s): groundwater (Wells #3 & #4)

Drinking Water Source Assessment information: n/a

Time and place of regularly scheduled board meetings for public participation: n/a

For more information, contact: Charina Gaspay, REM Phone: (916) 858-3100

TERMS USED IN THIS REPORT

Maximum Contaminant Level (MCL): The highest level of a contaminant that is allowed in drinking water. Primary MCLs are set as close to the PHGs (or MCLGs) as is economically and technologically feasible. Secondary MCLs are set to protect the odor, taste, and appearance of drinking water.

Maximum Contaminant Level Goal (MCLG): The level of a contaminant in drinking water below which there is no known or expected risk to health. MCLGs are set by the U.S. Environmental Protection Agency (USEPA).

Public Health Goal (PHG): The level of a contaminant in drinking water below which there is no known or expected risk to health. PHGs are set by the California Environmental Protection Agency.

Maximum Residual Disinfectant Level (MRDL): The highest level of a disinfectant allowed in drinking water. There is convincing evidence that addition of a disinfectant is necessary for control of microbial contaminants.

Maximum Residual Disinfectant Level Goal (MRDLG): The level of a drinking water disinfectant below which there is no known or expected risk to health. MRDLGs do not reflect the benefits of the use of disinfectants to control microbial contaminants.

Primary Drinking Water Standards (PDWS): MCLs and MRDLs for contaminants that affect health along with their monitoring and reporting requirements, and water treatment requirements.

Secondary Drinking Water Standards (SDWS): MCLs for contaminants that affect taste, odor, or appearance of the drinking water. Contaminants with SDWSs do not affect the health at the MCL levels.

Treatment Technique (TT): A required process intended to reduce the level of a contaminant in drinking water.

Regulatory Action Level (AL): The concentration of a contaminant which, if exceeded, triggers treatment or other requirements that a water system must follow.

Variances and Exemptions: State Board permission to exceed an MCL or not comply with a treatment technique under certain conditions.

ND: not detectable at testing limit

ppm: parts per million or milligrams per liter (mg/L)

ppb: parts per billion or micrograms per liter (µg/L)

ppt: parts per trillion or nanograms per liter (ng/L)

ppq: parts per quadrillion or picogram per liter (pg/L)

pCi/L: picocuries per liter (a measure of radiation)

The sources of drinking water (both tap water and bottled water) include rivers, lakes, streams, ponds, reservoirs, springs, and wells. As water travels over the surface of the land or through the ground, it dissolves naturally-occurring minerals and, in some cases, radioactive material, and can pick up substances resulting from the presence of animals or from human activity.

Contaminants that may be present in source water include:

- *Microbial contaminants*, such as viruses and bacteria, that may come from sewage treatment plants, septic systems, agricultural livestock operations, and wildlife.
- *Inorganic contaminants*, such as salts and metals, that can be naturally-occurring or result from urban stormwater runoff, industrial or domestic wastewater discharges, oil and gas production, mining, or farming.
- *Pesticides and herbicides*, that may come from a variety of sources such as agriculture, urban stormwater runoff, and residential uses.
- *Organic chemical contaminants*, including synthetic and volatile organic chemicals, that are by-products of industrial processes and petroleum production, and can also come from gas stations, urban stormwater runoff, agricultural application, and septic systems.
- *Radioactive contaminants*, that can be naturally-occurring or be the result of oil and gas production and mining activities.

In order to ensure that tap water is safe to drink, the USEPA and the State Water Resources Control Board (State Board) prescribe regulations that limit the amount of certain contaminants in water provided by public water systems. State Board regulations also establish limits for contaminants in bottled water that provide the same protection for public health.

Tables 1, 2, 3, 4, 5, 7, and 8 list all of the drinking water contaminants that were detected during the most recent sampling for the constituent. The presence of these contaminants in the water does not necessarily indicate that the water poses a health risk. The State Board allows us to monitor for certain contaminants less than once per year because the concentrations of these contaminants do not change frequently. Some of the data, though representative of the water quality, are more than one year old.

TABLE 1 – SAMPLING RESULTS SHOWING THE DETECTION OF COLIFORM BACTERIA

Microbiological Contaminants (complete if bacteria detected)	**Highest No. of Detections**	**No. of months in violation**	**MCL**	**MCLG**	**Typical Source of Bacteria**
Total Coliform Bacteria	**ND**	0	More than 1 sample in a month with a detection	0	Naturally present in the environment
Fecal Coliform or *E. coli*	**ND**	0	A routine sample and a repeat sample detect total coliform and either sample also detects fecal coliform or *E. coli*	0	Human and animal fecal waste

TABLE 2 – SAMPLING RESULTS SHOWING THE DETECTION OF LEAD AND COPPER

Lead and Copper (complete if lead or copper detected in the last sample set)	**Sample Date**	**No. of samples collected**	**90th percentile level detected**	**No. sites exceeding AL**	**AL**	**PHG**	**Typical Source of Contaminant**
Lead (ppb)	**8/04/2015**	**5**	**0.065 mg/L**	**0**	**1.0 mg/L**		Internal corrosion of plumbing systems; discharges from industrial manufacturers, erosion of natural deposits;
Copper (ppm)	**8/04/2015**	**5**	**0.0012 mg/L**	**0**	**0.015 mg/L**		Internal corrosion of plumbing systems; erosion of natural deposits; leaching from wood preservatives

TABLE 3 – SAMPLING RESULTS FOR SODIUM AND HARDNESS

Chemical or Constituent (and reporting units)	**Sample Date**	**Level Detected**	**Range of Detections**	**MCL**	**PHG (MCLG)**	**Typical Source of Contaminant**
Sodium (ppm)	(Was Not Analyzed For In 2015)			none	none	Salt present in the water and is generally naturally occurring
Hardness (ppm)				none	none	Sum of polyvalent cations present in the water, generally magnesium and calcium, and are usually naturally occurring

Any violation of an MCL or AL is asterisked. Additional information regarding the violation is provided later in this report.

TABLE 4 – DETECTION OF CONTAMINANTS WITH A PRIMARY DRINKING WATER STANDARD						
Chemical or Constituent (mg/L)	**Sample Date**	**Level Detected**	**Range of Detections**	**MCL [MRDL]**	**PHG (MCLG) [MRDLG]**	**Typical Source of Contaminant**
Fluoride	6/22/2015	0.3 mg/L	n/a	2 mg/L	1 mg/L	Internal corrosion of plumbing systems; discharges from industrial manufacturers, pesticides;
Nitrate (as NO_3)	6/22/2015	28.5 mg/L	n/a	45 mg/L	45 mg/L	Internal corrosion of plumbing systems; agricultural activities (fertilizers/animal wastes)
Nitrate Nitrogen (as N)	09/28/2015	11.5 mg/L	n/a	10 mg/L	10 mg/L	Internal corrosion of plumbing systems; agricultural activities (fertilizers/animal wastes)
Nitrate Nitrogen (as N) (confirmation sample)	10/09/2015	6 mg/L	n/a	10 mg/L	10 mg/L	Internal corrosion of plumbing systems; agricultural activities (fertilizers/animal wastes)

TABLE 5 – DETECTION OF CONTAMINANTS WITH A SECONDARY DRINKING WATER STANDARD						
Chemical or Constituent (and reporting units)	**Sample Date**	**Level Detected**	**Range of Detections**	**MCL**	**PHG (MCLG)**	**Typical Source of Contaminant**
(none)						

TABLE 6 – DETECTION OF UNREGULATED CONTAMINANTS					
Chemical or Constituent (and reporting units)	**Sample Date**	**Level Detected**	**Range of Detections**	**Notification Level**	**Health Effects Language**
(none)					

***Any violation of an MCL, MRDL, or TT is asterisked. Additional information regarding the violation is provided later in this report.*

Additional General Information on Drinking Water

Drinking water, including bottled water, may reasonably be expected to contain at least small amounts of some contaminants. The presence of contaminants does not necessarily indicate that the water poses a health risk. More information about contaminants and potential health effects can be obtained by calling the USEPA's Safe Drinking Water Hotline (1-800-426-4791).

Some people may be more vulnerable to contaminants in drinking water than the general population. Immuno-compromised persons such as persons with cancer undergoing chemotherapy, persons who have undergone organ transplants, people with HIV/AIDS or other immune system disorders, some elderly, and infants can be particularly at risk from infections. These people should seek advice about drinking water from their health care providers. USEPA/Centers for Disease Control (CDC) guidelines on appropriate means to lessen the risk of infection by *Cryptosporidium* and other microbial contaminants are available from the Safe Drinking Water Hotline (1-800-426-4791).

Lead-Specific Language for Community Water Systems: If present, elevated levels of lead can cause serious health problems, especially for pregnant women and young children. Lead in drinking water is primarily from materials and components associated with service lines and home plumbing. Pick-n-Pull is responsible for providing quality drinking water, but cannot control the variety of materials used in plumbing components. When your water has been sitting for several hours, you can minimize the potential for lead exposure by flushing your tap for 30 seconds to 2 minutes before using water for drinking or cooking. If you are concerned about lead in your water, you may wish to have your water tested. Information on lead in drinking water, testing methods, and steps you can take to minimize exposure is available from the Safe Drinking Water Hotline or at http://www.epa.gov/safewater/lead.

Lead was detected well below the maximum contaminant level for lead.

Summary Information for Violation of a MCL, MRDL, AL, TT, or Monitoring and Reporting Requirement

VIOLATION OF A MCL, MRDL, AL, TT, OR MONITORING AND REPORTING REQUIREMENT				
Violation	**Explanation**	**Duration**	**Actions Taken to Correct the Violation**	**Health Effects Language**
No Violation but Nitrate Nitrite (as N) was detected at 11.5 mg/L	Nitrate Nitrite (as N) was detected slightly above the MCL but a confirmation sample collected confirmed levels to be below the MCL.	Observed in October 2015	Site will continue to monitor nitrate nitrite (as N) on a quarterly basis. In addition, the area around the wells have been cleared of material storage and re-graded to limit runoff from puddling at well pad. Water at the Site is not used as a drinking source.	High levels of nitrate in drinking water has been linked to methemoglobinemia to infants. High levels of nitrate may also be an indication of possible contaminants such as bacteria or pesticides.

For Water Systems Providing Ground Water as a Source of Drinking Water

TABLE 7 – SAMPLING RESULTS SHOWING FECAL INDICATOR-POSITIVE GROUND WATER SOURCE SAMPLES					
Microbiological Contaminants (complete if fecal-indicator detected)	**Total No. of Detections**	**Sample Dates**	**MCL [MRDL]**	**PHG (MCLG) [MRDLG]**	**Typical Source of Contaminant**
E. coli	0	monthly	0	(0)	Human and animal fecal waste
Enterococci	0	monthly	TT	n/a	Human and animal fecal waste
Coliphage	0	monthly	TT	n/a	Human and animal fecal waste

Summary Information for Fecal Indicator-Positive Ground Water Source Samples, Uncorrected Significant Deficiencies, or Ground Water TT

SPECIAL NOTICE OF FECAL INDICATOR-POSITIVE GROUND WATER SOURCE SAMPLE
N/A – no detections of bacteria in groundwater

SPECIAL NOTICE FOR UNCORRECTED SIGNIFICANT DEFICIENCIES
N/A – no deficiencies

VIOLATION OF GROUND WATER TT				
TT Violation	**Explanation**	**Duration**	**Actions Taken to Correct the Violation**	**Health Effects Language**
N/A – no deficiencies				

For Systems Providing Surface Water as a Source of Drinking Water

TABLE 8 - SAMPLING RESULTS SHOWING TREATMENT OF SURFACE WATER SOURCES	
Treatment Technique [(a)] (Type of approved filtration technology used)	**N/A - No treatment of surface water sources**
Turbidity Performance Standards [(b)] (that must be met through the water treatment process)	Turbidity of the filtered water must: 1 – Be less than or equal to _____ NTU in 95% of measurements in a month. ore than eight consecutive hours. time.
Lowest monthly percentage of sa Performance Standard No. 1.	
Highest single turbidity measurement during the year	
Number of violations of any surface water treatment requirements	

N/A – surface water is not a source for this system

(a) A required process intended to reduce the level of a contaminant in drinking water.

(b) Turbidity (measured in NTU) is a measurement of the cloudiness of water and is a good indicator of water quality and filtration performance. Turbidity results which meet performance standards are considered to be in compliance with filtration requirements.

** Any violation of a TT is marked with an asterisk. Additional information regarding the violation is provided below.*

Summary Information for Violation of a Surface Water TT

VIOLATION OF A SURFACE WATER TT				
TT Violation	**Explanation**	**Duration**	**Actions Taken to Correct the Violation**	**Health Effects Language**

N/A – surface water is not a source for this system

Summary Information for Operating Under a Variance or Exemption

N/A – does not operate under a variance or exemption

Certification Form

CWS name:____**Stockton Pick-n-Pull**_____

PWS I.D. no:___**3901481**_____

The community water system named above hereby confirms that its consumer confidence report has been distributed to customers (and appropriate notices of availability have been given). Further, the system certifies that the information contained in the report is correct and consistent with the compliance monitoring data previously submitted to the primacy agency.

Certified by:

Signature:_____

Name__Charina Gaspay_____

Title___Regional Environmental Manager_____

Phone #__916-858-3100_____ Date___06/22/2016_____

***You are not required by EPA rules to report the following information, but you may want to provide it to your state. Check all items that apply. ***

_**X**_ CCR was distributed by mail or other direct delivery. Specify other direct delivery methods:

Copies of the 2015 Consumer Confidence Report was emailed to Site Management and posted in the employee break rooms._____

_**X**_ "Good faith" efforts were used to reach non-bill paying consumers. Those efforts included the following methods as recommended by the primacy agency:

____posting the CCR on the Internet at www._____

____mailing the CCR to postal patrons within the service area. (attach zip codes used)

____advertising availability of the CCR in news media (attach copy of announcement)

____publication of CCR in local newspaper (attach copy)

_**X**_posting the CCR in public places (attach a list of locations) – S**ite offices and break rooms.**

____delivery of multiple copies to single bill addresses serving several persons such as: apartments, businesses, and large private employers

____delivery to community organizations (attach a list)

____(for systems serving at least 100,000 persons) Posted CCR on a publicly-accessible Internet site at the address: www._____

____Delivered CCR to other agencies as required by the primacy agency (attach a list)

Public Health Goals for
NITRATE AND NITRITE
in Drinking Water

Prepared by

Pesticide and Environmental Toxicology Section
Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

December 1997

LIST OF CONTRIBUTORS

PHG PROJECT MANAGEMENT	REPORT PREPARATION	SUPPORT
Project Officer Anna Fan, Ph.D. ***Chemical Prioritization Report Outline*** Joseph Brown, Ph.D. Coordinator David Morry, Ph.D. Yi Wang, Ph.D. ***Document Development*** Michael DiBartolomeis, Ph.D. Coordinator George Alexeeff, Ph.D. Hanafi Russell, M.S. Yi Wang, Ph.D. ***Public Workshop*** Michael DiBartolomeis, Ph.D. Coordinator Judy Polakoff, M.S. Organizer ***Methodology/Approaches/Review Comments*** Joseph Brown, Ph.D. Robert Howd, Ph.D. Coordinators Lubow Jowa, Ph.D. David Morry, Ph.D. Rajpal Tomar, Ph.D. Yi Wang, Ph.D.	***Author*** Judy Polakoff, M.S. ***Primary Reviewer*** Frank Mycroft, Ph.D. ***Secondary Reviewer*** Michael DiBartolomeis, Ph.D. ***Final Reviewers*** Anna Fan, Ph.D. William Vance, Ph.D. ***Editor*** Michael DiBartolomeis, Ph.D.	***Administrative Support*** Edna Hernandez Coordinator Laurie Bliss Sharon Davis Kathy Elliott Vickie Grayson Michelle Johnson Genevieve Shafer Tonya Turner ***Library Support*** Mary Ann Mahoney Valerie Walter ***Website Posting*** Robert Brodberg, Ph.D. Edna Hernandez Laurie Monserrat, M.S. Judy Polakoff, M.S. Hanafi Russell, M.S.

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PREFACE

Drinking Water Public Health Goal of the Office of Environmental Health Hazard Assessment

This Public Health Goal (PHG) technical support document provides information on health effects from contaminants in drinking water. The PHG describes concentrations of contaminants at which adverse health effects would not be expected to occur, even over a lifetime of exposure. PHGs are developed for chemical contaminants based on the best available toxicological data in the scientific literature. These documents and the analyses contained in them provide estimates of the levels of contaminants in drinking water that would pose no significant health risk to individuals consuming the water on a daily basis over a lifetime.

The California Safe Drinking Water Act of 1996 (amended Health and Safety Code, Section 116365) requires the Office of Environmental Health Hazard Assessment (OEHHA) to adopt PHGs for contaminants in drinking water based exclusively on public health considerations. The Act requires OEHHA to adopt PHGs that meet the following criteria:

1. PHGs for acutely toxic substances shall be set at levels at which scientific evidence indicates that no known or anticipated adverse effects on health will occur, plus an adequate margin-of-safety.
2. PHGs for carcinogens or other substances which can cause chronic disease shall be based solely on health effects without regard to cost impacts and shall be set at levels which OEHHA has determined do not pose any significant risk to health.
3. To the extent the information is available, OEHHA shall consider possible synergistic effects resulting from exposure to two or more contaminants.
4. OEHHA shall consider the existence of groups in the population that are more susceptible to adverse effects of the contaminants than a normal healthy adult.
5. OEHHA shall consider the contaminant exposure and body burden levels that alter physiological function or structure in a manner that may significantly increase the risk of illness.
6. In cases of scientific ambiguity, OEHHA shall use criteria most protective of public health and shall incorporate uncertainty factors of noncarcinogenic substances for which scientific research indicates a safe dose-response threshold.
7. In cases where scientific evidence demonstrates that a safe dose-response threshold for a contaminant exists, then the PHG should be set at that threshold.
8. The PHG may be set at zero if necessary to satisfy the requirements listed above.
9. OEHHA shall consider exposure to contaminants in media other than drinking water, including food and air and the resulting body burden.
10. PHGs adopted by OEHHA shall be reviewed periodically and revised as necessary based on the availability of new scientific data.

PHGs adopted by OEHHA are for use by the California Department of Health Services (DHS) in establishing primary drinking water standards (State Maximum Contaminant Levels, or MCLs). Whereas PHGs are to be based solely on scientific and public health considerations without regard to economic cost considerations, drinking water standards adopted by DHS are to consider economic factors and technical feasibility. For this reason PHGs are only one part of the information used by DHS for establishing drinking water standards. PHGs established by

OEHHA exert no regulatory burden and represent only non-mandatory goals. By federal law, MCLs established by DHS must be at least as stringent as the federal MCL if one exists.

PHG documents are developed for technical assistance to DHS, but may also benefit federal, state and local public health officials. While the PHGs are calculated for single chemicals only, they may, if the information is available, address hazards associated with the interactions of contaminants in mixtures. Further, PHGs are derived for drinking water only and are not to be utilized as target levels for the contamination of environmental waters where additional concerns of bioaccumulation in fish and shellfish may pertain. Often environmental water contaminant criteria are more stringent than drinking water PHGs, to account for human exposures to a single chemical in multiple environmental media and from bioconcentration by plants and animals in the food chain.

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SUMMARY

OEHHA developed Public Health Goals (PHGs) of 45 ppm for nitrate (equivalent to 10 ppm nitrate-nitrogen), 1 ppm for nitrite-nitrogen and 10 ppm for joint nitrate/nitrite (expressed as nitrogen) in drinking water. The calculation of these PHGs is based on the protection of infants from the occurrence of methemoglobinemia, the principal toxic effect observed in humans exposed to nitrate or nitrite. The PHGs are equivalent to California's current drinking water standards for nitrate (45 mg/L nitrate), nitrite (1 mg/L nitrite-nitrogen) and 10 mg/L (joint nitrate/nitrite expressed as nitrogen) which were adopted by the California Department of Health Services (DHS) in 1994 from the U.S. Environmental Protection Agency's (U.S. EPA's) Maximum Contaminant Levels (MCLs) promulgated in 1991. Following our review of the current literature and a reevaluation of the bases for calculating the MCLs for these compounds, we determined that there was no scientific basis to propose alternative PHGs. Therefore, OEHHA adopts PHGs of 45 mg/L (45 ppm) for nitrate, 1 mg/L (1 ppm) for nitrite-nitrogen and 10 mg/L (10 ppm) for joint nitrate/nitrite (expressed as nitrogen) in drinking water.

INTRODUCTION

California's current drinking water standard for nitrate is 45 mg nitrate/L (equivalent to 10 mg nitrate-nitrogen/L) and for nitrite is 1 mg nitrite-nitrogen/L. In addition, there is a joint nitrate/nitrite standard of 10 mg/L, expressed on a nitrogen basis. These values were adopted by the DHS in 1994 from U.S. EPA's MCLs promulgated in 1991. The standards are based on the occurrence of methemoglobinemia, the principal toxic effect observed in humans exposed to nitrate or nitrite. In developing PHGs for nitrate and nitrite, we evaluated the basis for U.S. EPA's MCLs. In addition, a search of the scientific literature was conducted to determine if there are any new data that would support the development of numbers different from the current values[1]. It is important to note that Office of Environmental Health Hazard Assessment (OEHHA) staff have conducted peer-reviewed, published evaluations of the hazards of nitrate and nitrite in drinking water that were important to the development of PHGs (Fan *et al.*, 1987; Fan and Steinberg, 1996). The results of our present evaluation are described below.

HUMAN EXPOSURE

Human exposure to nitrates and nitrites results primarily from dietary ingestion, particularly from vegetables and cured meats. The average adult daily intake from food in the United States has been estimated to be 40 to 100 mg for nitrate, and 0.3 to 2.6 mg for nitrite. Exposure estimates indicate that for more than 99% of the adult population in the United States, only 1 to 3% of nitrate and nitrite intake comes from drinking water. Drinking water becomes an important contributor to total nitrate exposure only in areas of notable contamination (NRC, 1981; 1995). For infants, the exposure scenarios are somewhat different. For breast-fed infants, total nitrate exposure is negligible. For bottle-fed infants consuming drinking water used to prepare their formula, drinking water can be a substantial exposure pathway. At the MCL of 10 mg nitrate-nitrogen/L, the daily intake for a 4 kg infant consuming 0.64 L/day of water used to prepare formula would be approximately 1.6 mg/kg-day.

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[1] A copy of the literature review is available to the public upon request.

TOXICOLOGY

Methemoglobinemia

Methemoglobinemia is the primary adverse health effect associated with human exposure to nitrate or nitrite. To cause methemoglobinemia, nitrate must be converted to nitrite. Nitrite causes the oxidation of normal hemoglobin to methemoglobin which is unable to transport oxygen from lungs to tissues. Bacteria in the gastrointestinal system mediate the conversion of nitrate to nitrite. Consequently the risk of methemoglobinemia from ingestion of nitrate depends not only on the dose of nitrate by also on the number and type of enteric bacteria. Low levels of methemoglobin occur in individuals with typical values ranging from 0.5 to 2.0% (NRC, 1981). While methemoglobinemia can be clinically diagnosed at levels of 1%, methemoglobin levels up to 10% are generally not considered adverse. At levels above 10%, methemoglobinemia causes cyanosis, and at higher concentrations, asphyxia (WHO, 1996; U.S. EPA, 1997).

Infants are generally recognized as the subpopulation most susceptible to nitrate induced methemoglobinemia (U.S. EPA, 1990; U.S. EPA, 1997). Several factors make infants particularly sensitive to methemoglobinemia including: 1) infants have a greater total fluid intake per unit body weight than adults, 2) the gastrointestinal system in infants normally has a high pH that allows bacterial proliferation and nitrate catalysis to nitrite, 3) frequent occurrences of infant gastroenteritis may favor the development of methemoglobin when the upper gastrointestinal tract becomes colonized with bacteria, 4) fetal hemoglobin is more readily oxidized than adult hemoglobin and 5) infants have half the methemoglobin reductase activity of older children and adults and are therefore less capable of metabolizing excess methemoglobin (Winton *et al.*, 1971; NRC, 1981; Kross *et al.*, 1992).

There are other individuals who may be predisposed to the development of nitrate-induced methemoglobinemia due to altered physiological states. These include pregnant women and possibly others with glucose-6-phosphate dehydrogenase deficiency, adults with reduced gastric acidity (e.g., from diseases including achlorohydria or atrophic gastritis) and those with a lack of methemoglobin reductase (U.S. EPA, 1997; NRC, 1981).

Since the mid 1950's, more than 2,000 cases of infantile methemoglobinemia have been reported world-wide. Most cases are associated with exposure concentrations greater than 20 mg nitrate-nitrogen/L in drinking water that was used to prepare infant formula. Cases of infantile methemoglobinemia associated with nitrate exposure concentrations of 11 to 20 mg nitrate-nitrogen/L are usually associated with concomitant exposure to water contaminated with bacteria. Thorough reviews of the literature on the occurrence of methemoglobinemia in infants are provided by U.S. EPA (1990), Fan *et al.* (1987), and Fan and Steinberg (1996).

Only two reports were found documenting cases of infantile methemoglobinemia resulting from ingestion of nitrate-contaminated water since the development of the federal standard in 1991. In Wisconsin, a six-week-old girl was found to have a methemoglobin level of 21.4% after having been hospitalized twice, first for dehydration and vomiting, and later for acute weight loss and limited consumption of formula. Water samples taken during the infant's hospitalization contained 9.9 and 58 mg nitrate-nitrogen/L as collected from the reverse-osmosis unit and from the well that supplied water to the house, respectively. In addition, an early morning first draw sample collected from the kitchen faucet contained copper levels six-times the federal MCL. The infant's condition

was attributed to her being given formula mixed with water contaminated with both nitrate and copper (MMWR, 1993). In a recent report from Poland (Lutynski *et al.*, 1996), it was noted that from 1979 to 1992, the Poison Information Center in Krakow documented 239 cases of methemoglobinemia from Krakow and neighboring provinces. Of the 239 cases, 216 involved infants who were fed with nitrate-contaminated well water and carrot soup. Exposure concentrations were not given, and water supplies in the area were characterized as being of very poor quality.

Reproductive and Developmental Toxicity

In a recent report by the National Research Council (NRC, 1995), the authors concluded that while data from recent epidemiological studies have suggested an association between maternal nitrate exposure from drinking water and developmental effects in offspring, a definite conclusion on the cause-and-effect relationship cannot be drawn.

Most of the studies in experimental animals have been conducted with nitrite. Data have shown reproductive and developmental toxicity associated with exposure to nitrite, but the effects occurred mostly at very high exposure concentrations which can also produce maternal methemoglobinemia. Teratogenic effects have not been observed in rats, mice, hamsters or rabbits (Fan *et al.*, 1987; Fan and Steinberg, 1996; NRC, 1995).

Since the development of the federal standard in 1991, one report was identified of spontaneous abortions possibly related to consumption of nitrate-contaminated well water in Indiana (MMWR, 1996). Four women living in close proximity reported a total of eight spontaneous abortions from 1991 to 1994. Nitrate concentrations of 19 to 29 mg nitrate-nitrogen/L were measured in well-water sources. Term births occurred before or after the period when each of the four women consumed nitrate-contaminated water. The investigation did not establish a causal link between nitrate exposure from drinking water and spontaneous abortion, but the findings indicate the need for further assessment of the possible effects of nitrate on human reproduction. Developmental effects were reported in rat pups from dams consuming sodium nitrite (2,000 ppm) in drinking water (Nyakas *et al.*, 1994). The observed effects on behavior and adrenal function were attributed to fetal hypoxia caused by severe methemoglobinemia in the pregnant rats.

Carcinogenicity

Concern has been raised about a possible increased risk of cancer in humans from the endogenous and exogenous formation of N-nitroso compounds from nitrite, many of which are carcinogenic in animals. However, recent epidemiological studies have not supported an association between nitrate or nitrite exposure from drinking water and increased cancer rates in humans. In experimental animals, nitrate and nitrite have not been shown to be carcinogenic. Nitrite has only been shown to be carcinogenic in animals when administered concurrently with nitrosable amines, apparently as a result of the endogenous formation of carcinogenic amines (WHO, 1996; NRC, 1995; Fan and Steinberg, 1996).

In addition to the cancer studies evaluated by the World Health Organization (WHO, 1996), the National Research Council (NRC, 1995) and U.S. EPA (1991), three recent epidemiological studies were identified examining the effects of nitrate exposure on human cancer risk. The results of one case-control study from Germany (Steindorf *et al.*, 1994) suggest the absence of an association between low nitrate levels in drinking water (16 mg nitrate-nitrogen/L) and the risk of

brain tumors. Two studies by Morales-Suarez-Varela were published on the impact of elevated nitrate concentrations in drinking water (greater than 50 ppm) on cancer incidence and mortality in Valencia, Spain (Morales-Suarez-Varela *et al.*, 1993; Morales-Suarez-Varela *et al.*, 1995). The authors suggest a possible association between elevated nitrate concentrations in drinking water and cancer of the bladder and stomach. However, a definite cause-and-effect relationship was not shown. Another case-control study conducted in Nebraska examined nitrate in drinking water and the risk of non-Hodgkin's lymphoma (Ward *et al.*, 1996). Authors reported that long-term exposure to elevated nitrate in drinking water (greater than 50 ppm) may contribute to the risk of non-Hodgkin's lymphoma, but again, no cause-and-effect relationship was shown.

Other Health Effects

Inorganic and organic nitrite compounds can produce hypotension in humans as a result of direct action on smooth muscle. Therapeutic doses of 30 to 60 mg sodium nitrite are typically used to treat angina pectorus. Organic nitrates can also produce hypotension, although through an indirect mechanism of action, while inorganic nitrate does not produce the effect (U.S. EPA, 1990).

DOSE-RESPONSE ASSESSMENT

The current state and federal MCLs for nitrates and nitrites are based on the occurrence of infantile methemoglobinemia resulting from ingestion of nitrate-contaminated water. The two principal studies used as the basis of these standards are described below.

Bosch *et al.* (1950) evaluated 139 cases of cyanosis due to methemoglobinemia reported in Minnesota from 1947 to 1949. The 139 cases, which included 14 deaths, occurred in infants under six months of age. Nitrate concentrations were measured in 129 wells used to supply water to the infants with methemoglobinemia. None of the wells contained nitrate concentrations less than 10 mg nitrate-nitrogen/L. Only two of the wells contained 10 to 20 mg nitrate-nitrogen/L, however in both of these cases the diagnosis of methemoglobinemia was considered questionable. Coliform organisms were detected in 45 of 51 samples tested for bacterial contamination.

Walton (1951) reported results of a survey conducted by the American Public Health Association that identified 278 cases of infantile methemoglobinemia associated with consumption of nitrate-contaminated water. Water nitrate concentrations were available for 214 of the 278 cases. Of the 214 cases, none occurred from water nitrate concentrations less than 10 mg nitrate-nitrogen/L, and only five cases (2%) involved infants exposed to 11 to 20 mg nitrate-nitrogen/L. More than 80% of the infants were exposed to nitrate concentrations greater than 50 mg nitrate-nitrogen/L. Data on bacteriological contamination of the water and/or incidence of gastrointestinal disease among the infants were not reported.

Nitrate

Based primarily on the studies of Bosch *et al.* (1950) and Walton (1951), with support from a large number of additional epidemiological and case studies in humans (including Cornblath and Hartmann, 1948; Simon *et al.*, 1964; Toussaint and Selenka, 1970; Craun *et al.*, 1981; see U.S. EPA, 1990 for descriptions of these and other studies) a no-observed-adverse-effect-level (NOAEL) is identified to be 10 mg nitrate-nitrogen/L. An uncertainty factor of one was applied because the available data provide an adequate NOAEL for the critical toxic effect (methemoglobinemia) in the most sensitive human subpopulation (infants). Therefore, the MCL is

10 mg nitrate-nitrogen/L, or 45 mg nitrate/L (U.S. EPA, 1997), based on early clinical signs of cyanosis associated with methemoglobinemia in infants.

Nitrite

There are no reliable quantitative data on nitrite and methemoglobinemia in humans. While nitrite has been shown to cause methemoglobinemia in animals, humans appear to be more sensitive to nitrite-induced methemoglobin formation. Therefore, the MCL for nitrite was derived by extrapolation of nitrate toxicity data in humans. Based on data which indicate that the fraction of nitrate reduced to nitrite in adults is at least 5%, and knowing that nitrate reduction in infants is significantly greater than in adults (due to the presence of gastrointestinal bacteria), the conversion rate is estimated to be at least 10% in infants (U.S. EPA, 1990).

Using the same NOAEL selected for nitrate of 10 mg nitrate-nitrogen/L (from Bosch *et al.*, 1950; Walton, 1951), an uncertainty factor of one was applied because the NOAEL was of the critical toxic effect (methemoglobinemia) in the most sensitive human subpopulation (infants). In addition, the NOAEL was multiplied by 0.1 to account for the estimated conversion rate of nitrate to nitrite by gastrointestinal tract bacteria in infants, as described above. Therefore, the MCL for nitrite is 1 mg nitrite-nitrogen/L (U.S. EPA, 1990; U.S. EPA, 1997).

Joint Nitrate and Nitrite

U.S. EPA set a joint standard for the sum of the concentration of nitrate and nitrite at 10 mg/L as nitrogen. The combined standard does not replace the individual MCLs for nitrate or nitrite, therefore the maximum contribution from nitrite cannot exceed 1 mg nitrite-nitrogen/L (U.S. EPA, 1991).

CALCULATION OF PHGS

Public health-protective concentrations (C, in mg/L) are calculated using the general formula for noncarcinogenic endpoints:

$$C = \frac{\text{NOAEL} \times \text{BW} \times \text{RSC}}{\text{UF} \times \text{L/day}} = \text{mg/L}$$

where,

NOAEL	=	No-observed-adverse-effect-level (10 mg nitrate-nitrogen/L)
BW	=	Body weight (not applicable)
RSC	=	Relative source contribution (not applicable)
UF	=	Uncertainty factor (one)
L/day	=	Volume of daily drinking water consumption (not applicable).

In the case of nitrate and nitrite, the NOAEL is based on human data for nitrate exposure from drinking water in the most sensitive population (infants). Therefore, the uncertainty factor for human variability is one. No other uncertainty factors are applied. For nitrite, the NOAEL is multiplied by 0.1 to account for the estimated conversion rate of nitrate to nitrite by gastrointestinal tract bacteria in infants, as previously described.

OEHHA staff support U.S. EPA's previous analysis on nitrate and nitrite. The values consider the conversion of nitrate to nitrite, nitrite-induced formation of methemoglobin, and the general absence of methemoglobinemia occurring at nitrate-nitrogen levels below 10 mg/L based on world-wide epidemiological evidence. The values are protective for infants, the most sensitive population. Review of updated information compiled since the development of the MCLs in 1991 support the earlier findings. In addition, no cases of methemoglobinemia occurring at or below the MCL have been identified in the United States. Therefore, OEHHA adopts the following PHGs:

nitrate	=	10 mg nitrate-nitrogen/L (10 ppm nitrate-nitrogen)
	=	45 mg nitrate/L (45 ppm nitrate),
nitrite	=	1 mg nitrite-nitrogen/L (1 ppm) and
joint nitrate/nitrite	=	10 mg/L (10 ppm), expressed on a nitrogen basis.

These PHGs, as well as current federal MCLs for nitrate and nitrite, should be adequately protective against methemoglobinemia in infants, as well as any potential reproductive or developmental effects.

RISK CHARACTERIZATION

Although there are an enormous amount of data on the occurrence of methemoglobinemia in infants ingesting nitrate-contaminated water, there are some uncertainties in the database used for the development for the MCL:

1. The concentration of nitrate is not known in all reported cases of methemoglobinemia, and cases of nitrate-induced infantile methemoglobinemia have been reported in other countries at levels below the MCL. While the reports that indicate cases occurring below the MCL are of uncertain quality (largely due to a lack of controls for the presence of confounding factors), there is the possibility that methemoglobinemia resulting from nitrate ingestion could occur at a nitrate level lower than the MCL.

2. It is possible that methemoglobinemia is more likely associated with nitrate plus bacterial contamination of drinking water. Bacterial contamination favors the conversion of nitrate to nitrite as well as the occurrence of diarrhea, which in infants can increase the risk of methemoglobinemia.

3. There could be unidentified differences among the study populations, including nutritional status, that could contribute to differences in the reported findings. Vitamin C can help reduce the risk of methemoglobinemia from nitrate (NRC, 1995) and some populations have a higher vitamin C intake level than others.

4. There are no known requirements for reporting cases of methemoglobinemia.

5. Infant illness or death from nitrate-induced methemoglobinemia may be misdiagnosed, perhaps as sudden-infant-death-syndrome (Johnson and Kross, 1990).

In light of the above, a particular public health concern regarding the methemoglobinemia data is the finding of a lowest-observed-adverse-effect-level (LOAEL) of 11 to 20 mg nitrate-nitrogen/L with no clear delineation of a NOAEL. It is important to note that, in the United States no cases of methemoglobinemia have been reported at a nitrate concentration at or below the MCL.

Animal experimental data have shown reproductive and developmental toxicity associated with high exposure levels to nitrate and nitrite, which are not likely to be encountered in drinking water. The lowest concentrations associated with developmental effects were reported at 1,000 ppm sodium nitrite in water, with a NOAEL at or around 500 ppm (Roth *et al.*, 1987; Roth and Smith, 1988). At an MCL of 45 ppm nitrate, and assuming a 10% conversion of nitrate to nitrite, the amount of nitrate ingestion from two liters of water would be several hundred times less than this NOAEL (Fan and Steinberg, 1996).

U.S. EPA has adopted a joint standard to account for the possibility of the simultaneous occurrence of nitrate and nitrite in drinking water. The equilibrium of nitrate and nitrite in aqueous environments greatly favors nitrate. Nitrite levels can become significant with bacterial contamination and anaerobic conditions (U.S. EPA, 1990); in such cases nitrite levels would be expected to be high, exceeding the nitrite MCL of 1 mg/L. In light of the environmental characteristics of nitrate and nitrite, and in keeping with U.S. EPA, we have concluded that the joint MCL of 10 mg/L (with the contribution from nitrite not exceeding 1 mg nitrite-nitrogen/L) adequate for the protection of public health.

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