Table 5. Percent Differences of Quantitation Limits to the EPA/ACS QL for the Episode 6000 Dataset

Analyte	Method	Procedure	ISO LOQ/ML	SL-IQE/ML
1,2-dichloropropane	524.2		-29.8%	-1.9%
1,3,5-tmb+4-chlorotoluene	502.2	PID	25.7%	-5.5%
1,3,5-trimethylbenzene	524.2		-13.6%	199.2%
1,3-dichlorobenzene	502.2	ELCD	-114.2%	161.4%
1,3-dichlorobenzene	502.2	PID	74.5%	79.7%
1,3-dichlorobenzene	524.2		-22.2%	-27.3%
1,3-dichloropropane	502.2	ELCD	-22.9%	7.5%
1,3-dichloropropane	524.2		13.0%	32.7%
1,4-dichlorobenzene	502.2	ELCD	-120.8%	1.0%
1,4-dichlorobenzene	524.2		-37.2%	-24.2%
1-chlorobutane	524.2		48.8%	199.3%
2,2-dichloropropane	524.2		-178.4%	116.7%
2-butanone	524.2		-34.2%	-76.6%
2-chlorotoluene	502.2	ELCD	-109.9%	-1.4%
2-chlorotoluene	502.2	PID	-24.6%	-16.4%
2-chlorotoluene	524.2		-7.6%	6.3%
2-hexanone	524.2		-152.8%	-167.5%
2-nitropropane	524.2		-43.9%	-108.9%
4-chlorotoluene	502.2	ELCD	-116.3%	-111.5%
4-chlorotoluene	524.2		-29.1%	199.2%
4-isopropyltoluene	524.2		-15.4%	-101.7%
4-methyl-2-pentanone	524.2		3.2%	-11.3%
Acetone	524.2		5.5%	31.3%
Acrylonitrile	524.2		-9.7%	173.4%
Allyl chloride	524.2		25.5%	198.7%
Aluminum	1620		-27.0%	129.1%
Aluminum	200.8	ICP/MS	-136.6%	-51.0%
Ammonia as nitrogen	350.3		-30.9%	-34.1%
Antimony	1620		-4.4%	62.6%
Antimony	200.8	ICP/MS	-186.6%	-174.7%
Arsenic	1620		-30.3%	-47.0%
Arsenic	200.8	ICP/MS	-32.5%	-22.5%

Table 5. Percent Differences of Quantitation Limits to the EPA/ACS QL for the Episode 6000 Dataset

Analyte	Method	Procedure	ISO LOQ/ML	SL-IQE/ML
Barium	1620		-5.7%	-19.3%
Barium	200.8	ICP/MS	46.6%	71.5%
Benzene	502.2	PID	53.7%	58.1%
Benzene	524.2		40.5%	-13.1%
Beryllium	1620		-61.9%	-68.5%
Beryllium	200.8	ICP/MS	-9.9%	75.0%
Boron	1620		-8.2%	2.2%
Bromobenzene	502.2	ELCD	18.0%	150.4%
Bromobenzene	502.2	PID	-0.9%	67.0%
Bromobenzene	524.2		-18.1%	-35.4%
Bromochloromethane	502.2	ELCD	25.8%	187.9%
Bromochloromethane	524.2		9.3%	-30.3%
Bromodichloromethane	502.2	ELCD	-25.6%	182.0%
Bromodichloromethane	524.2		-38.7%	-43.9%
Bromoform	502.2	ELCD	-12.0%	197.7%
Bromoform	524.2		-54.2%	-3.7%
Bromomethane	502.2	ELCD	N/A	176.9%
Bromomethane	524.2		23.2%	12.2%
Cadmium	1620		-36.4%	-19.7%
Cadmium	200.8	ICP/MS	79.2%	103.6%
Calcium	1620		60.4%	-0.0%
Carbon disulfide	524.2		-26.2%	1.3%
Carbon tetrachloride	524.2		23.9%	33.4%
Carbontet+1,1-dcp	502.2	ELCD	-74.3%	-37.3%
Chloroacetonitrile	524.2		70.3%	49.3%
Chlorobenzene	502.2	ELCD	15.7%	189.0%
Chlorobenzene	502.2	PID	35.2%	17.4%
Chlorobenzene	524.2		7.4%	-50.8%
Chloroethane	502.2	ELCD	-161.8%	168.4%
Chloroethane	524.2		-8.0%	24.2%
Chloroform	502.2	ELCD	-149.4%	-155.3%
Chloroform	524.2		31.7%	19.2%

Table 5. Percent Differences of Quantitation Limits to the EPA/ACS QL for the Episode 6000 Dataset

Analyte	Method	Procedure	ISO LOQ/ML	SL-IQE/ML
Chloromethane	502.2	ELCD	52.5%	158.6%
Chloromethane	524.2		-9.8%	-34.9%
Chromium	1620		-0.7%	22.9%
Chromium	200.8	ICP/MS	49.3%	134.9%
Cis-1,2-dce+2,2-dcp	502.2	ELCD	-9.5%	-24.7%
Cis-1,2-dichloroethene	524.2		42.4%	36.1%
Cis-1,3-dichloropropene	502.2	ELCD	-45.8%	181.6%
Cis-1,3-dichloropropene	502.2	PID	23.8%	-168.7%
Cis-1,3-dichloropropene	524.2		15.3%	34.2%
Cobalt	1620		-82.0%	-20.2%
Cobalt	200.8	ICP/MS	N/A	N/A
Copper	1620		31.6%	81.5%
Copper	200.8	ICP/MS	34.6%	179.2%
Dibromochloromethane	502.2	ELCD	41.1%	193.7%
Dibromochloromethane	524.2		-29.0%	36.0%
Dibromomethane	502.2	ELCD	34.7%	194.3%
Dibromomethane	524.2		-22.2%	-8.3%
Dichlorodifluoromethane	502.2	ELCD	-53.2%	192.8%
Dichlorodifluoromethane	524.2		36.7%	82.3%
Diethyl ether	524.2		11.9%	-21.3%
Ethyl methacrylate	524.2		-35.7%	-8.9%
Ethylbenzene	502.2	PID	-11.5%	44.6%
Ethylbenzene	524.2		20.4%	-25.4%
Hardness	130.2		39.1%	92.8%
Hexachlorobutadiene	502.2	ELCD	-114.4%	19.4%
Hexachlorobutadiene	524.2		-22.3%	13.3%
Hexachloroethane	524.2		14.8%	-17.7%
Hexchlobutadiene+naphthalene	502.2	PID	-82.3%	-25.9%
Iron	1620		152.7%	133.1%
Isopropylbenzene	502.2	PID	-10.4%	25.3%
Isopropylbenzene	524.2		11.9%	199.2%
Lead	1620		1.2%	13.1%

Table 5. Percent Differences of Quantitation Limits to the EPA/ACS QL for the Episode 6000 Dataset

Analyte	Method	Procedure	ISO LOQ/ML	SL-IQE/ML
Lead	200.8	ICP/MS	-145.2%	-98.0%
M+p xylene	502.2	PID	-98.3%	10.6%
M+p xylene	524.2		-17.7%	199.2%
Magnesium	1620		-9.6%	-60.7%
Manganese	1620		-86.2%	-26.9%
Manganese	200.8	ICP/MS	28.0%	84.1%
Mercury	200.8	ICP/MS	94.2%	63.6%
Methacrylonitrile	524.2		6.4%	180.1%
Methyl iodide	524.2		7.3%	-18.1%
Methyl tert-butyl ether	524.2		-31.2%	20.1%
Methyla crylate	524.2		-3.5%	-31.7%
Methylene chloride	502.2	ELCD	N/A	169.4%
Methylene chloride	524.2		55.6%	73.6%
Methylm ethacrylate	524.2		-89.5%	181.6%
Molybdenum	1620		-2.5%	-27.3%
Molybdenum	200.8	ICP/MS	135.3%	193.5%
N-butylbenzene	502.2	PID	24.5%	152.7%
N-butylbenzene	524.2		42.2%	29.5%
N-propylbenzene	502.2	PID	-44.1%	-7.1%
N-propylbenzene	524.2		9.9%	198.7%
Naphthalene	524.2		-8.2%	-59.5%
Nickel	1620		-40.3%	-39.2%
Nickel	200.8	ICP/MS	-54.2%	-92.9%
O-xylene	524.2		21.3%	-21.4%
O-xylene+styrene	502.2	PID	4.9%	-10.0%
P-isopropitol+1,4-dcb	502.2	PID	45.6%	78.1%
Pentachloroethane	524.2		-183.5%	-113.6%
Sec-butylbenzene	502.2	PID	-3.4%	-24.2%
Sec-butylbenzene	524.2		22.8%	-5.2%
Selenium	1620		63.5%	89.4%
Selenium	200.8	ICP/MS	46.8%	70.6%
Silver	1620		-17.8%	25.5%

Table 5. Percent Differences of Quantitation Limits to the EPA/ACS QL for the Episode 6000 Dataset

Analyte	Method	Procedure	ISO LOQ/ML	SL-IQE/ML
Silver	200.8	ICP/MS	-59.2%	94.7%
Sodium	1620		22.8%	51.2%
Styrene	524.2		6.9%	-20.6%
Tert-butylbenzene	502.2	PID	19.2%	67.9%
Tert-butylbenzene	524.2		-44.8%	-30.6%
Tetrachloroethene	502.2	ELCD	40.9%	83.5%
Tetrachloroethene	502.2	PID	19.6%	115.8%
Tetrachloroethene	524.2		61.5%	197.4%
Thallium	1620		60.8%	33.3%
Thallium	200.8	ICP/MS	3.8%	16.3%
Thorium	200.8	ICP/MS	90.3%	74.9%
Tin	1620		-7.9%	-6.1%
Titanium	1620		4.0%	-33.7%
Toluene	502.2	PID	-21.1%	-3.0%
Toluene	524.2		-57.9%	-9.1%
Total phosphorus	365.2		17.2%	39.9%
Total suspended solids	160.2		0.2%	29.5%
Trans-1,2-dichloroethene	502.2	ELCD	15.7%	-4.9%
Trans-1,2-dichloroethene	524.2		33.7%	41.7%
Trans-1,3-dichloropropene	502.2	ELCD	-101.5%	174.3%
Trans-1,3-dichloropropene	502.2	PID	19.8%	-13.4%
Trans-1,3-dichloropropene	524.2		-49.5%	8.7%
Trans-1,4-dichloro-2-butene	524.2		-10.4%	175.1%
Trichloroethene	502.2	ELCD	-144.3%	193.8%
Trichloroethene	502.2	PID	7.8%	120.2%
Trichloroethene	524.2		34.6%	-17.8%
Trichlorofluoromethane	502.2	ELCD	105.3%	161.3%
Trichlorofluoromethane	524.2		33.0%	198.1%
Uranium	200.8	ICP/MS	-33.2%	2.6%
Vanadium	1620		7.6%	19.6%
Vanadium	200.8	ICP/MS	27.1%	-3.4%
Vinyl chloride	502.2	ELCD	-116.4%	156.7%

Table 5. Percent Differences of Quantitation Limits to the EPA/ACS QL for the Episode 6000 Dataset

Analyte	Method	Procedure	ISO LOQ/ML	SL-IQE/ML
Vinyl chloride	524.2		-35.7%	9.3%
Weak cyanide	1677	WADCN	-80.5%	-20.8%
Xylene (total)	524.2		28.4%	199.7%
Yttrium	1620		27.2%	56.8%
Zinc	1620		-4.3%	4.4%
Zinc	200.8	ICP/MS	7.1%	111.4%

Note: ELCD or PID in the Procedure column indicates the photo-ionization detector (PID) or electrolytic conductivity detector (ELCD) in EPA Method 502.2

Summary Statistics for Table 5

	ISO LOQ/QL	SL-IQE/QL
Minimum	-194.7%	-174.7%
25th percentile	-35.0%	-18.1%
Median	-4.2%	19.6%
75th percentile	23.0%	111.4%
Maximum	152.7%	199.7%

Comparison	Sign Test p-value	Wilcoxon p-value
LOQ vs. QL	0.390	0.043
SL-IQE vs. QL	0.0001	<0.0001

Comparison	# analytes	Median % Difference	Sign Test p-value	Wilcoxon p-value
SL-IQE vs. QL (constant model used for SL-IQE)	32	179.6%	<0.0001	<0.0001
SL-IQE vs. QL (Linear model used for SL-IQE)	65	67.9%	<0.0001	<0.0001
SL-IQE vs. QL (Hybrid model used for SL-IQE)	100	-7.7%	0.533	0.160

Table 6. Detection and Quantitation Limits for EPA Methods 1631 and 1638 as Computed by EPA and by EPRI (ng/L)

Element	Ambient WQC[2]	Detection limits			Quantitation limits		
		MDL in Method	IDE computed by		ML in Method	IQE computed by	
			EPA	EPRI		EPA	EPRI
Antimony	14000	9.7	170	110	20	270	270
Cadmium	370	25	160	150	100	540	380
Copper	2400	87	800	770	200	3800	3000
Lead	540	15	140	160	50	420	370
Mercury	12	0.2	0.81	0.43	0.5	0.55	1.6
Nickel	8200	330	230	130	1000	15000	330
Selenium	5000	450	810	600	1000	630	720
Silver	320	29	440	---	100	5500	---
Thallium	1700	7.9	28	20	20	88	50
Zinc	32000	140	1800	2100	500	21000	26100

[1] Mercury determined by EPA Method 1631; as others by EPA Method 1638

[2] Lowest ambient water quality criterion (WQC) in the National Toxics Rule (40 CFR 131.36)

Table 7. Comparison of IDEs and IQEs resulting from all model types for EPA Methods 1631 and 1638

Calculated IDEs					
Analyte	IDE, Based on Given Model				RSD (%)
	Constant	Linear	Exponential	Hybrid	
Antimony	2500	-80 [1]	170	100	148%
Cadmium	1200	130	160	150	129%
Copper	2700	1000	800	720	72%
Lead	400	150	140	150	61%
Mercury	8.3	0.058	0.81	0.52	162%
Nickel	7000	-48 [1]	230	120	161%
Selenium	4500	720	810	530	117%
Silver	2500	710	440	650	89%
Thallium	230	22	28	17	140%
Zinc	10,000	1600	1800	1700	110%
Calculated IQEs (10%)					
Analyte	IQE, Based on Given Model				RSD(%)
	Constant	Linear	Exponential	Hybrid	
Amimony	5400	-570 [1]	380	270	145%
Cadmium	2600	540	380	380	112%
Copper	5900	3800	2100	2300	50%
Lead	860	420	340	330	52%
Mercury	18	0.55	2.1	1.6	150%
Nickel	15,000	-160 [1]	500	270	190%
Selenium	9600	7600	2200	630 [3]	86%
Silver	5500	1500 [4]	1500	undefined [2]	82%
Thallium	500	88	67	47	124%
Zinc	22,000	21,000	4800	6700	67%

[1] Negative due to negative intercept estimate in precision model.
[2] IDE or IQE did not converge to a single value for estimated models.
[3] IQE 10% undefined, IQE 20% reported
[4] IQE 10% negative, IQE 20% reported

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
1,1,1,2-tetrachloroethane	502.2	ELCD	0.034	0.011	-99.6%	Exponential	Linear
1,1,1,2-tetrachloroethane	524.2		0.244	0.170	-35.8%	Exponential	Exponential
1,1,1-trichloroethane	502.2	ELCD	0.041	0.044	6.2%	Exponential	Exponential
1,1,1-trichloroethane	524.2		0.308	0.035	-159.4%	Exponential	Hybrid
1,1,2,2-tce+1,2,3-tcp	502.2	ELCD	0.179	3.548	180.8%	Exponential	Constant
1,1,2,2-tetrachloroethane	524.2		0.436	0.538	20.8%	Exponential	Exponential
1,1,2-trichloroethane	502.2	ELCD	0.032	0.013	-86.7%	Exponential	Linear
1,1,2-trichloroethene	524.2		0.319	0.229	-32.8%	Exponential	Exponential
1,1-dichloroethane	502.2	ELCD	0.083	0.036	-78.8%	Exponential	Exponential
1,1-dichloroethene	524.2		0.229	0.084	-92.7%	Exponential	Exponential
1,1-dichloroethene	502.2	ELCD	0.234	0.120	-64.0%	Exponential	Exponential
1,1-dichloroethene	524.2		0.335	0.080	-122.6%	Exponential	Hybrid
1,1-dichloropropanone	524.2		6.372	8.941	33.6%	Exponential	Exponential
1,1-dichloropropene	524.2		0.287	4.435	175.7%	Exponential	Constant [1]
1,2,3-trichlorobenzene	502.2	ELCD	0.134	0.169	23.1%	Exponential	Constant
1,2,3-trichlorobenzene	502.2	PID	0.115	0.069	-49.9%	Exponential	Exponential
1,2,3-trichlorobenzene	524.2		0.275	0.150	-59.2%	Exponential	Exponential
1,2,3-trichloropropane	524.2		1.263	16.238	171.1%	Exponential	Constant [1]
1,2,4-trichlorobenzene	502.2	ELCD	0.088	0.100	13.1%	Exponential	Constant
1,2,4-trichlorobenzene	502.2	PID	0.124	0.075	-48.9%	Exponential	Exponential
1,2,4-trichlorobenzene	524.2		0.224	0.115	-64.6%	Exponential	Exponential
1,2,4-trimethylbenzene	502.2	PID	0.125	0.143	12.8%	Exponential	Constant
1,2,4-trimethylbenzene	524.2		0.144	0.059	-84.6%	Exponential	Exponential
1,2-dibromo-3-chloropropane	524.2		1.749	0.432	-120.8%	Exponential	Hybrid
1,2-dibromoethane	502.2	ELCD	0.164	0.025	-147.8%	Exponential	Linear
1,2-dibromoethane	524.2		0.326	0.316	-3.1%	Exponential	Exponential
1,2-dichlorobenzene	502.2	ELCD	0.065	0.057	-13.4%	Exponential	Linear
1,2-dichlorobenzene	502.2	PID	0.148	0.077	-62.5%	Exponential	Exponential
1,2-dichlorobenzene	524.2		0.130	0.069	-61.3%	Exponential	Exponential
1,2-dichloroethane	502.2	ELCD	0.042	0.026	-48.3%	Exponential	Exponential

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
1,2-dichloroethane	524.2		0.258	0.211	-19.9%	Exponential	Exponential
1,2-dichloropropane	502.2	ELCD	0.043	0.087	67.5%	Exponential	Constant
1,2-dichloropropane	524.2		0.247	0.221	-11.1%	Exponential	Exponential
1,3,5-tmb+4-chlorotoluene	502.2	PID	0.114	0.141	21.4%	Exponential	Constant
1,3,5-trimethylbenzene	524.2		0.135	0.049	-94.1%	Exponential	Exponential
1,3-dichlorobenzene	502.2	ELCD	0.118	0.615	135.5%	Exponential	Constant
1,3-dichlorobenzene	502.2	PID	0.126	0.197	43.9%	Exponential	Constant
1,3-dichlorobenzene	524.2		0.143	0.038	-116.4%	Exponential	Exponential
1,3-dichloropropane	502.2	ELCD	0.047	0.020	-81.3%	Exponential	Exponential
1,3-dichloropropane	524.2		0.202	0.122	-49.2%	Exponential	Exponential
1,4-dichlorobenzene	502.2	ELCD	0.061	0.040	-40.5%	Exponential	Linear
1,4-dichlorobenzene	524.2		0.140	0.051	-93.7%	Exponential	Exponential
1-chlorobutane	524.2		0.220	0.061	-113.5%	Exponential	Linear
2,2-dichloropropane	524.2		0.691	0.122	-139.9%	Exponential	Hybrid
2-butanone	524.2		0.833	1.441	53.5%	Exponential	Exponential
2-chlorotoluene	502.2	ELCD	0.175	0.117	-40.2%	Exponential	Exponential
2-chlorotoluene	502.2	PID	0.230	0.409	56.2%	Exponential	Constant
2-chlorotoluene	524.2		0.136	0.039	-111.2%	Exponential	Exponential
2-hexanone	524.2		0.902	0.904	0.3%	Exponential	Exponential
2-nitropropane	524.2		1.082	9.354	158.5%	Exponential	Constant
4-chlorotoluene	502.2	ELCD	0.149	0.145	-3.2%	Exponential	Linear
4-chlorotoluene	524.2		0.123	0.038	-105.5%	Exponential	Exponential
4-isopropyltoluene	524.2		0.117	0.038	-101.3%	Exponential	Exponential
4-methyl-2-pentanone	524.2		1.195	1.088	-9.3%	Exponential	Exponential
Acetone	524.2		2.120	30.183	173.8%	Exponential	Constant
Acrylonitrile	524.2		1.333	1.077	-21.3%	Exponential	Exponential
Allyl Chloride	524.2		0.229	0.073	-103.6%	Exponential	Hybrid
Aluminum	1620		206.975	73.421	-95.3%	Constant	Constant
Aluminum	200.8		12.747	22.654	56.0%	Exponential	Constant
Ammonia as Nitrogen[2]	350.3		0.014	0.040	94.0%	Exponential	Constant

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
Antimony	1620		4.260	6.467	41.2%	Constant	Linear
Antimony	200.8		0.019	0.304	176.5%	Exponential	Constant
Arsenic	1620		1.410	2.268	46.6%	Exponential	Constant
Arsenic	200.8		0.366	0.374	2.1%	Exponential	Exponential
Barium	1620		1.837	1.624	-12.3%	Constant	Constant
Barium	200.8		0.084	0.073	-13.7%	Exponential	Constant
Benzene	502.2	PID	0.079	0.061	-25.0%	Exponential	Exponential
Benzene	524.2		0.125	0.030	-122.6%	Exponential	Exponential
Beryllium	1620		0.448	0.438	-2.2%	Exponential	Exponential
Beryllium	200.8		0.024	0.017	-34.2%	Exponential	Constant
Boron	1620		21.161	22.333	5.4%	Exponential	Exponential
Bromobenzene	502.2	ELCD	0.765	0.348	-75.0%	Linear	Exponential
Bromobenzene	502.2	PID	0.050	0.025	-65.4%	Exponential	Exponential
Bromobenzene	524.2		0.211	0.165	-24.1%	Exponential	Exponential
Bromochloromethane	502.2	ELCD	0.482	0.044	-166.9%	Linear	Exponential
Bromochloromethane	524.2		0.345	0.507	38.1%	Exponential	Exponential
Bromodichloromethane	502.2	ELCD	0.075	0.026	-95.5%	Exponential	Exponential
Bromodichloromethane	524.2		0.205	0.088	-79.7%	Exponential	Exponential
Bromoform	502.2	ELCD	1.513	0.025	-193.5%	Constant	Linear
Bromoform	524.2		0.400	0.336	-17.4%	Exponential	Exponential
Bromomethane	502.2	ELCD	7.293	0.760	-162.3%	Constant	Exponential
Bromomethane	524.2		0.280	0.154	-57.8%	Exponential	Linear
Cadmium	1620		0.191	0.211	9.8%	Exponential	Exponential
Cadmium	200.8		0.022	0.016	-33.8%	Exponential	Constant
Calcium	1620		41.358	53.375	25.4%	Linear	Constant
Carbon Disulfide	524.2		0.239	0.087	-93.6%	Exponential	Linear
Carbon Tetrachloride	524.2		0.314	0.174	-57.3%	Exponential	Linear
Carbontet+1,1-dcp	502.2	ELCD	0.072	0.061	-15.5%	Exponential	Exponential
Chloroacetonitrile	524.2		1.569	2.079	28.0%	Exponential	Exponential
Chlorobenzene	502.2	ELCD	0.460	0.064	-151.5%	Linear	Exponential

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
Chlorobenzene	502.2	PID	0.064	0.059	-7.8%	Exponential	Exponential
Chlorobenzene	524.2		0.133	0.034	-118.1%	Exponential	Exponential
Chloroethane	502.2	ELCD	2.598	0.096	-185.7%	Constant	Linear
Chloroethane	524.2		0.395	0.303	-26.3%	Exponential	Exponential
Chloroform	502.2	ELCD	0.032	0.008	-117.3%	Exponential	Linear
Chloroform	524.2		0.225	0.104	-73.4%	Exponential	Exponential
Chloromethane	502.2	ELCD	0.250	0.520	70.3%	Exponential	Constant
Chloromethane	524.2		0.253	0.150	-51.2%	Exponential	Exponential
Chromium	1620		0.496	0.759	41.8%	Exponential	Constant
Chromium	200.8		0.408	0.491	18.5%	Linear	Constant
Cis-1,2-dce+2,2-dcp	502.2	ELCD	0.055	0.039	-35.0%	Exponential	Exponential
Cis-1,2-dichloroethene	524.2		0.234	0.201	-15.2%	Exponential	Exponential
Cis-1,3-dichloropropene	502.2	ELCD	0.074	0.024	-102.4%	Exponential	Exponential
Cis-1,3-dichloropropene	502.2	PID	0.082	0.111	30.2%	Exponential	Exponential
Cis-1,3-dichloropropene	524.2		0.173	0.119	-37.1%	Exponential	Exponential
Cobalt	1620		16.463	12.267	-29.2%	Exponential	Exponential
Cobalt	200.8		0.074	0.001	-195.2%	Constant	Exponential
Copper	1620		21.189	15.897	-28.5%	Constant	Constant
Copper	200.8		0.798	0.905	12.6%	Constant	Constant
Dibromochloromethane	502.2	ELCD	0.436	0.394	-10.1%	Linear	Constant
Dibromochloromethane	524.2		0.287	0.203	-34.3%	Exponential	Exponential
Dibromomethane	502.2	ELCD	0.460	0.298	-42.8%	Linear	Constant
Dibromomethane	524.2		0.388	0.439	12.5%	Exponential	Exponential
Dichlorodifluoromethane	502.2	ELCD	0.240	1.225	134.5%	Exponential	Constant
Dichlorodifluoromethane	524.2		0.560	0.591	5.4%	Exponential	Exponential
Diethyl Ether	524.2		0.376	0.330	-12.9%	Exponential	Exponential
Ethyl Methacrylate	524.2		0.273	0.259	-5.2%	Exponential	Exponential
Ethylbenzene	502.2	PID	0.078	0.050	-44.2%	Exponential	Exponential
Ethylbenzene	524.2		0.198	0.107	-59.5%	Exponential	Exponential
Hardness [2]	130.2		2.258	4.886	73.6%	Exponential	Constant

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
Hexachlorobutadiene	502.2	ELCD	0.094	0.073	-24.8%	Exponential	Linear
Hexachlorobutadiene	524.2		0.308	0.237	-26.0%	Exponential	Exponential
Hexachloroethane	524.2		0.288	0.260	-10.1%	Exponential	Exponential
Hexchlobutadiene+naphthalene	502.2	PID	0.597	0.592	-1.0%	Exponential	Constant
Iron	1620		373.590	1064.987	96.1%	Linear	Constant
Isopropylbenzene	502.2	PID	0.060	0.041	-37.0%	Exponential	Exponential
Isopropylbenzene	524.2		0.120	0.037	-104.7%	Exponential	Exponential
Lead	1620		2.423	2.951	19.6%	Exponential	Constant
Lead	200.8		0.204	2.872	173.5%	Exponential	Constant
M+p Xylene	502.2	PID	0.121	0.119	-1.2%	Exponential	Constant
M+p Xylene	524.2		0.142	0.031	-127.3%	Exponential	Exponential
Magnesium	1620		105.998	184.221	53.9%	Exponential	Constant
Manganese	1620		6.808	4.548	-39.8%	Constant	Constant
Manganese	200.8		0.109	0.077	-34.7%	Constant	Constant
Mercury	200.8		0.027	0.014	-63.8%	Exponential	Hybrid
Methacrylonitrile	524.2		0.718	0.552	-26.2%	Exponential	Hybrid
Methyl Iodide	524.2		0.193	0.109	-55.5%	Exponential	Exponential
Methyl Tert-butyl Ether	524.2		0.225	0.173	-26.3%	Exponential	Exponential
Methylacrylate	524.2		0.601	0.569	-5.5%	Exponential	Exponential
Methylene Chloride	502.2	ELCD	2.841	-1.381	-578.5%	Constant	Constant
Methylene Chloride	524.2		0.314	0.158	-66.1%	Exponential	Exponential
Methylmethacrylate	524.2		0.535	0.382	-33.3%	Exponential	Linear
Molybdenum	1620		3.034	6.028	66.1%	Exponential	Constant
Molybdenum	200.8		0.271	0.006	-191.8%	Constant	Constant
N-butylbenzene	502.2	PID	0.152	0.056	-93.0%	Exponential	Exponential
N-butylbenzene	524.2		0.092	0.105	13.9%	Exponential	Constant
N-propylbenzene	502.2	PID	25.560	41.908	48.5%	Exponential	Constant
N-propylbenzene	524.2		0.083	0.070	-16.1%	Exponential	Constant
Naphthalene	524.2		0.141	0.052	-91.4%	Exponential	Linear
Nickel	1620		0.284	0.052	-137.6%	Exponential	Hybrid

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
Nickel	200.8		0.186	0.194	4.1%	Exponential	Exponential
o-xylene	524.2		0.198	0.082	-82.9%	Exponential	Exponential
o-xylene+styrene	502.2	PID	0.116	0.151	26.8%	Exponential	Constant
P-Isopropotol+1,4-deb	502.2	PID	0.408	0.437	7.0%	Exponential	Linear
Pentachloroethane	524.2		0.159	0.150	-5.8%	Exponential	Constant
Sec-butylbenzene	502.2	PID	0.081	0.057	-35.3%	Exponential	Exponential
Sec-butylbenzene	524.2		0.140	0.040	-111.6%	Exponential	Exponential
Selenium	1620		1.975	1.801	-9.2%	Exponential	Exponential
Selenium	200.8		0.416	0.342	-19.5%	Exponential	Exponential
Silver	1620		10.668	11.589	8.3%	Exponential	Constant
Silver	200.8		0.012	-0.084	269.8%	Exponential	Constant [1]
Sodium	1620		138.768	140.860	1.5%	Exponential	Exponential
Styrene	524.2		0.141	0.048	-98.2%	Exponential	Exponential
Tert-butylbenzene	502.2	PID	0.074	0.051	-35.9%	Exponential	Exponential
Tert-butylbenzene	524.2		0.186	0.057	-106.6%	Exponential	Exponential
Tetrachloroethene	502.2	ELCD	0.061	0.054	-11.0%	Exponential	Exponential
Tetrachloroethene	502.2	PID	0.156	0.103	-40.6%	Exponential	Linear
Tetrachloroethene	524.2		0.469	0.550	15.9%	Exponential	Linear
Thallium	1620		1.153	1.249	8.0%	Exponential	Linear
Thallium	200.8		0.001	0.000	-76.1%	Exponential	Exponential
Thorium	200.8		0.001	0.000	-93.4%	Exponential	Constant
Tin	1620		3.932	4.651	16.8%	Exponential	Exponential
Thanium	1620		5.376	20.828	117.9%	Exponential	Constant
Toluene	502.2	PID	0.064	0.064	-1.3%	Exponential	Constant
Toluene	524.2		0.146	0.558	117.1%	Exponential	Constant [1]
Total Phosphorus [2]	365.2		0.013	0.011	-18.1%	Exponential	Exponential
Total Suspended Solids [2]	160.2		3.005	2.370	-23.6%	Exponential	Exponential
Trans-1,2-dichloroethene	502.2	ELCD	0.081	0.066	-21.7%	Exponential	Linear
Trans-1,2-dichloroethene	524.2		0.300	0.075	-119.7%	Exponential	Hybrid
Trans-1,3-dichloropropene	502.2	ELCD	0.098	0.033	-98.9%	Exponential	Exponential

Table 8. Comparison of 16-point and 5-point Single-laboratory IDEs (SL-IDEs) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE (16)	SL-IDE (5)	Percent Difference	SL-IDE 16 Model	SL-IDE 5 Model
Trans-1,3-dichloropropene	502.2	PID	0.092	0.116	22.7%	Exponential	Exponential
Trans-1,3-dichloropropene	524.2		0.223	0.132	-51.1%	Exponential	Exponential
Trans-1,4-dichloro-2-butene	524.2		1.250	1.448	14.7%	Exponential	Exponential
Trichloroethene	502.2	ELCD	0.059	0.020	-99.6%	Exponential	Exponential
Trichloroethene	502.2	PID	0.097	0.089	-8.5%	Exponential	Exponential
Trichloroethene	524.2		0.332	0.344	3.6%	Exponential	Linear
Trichlorofluoromethane	502.2	ELCD	2.079	0.688	-100.5%	Constant	Constant
Trichlorofluoromethane	524.2		0.384	0.384	0.1%	Exponential	Exponential
Uranium	200.8		0.000	0.000	-70.8%	Exponential	Exponential
Vanadium	1620		10.630	9.082	-15.7%	Exponential	Exponential
Vanadium	200.8		0.864	1.023	16.9%	Exponential	Linear
Vinyl Chloride	502.2	ELCD	3.672	0.387	-161.9%	Constant	Linear
Vinyl Chloride	524.2		0.365	0.188	-63.8%	Exponential	Linear
WAD Cyanide	1677		0.701	1.296	59.6%	Linear	Constant
Xylene (Total)	524.2		0.128	0.029	-126.9%	Exponential	Exponential
Yttrium	1620		3.247	13.972	124.6%	Exponential	Constant
Zinc	1620		4.500	6.943	42.7%	Exponential	Constant
Zinc	200.8		1.598	5.245	106.6%	Exponential	Constant

Note: ELCD or PID in the Procedure column indicates the photoionization detector (PID) or electrolytic conductivity detector (ELCD) in EPA Method 502.2

[1] Original model picked was Hybrid, but failed to converge

[2] Results reported as mg/L

Summary Statistics for Table 8

	SL-IDE(16) vs. SL-IDE (5) (all analytes)	SL-IDE(16) vs. SL-IDE (5) (same model used)	SL-IDE(16) vs. SL-IDE (5) (different models used)
Number of Analytes	198	108	90
Minimum:	-578.5%	-578.5%	-195.2%
25th percentile:	-79.5%	-80.1%	-72.2%
Median:	-24.9%	-35.6%	1.3%
75th percentile:	12.8%	-9.3%	55.5%
Maximum:	269.8%	53.5%	269.8%

	Number of analytes	Median % Difference	Sign Test p-value	Wilcoxon p-value
SL-IDE (16) vs. SL-IDE (5) (all analytes)	198	-24.9%	<0.0001	<0.0001
SL-IDE(16) vs. SL-IDE (5) (same model used)	108	-35.6%	<0.0001	<0.0001
SL-IDE(16) vs. SL-IDE (5) (different models used)	90	1.3%	>0.999	0.847

Table 9. Comparison of 16-point and 5-point Single-laboratory IQEs at 10% RSD (SL-IQEs 10%) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE10% (16)	SL-IQE10% (5)	Percent Difference	SL-IQE Model (16)	SL-IQE Model (5)
1,1,1,2-tetrachloroethane	502.2	ELCD	0.030	0.048	45.7%	Hybrid	Linear
1,1,1,2-tetrachloroethane	524.2		0.181	0.320	55.3%	Hybrid	Linear
1,1,1-trichloroethane	502.2	ELCD	0.830	0.055	-175.2%	Linear	Hybrid
1,1,1-trichloroethane	524.2		0.240	0.081	-98.6%	Hybrid	Hybrid
1,1,2,2-tcs+1,2,3-tcp	502.2	ELCD	5.514	6.984	23.5%	Constant	Constant
1,1,2,2-tetrachloroethane	524.2		0.569	0.942	49.4%	Hybrid	Linear
1,1,2-trichloroethane	502.2	ELCD	0.060	0.046	-26.2%	Linear	Linear
1,1,2-trichloroethane	524.2		0.290	0.344	17.1%	Hybrid	Linear
1,1-dichloroethane	502.2	ELCD	0.527	0.058	-160.5%	Linear	Hybrid
1,1-dichloroethane	524.2		0.115	0.099	-14.8%	Hybrid	Hybrid
1,1-dichloroethene	502.2	ELCD	3.796	0.305	-170.3%	Linear	Hybrid
1,1-dichloroethene	524.2		0.129	0.199	42.6%	Hybrid	Hybrid
1,1-dichloropropanone	524.2		12.705	16.447	25.7%	Linear	Hybrid
1,1-dichloropropene	524.2		0.180	9.106[6]	192.2%	Hybrid	Constant
1,2,3-trichlorobenzene	502.2	ELCD	0.851	0.341	-85.6%	Linear	Constant
1,2,3-trichlorobenzene	502.2	PID	0.248	0.246	-0.9%	Hybrid	Hybrid
1,2,3-trichlorobenzene	524.2		0.216	0.147	-38.1%	Hybrid	Linear
1,2,3-trichloropropane	524.2		11.316	33.343[6]	98.6%	Linear	Constant
1,2,4-trichlorobenzene	502.2	ELCD	0.401	0.202	-65.9%	Linear	Constant
1,2,4-trichlorobenzene	502.2	PID	0.439	0.207	-72.0%	Linear	Hybrid
1,2,4-trichlorobenzene	524.2		0.141	3.760	185.6%	Hybrid	Constant
1,2,4-trimethylbenzene	502.2	PID	0.653	0.293	-76.2%	Linear	Constant
1,2,4-trimethylbenzene	524.2		20.896	0.119	-197.7%	Constant	Linear
1,2-dibromo-3-chloropropane	524.2		71.182[6]	0.877	-195.1%	Constant	Hybrid
1,2-dibromoethane	502.2	ELCD	0.592	0.065	-160.2%	Linear	Linear
1,2-dibromoethane	524.2		0.417	0.579	32.5%	Hybrid	Linear
1,2-dichlorobenzene	502.2	ELCD	0.183	0.109[3]	-50.9%	Linear	Linear
1,2-dichlorobenzene	502.2	PID	0.346	0.123	-94.7%	Hybrid	Hybrid
1,2-dichlorobenzene	524.2		0.085	0.117	32.3%	Hybrid	Linear
1,2-dichloroethane	502.2	ELCD	0.065	0.727[6]	167.2%	Hybrid	Constant
1,2-dichloroethane	524.2		0.222	0.327	38.4%	Hybrid	Linear
1,2-dichloropropane	502.2	ELCD	0.102	0.178	54.1%	Linear	Constant
1,2-dichloropropane	524.2		0.196	0.219	10.9%	Hybrid	Linear
1,3,5-tmb+4-chlorotoluene	502.2	PID	0.189	0.289	41.7%	Hybrid	Constant
1,3,5-trimethylbenzene	524.2		23.744	0.086	-198.6%	Constant	Linear
1,3-dichlorobenzene	502.2	ELCD	0.936	1.239	27.9%	Linear	Constant
1,3-dichlorobenzene	502.2	PID	0.465	0.404	-14.2%	Linear	Constant
1,3-dichlorobenzene	524.2		0.076	0.081	7.0%	Hybrid	Hybrid
1,3-dichloropropane	502.2	ELCD	0.054	0.448	157.0%	Linear	Constant
1,3-dichloropropane	524.2		0.139	0.154	10.0%	Hybrid	Hybrid
1,4-dichlorobenzene	502.2	ELCD	0.101	0.100	-1.3%	Hybrid	Linear
1,4-dichlorobenzene	524.2		0.078	0.068	-14.1%	Hybrid	Linear
1-chlorobutane	524.2		29.943	0.170	-197.7%	Constant	Linear
2,2-dichloropropane	524.2		38.009	0.361	-196.2%	Constant	Hybrid
2-butanone	524.2		0.893	39.665	191.2%		Constant
2-chlorotoluene	502.2	ELCD	0.493	0.357	-32.1%	Hybrid	Linear

Table 9. Comparison of 16-point and 5-point Single-laboratory IQEs at 10% RSD (SL-IQEs 10%) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE10% (16)	SL-IQE10% (5)	Percent Difference	SL-IQE Model (16)	SL-IQE Model (5)
2-chlorotoluene	502.2	PID	0.849	0.806	-5.2%	Hybrid	Constant
2-chlorotoluene	524.2		0.053	0.044	-19.1%	Hybrid	Linear
2-hexanone	524.2		0.442	61.796	197.2%	Hybrid	Constant
2-nitropropane	524.2		0.590	17.783	187.2%	Hybrid	Constant
4-chlorotoluene	502.2	ELCD	0.142[1]	0.485	109.4%	Hybrid	Linear
4-chlorotoluene	524.2		23.810	0.837	-186.4%	Constant	Constant
4-isopropyltoluene	524.2		0.016	1.194	194.6%	Hybrid	Constant
4-methyl-2-pentanone	524.2		1.785	14.514	156.2%	Hybrid	Constant
Acetone	524.2		2.741	59.415	182.4%	Hybrid	Constant
Acrylonitrile	524.2		28.056	19.275	-37.1%	Constant	Constant
Allyl Chloride	524.2		29.674	0.164	-197.8%	Constant	Hybrid
Aluminum	1620		464.069	144.530	-105.0%	Constant	Constant
Aluminum	200.8	ICP/MS	29.684	47.196	45.6%	Hybrid	Constant
Ammonia as Nitrogen[2]	350.3		0.035	0.082	78.8%	Hybrid	Constant
Antimony	1620		9.551	8.364[5]	-3.6%	Constant	Constant
Antimony	200.8	ICP/MS	0.034	0.633	179.8%	Hybrid	Constant
Arsenic	1620		3.097	4.656	40.2%	Hybrid	Constant
Arsenic	200.8	ICP/MS	0.798	0.847	6.1%	Hybrid	Hybrid
Barium	1620		4.118	3.334	-21.1%	Constant	Constant
Barium	200.8	ICP/MS	0.211	0.153	-32.1%	Linear	Constant
Benzene	502.2	PID	0.182	0.130	-33.2%	Linear	Linear
Benzene	524.2		0.044	0.029	-41.0%	Hybrid	Linear
Beryllium	1620		0.980	0.985	0.6%	Hybrid	Linear
Beryllium	200.8	ICP/MS	0.044	0.036	-19.9%	Hybrid	Constant
Boron	1620		51.134	46.392	-9.7%	Linear	Hybrid
Bromobenzene	502.2	ELCD	3.529	29.488	157.2%	Linear	Linear
Bromobenzene	502.2	PID	0.100	0.057	-55.4%	Linear	Hybrid
Bromobenzene	524.2		0.140	0.187	28.7%	Hybrid	Hybrid
Bromochloromethane	502.2	ELCD	1.598	0.057	-186.1%	Linear	Hybrid
Bromochloromethane	524.2		0.368	0.592	46.5%	Hybrid	Hybrid
Bromodichloromethane	502.2	ELCD	0.424	0.465	9.1%	Linear	Constant
Bromodichloromethane	524.2		0.128	0.111	-13.8%	Hybrid	Linear
Bromoform	502.2	ELCD	3.393	0.068	-192.1%	Constant	Linear
Bromoform	524.2		0.482	0.406	-17.1%	Hybrid	Hybrid
Bromomethane	502.2	ELCD	16.351	2.195	-152.7%	Constant	Hybrid
Bromomethane	524.2		0.226	0.412	58.4%	Hybrid	Linear
Cadmium	1620		0.410	0.400	-2.6%	Hybrid	Linear
Cadmium	200.8	ICP/MS	0.063	0.033	-63.4%	Hybrid	Constant
Calcium	1620		99.975	109.600	9.2%	Linear	Constant
Carbon Disulfide	524.2		0.101	0.268	90.3%	Hybrid	Linear
Carbon Tetrachloide	524.2		0.140	0.520	115.1%	Hybrid	Linear
Carbontet+1,1-dcp	502.2	ELCD	0.069	1.553	183.1%	Hybrid	Constant
Chloroacetonitrile	524.2		3.310	31.753	162.2%	Hybrid	Constant
Chlorobenzene	502.2	ELCD	1.766	1.558	-12.5%	Linear	Constant
Chlorobenzene	502.2	PID	0.119	0.034[3]	-110.6%	Hybrid	Linear
Chlorobenzene	524.2		0.059	0.831	173.3%	Hybrid	Constant

Table 9. Comparison of 16-point and 5-point Single-laboratory IQEs at 10% RSD (SL-IQEs 10%) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE10% (16)	SL-IQE10% (5)	Percent Difference	SL-IQE Model (16)	SL-IQE Model (5)
Chloroethane	502.2	ELCD	5.826	0.644	-160.2%	Constant	Linear
Chloroethane	524.2		0.255	0.207	-20.8%	Hybrid	Hybrid
Chloroform	502.2	ELCD	0.025	0.033	26.1%	Linear	Linear
Chloroform	524.2		0.121	0.092	-27.7%	Hybrid	Linear
Chloromethane	502.2	ELCD	1.734	1.049	-49.2%	Linear	Constant
Chloromethane	524.2		0.141	0.191	30.4%	Hybrid	Linear
Chromium	1620		1.259	1.558	21.2%	Linear	Constant
Chromium	200.8	ICP/MS	1.028	1.022	-0.6%	Linear	Constant
Cis-1,2-dce+2,2-dcp	502.2	ELCD	0.039	1.055	185.7%	Hybrid	Constant
Cis-1,2-dichloroethene	524.2		0.144	0.151	4.9%	Hybrid	Hybrid
Cis-1,3-dichloropropene	502.2	ELCD	0.415	0.447[6]	7.4%	Linear	Constant
Cis-1,3-dichloropropene	502.2	PID	0.017[1]	0.226	172.0%	Hybrid	Linear
Cis-1,3-dichloropropene	524.2		0.141	0.085	-49.3%	Hybrid	Linear
Cobalt	1620		40.837	25.933	-44.6%	Linear	Linear
Cobalt	200.8	ICP/MS	N/A[4]	0.001	0.0%	Linear	Hybrid
Copper	1620		47.509	32.643	-37.1%	Constant	Constant
Copper	200.8	ICP/MS	1.825	1.885	3.2%	Constant	Constant
Dibromochloromethane	502.2	ELCD	1.252	0.809	-43.0%	Linear	Constant
Dibromochloromethane	524.2		0.288	0.167	-53.2%	Hybrid	Hybrid
Dibromomethane	502.2	ELCD	1.395	0.587	-81.6%	Linear	Constant
Dibromomethane	524.2		0.460	0.498	7.9%	Hybrid	Hybrid
Dichlorodifluoromethane	502.2	ELCD	1.091[5]	2.470	77.4%	Linear	Constant
Dichlorodifluoromethane	524.2		0.480	0.442	-8.1%	Hybrid	Hybrid
Diethyl ether	524.2		0.404	0.525	26.0%	Hybrid	Hybrid
Ethyl methacrylate	524.2		0.183	0.141	-26.0%	Hybrid	Linear
Ethylbenzene	502.2	PID	0.157	0.007[3]	-182.9%	Hybrid	Linear
Ethylbenzene	524.2		0.077	0.064	-19.2%	Hybrid	Linear
Hardness[2]	130.2		5.465	10.032	58.9%	Linear	Constant
Hexachlorobutadiene	502.2	ELCD	0.243	0.582	82.2%	Hybrid	Linear
Hexachlorobutadiene	524.2		0.228	0.232	1.7%	Hybrid	Linear
Hexachloroethane	524.2		0.167	0.386	78.9%	Hybrid	Linear
Hexchlobutadiene+naphthalene	502.2	PID	1.542	1.193	-25.6%	Hybrid	Constant
Iron	1620		996.565[5]	2186.832	74.8%	Linear	Constant
Isopropylbenzene	502.2	PID	0.129	0.032	-120.6%	Linear	Linear
Isopropylbenzene	524.2		25.592	1.157	-182.7%	Constant	Constant
Lead	1620		5.698	6.059	6.1%	Linear	Constant
Lead	200.8	ICP/MS	0.685	5.983	158.9%	Linear	Constant
M+p xylene	502.2	PID	0.222	0.240	7.6%	Hybrid	Constant
M+p xylene	524.2		24.651	0.034	-199.4%	Constant	Hybrid
Magnesium	1620		267.199	378.277	34.4%	Linear	Constant
Manganese	1620		15.264	9.339	-48.2%	Constant	Constant
Manganese	200.8	ICP/MS	0.245	0.160	-41.8%	Constant	Constant
Mercury	200.8	ICP/MS	0.039	0.017[1]	-79.4%	Hybrid	Hybrid
Methacrylonitrile	524.2		19.062	1.111	-178.0%	Constant	Hybrid
Methyl iodide	524.2		0.083	3.681	191.1%	Hybrid	Constant
Methyl tertbutyl ether	524.2		0.122	15.132[6]	196.8%	Hybrid	Constant

Table 9. Comparison of 16-point and 5-point Single-laboratory IQEs at 10% RSD (SL-IQEs 10%) for the Episode 6000 Dataset (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE10% (16)	SL-IQE10% (5)	Percent Difference	SL-IQE Model (16)	SL-IQE Model (5)
Methyla crylate	524.2		0.727	0.853	16.0%	Hybrid	Linear
Methylene Chloride	502.2	ELCD	6.033	N/A [4]	N/A	Constant	Constant
Methylene Chloride	524.2		0.433	0.293	-38.5%	Hybrid	Linear
Methylm ethacrylate	524.2		20.773	0.873	-183.9%	Constant	Linear
Molybdenum	1620		7.597	11.866	43.9%	Linear	Constant
Molybdenum	200.8	ICP/MS	0.608	0.012	-192.4%	Constant	Constant
N-butylbenzene	502.2	PID	0.745	0.586	-24.0%	Linear	Linear
N-butylbenzene	524.2		0.067	1.287	180.1%	Hybrid	Constant
N-propylbenzene	502.2	PID	0.186	0.212	13.0%	Hybrid	Constant
N-propylbenzene	524.2		29.878	0.118	-198.4%	Constant	Hybrid
Naphthalene	524.2		0.108	0.256	81.1%	Hybrid	Hybrid
Nickel	1620		67.206	86.054	24.6%	Linear	Constant
Nickel	200.8	ICP/MS	0.183	0.147	-21.9%	Hybrid	Constant
O-xylene	524.2		0.040	0.016	-85.5%	Hybrid	Linear
O-xylene+styrene	502.2	PID	0.181	0.305	51.0%	Linear	Constant
P-isopropitol+1,4-dcb	502.2	PID	0.456	0.302	-40.8%	Linear	Constant
Pentachloroethane	524.2		0.551	1.036	61.1%	Hybrid	Linear
Sec-butylbenzene	502.2	PID	0.157	0.754	131.1%	Hybrid	Constant
Sec-butylbenzene	524.2		0.047	1.266	185.5%	Hybrid	Constant
Selenium	1620		5.235	4.076	-24.9%	Linear	Linear
Selenium	200.8	ICP/MS	1.045	0.707	-38.6%	Linear	Hybrid
Silver	1620		25.842	22.813	-12.5%	Linear	Constant
Silver	200.8	ICP/MS	0.056	N/A [4]	N/A	Linear	Linear
Sodium	1620		337.755	333.796	-1.2%	Linear	Linear
Styrene	524.2		0.041	0.067	49.3%	Hybrid	Linear
Tert-butylbenzene	502.2	PID	0.203	0.111	-58.9%	Linear	Hybrid
Tert-butylbenzene	524.2		0.073	0.074	1.1%	Hybrid	Linear
Tetrachloroethene	502.2	ELCD	0.122	0.182	39.7%	Hybrid	Linear
Tetrachloroethene	502.2	PID	0.750	0.385	-64.4%	Linear	Linear
Tetrachloroethene	524.2		30.554 [5]	1.643	-179.6%	Constant	Linear
Thallium	1620		2.799	2.745	-1.9%	Linear	Linear
Thallium	200.8	ICP/MS	0.002	0.001	-76.8%	Linear	Linear
Thorium	200.8	ICP/MS	0.004	0.001	-134.2%	Linear	Constant
Tin	1620		9.406	9.772	3.8%	Linear	Linear
Titanium	1620		14.236	42.768	100.1%	Linear	Constant
Toluene	502.2	PID	0.194	0.131	-39.1%	Linear	Constant
Toluene	524.2		0.046	1.145 [5]	184.7%	Hybrid	Constant
Total Phosphorus [2]	365.2		0.030	0.026	-15.8%	Hybrid	Linear
Total Suspended Solids [2]	160.2		6.729	6.929	2.9%	Hybrid	Linear
Trans-1,2-dichloroethene	502.2	ELCD	0.191	0.081 [5]	-80.6%	Hybrid	Linear
Trans-1,2-dichloroethene	524.2		0.153	0.171	11.3%	Hybrid	Hybrid
Trans-1,3-dichloropropene	502.2	ELCD	0.729	0.485	-40.2%	Linear	Constant
Trans-1,3-dichloropropene	502.2	PID	0.175	0.238	30.7%	Hybrid	Linear
Trans-1,3-dichloropropene	524.2		0.218	0.101	-73.5%	Hybrid	Hybrid
Trans-1,4-dichloro-2-butene	524.2		30.108	1.768	-177.8%	Constant	Hybrid
Trichloroethene	502.2	ELCD	3.169	1.010	-103.3%	Linear	Constant

Table 9. Comparison of 16-point and 5-point
Single-laboratory IQEs at 10% RSD (SL-IQEs 10%) for the Episode 6000 Dataset
(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE10% (16)	SL-IQE10% (5)	Percent Difference	SL-IQE Model (16)	SL-IQE Model (5)
Trichloroethene	502.2	PID	0.401	0.079	-134.4%	Linear	Linear
Trichloroethene	524.2		0.167	1.068	145.8%	Hybrid	Linear
Trichlorofluoromethane	502.2	ELCD	4.662	1.355	-109.9%	Constant	Constant
Trichlorofluoromethane	524.2		42.490[6]	0.301	-197.2%	Constant	Hybrid
Uranium	200.8	ICP/MS	0.001	0.000	-69.1%	Linear	Linear
Vanadium	1620		24.338	17.798	-31.0%	Hybrid	Linear
vVnadium	200.8	ICP/MS	1.933	2.225	14.1%	Hybrid	Linear
Vinyl Chloride	502.2	ELCD	8.234	3.258	-86.6%	Constant	Linear
Vinyl Chloride	524.2		0.219	0.652	99.2%	Hybrid	Linear
Wad Cyanide	1677	WADCN	1.624	2.661	48.4%	Linear	Constant
Xylene (total)	524.2		23.520	0.017	-199.7%	Constant	Hybrid
Yttrium	1620		8.962	28.689	104.8%	Linear	Constant
Zinc	1620		10.452	14.257	30.8%	Hybrid	Constant
Zinc	200.8	ICP/MS	7.024	10.927	43.5%	Linear	Constant

[1] IQE 10% undefined, IQE 20% reported

[2] Results reported as mg/L

[3] IQE 10% negative, IQE 20% reported

[4] IQE 10%, IQE 20%, IQE30% all negative based on chosen model (linear)

[5] IQE 10% and IQE 20% both negative, IQE 30% reported

[6] Hybrid model selected but did not converge; IQE 10% based on constant model instead

Summary Statistics for Table 9

	SL-IQE10 (16) vs. SL-IQE10 (5) (all analytes)	SL-IQE10 (16) vs. SL-IQE10 (5) (same model used)	SL-IQE10 (16) vs. SL-IQE10 (5) (different models used)
Number of Analytes	195	50	145
Minimum:	-19,971.5%	-19,237.7%	-19,971.5%
25th percentile:	-6,115.2%	-7,243.8%	-4,927.0%
Median:	-194.6%	-2,442.7%	613.9%
75th percentile:	4,562.6%	576.4%	6109.3%
Maximum:	19,715.8%	15724.6%	19,715.8%

	Number of analytes	Median % Difference	Sign Test p-value	Wilcoxon p-value
SL-IQE10 (16) vs. SL-IQE10 (5) (all analytes)	195	-194.600	0.567	0.345
SL-IQE10 (16) vs. SL-IQE10 (5) (same model used)	50	-2,442.7%	0.015	0.001
SL-IQE10 (16) vs. SL-IQE10 (5) (different models used)	145	613.9%	0.507	0.606

Table 10. Comparison of ACIL, USGS and EPA Limits Calculating using USGS Blank and Spiked data

Analyte	# blanks	# spikes	ACIL CRV		USGS LT-MDL (adding median)		USGS LT-MDL (adding mean)		EPA MDL (Randomly selected from simulated 7-replicate MDLs)	
			Limit	% exceeding	Limit	% exceeding	Limit	% exceeding	Limit	% exceeding
Ammonia (FCA)	52	24	0.022	0.0%	0.021	0.0%	0.021	0.0%	0.062	0.0%
Ammonia (FCC)	52	24	0.023	1.9%	0.011	11.5%	0.011	11.5%	0.012	9.6%
Ammonia Low Level (FCC)	52	15	0.006	1.9%	0.003	21.2%	0.004	7.7%	0.006	1.9%
Arsenic, Dissolved	26	24	1.068	3.8%	1.005	3.8%	1.071	3.8%	0.895	3.8%
Arsenic, Total	26	24	1.493	3.8%	0.829	7.7%	0.825	7.7%	1.298	3.8%
Cadmium, Dissolved by GFAA	26	24	0.082	0.0%	0.099	0.0%	0.095	0.0%	0.121	0.0%
Cadmium, Total by GFAA	26	24	0.075	0.0%	0.084	0.0%	0.089	0.0%	0.130	0.0%
Chromium, Dissolved by GFAA	26	24	0.441	3.8%	0.466	0.0%	0.475	0.0%	0.473	0.0%
Chromium, Total by GFAA	26	24	0.316	3.8%	0.341	3.8%	0.340	3.8%	3.540	0.0%
Cobalt, Dissolved by GFAA	26	24	1.287	0.0%	1.911	0.0%	1.847	0.0%	1.451	0.0%
Cobalt, Total by GFAA	26	24	1.639	3.8%	1.053	3.8%	1.093	3.8%	1.076	3.8%
Copper, Dissolved by GFAA	26	24	0.536	3.8%	0.408	3.8%	0.421	3.8%	1.102	0.0%
Copper, Total by GFAA	26	24	0.684	0.0%	0.766	0.0%	0.764	0.0%	26.384	0.0%
Lead, Dissolved by GFAA	26	24	0.609	3.8%	0.861	0.0%	0.857	0.0%	0.860	0.0%
Lead, Total by GFAA	26	24	0.774	3.8%	0.780	3.8%	0.736	3.8%	0.678	3.8%
Molybdenum (Wastewater) by GFAA	25	24	0.906	0.0%	0.779	0.0%	0.778	0.0%	0.639	4.0%
Molybdenum, Dissolved by GFAA	26	24	0.862	3.8%	1.098	0.0%	1.082	3.8%	1.190	0.0%
Nickel, Dissolved by GFAA	26	24	0.991	0.0%	1.014	0.0%	0.909	0.0%	2.568	0.0%
Nickel, Total by GFAA	26	24	1.765	0.0%	0.936	19.2%	1.167	11.5%	2.076	0.0%
Nitrate/Nitrite (FCA)	52	24	0.018	0.0%	0.009	21.2%	0.010	17.3%	0.009	21.2%

Table 10. Comparison of ACIL, USGS and EPA Limits Calculating using USGS Blank and Spiked data

Analyte	# blanks	# spikes	ACIL CRV		USGS LT-MDL (adding median)		USGS LT-MDL (adding mean)		EPA MDL (Randomly selected from simulated 7-replicate MDLs)	
			Limit	% exceeding	Limit	% exceeding	Limit	% exceeding	Limit	% exceeding
Nitrate/Nitrite (FCC)	52	15	0.023	3.8%	0.025	1.9%	0.026	1.9%	0.019	5.8%
Nitrate/Nitrite Low Level (FCC)	52	24	0.007	0.0%	0.008	0.0%	0.008	0.0%	0.006	11.5%
Nitrite (FCC)	52	15	0.003	0.0%	0.002	1.9%	0.002	1.9%	0.003	0.0%
Nitrite Low Level (FCC)	52	24	0.001	0.0%	0.002	0.0%	0.002	0.0%	0.002	0.0%
Orthophosphate (FCC)	52	24	0.022	3.8%	0.008	19.2%	0.010	15.4%	0.010	15.4%
Orthophosphate Low Level (FCC)	52	24	0.002	0.0%	0.000	26.9%	0.000	26.9%	0.001	0.0%
Phosphorus, Low Level Filtered	52	24	0.003	1.9%	0.003	0.0%	0.003	0.0%	0.003	0.0%
Phosphorus, Low Level Filtered	52	24	0.003	0.0%	0.003	0.0%	0.003	0.0%	0.004	0.0%
Phosphorus, Low Level in Wastewater	52	24	0.003	3.8%	0.004	1.9%	0.004	1.9%	0.009	0.0%
Selenium, Dissolved	26	24	1.174	0.0%	1.434	0.0%	1.410	0.0%	1.334	0.0%
Selenium, Total	26	24	2.123	3.8%	1.211	7.7%	1.324	7.7%	1.130	11.5%
Silver, Dissolved by GFAA	26	24	0.088	3.8%	0.159	0.0%	0.158	0.0%	0.122	0.0%
Silver, Total by GFAA	26	24	0.140	3.8%	0.125	3.8%	0.131	3.8%	0.196	0.0%
TKN/ Ammonia (FCA)	52	24	0.070	0.0%	0.092	0.0%	0.091	0.0%	0.071	0.0%
TKN/ Ammonia (FCC)	52	24	0.083	1.9%	0.056	3.8%	0.059	3.8%	0.049	7.7%
TKN/ Ammonia (WCA)	52	24	0.483	1.9%	0.081	1.9%	0.104	1.9%	0.071	1.9%
Total Phosphorus (FCA)	52	24	0.021	3.8%	0.026	0.0%	0.026	0.0%	0.022	1.9%
Total Phosphorus (FCC)	52	24	0.026	0.0%	0.025	0.0%	0.025	0.0%	0.023	0.0%
Total Phosphorus (WCA)	52	24	0.027	1.9%	0.023	1.9%	0.023	1.9%	0.021	3.8%

Summary Statistics for Table 10.

Limit Type	% of Blanks Exceeding Limit for Dataset	
	Mean	Standard Error
ACIL CRV	1.9%	0.3%
USGS LT-MDL (adding median)	4.4%	1.2%
USGS LT-MDL (adding mean)	3.7%	0.9%
EPA MDL	2.9%	0.8%

Table 11. Comparison of SL-IDEs and MDLs calculated With and Without Outlier Removal, Episode 6000 Data

(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE			MDL	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers dropped
1,1,1,2-tetrachloroethane	502.2	ELCD	0.034	0.024	E/E	0.041	0.006
1,1,1,2-tetrachloroethene	524.2		0.244	0.211	E/E	0.052	0.052
1,1,1-trichloroethane	502.2	ELCD	0.041	0.038	E/E	0.012	0.012
1,1,1-trichloroethane	524.2		0.308	0.311	E/E	0.055	0.055
1,1,2,2-tce+1,2,3-tcp	502.2	ELCD	0.179	0.123	E/E	0.064	0.064
1,1,2,2-tetrachloroethane	524.2		0.436	0.296	E/E	0.132	0.132
1,1,2-trichloroethane	502.2	ELCD	0.032	0.026	E/E	0.024	0.018
1,1-dichloroethane	502.2	ELCD	0.083	0.060	E/E	0.010	0.014
1,1-dichloroethane	524.2		0.229	0.187	E/E	0.033	0.033
1,1-dichloroethene	502.2	ELCD	0.234	0.165	E/E	0.038	0.028
1,1-dichloropropene	524.2		0.287	0.294	E/E	0.045	0.045
1,2,3-trichlorobenzene	502.2	ELCD	0.134	0.066	E/E	0.048	0.021
1,2,3-trichlorobenzene	502.2	PID	0.115	0.095	E/E	0.057	0.057
1,2,3-trichlorobenzene	524.2		0.275	0.256	E/E	0.070	0.070
1,2,3-trichloropropane	524.2		1.263	1.046	E/E	7.328	4.014
1,2,4-trichlorobenzene	502.2	ELCD	0.088	0.076	E/E	0.022	0.022
1,2,4-trichlorobenzene	502.2	PID	0.124	0.117	E/E	0.070	0.070
1,2,4-trimethylbenzene	502.2	PID	0.125	0.107	E/E	0.095	0.095
1,2,4-trimethylbenzene	524.2		0.144	0.134	E/E	0.012	0.026
1,2-dibromo-3-chloropropane	524.2		1.749	1.368	E/E	1.457	1.457
1,2-dibromoethane	502.2	ELCD	0.164	0.146	E/E	0.096	0.095
1,2-dibromoethane	524.2		0.326	0.290	E/E	0.127	0.127
1,2-dichlorobenzene	502.2	ELCD	0.065	0.061	E/E	0.035	0.035
1,2-dichlorobenzene	524.2		0.130	0.133	E/E	0.030	0.025
1,2-dichloroethane	502.2	ELCD	0.042	0.029	E/E	0.017	0.017
1,2-dichloroethane	524.2		0.258	0.237	E/E	0.039	0.059
1,2-dichloropropane	502.2	ELCD	0.043	0.031	E/E	0.023	0.029
1,2-dichloropropane	524.2		0.247	0.175	E/E	0.056	0.026
1,3,5-trimethylbenzene	524.2		0.135	0.127	E/E	0.011	0.011
1,3-dichlorobenzene	502.2	ELCD	0.118	0.073	E/E	0.035	0.014
1,3-dichlorobenzene	502.2	PID	0.126	0.106	E/E	0.093	0.067
1,3-dichloropropene	502.2	ELCD	0.047	0.037	E/E	0.016	0.014
1,3-dichloropropane	524.2		0.202	0.182	E/E	0.038	0.038
1,4-dichlorobenzene	502.2	ELCD	0.061	0.053	E/E	0.026	0.026
1,4-dichlorobenzene	524.2		0.140	0.130	E/E	0.023	0.023
2,2-dichloropropane	524.2		0.691	0.630	E/E	2.376	2.376
2-butanone	524.2		0.833	0.696	E/E	0.417	0.874
2-chlorotoluene	502.2	ELCD	0.175	0.161	E/E	0.108	0.108
2-chlorotoluene	502.2	PID	0.230	0.143	E/E	0.238	0.086
2-hexanone	524.2		0.902	0.753	E/E	1.316	0.426
4-chlorotoluene	502.2	ELCD	0.149	0.134	E/E	0.110	0.083
4-chlorotoluene	524.2		0.123	0.114	E/E	0.010	0.010
Allyl Chloride	524.2		0.229	0.213	E/E	0.032	0.029
Aluminum	1620		206.975	47.299	C/E	29.555	19.524
Aluminum	200.8	ICP/MS	12.747	9.371	E/E	19.145	0.839
Ammonia as Nitrogen[2]	350.3		0.014	0.013	E/E	0.010	0.010

Table 11. Comparison of SL-IDEs and MDLs calculated With and Without Outlier Removal, Episode 6000 Data

(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE			MDL	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers dropped
Antimony	200.8	ICP/MS	0.019	0.014	E/E	0.178	0.008
Arsenic	200.8	ICP/MS	0.366	0.347	E/E	0.226	0.226
Barium	1620		1.837	1.441	C/C	1.702	1.702
Barium	200.8	ICP/MS	0.084	0.068	E/E	0.033	0.018
Benzene	502.2	PID	0.079	0.074	E/E	0.030	0.030
Beryllium	1620		0.448	0.430	E/E	0.528	0.528
Beryllium	200.8	ICP/MS	0.024	0.021	E/E	0.007	0.007
Bromobenzene	502.2	ELCD	0.765	0.242	L/E	0.131	0.131
Bromobenzene	502.2	PID	0.050	0.046	E/E	0.012	0.012
Bromobenzene	524.2		0.211	0.195	E/E	0.044	0.044
Bromochloromethane	502.2	ELCD	0.482	0.390	L/L	0.013	0.013
Bromodichloromethane	502.2	ELCD	0.075	0.065	E/E	0.004	0.004
Bromodichloromethane	524.2		0.205	0.190	E/E	0.043	0.043
Bromoform	502.2	ELCD	1.513	1.504	C/C	0.006	0.006
Bromoform	524.2		0.400	0.363	E/E	0.123	0.123
Bromomethane	502.2	ELCD	7.293	7.427	C/C	0.267	0.477
Cadmium	1620		0.191	0.159	E/E	0.127	0.127
Cadmium	200.8	ICP/MS	0.022	0.022	E/E	0.004	0.004
Calcium	1620		41.358	36.054	L/L	36.726	36.726
Carbon Tetrachloide	524.2		0.314	0.288	E/E	0.038	0.038
Carbontet+1,1-dcp	502.2	ELCD	0.072	0.068	E/E	0.029	0.029
Chlorobenzene	502.2	ELCD	0.460	0.378	L/L	0.011	0.011
Chlorobenzene	502.2	PID	0.064	0.055	E/E	0.030	0.026
Chloroethane	502.2	ELCD	2.598	2.357	C/C	0.108	0.011
Chloroethane	524.2		0.395	0.362	E/E	0.066	0.048
Chloroform	502.2	ELCD	0.032	0.026	E/E	0.043	0.043
Chloromethane	502.2	ELCD	0.250	0.150	E/E	0.070	0.070
Chloromethane	524.2		0.253	0.302	E/E	0.045	0.045
Chromium	1620		0.496	0.464	E/E	0.310	0.310
Chromium	200.8	ICP/MS	0.408	0.207	L/E	0.073	0.073
Cis-1,2-dce+2,2-dcp	502.2	ELCD	0.055	0.052	E/E	0.013	0.013
Cis-1,3-dichloropropene	502.2	ELCD	0.074	0.062	E/E	0.007	0.007
Cis-1,3-dichloropropene	502.2	PID	0.082	0.138	E/E	0.057	0.057
Cis-1,3-dichloropropene	524.2		0.173	0.145	E/E	0.038	0.036
Cobalt	1620		16.463	15.625	E/E	9.820	9.820
Cobalt	200.8	ICP/MS	0.074	0.074	C/C	0.001	0.001
Copper	1620		21.189	14.718	C/C	6.046	6.046
Copper	200.8	ICP/MS	0.798	0.160	C/E	0.037	0.037
Dibromochloromethane	502.2	ELCD	0.436	0.413	L/L	0.009	0.006
Dibromochloromethane	524.2		0.287	0.210	E/E	0.051	0.051
Dibromomethane	502.2	ELCD	0.460	0.344	L/L	0.007	0.007
Dibromomethane	524.2		0.388	0.319	E/E	0.102	0.102
Dichlorodifluoromethane	502.2	ELCD	0.240	0.069	E/E	0.009	0.071
Diethyl Ether	524.2		0.376	0.301	E/E	0.120	0.120
Ethyl Methacrylate	524.2		0.273	0.246	E/E	0.045	0.035
Ethylbenzene	502.2	PID	0.078	0.073	E/E	0.021	0.021

Table 11. Comparison of SL-IDEs and MDLs calculated With and Without Outlier Removal, Episode 6000 Data

(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE			MDL	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers dropped
Ethylbenzene	524.2		0.198	0.184	E/E	0.033	0.023
Hexachlorobutadiene	502.2	ELCD	0.094	0.081	E/E	0.043	0.043
Hexchlobutadiene+naphthalene	502.2	PID	0.597	0.490	E/E	0.649	0.649
Iron	1620		373.590	42.840	L/E	90.409	19.188
Isopropylbenzene	502.2	PID	0.060	0.047	E/E	0.020	0.020
Isopropylbenzene	524.2		0.120	0.107	E/E	0.011	0.010
Lead	1620		2.423	1.855	E/E	1.647	1.288
Lead	200.8	ICP/MS	0.204	0.133	E/E	0.655	0.131
M+p xylene	502.2	PID	0.121	0.114	E/E	0.090	0.090
Magnesium	1620		105.998	100.489	E/E	103.033	103.033
Manganese	1620		6.808	2.183	C/E	6.856	1.176
Manganese	200.8	ICP/MS	0.109	0.018	C/E	0.031	0.012
Mercury	200.8	ICP/MS	0.027	0.024	E/E	0.004	0.004
Methacrylonitrile	524.2		0.718	0.492	E/E	0.356	0.336
Methylacrylate	524.2		0.601	0.477	E/E	0.220	0.220
Methylene Chloride	524.2		0.314	0.279	E/E	0.082	0.082
Methylmethacrylate	524.2		0.535	0.480	E/E	0.225	0.225
Molybdenum	1620		3.034	2.683	E/E	2.455	2.455
Molybdenum	200.8	ICP/MS	0.271	0.027 [1]	C/C	0.004	0.002
N-butylbenzene	502.2	PID	0.141	0.105	E/E	0.030	0.083
N-propylbenzene	502.2	PID	0.092	0.071	E/E	0.040	0.040
Naphthalene	524.2		0.186	0.219	E/E	0.048	0.048
Nickel	1620		25.560	23.853	E/E	20.219	20.219
Nickel	200.8	ICP/MS	0.083	0.057	E/E	0.146	0.075
O-xylene+styrene	502.2	PID	0.116	0.087	E/E	0.059	0.043
P-isopropol+1,4-dcb	502.2	PID	0.159	0.131	E/E	0.073	0.054
Pentachloroethane	524.2		0.408	0.351	E/E	0.553	0.207
Sec-butylbenzene	502.2	PID	0.081	0.068	E/E	0.055	0.036
Selenium	200.8	ICP/MS	0.416	0.324	E/E	0.192	0.192
Silver	1620		10.668	10.718	E/L	4.907	4.250
Silver	200.8	ICP/MS	0.012	0.010	E/E	0.004	0.004
Tert-butylbenzene	502.2	PID	0.074	0.082	E/E	0.029	0.029
Tetrachloroethene	502.2	ELCD	0.061	0.054	E/E	0.018	0.018
Tetrachloroethene	502.2	PID	0.156	0.131	E/E	0.062	0.062
Tetrachloroethene	524.2		0.469	0.393	E/E	0.085	0.027
Thallium	200.8	ICP/MS	0.001	0.001	E/E	0.000	0.000
Thorium	200.8	ICP/MS	0.001	0.001	E/E	0.001	0.001
Tin	1620		3.932	3.700	E/E	3.670	3.670
Titanium	1620		5.376	4.732	E/E	4.777	4.663
Toluene	502.2	PID	0.064	0.056	E/E	0.070	0.071
Toluene	524.2		0.146	0.136	E/E	0.020	0.018
Total Suspended Solids [2]	160.2		3.005	3.060	E/E	1.170	0.980
Trans-1,2-dichloroethene	502.2	ELCD	0.081	0.073	E/E	0.041	0.041
Trans-1,3-dichloroPropene	502.2	ELCD	0.098	0.083	E/E	0.012	0.012
Trans-1,3-dichloropropene	502.2	PID	0.092	0.088	E/E	0.058	0.058
Trans-1,3-dichloropropene	524.2		0.223	0.188	E/E	0.051	0.051

Table 11. Comparison of SL-IDEs and MDLs calculated With and Without Outlier Removal, Episode 6000 Data

(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE			MDL	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers dropped
Trichloroethene	502.2	ELCD	0.059	0.049	E/E	0.012	0.012
Trichloroethene	502.2	PID	0.097	0.078	E/E	0.027	0.027
Trichloroethene	524.2		0.332	0.333	E/E	0.061	0.061
Trichlorofluoromethane	502.2	ELCD	2.079	1.762	C/C	0.108	0.012
Trichlorofluoromethane	524.2		0.384	0.528	E/E	0.087	0.087
Uranium	200.8	ICP/MS	0.000	0.000	E/E	0.000	0.000
Vinyl Chloride	502.2	ELCD	3.672	3.577	C/C	0.270	0.270
Wad Cyanide	1677	WADCN	0.701	0.665	L/L	0.572	0.550
Yttrium	1620		3.247	3.078	E/E	1.923	1.923
Zinc	1620		4.500	4.135	E/E	2.597	2.597
Zinc	200.8	ICP/MS	1.598	1.016	E/E	0.900	0.585

[1] Constant model used because IDE did not converge for chosen model (Exponential)

[2] Results reported as mg/L

Summary Statistics for Table 11.

Percent Difference (Positive if limit with outliers kept>limit with outliers removed)	# Analytes	Minimum	25th Percentile	Median	75th Percentile	Maximum
SL-IDE (all)	149	-51.6%	7.1%	14.3%	24.4%	164.2%
SL-IDE (same model used)	141	-51.6%	6.9%	13.7%	22.2%	164.2%
SL-IDE (different model used)	8	-0.5%	93.4%	114.7%	135.9%	158.9%
MDL	60	-115.4%	4.4%	30.2%	75.6%	183.7%

Table 12. Comparison of SL-IQEs and MLs calculated With and Without Outlier Removal, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE (10%)			ML	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers Dropped
1,1,1,2-tetrachloroethane	502.2	ELCD	0.030	0.023	H/H	0.2	0.02
1,1,1,2-tetrachloroethane	524.2		0.181	0.142	H/H	0.2	0.2
1,1,1-trichloroethene	502.2	ELCD	0.830	2.207	L/C	0.05	0.05
1,1,1-trichloroethane	524.2		0.240	0.157	H/H	0.2	0.2
1,1,2,2-tce+1,2,3-tcp	502.2	ELCD	5.514	5.290 [5]	C/C	0.2	0.2
1,1,2,2-tetrachloroethane	524.2		0.569	0.318	H/H	0.5	0.5
1,1,2-trichloroethane	502.2	ELCD	0.060	0.030	L/H	0.1	0.05
1,1-dichloroethane	502.2	ELCD	0.527	0.311	L/L	0.05	0.05
1,1-dichloroethane	524.2		0.115	25.620 [5]	H/C	0.1	0.1
1,1-dichloroethene	502.2	ELCD	3.796	3.827	L/L	0.1	0.1
1,1-dichloropropene	524.2		0.180	0.090	H/H	0.2	0.2
1,2,3-trichlorobenzene	502.2	ELCD	0.851	0.117	L/L	0.2	0.1
1,2,3-trichlorobenzene	502.2	PID	0.248	0.190	H/H	0.2	0.2
1,2,3-trichlorobenzene	524.2		0.216	0.217	H/H	0.2	0.2
1,2,3-trichloropropane	524.2		11.316	5.134	L/L	20	10
1,2,4-trichlorobenzene	502.2	ELCD	0.401	0.226	L/L	0.1	0.1
1,2,4-trichlorobenzene	502.2	PID	0.439	0.429	L/L	0.2	0.2
1,2,4-trimethylbenzene	502.2	PID	0.653	0.621	L/L	0.5	0.5
1,2,4-trimethylbenzene	524.2		20.896	21.013	C/C	0.05	0.1
1,2-dibromo-3-chloropropane	524.2		71.182 [5]	72.198 [5]	C/C	5	5
1,2-dibromoethane	502.2	ELCD	0.592	0.560	L/L	0.5	0.2
1,2-dibromoethane	524.2		0.417	0.418	H/H	0.5	0.5
1,2-dichlorobenzene	502.2	ELCD	0.183	0.114	L/H	0.1	0.1
1,2-dichlorobenzene	524.2		0.085	0.067	H/H	0.1	0.1
1,2-dichloroethane	502.2	ELCD	0.065	0.031	H/H	0.05	0.05
1,2-dichloroethane	524.2		0.222	0.168	H/H	0.1	0.2
1,2-dichloropropane	502.2	ELCD	0.102	0.038	L/H	0.1	0.1
1,2-dichloropropane	524.2		0.196	0.085	H/H	0.2	0.1
1,3,5-trimethylbenzene	524.2		23.744	23.877	C/C	0.05	0.05
1,3-dichlorobenzene	502.2	ELCD	0.936	0.463	L/L	0.1	0.05
1,3-dichlorobenzene	502.2	PID	0.465	0.401	L/L	0.2	0.2
1,3-dichloropropane	502.2	ELCD	0.054	0.059	L/H	0.05	0.05
1,3-dichloropropane	524.2		0.139	0.151	H/H	0.1	0.1
1,4-dichlorobenzene	502.2	ELCD	0.101	0.079	H/H	0.1	0.1
1,4-dichlorobenzene	524.2		0.078	0.077	H/H	0.1	0.1
2,2-dichloropropane	524.2		38.009	38.299	C/C	10	10
2-butanone	524.2		0.893	0.534	H/H	2	2
2-chlorotoluene	502.2	ELCD	0.493	0.439	H/H	0.5	0.5
2-chlorotoluene	502.2	PID	0.849	0.770	H/L	1	0.2
2-hexanone	524.2		0.442	0.518	H/H	5	2
4-chlorotoluene	502.2	ELCD	0.142 [1]	0.517	H/H	0.5	0.2
4-chlorotoluene	524.2		23.810	23.941	C/C	0.05	0.05
Allyl Chloride	524.2		29.674	29.866	C/C	0.1	0.1
Aluminum	1620		464.069	156.043	C/L	100	50
Aluminum	200.8	ICP/MS	29.684	31.466	H/L	50	2
Ammonia as Nitrogen [2]	350.3		0.035	0.032	H/H	0.05	0.05
Antimony	200.8	ICP/MS	0.034	0.020	H/H	0.5	0.02

Table 12. Comparison of SL-IQEs and MLs calculated With and Without Outlier Removal, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE (10%)			ML	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers Dropped
Arsenic	200.8	ICP/MS	0.798	0.747	H/H	1	1
Barium	1620		4.118	3.231	C/C	5	5
Barium	200.8	ICP/MS	0.211	0.191	L/L	0.1	0.05
Benzene	502.2	PID	0.182	0.149	L/H	0.1	0.1
Beryllium	1620		0.980	0.975	H/H	2	2
Beryllium	200.8	ICP/MS	0.044	0.038	H/H	0.02	0.02
Bromobenzene	502.2	ELCD	3.529	0.594	L/H	0.5	0.5
Bromobenzene	502.2	PID	0.100	0.022	L/L	0.05	0.05
Bromobenzene	524.2		0.140	0.143	H/H	0.2	0.2
Bromochloromethane	502.2	ELCD	1.598	1.344	L/L	0.05	0.05
Bromodichloromethane	502.2	ELCD	0.424	0.323	L/L	0.02	0.02
Bromodichloromethane	524.2		0.128	0.131	H/H	0.2	0.2
Bromoform	502.2	ELCD	3.393	3.350	C/C	0.02	0.02
Bromoform	524.2		0.482	0.484	H/H	0.5	0.5
Bromomethane	502.2	ELCD	16.351	16.541	C/C	1	2
Cadmium	1620		0.410	0.422	H/L	0.5	0.5
Cadmium	200.8	ICP/MS	0.063	0.068	H/H	0.02	0.02
Calcium	1620		99.975	88.075	L/L	100	100
Carbon Tetrachloride	524.2		0.140	0.061	H/H	0.1	0.1
Carbontet+1,1-dcp	502.2	ELCD	0.069	4.481	H/C	0.1	0.1
Chlorobenzene	502.2	ELCD	1.766	1.514	L/L	0.05	0.05
Chlorobenzene	502.2	PID	0.119	0.100	H/H	0.1	0.1
Chloroethane	502.2	ELCD	5.826	5.285	C/C	0.5	0.05
Chloroethane	524.2		0.255	0.202	H/H	0.2	0.2
Chloroform	502.2	ELCD	0.025	0.006	L/H	0.2	0.2
Chloromethane	502.2	ELCD	1.734	0.766	L/L	0.2	0.2
Chloromethane	524.2		0.141	0.187	H/H	0.2	0.2
Chromium	1620		1.259	1.072	L/L	1	1
Chromium	200.8	ICP/MS	1.028	0.636	L/L	0.2	0.2
Cis-1,2-dce+2,2-dcp	502.2	ELCD	0.039	0.038	H/H	0.05	0.05
Cis-1,3-dichloropropane	502.2	ELCD	0.415	0.131	L/H	0.02	0.02
Cis-1,3-dichloropropene	502.2	PID	0.017 [1]	0.262	H/H	0.2	0.2
Cis-1,3-dichloropropene	524.2		0.141	0.070	H/H	0.1	0.1
Cobalt	1620		40.837	39.614	L/L	50	50
Cobalt	200.8	ICP/MS	N/A [3]	N/A [3]	N/A	0.005	0.005
Copper	1620		47.509	33.000	C/C	20	20
Copper	200.8	ICP/MS	1.825	1.706	C/C	0.1	0.1
Dibromochloromethane	502.2	ELCD	1.252	1.189	L/L	0.02	0.02
Dibromochloromethane	524.2		0.288	0.177	H/H	0.2	0.2
Dibromomethane	502.2	ELCD	1.395	1.099	L/L	0.02	0.02
Dibromomethane	524.2		0.460	0.473	H/H	0.5	0.5
Dichlorodifluoromethane	502.2	ELCD	1.091 [4]	5.023	L/C	0.02	0.2
Diethyl Ether	524.2		0.404	0.400	H/H	0.5	0.5
Ethyl Methacrylate	524.2		0.183	0.109	H/H	0.2	0.1
Ethylbenzene	502.2	PID	0.157	0.149	H/H	0.1	0.1
Ethylbenzene	524.2		0.077	0.047	H/H	0.1	0.1
Hexachlorobutadiene	502.2	ELCD	0.243	0.194	H/H	0.2	0.2

Table 12. Comparison of SL-IQEs and MLs calculated With and Without Outlier Removal, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE (10%)			ML	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers Dropped
Hexchlobutadiene+naphthalene	502.2	PID	1.542	1.216	H/H	2	2
Iron	1620		996.565 [4]	151.265	L/H	200	50
Isopropylbenzene	502.2	PID	0.129	1.928	L/C	0.1	0.1
Isopropylbenzene	524.2		25.592	25.726	C/C	0.05	0.05
Lead	1620		5.698	4.449	L/L	5	5
Lead	200.8	ICP/MS	0.685	0.281	L/H	2	0.5
M+p xylene	502.2	PID	0.222	0.217	H/H	0.2	0.2
Magnesium	1620		267.199	259.424	L/L	500	500
Manganese	1620		15.264	5.629	C/L	20	5
Manganese	200.8	ICP/MS	0.245	0.071	C/L	0.1	0.05
Mercury	200.8	ICP/MS	0.039	0.033	H/H	0.02	0.02
Methacrylonitrile	524.2		19.062	19.451	C/C	1	1
Methyla crylate	524.2		0.727	0.586	H/H	1	1
Methylene Chloride	524.2		0.433	0.390	H/H	0.2	0.2
Methylm ethacryl ate	524.2		20.773	20.951	C/C	1	1
Molybdenum	1620		7.597	6.737	L/L	10	10
Molybdenum	200.8	ICP/MS	0.608	0.011	C/H	0.01	0.005
N-butylbenzene	502.2	PID	0.745	0.397	L/L	0.1	0.2
N-propylbenzene	502.2	PID	0.186	0.128	H/H	0.2	0.2
Naphthalene	524.2		0.108	0.166	H/H	0.2	0.2
Nickel	1620		67.206	58.049	L/L	100	100
Nickel	200.8	ICP/MS	0.183	0.116	H/H	0.5	0.2
O-xylene+styrene	502.2	PID	0.181	0.140	L/H	0.2	0.2
P-isopropitol+1,4-dcb	502.2	PID	0.456	0.330	L/L	0.2	0.2
Pentachloroethane	524.2		0.551	0.406	H/H	2	1
Sec-butylbenzene	502.2	PID	0.157	0.101	H/H	0.2	0.1
Selenium	200.8	ICP/MS	1.045	0.607	L/H	0.5	0.5
Silver	1620		25.842	25.005	L/L	20	20
Silver	200.8	ICP/MS	0.056	0.027	L/L	0.02	0.02
Tert-butylbenzene	502.2	PID	0.203	0.121	L/L	0.1	0.1
Tetrachloroethene	502.2	ELCD	0.122	0.092	H/H	0.05	0.05
Tetrachloroethene	502.2	PID	0.750	0.664	L/L	0.2	0.2
Tetrachloroethene	524.2		30.554 [5]	0.275	C/H	0.2	0.1
Thallium	200.8	ICP/MS	0.002	0.002	L/L	0.002	0.002
Thorium	200.8	ICP/MS	0.004	0.001	L/H	0.002	0.002
Tin	1620		9.406	8.651	L/L	10	10
Titanium	1620		14.236	13.166	L/L	20	20
Toluene	502.2	PID	0.194	0.084	L/L	0.2	0.2
Toluene	524.2		0.046	0.039	H/H	0.05	0.05
Total Suspended Solids [2]	160.2		6.729	7.441	H/L	5	5
Trans-1,2-dichloroethene	502.2	ELCD	0.191	0.159	H/H	0.2	0.2
Trans-1,3-dichloropropene	502.2	ELCD	0.729	0.610	L/L	0.05	0.05
Trans-1,3-dichloropropene	502.2	PID	0.175	0.173	H/H	0.2	0.2
Trans-1,3-dichloroprepene	524.2		0.218	0.124	H/H	0.2	0.2
Trichloroethene	502.2	ELCD	3.169	0.041 [1]	L/L	0.05	0.05
Trichloroethene	502.2	PID	0.401	0.332	L/L	0.1	0.1
Trichloroethene	524.2		0.167	0.237	H/H	0.2	0.2

Table 12. Comparison of SL-IQEs and MLs calculated With and Without Outlier Removal, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE (10%)			ML	
			Outliers Kept	Outliers Dropped	Model Used (Kept/Dropped)	Outliers Kept	Outliers Dropped
Trichlorofluoromethane	502.2	ELCD	4.662	3.950	C/C	0.5	0.05
Trichlorofluoromethane	524.2		42.490[5]	0.228	C/H	0.2	0.2
Uranium	200.8	ICP/MS	0.001	0.001	L/H	0.001	0.001
Vinyl Chloride	502.2	ELCD	8.234	8.020	C/C	1	1
Weak Cyanide	1677	WADCN	1.624	1.543	L/L	2	2
Yttrium	1620		8.962	8.501	L/L	5	5
Zinc	1620		10.452	11.630	H/L	10	10
Zinc	200.8	ICP/MS	7.024	2.291	L/H	2	2

[1] IQE 10% undefined, IQE 20% reported
[2] Results reported as mg/L
[3] IQE 10%, IQE 20% and IQE 30% all negative based on chosen model (linear)
[4] IQE 10% and IQE 20% both negative, IQE 30% reported
[5] Hybrid model selected but did not converge; IQE 10% based on constant model instead

Summary Statistics for Table 12

Percent Difference (Positive if limit with outliers kept> limit with outliers removed)	# Analytes	Minimum	25th Percentile	Median	75th Percentile	Maximum
SL-IQE (all)	148	-198.2%	1.0%	16.3%	50.2%	197.9%
SL-IQE (same model used)	117	-176.3%	0.0%	2.8%	23.7%	194.9%
SL-IQE (different model used)	31	-198.2%	-7.7%	53.1%	107.1%	197.9%
ML	31	-163.6%	66.7%	66.7%	120.0%	184.6%

Table 13. Comparison of SL-IDEs calculated using different Model Types, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE, Based on Given Model				RSD
			Constant	Linear	Exponential	Hybrid	
1,1,1,2-tetrachloroethane	502.2	ELCD	0.687	0.000	0.034	0.010	184%
1,1,1,2-tetrachloroethane	524.2		11.051	-1.234	0.244	0.078	166%
1,1,1-trichloroethane	502.2	ELCD	0.985	0.016	0.041	0.010	183%
1,1,1-trichloroethane	524.2		14.141	-0.836	0.308	0.098	166%
1,1,2,2-tcp+1,2,3-tcp	502.2	ELCD	2.597	-0.222	0.179	N/A [1]	123%
1,1,2,2-tetrachloroethane	524.2		12.456	-1.517	0.436	0.248	160%
1,1,2-trichloroethane	502.2	ELCD	0.476	0.016	0.032	0.016	169%
1,1,2-trichloroethane	524.2		7.245	-0.407	0.319	0.127	158%
1,1-dichloroethane	502.2	ELCD	0.801	0.083	0.083	0.067	140%
1,1-dichloroethane	524.2		11.355	-0.642	0.229	0.049	167%
1,1-dichloroethene	502.2	ELCD	1.167	0.305	0.234	0.213	96%
1,1-dichloroethene	524.2		18.473	-2.042	0.335	0.050	168%
1,1-dichloropropanone	524.2		15.292	4.713	6.372	6.513	58%
1,1-dichloropropene	524.2		13.573	-0.554	0.287	0.073	167%
1,2,3-trichlorobenzene	502.2	ELCD	0.942	0.117	0.134	0.117	125%
1,2,3-trichlorobenzene	502.2	PID	0.640	0.134	0.115	0.083	109%
1,2,3-trichlorobenzene	524.2		18.047	-1.759	0.275	0.090	168%
1,2,3-trichloropropane	524.2		12.464	3.599	1.263	0.041	129%
1,2,4-trichlorobenzene	502.2	ELCD	0.739	0.082	0.088	0.069	135%
1,2,4-trichlorobenzene	502.2	PID	0.688	0.113	0.124	0.100	112%
1,2,4-trichlorobenzene	524.2		14.387	-1.058	0.224	0.059	168%
1,2,4-trimethylbenzene	502.2	PID	0.889	0.125	0.125	0.108	123%
1,2,4-trimethylbenzene	524.2		9.319	-0.074	0.144	0.020	169%
1,2-dibromo-3-chloropropane	524.2		34.167	-7.305	1.749	N/A [1]	128%
1,2-dibromoethane	502.2	ELCD	0.543	0.184	0.164	0.160	71%
1,2-dibromoethane	524.2		8.173	-0.811	0.326	0.184	158%
1,2-dichlorobenzene	502.2	ELCD	0.653	0.037	0.065	0.045	151%
1,2-dichlorobenzene	502.2	PID	0.895	0.136	0.148	0.121	117%
1,2-dichlorobenzene	524.2		12.369	-1.392	0.130	0.036	170%
1,2-dichloroethane	502.2	ELCD	0.951	-0.041	0.042	0.022	157%
1,2-dichloroethane	524.2		7.061	-0.485	0.258	0.097	161%
1,2-dichloropropane	502.2	ELCD	0.733	0.015	0.043	0.024	173%
1,2-dichloropropane	524.2		9.388	-0.729	0.247	0.085	164%
1,3,5-mb+4-chlorotoluene	502.2	PID	1.526	0.084	0.114	0.073	160%
1,3,5-trimethylbenzene	524.2		10.590	-0.059	0.135	0.016	170%
1,3-dichlorobenzene	502.2	ELCD	0.775	0.230	0.118	0.103	103%
1,3-dichlorobenzene	502.2	PID	0.773	0.102	0.126	0.099	121%
1,3-dichlorobenzene	524.2		12.273	-1.099	0.143	0.033	170%
1,3-dichloropropane	502.2	ELCD	0.578	0.015	0.047	0.028	164%
1,3-dichloropropane	524.2		6.432	-0.320	0.202	0.061	163%
1,4-dichlorobenzene	502.2	ELCD	0.654	0.050	0.061	0.033	152%
1,4-dichlorobenzene	524.2		11.443	-1.116	0.140	0.034	169%
1-chlorobutane	524.2		13.444	-0.406	0.220	0.024	169%
2,2-dichloropropane	524.2		17.294	-0.134	0.691	0.152	161%
2-butanone	524.2		14.170	-1.296	0.833	0.384	153%
2-chlorotoluene	502.2	ELCD	1.533	0.051	0.175	0.166	146%
2-chlorotoluene	502.2	PID	0.977	0.272	0.230	0.187	90%
2-chlorotoluene	524.2		11.146	-0.639	0.136	0.023	170%

Table 13. Comparison of SL-IDEs calculated using different Model Types, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE, Based on Given Model				RSD
			Constant	Linear	Exponential	Hybrid	
2-hexanone	524.2		22.744	-5.136	0.902	0.188	161%
2-nitropropane	524.2		18.337	-3.854	1.082	0.254	156%
4-chlorotoluene	502.2	ELCD	1.792	-0.022	0.149	0.112	140%
4-chlorotoluene	524.2		10.619	-0.329	0.123	0.013	170%
4-isopropyltoluene	524.2		9.108	0.162	0.117	0.007	192%
4-methyl-2-pentanone	524.2		20.121	-5.006	1.195	0.773	150%
Acetone	524.2		22.659	-1.723	2.120	1.092	141%
Acrylonitrile	524.2		13.467	-1.190	1.333	0.715	139%
Allyl Chloride	524.2		13.324	-0.815	0.229	0.051	168%
Aluminum	1620		206.975	88.830	51.697	N/A [1]	70%
Aluminum	200.8	ICP/MS	41.919	12.689	12.747	12.961	73%
Ammonia as Nitrogen [2]	350.3		0.078	0.009	0.014	0.013	114%
Antimony	1620		4.260	3.728	3.562	3.596	9%
Antimony	200.8	ICP/MS	0.229	0.027	0.019	0.015	144%
Arsenic	1620		2.131	1.510	1.410	1.390	22%
Arsenic	200.8	ICP/MS	2.023	0.257	0.366	0.345	114%
Barium	1620		1.837	1.522	1.300	1.306	17%
Barium	200.8	ICP/MS	0.257	0.085	0.084	0.079	69%
Benzene	502.2	PID	0.802	0.036	0.079	0.060	152%
Benzene	524.2		8.619	-0.122	0.125	0.019	169%
Beryllium	1620		1.587	0.365	0.448	0.431	83%
Beryllium	200.8	ICP/MS	0.170	0.013	0.024	0.018	134%
Boron	1620		38.617	20.625	21.161	20.805	35%
Bromobenzene	502.2	ELCD	1.685	0.765	0.499	0.515	65%
Bromobenzene	502.2	PID	0.569	0.028	0.050	0.032	157%
Bromobenzene	524.2		12.851	-1.691	0.211	0.060	168%
Bromochloromethane	502.2	ELCD	0.939	0.482	0.162	0.157	85%
Bromochloromethane	524.2		8.929	-0.807	0.345	0.161	159%
Bromodichloromethane	502.2	ELCD	0.617	0.111	0.075	0.060	125%
Bromodichloromethane	524.2		8.020	-0.455	0.205	0.056	165%
Bromoform	502.2	ELCD	1.513	1.161	0.381	0.381	66%
Bromoform	524.2		10.207	-1.309	0.400	0.211	159%
Bromomethane	502.2	ELCD	7.293	5.796	4.313	N/A [1]	26%
Bromomethane	524.2		12.379	-1.072	0.280	0.096	166%
Cadmium	1620		0.364	0.208	0.191	0.180	37%
Cadmium	200.8	ICP/MS	0.040	0.022	0.022	0.026	31%
Calcium	1620		54.321	41.358	37.020	37.410	19%
Carbon Disulfide	524.2		14.835	-1.181	0.239	0.040	168%
Carbon Tetrachloride	524.2		15.266	-1.197	0.314	0.056	167%
Carbontet+1,1-dcp	502.2	ELCD	1.998	-0.007	0.072	0.020	162%
Chloroacetonitrile	524.2		11.548	-0.814	1.569	1.453	119%
Chlorobenzene	502.2	ELCD	0.982	0.460	0.189	0.183	83%
Chlorobenzene	502.2	PID	0.749	0.020	0.064	0.048	160%
Chlorobenzene	524.2		10.276	-0.665	0.133	0.026	169%
Chloroethane	502.2	ELCD	2.598	2.161	1.091	1.053	45%
Chloroethane	524.2		14.465	-0.836	0.395	0.104	165%
Chloroform	502.2	ELCD	0.732	0.006	0.032	0.004	185%
Chloroform	524.2		9.385	-0.399	0.225	0.051	166%

Table 13. Comparison of SL-IDEs calculated using different Model Types, Episode 6000 Data
(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE, Based on Given Model				RSD
			Constant	Linear	Exponential	Hybrid	
Chloromethane	502.2	ELCD	1.130	0.453	0.250	0.233	82%
Chloromethane	524.2		19.617	-2.484	0.253	0.056	169%
Chromium	1620		1.090	0.528	0.496	0.471	46%
Chromium	200.8	ICP/MS	0.672	0.408	0.284	0.290	44%
Cis-1,2-dce+2,2-dcp	502.2	ELCD	1.893	-0.048	0.055	0.012	164%
Cis-1,2-dichloroethene	524.2		11.249	-0.960	0.234	0.062	167%
Cis-1,3-dichloropropene	502.2	ELCD	0.716	0.083	0.074	0.061	138%
Cis-1,3-dichloropropene	502.2	PID	0.933	0.039	0.082	0.013	167%
Cis-1,3-dichloropropene	524.2		7.072	-0.454	0.173	0.062	165%
Cobalt	1620		30.100	16.339	16.463	16.102	35%
Cobalt	200.8	ICP/MS	0.074	-0.012	-0.004	-0.001	192%
Copper	1620		21.189	16.989	14.754	14.861	18%
Copper	200.8	ICP/MS	0.798	0.404	0.205	0.207	69%
Dibromochloromethane	502.2	ELCD	0.784	0.436	0.144	0.141	81%
Dibromochloromethane	524.2		8.159	-0.667	0.287	0.126	161%
Dibromomethane	502.2	ELCD	0.836	0.460	0.192	0.184	73%
Dibromomethane	524.2		7.135	-0.585	0.388	0.203	153%
Dichlorodifluoromethane	502.2	ELCD	2.194	0.348	0.240	0.153	133%
Dichlorodifluoromethane	524.2		24.275	-4.798	0.560	0.183	166%
Diethyl Ether	524.2		12.008	-1.243	0.376	0.175	162%
Ethyl Methacrylate	524.2		10.053	-0.957	0.273	0.079	164%
Ethylbenzene	502.2	PID	0.888	0.020	0.078	0.060	160%
Ethylbenzene	524.2		11.939	-0.776	0.198	0.032	168%
Hardness [2]	130.2		3.658	2.362	2.258	2.385	25%
Hexachlorobutadiene	502.2	ELCD	0.997	0.105	0.094	0.065	144%
Hexachlorobutadiene	524.2		17.734	-2.203	0.308	0.092	167%
Hexachloroethane	524.2		18.095	-2.155	0.288	0.069	168%
Hexchlobutadiene+naphthalene	502.2	PID	1.442	0.793	0.597	0.523	50%
Iron	1620		486.971	373.590	125.364	124.648	66%
Isopropylbenzene	502.2	PID	0.856	0.025	0.060	0.033	168%
Isopropylbenzene	524.2		11.414	-0.141	0.120	0.012	170%
Lead	1620		3.976	2.396	2.423	2.437	28%
Lead	200.8	ICP/MS	1.007	0.265	0.204	0.200	94%
M+p xylene	502.2	PID	1.701	0.005	0.121	0.088	170%
M+p xylene	524.2		10.994	-0.206	0.142	0.016	170%
Magnesium	1620		145.717	112.074	105.998	106.575	16%
Manganese	1620		6.808	4.201	2.993	3.033	42%
Manganese	200.8	ICP/MS	0.109	0.065	0.034	0.034	59%
Mercury	200.8	ICP/MS	0.827	0.006	0.027	0.016	185%
Methacrylonitrile	524.2		8.883	-0.181	0.718	0.356	145%
Methyl Iodide	524.2		12.103	-0.866	0.193	0.035	168%
Methyl tert-butyl ether	524.2		10.845	-1.117	0.225	0.053	167%
Methylacrylate	524.2		13.820	-1.522	0.601	0.315	157%
Methylene Chloride	502.2	ELCD	2.841	1.822	-3.178	N/A [1]	651%
Methylene Chloride	524.2		8.787	-0.455	0.314	0.188	159%
Methylmethacrylate	524.2		9.597	-0.342	0.535	0.244	154%
Molybdenum	1620		4.908	3.163	3.034	3.042	26%
Molybdenum	200.8	ICP/MS	0.271	0.096	0.180	-0.007	88%

Table 13. Comparison of SL-IDEs calculated using different Model Types, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE, Based on Given Model				RSD
			Constant	Linear	Exponential	Hybrid	
Naphthalene	524.2		14.829	-0.891	0.186	0.044	169%
N-butylbenzene	502.2	PID	0.714	0.215	0.141	0.135	92%
N-butylbenzene	524.2		10.237	-0.145	0.152	0.028	169%
Nickel	1620		50.587	26.333	25.560	24.898	39%
Nickel	200.8	ICP/MS	1.023	0.176	0.083	0.072	136%
N-propylbenzene	502.2	PID	0.785	0.075	0.092	0.066	139%
N-propylbenzene	524.2		13.415	-0.751	0.284	0.061	167%
o-xylene	524.2		11.622	-0.802	0.198	0.017	168%
o-xylene+styrene	502.2	PID	1.372	0.043	0.116	0.082	160%
Pentachloroethane	524.2		11.186	-0.793	0.408	0.237	159%
P-isopropitol+1,4-dcb	502.2	PID	1.583	0.091	0.159	0.118	150%
Sec-butylbenzene	502.2	PID	0.942	0.053	0.081	0.052	156%
Sec-butylbenzene	524.2		11.240	0.080	0.140	0.020	194%
Selenium	1620		4.161	2.054	1.975	1.971	43%
Selenium	200.8	ICP/MS	2.090	0.406	0.416	0.364	104%
Silver	1620		13.219	11.098	10.668	10.801	10%
Silver	200.8	ICP/MS	0.048	0.020	0.012	0.010	77%
Sodium	1620		169.136	141.290	138.768	140.811	10%
Styrene	524.2		10.516	-0.600	0.141	0.017	169%
Tert-butylbenzene	502.2	PID	0.854	0.038	0.074	0.050	158%
Tert-butylbenzene	524.2		11.706	-0.323	0.186	0.030	169%
Tetrachloroethene	502.2	ELCD	0.927	0.029	0.061	0.031	169%
Tetrachloroethene	502.2	PID	1.027	0.114	0.156	0.127	126%
Tetrachloroethene	524.2		13.627	-0.451	0.469	N/A [1]	132%
Thallium	1620		1.726	1.185	1.153	1.161	21%
Thallium	200.8	ICP/MS	0.003	0.001	0.001	0.001	73%
Thorium	200.8	ICP/MS	0.032	0.002	0.001	0.000	176%
Tin	1620		5.755	3.991	3.932	3.986	20%
Titanium	1620		8.500	6.012	5.376	5.419	23%
Toluene	502.2	PID	0.731	0.044	0.064	0.051	152%
Toluene	524.2		9.778	-0.303	0.146	0.019	169%
Total Phosphorus [2]	365.2		0.018	0.014	0.013	0.013	16%
Total Suspended Solids [2]	160.2		4.317	3.195	3.005	2.977	19%
trans-1,2-dichloroethene	502.2	ELCD	0.922	0.067	0.081	0.060	151%
trans-1,2-dichloroethene	524.2		13.734	-0.953	0.300	0.062	167%
trans-1,3-dichloropropene	502.2	ELCD	0.666	0.201	0.098	0.087	104%
trans-1,3-dichloropropene	502.2	PID	0.650	0.052	0.092	0.068	135%
trans-1,3-dichloropropene	524.2		6.714	-0.432	0.223	0.096	161%
trans-1,4-dichloro-2-butene	524.2		14.301	-1.059	1.250	0.782	141%
Trichloroethene	502.2	ELCD	1.006	0.035	0.059	0.038	169%
Trichloroethene	502.2	PID	0.914	0.066	0.097	0.069	146%
Trichloroethene	524.2		12.510	-0.619	0.332	0.065	165%
Trichlorofluoromethane	502.2	ELCD	2.079	1.656	1.107	1.076	32%
Trichlorofluoromethane	524.2		19.248	-2.147	0.384	N/A [1]	136%
Uranium	200.8	ICP/MS	0.002	0.000	0.000	0.000	116%
Vanadium	1620		22.721	9.967	10.630	10.693	46%
Vanadium	200.8	ICP/MS	2.762	0.730	0.864	0.840	75%
Vinyl Chloride	502.2	ELCD	3.672	3.036	1.756	1.690	39%

Table 13. Comparison of SL-IDEs calculated using different Model Types, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IDE, Based on Given Model				RSD
			Constant	Linear	Exponential	Hybrid	
Vinyl Chloride	524.2		22.292	-3.345	0.365	0.083	168%
Wad Cyanide	1677	WADCN	1.023	0.701	0.620	0.638	25%
Xylene (total)	524.2		10.490	-0.264	0.128	0.008	170%
Yttrium	1620		4.569	3.520	3.247	3.279	17%
Zinc	1620		14.628	3.804	4.500	4.425	76%
Zinc	200.8	ICP/MS	7.561	2.537	1.598	1.610	86%

[1] Hybrid model failed to converge

[2] Results reported as mg/L

Summary Statistics for Table 13

Method	# Analytes	Minimum	25th Percentile	Median	75th Percentile	Maximum
All	198	8.5%	81.8%	151.1%	166.7%	650.6%
502.2	65	25.7%	103.5%	140.1%	159.9%	650.6%
524.2	81	58.2%	159.2%	166.0%	168.5%	194.5%
1620	26	8.5%	18.1%	26.8%	42.4%	83.0%
200.8	21	31.0%	72.5%	88.0%	134.5%	191.6%

Table 14. Comparison of SL-IQEs calculated using different Model Types, Episode 6000 Data
(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE 10%, Based on Given Model				RSD [1]
			Constant	Linear	Exponential	Hybrid	
1,1,1,2-tetrachloroethane	502.2	ELCD	1.541	0.000	0.078	0.030	182.6%
1,1,1,2-tetrachloroethane	524.2		24.612	-4.974	0.556	0.181	165.7%
1,1,1-trichloroethane	502.2	ELCD	2.208	0.830	0.096	0.058	126.0%
1,1,1-trichloroethane	524.2		31.494	-4.112	0.704	0.240	165.7%
1,1,2,2-tca+1,2,3-tcp	502.2	ELCD	5.514	-1.416	0.430	N/A [2]	120.9%
1,1,2,2-tetrachloroethane	524.2		27.377	-5.971	1.001	0.569	159.1%
1,1,2-trichloroethane	502.2	ELCD	1.067	0.060	0.075	0.040	162.6%
1,1,2-trichloroethane	524.2		15.923	-1.175	0.726	0.290	157.7%
1,1-dichloroethane	502.2	ELCD	1.795	0.527	0.200	0.178	113.2%
1,1-dichloroethane	524.2		25.290	-2.390	0.521	0.115	166.8%
1,1-dichloroethene	502.2	ELCD	2.617	3.796	0.627	0.886	75.6%
1,1-dichloroethene	524.2		41.142	-28.559	0.767	0.129	167.7%
1,1-dichloropropanone	524.2		30.102	12.705	15.558	15.041	43.2%
1,1-dichloropropene	524.2		30.229	-2.582	0.655	0.180	166.2%
1,2,3-trichlorobenzene	502.2	ELCD	2.113	0.851	0.334	0.341	92.1%
1,2,3-trichlorobenzene	502.2	PID	1.435	0.482	0.279	0.248	91.5%
1,2,3-trichlorobenzene	524.2		40.193	-12.045	0.628	0.216	167.9%
1,2,3-trichloropropane	524.2		27.394	11.316	2.981	0.166	117.0%
1,2,4-trichlorobenzene	502.2	ELCD	1.658	0.401	0.212	0.186	114.4%
1,2,4-trichlorobenzene	502.2	PID	1.544	0.439	0.303	0.276	94.7%
1,2,4-trichlorobenzene	524.2		32.041	-5.251	0.510	0.141	168.0%
1,2,4-trimethylbenzene	502.2	PID	1.993	0.653	0.309	0.291	99.2%
1,2,4-trimethylbenzene	524.2		20.896	-0.243	0.326	0.048	168.6%
1,2-dibromo-3-chloropropane	524.2		71.182	-145.715	4.217	N/A [2]	125.6%
1,2-dibromoethane	502.2	ELCD	1.218	0.592	0.401	0.381	60.5%
1,2-dibromoethane	524.2		17.963	-2.444	0.743	0.417	157.5%
1,2-dichlorobenzene	502.2	ELCD	1.465	0.183	0.154	0.121	136.6%
1,2-dichlorobenzene	502.2	PID	1.992	0.638	0.367	0.346	93.6%
1,2-dichlorobenzene	524.2		27.734	-6.758	0.294	0.085	169.7%
1,2-dichloroethane	502.2	ELCD	2.132	0.266	0.100	0.065	155.8%
1,2-dichloroethane	524.2		15.586	-1.407	0.585	0.222	160.5%
1,2-dichloropropane	502.2	ELCD	1.643	0.102	0.101	0.065	162.6%
1,2-dichloropropane	524.2		20.909	-2.433	0.562	0.196	164.1%
1,3,5-tmb+4-chlorotoluene	502.2	PID	3.422	0.396	0.268	0.189	147.0%
1,3,5-trimethylbenzene	524.2		23.744	-0.208	0.305	0.037	169.5%
1,3-dichlorobenzene	502.2	ELCD	1.738	0.936	0.289	0.267	85.9%
1,3-dichlorobenzene	502.2	PID	1.732	0.465	0.309	0.288	99.3%
1,3-dichlorobenzene	524.2		27.518	-4.866	0.324	0.076	169.5%
1,3-dichloropropane	502.2	ELCD	1.287	0.054	0.110	0.067	159.5%
1,3-dichloropropane	524.2		14.324	-0.934	0.458	0.139	162.8%
1,4-dichlorobenzene	502.2	ELCD	1.467	0.218	0.144	0.101	136.4%
1,4-dichlorobenzene	524.2		25.657	-5.226	0.316	0.078	169.3%
1-chlorobutane	524.2		29.943	-1.682	0.499	0.060	168.5%
2,2-dichloropropane	524.2		38.009	-15.752	1.607	0.464	159.8%
2-butanone	524.2		30.407	-4.569	1.934	0.893	151.2%
2-chlorotoluene	502.2	ELCD	3.438	1.364	0.452	0.493	97.4%
2-chlorotoluene	502.2	PID	2.176	1.249	0.597	0.849	56.9%
2-chlorotoluene	524.2		24.990	-2.436	0.308	0.053	169.5%

Table 14. Comparison of SL-IQEs calculated using different Model Types, Episode 6000 Data
(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE 10%, Based on Given Model				RSD [1]
			Constant	Linear	Exponential	Hybrid	
2-hexanone	524.2		47.881	-30.174	2.102	0.442	160.2%
2-nitropropane	524.2		38.203	-16.221	2.531	0.590	153.7%
4-chlorotoluene	502.2	ELCD	4.017	0.161	0.383	N/A [3]	142.4%
4-chlorotoluene	524.2		23.810	-1.231	0.278	0.032	169.9%
4-isopropyltoluene	524.2		20.421	0.528	0.265	0.016	189.9%
4-methyl-2-pentanone	524.2		41.919	-23.810	2.804	1.785	147.6%
Acetone	524.2		47.703	-8.481	5.137	2.741	136.5%
Acrylonitrile	524.2		28.056	-3.845	3.129	1.651	135.6%
Allyl Chloride	524.2		29.674	-3.694	0.521	0.121	167.7%
Aluminum	1620		464.069	255.899	130.746	N/A [2]	59.4%
Aluminum	200.8	ICP/MS	93.989	37.673	30.404	29.684	64.5%
Ammonia as Nitrogen [4]	350.3		0.175	0.052	0.035	0.035	90.3%
Antimony	1620		9.551	8.719	8.275	8.104	7.5%
Antimony	200.8	ICP/MS	0.525	0.073	0.044	0.034	140.8%
Arsenic	1620		4.705	3.542	3.240	3.097	20.0%
Arsenic	200.8	ICP/MS	4.629	0.692	0.859	0.798	110.3%
Barium	1620		4.118	3.475	2.973	2.934	16.4%
Barium	200.8	ICP/MS	0.589	0.211	0.197	0.183	66.6%
Benzene	502.2	PID	1.798	0.182	0.189	0.155	139.7%
Benzene	524.2		19.325	-0.385	0.284	0.044	168.9%
Beryllium	1620		3.559	0.964	1.044	0.980	78.3%
Beryllium	200.8	ICP/MS	0.382	0.041	0.057	0.044	127.8%
Boron	1620		86.584	51.134	49.514	47.266	31.9%
Bromobenzene	502.2	ELCD	3.704	3.529	1.408	1.417	50.7%
Bromobenzene	502.2	PID	1.277	0.100	0.118	0.079	149.8%
Bromobenzene	524.2		28.621	-7.963	0.479	0.140	167.7%
Bromochloromethane	502.2	ELCD	2.106	1.598	0.399	0.379	77.6%
Bromochloromethane	524.2		19.625	-2.531	0.787	0.368	158.8%
Bromodichloromethane	502.2	ELCD	1.384	0.424	0.178	0.148	108.8%
Bromodichloromethane	524.2		17.863	-1.404	0.465	0.128	164.9%
Bromoform	502.2	ELCD	3.393	2.540	0.922	0.877	64.3%
Bromoform	524.2		22.334	-4.327	0.914	0.482	157.9%
Bromomethane	502.2	ELCD	16.351	5.779	N/A [3]	N/A [2]	67.6%
Bromomethane	524.2		27.570	-5.134	0.637	0.226	165.3%
Cadmium	1620		0.816	0.505	0.445	0.410	34.1%
Cadmium	200.8	ICP/MS	0.090	0.065	0.054	0.063	23.1%
Calcium	1620		121.796	99.975	86.815	84.600	17.4%
Carbon Disulfide	524.2		33.263	-7.679	0.545	0.101	168.3%
Carbon Tetrachloride	524.2		34.000	-7.521	0.718	0.140	166.8%
Carbontet+1,1-dcp	502.2	ELCD	4.480	0.105	0.167	0.069	181.2%
Chloroacetonitrile	524.2		24.059	-2.331	3.679	3.310	114.7%
Chlorobenzene	502.2	ELCD	2.202	1.766	0.477	0.458	72.9%
Chlorobenzene	502.2	PID	1.679	0.092	0.151	0.119	152.8%
Chlorobenzene	524.2		23.041	-2.418	0.300	0.059	169.2%
Chloroethane	502.2	ELCD	5.826	4.368	2.730	2.613	39.2%
Chloroethane	524.2		31.932	-4.186	0.907	0.255	164.1%
Chloroform	502.2	ELCD	1.640	0.025	0.075	0.011	183.1%
Chloroform	524.2		20.902	-1.329	0.511	0.121	165.6%

Table 14. Comparison of SL-IQEs calculated using different Model Types, Episode 6000 Data
(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE 10%, Based on Given Model				RSD [1]
			Constant	Linear	Exponential	Hybrid	
Chloromethane	502.2	ELCD	2.533	1.734	0.650	0.678	65.0%
Chloromethane	524.2		43.690	-89.292	0.577	0.141	169.0%
Chromium	1620		2.444	1.259	1.141	1.062	44.0%
Chromium	200.8	ICP/MS	1.538	1.028	0.681	0.669	41.7%
Cis-1,2-dce+2,2-dcp	502.2	ELCD	4.244	0.218	0.127	0.039	178.0%
Cis-1,2-dichloroethane	524.2		25.054	-3.865	0.532	0.144	166.4%
Cis-1,3-dichloroPropene	502.2	ELCD	1.604	0.415	0.177	0.151	117.3%
Cis-1,3-dichloropropene	502.2	PID	2.077	0.222	0.196	N/A [3]	129.7%
Cis-1,3-dichloropropene	524.2		15.751	-1.358	0.391	0.141	164.7%
Cobalt	1620		67.490	40.837	38.691	36.682	31.5%
Cobalt	200.8	ICP/MS	0.166	-0.022	-0.009	0.002	138.6%
Copper	1620		47.509	39.683	34.348	33.546	16.6%
Copper	200.8	ICP/MS	1.825	0.984	0.487	0.477	67.2%
Dibromochloromethane	502.2	ELCD	1.757	1.252	0.349	0.330	76.3%
Dibromochloromethane	524.2		18.012	-2.066	0.653	0.288	160.3%
Dibromomethane	502.2	ELCD	1.874	1.395	0.475	0.447	67.3%
Dibromomethane	524.2		15.614	-1.663	0.885	0.460	152.6%
Dichlorodifluoromethane	502.2	ELCD	4.918	-0.244	0.732	0.654	116.1%
Dichlorodifluoromethane	524.2		53.352	30.938	1.297	0.480	118.6%
Diethyl Ether	524.2		26.391	-4.619	0.860	0.404	161.4%
Ethyl Methacrylate	524.2		22.094	-3.192	0.621	0.183	164.1%
Ethylbenzene	502.2	PID	1.991	0.128	0.188	0.157	148.8%
Ethylbenzene	524.2		26.591	-3.326	0.450	0.077	168.2%
Hardness [4]	130.2		8.005	5.465	5.109	5.258	23.0%
Hexachlorobutadiene	502.2	ELCD	2.236	0.753	0.228	0.243	109.3%
Hexachlorobutadiene	524.2		39.496	-21.961	0.703	0.228	167.2%
Hexachloroethane	524.2		40.301	-19.924	0.657	0.167	168.0%
Hexchlobutadiene+naphthalene	502.2	PID	3.234	2.358	1.524	1.542	37.5%
Iron	1620		1091.863	-281.500	N/A [3]	N/A [3]	N/A
Isopropylbenzene	502.2	PID	1.919	0.129	0.141	0.088	158.1%
Isopropylbenzene	524.2		25.592	-0.498	0.270	0.029	170.2%
Lead	1620		8.914	5.698	5.587	5.489	25.9%
Lead	200.8	ICP/MS	2.305	0.685	0.478	0.462	90.4%
M+p xylene	502.2	PID	3.813	0.031	0.285	0.222	167.3%
M+p xylene	524.2		24.651	-0.743	0.321	0.037	169.5%
Magnesium	1620		326.719	267.199	247.396	240.982	14.4%
Manganese	1620		15.264	10.195	7.113	6.899	39.5%
Manganese	200.8	ICP/MS	0.245	0.156	0.079	0.076	57.3%
Mercury	200.8	ICP/MS	1.854	0.019	0.063	0.039	183.8%
Methacrylonitrile	524.2		19.062	-0.518	1.655	0.815	143.5%
Methyl Iodide	524.2		26.956	-3.833	0.439	0.083	168.3%
Methyl terTbutyl ether	524.2		23.940	-4.171	0.511	0.122	166.5%
Methylacrylate	524.2		29.913	-5.560	1.386	0.727	156.1%
Methylene Chloride	502.2	ELCD	6.033	5.201	-4.095	N/A [2]	10.5%
Methylene Chloride	524.2		19.701	-1.528	0.717	0.433	158.9%
Methylmethacrylate	524.2		20.773	-1.043	1.228	0.561	152.7%
Molybdenum	1620		11.003	7.597	7.049	6.869	23.9%
Molybdenum	200.8	ICP/MS	0.608	0.260	N/A [3]	0.026	98.3%

Table 14. Comparison of SL-IQEs calculated using different Model Types, Episode 6000 Data
(µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE 10%, Based on Given Model				RSD [1]
			Constant	Linear	Exponential	Hybrid	
N-butylbenzene	502.2	PID	1.601	0.745	0.343	0.325	79.3%
N-butylbenzene	524.2		22.952	-0.521	0.345	0.067	168.6%
N-propylbenzene	502.2	PID	1.759	0.351	0.221	0.186	120.2%
N-propylbenzene	524.2		29.878	-3.650	0.647	0.148	166.5%
Nepthalene	524.2		33.249	-4.704	0.422	0.108	169.1%
Nickel	1620		113.424	67.206	60.455	57.072	35.2%
Nickel	200.8	ICP/MS	2.341	0.800	0.202	0.183	115.1%
O-xylene	524.2		25.884	-3.313	0.450	0.040	168.4%
O-xylene+styrene	502.2	PID	3.077	0.181	0.272	0.202	153.2%
P-isopreptol+1,4-dcb	502.2	PID	3.550	0.456	0.380	0.312	134.9%
Pentachloroethane	524.2		24.914	-3.372	0.934	0.551	158.6%
Sec-butylbenzene	502.2	PID	2.112	0.346	0.196	0.157	134.2%
Sec-butylbenzene	524.2		25.203	0.279	0.316	0.047	193.4%
Selenium	1620		9.268	5.235	4.657	4.474	38.3%
Selenium	200.8	ICP/MS	4.686	1.045	0.957	0.829	99.7%
Silver	1620		29.640	25.842	24.547	24.294	9.5%
Silver	200.8	ICP/MS	0.107	0.056	0.030	0.034	62.6%
Sodium	1620		379.229	337.755	323.935	317.747	8.1%
Styrene	524.2		23.420	-2.180	0.318	0.041	169.3%
Tert-butylbenzene	502.2	PID	1.916	0.203	0.177	0.135	143.6%
Tert-butylbenzene	524.2		26.246	-1.197	0.423	0.073	168.4%
Tetrachloroethene	502.2	ELCD	2.078	0.415	0.145	0.122	135.5%
Tetrachloroethene	502.2	PID	2.303	0.750	0.392	0.400	94.7%
Tetrachloroethene	524.2		30.554	-2.553	1.080	N/A [2]	131.8%
Thallium	1620		3.870	2.799	2.661	2.614	19.9%
Thallium	200.8	ICP/MS	0.007	0.002	0.002	0.002	70.9%
Thorium	200.8	ICP/MS	0.074	0.004	0.003	0.001	174.7%
Tin	1620		12.904	9.406	9.064	8.971	18.7%
Titanium	1620		19.058	14.236	12.443	12.213	21.9%
Toluene	502.2	PID	1.640	0.194	0.153	0.124	140.6%
Toluene	524.2		21.925	-1.050	0.330	0.046	168.8%
Total Phosphorus [4]	365.2		0.040	0.032	0.030	0.030	14.1%
Total Suspended Solids [4]	160.2		9.679	7.570	6.985	6.729	17.3%
Trans-1,2-dichloroethene	502.2	ELCD	2.068	0.795	0.197	0.191	108.7%
Trans-1,2-dichloroethene	524.2		30.588	-4.773	0.684	0.153	166.3%
Trans-1,3-dichloropropene	502.2	ELCD	1.492	0.729	0.237	0.212	89.8%
Trans-1,3-dichloropropene	502.2	PID	1.457	0.206	0.221	0.175	122.1%
Trans-1,3-dichloropropene	524.2		14.821	-1.254	0.506	0.218	161.1%
Trans-1,4-dichloro-2-butene	524.2		30.108	-3.685	2.938	1.819	137.8%
Trichloroethene	502.2	ELCD	2.256	3.169	0.141	0.120	108.1%
Trichloroethene	502.2	PID	2.049	0.401	0.235	0.209	122.7%
Trichloroethene	524.2		27.861	-2.666	0.759	0.167	164.9%
Trichlorofluoromethane	502.2	ELCD	4.662	5.166	3.222	3.308	23.8%
Trichlorofluoromethane	524.2		42.490	-50.543	0.881	N/A [2]	135.7%
Uranium	200.8	ICP/MS	0.005	0.001	0.001	0.001	112.1%
Vanadium	1620		50.943	26.049	25.112	24.338	40.8%
Vanadium	200.8	ICP/MS	6.320	1.828	2.022	1.933	72.6%
Vinyl Chlorkie	502.2	ELCD	8.234	4.775	3.544	3.828	42.3%

Table 14. Comparison of SL-IQEs calculated using different Model Types, Episode 6000 Data (µg/L except where footnoted)

Analyte	Method	Procedure	SL-IQE 10%, Based on Given Model				RSD [1]
			Constant	Linear	Exponential	Hybrid	
Vinyl Chloride	524.2		49.647	49.158	0.837	0.219	113.0%
Wad Cyanide	1677	WADCN	2.277	1.624	1.414	1.424	24.2%
Xylene (total)	524.2		23.520	-0.952	0.290	0.019	169.8%
Yttrium	1620		10.244	8.962	7.839	7.516	14.3%
Zinc	1620		32.799	12.850	10.999	10.452	64.0%
Zinc	200.8	ICP/MS	17.301	7.024	3.817	3.741	80.4%

[1] Calculation includes positive IQEs only

[2] Given model did not converge

[3] IQE 10% could not be calculated based on given model

[4] Results reported as mg/L

Summary Statistics for Table 14

Method	# analytes	Minimum	25th Percentile	Median	75th Percentile	Maximum
All	197	7.5%	72.6%	135.6%	165.3%	193.4%
502.2	65	10.5%	79.3%	114.4%	142.4%	183.1%
524.2	81	43.2%	157.9%	165.7%	168.4%	193.4%
1620	25	7.5%	16.6%	23.9%	38.3%	78.3%
200.8	21	23.1%	66.6%	90.4%	115.1%	183.8%

Table 15. Comparison of SL-IDEs and SL-IQEs Calculated Using Different Software

Analyte	Model Type	Limit	QCalc	Excel	SAS [1]
1,1-dichloroethene (502.2)	Hybrid	IDE	-0.0338	0.3180 [2]	0.2135
		IQE 10	-0.87	2.006	0.886
	Exponential	IDE	0.2307	0.2367	0.2337
		IQE 10		0.622	0.627
	Linear	IDE		0.3059	0.3051
		IQE 10	3.7	3.693	3.796
	Constant	IDE		1.169	1.167
		IQE 10		2.604	2.617
1,2,4-trichlorobenzene (502.2, ELCD)	Hybrid	IDE	0.0688	0.1072	0.0694
		IQE 10	0.19	0.297	0.186
	Exponential	IDE	0.0874	0.0888	0.0880
		IQE 10		0.212	0.212
	Linear	IDE		0.0821	0.0817
		IQE 10	0.40	0.399	0.401
	Constant	IDE		0.741	0.740
		IQE 10		1.651	1.658
1,3,5-trimethylbenzene (524.2)	Hybrid	IDE	0.0157	-4.10E-07	0.0157
		IQE 10	0.04	-6.00E-06	0.037
	Exponential	IDE	0.1345	0.1367	0.1349
		IQE 10		not calc [3]	0.305
	Linear	IDE		-0.0595	-0.0586
		IQE 10	not calc [3]	not calc [3]	-0.208
	Constant	IDE		10.448	10.590
		IQE 10		23.269	23.744
Antimony (1620) [4]	Hybrid	IDE	3.5724	3.8364	3.5960
		IQE 10	8.10	8.578	8.104
	Exponential	IDE	3.5380	3.5853	3.5616
		IQE 10		8.270	8.275
	Linear	IDE		3.7511	3.7283
		IQE 10	8.72	8.713	8.719
	Constant	IDE		4.266	4.260
		IQE 10		9.502	9.551
Arsenic (200.8)	Hybrid	IDE	0.3433	0.3675	0.3449
		IQE 10	0.80	0.837	0.798
	Exponential	IDE	0.3643	0.3734	0.3661
		IQE 10		0.858	0.859
	Linear	IDE		0.2623	0.2570
		IQE 10	0.69	0.691	0.692
	Constant	IDE		2.056	2.023
		IQE 10		4.611	4.629

Table 15. Comparison of SL-IDEs and SL-IQEs Calculated Using Different Software

Analyte	Model Type	Limit	QCalc	Excel	SAS [1]
Bromoform (524.2)	Hybrid	IDE	0.2165	-0.0094	0.2113
		IQE 10	0.48	-0.132	0.482
	Exponential	IDE	0.4097	0.4157	0.3998
		IQE 10		not calc [3]	0.914
	Linear	IDE		-1.3717	-1.3091
		IQE 10	not calc [3]	not calc [3]	-4.327
	Constant	IDE		10.355	10.207
		IQE 10		22.220	22.334
Chloroethane (524.2)	Hybrid	IDE	0.1048	-0.0035	0.1036
		IQE 10	0.25	-0.057	0.255
	Exponential	IDE	0.3999	0.4028	0.3953
		IQE 10		not calc [3]	0.907
	Linear	IDE		-0.8594	-0.8365
		IQE 10	not calc [3]	not calc [3]	-4.186
	Constant	IDE		14.518	14.465
		IQE 10		31.769	31.932
Cis-1,3-dichloropropene (502.2 ELCD)	Hybrid	IDE	0.0600	0.1254	0.0606
		IQE 10	0.15	0.351	0.151
	Exponential	IDE	0.0734	0.0750	0.0740
		IQE 10		0.176	0.177
	Linear	IDE		0.0833	0.0830
		IQE 10	0.41	0.412	0.415
	Constant	IDE		0.718	0.716
		IQE 10		1.598	1.604
Dibromochloromethane (502.2)	Hybrid	IDE	0.1397	0.4531	0.1406
		IQE 10	0.33	1.081	0.330
	Exponential	IDE	0.1430	0.1502	0.1441
		IQE 10		0.348	0.349
	Linear	IDE		0.4389	0.4359
		IQE 10	1.25	1.252	1.252
	Constant	IDE		0.786	0.784
		IQE 10		1.750	1.757
Lead (200.8)	Hybrid	IDE	0.2001	0.3318	0.2005
		IQE 10	0.46	0.752	0.462
	Exponential	IDE	0.2033	0.2086	0.2038
		IQE 10		0.477	0.478
	Linear	IDE		0.2705	0.2650
		IQE 10	0.68	0.684	0.685
	Constant	IDE		1.024	1.007
		IQE 10		2.296	2.305

Table 15. Comparison of SL-IDEs and SL-IQEs Calculated Using Different Software

Analyte	Model Type	Limit	QCalc	Excel	SAS 1
M+p Xylene (502.2)	Hybrid	IDE	0.0876	0.0872	0.0883
		IQE 10	0.22	0.255	0.222
	Exponential	IDE	0.1197	0.1208	0.1205
		IQE 10		0.285	0.285
	Linear	IDE		0.0053	0.0052
		IQE 10	0.03	0.030	0.031
	Constant	IDE		1.704	1.701
		IQE 10		3.795	3.813
Methylmethacrylate (524.2)	Hybrid	IDE	0.2522	-0.0267	0.2441
		IQE 10	0.56	-0.364	0.561
	Exponential	IDE	0.5528	0.5615	0.5350
		IQE 10		not calc [3]	1.228
	Linear	IDE		-0.3617	-0.3415
		IQE 10	not calc [3]	not calc [3]	-1.043
	Constant	IDE		9.734	9.597
		IQE 10		20.667	20.773
Sec-butylbenzene (524.2)	Hybrid	IDE	0.0194	0.0205	0.0195
		IQE 10	0.05	0.050	0.047
	Exponential	IDE	0.1388	0.1403	0.1397
		IQE 10		0.316	0.316
	Linear	IDE		0.0803	0.0798
		IQE 10	0.28	0.279	0.279
	Constant	IDE		11.258	11.240
		IQE 10		25.074	25.203
Selenium (200.8)	Hybrid	IDE	0.3565	0.4600	0.3637
		IQE 10	0.83	1.045	0.829
	Exponential	IDE	0.4076	0.4159	0.4159
		IQE 10		0.957	0.957
	Linear	IDE		0.4057	0.4059
		IQE 10	1.04	1.044	1.045
	Constant	IDE		2.082	2.090
		IQE 10		4.668	4.686
Selenium (1620)	Hybrid	IDE	-0.3256	2.2850	1.9709
		IQE 10	-4.47	5.107	4.474
	Exponential	IDE	1.9742	2.0045	1.9754
		IQE 10		4.653	4.657
	Linear	IDE		2.0809	2.0539
		IQE 10	5.23	5.231	5.235
	Constant	IDE		4.195	4.161
		IQE 10		9.221	9.268
Sodium (1620)	Hybrid	IDE	139.8852	145.2512	140.8112
		IQE 10	317.64	326.198	317.747
	Exponential	IDE	137.8479	139.6656	138.7678
		IQE 10		323.711	323.935

Table 15. Comparison of SL-IDEs and SL-IQEs Calculated Using Different Software

Analyte	Model Type	Limit	QCalc	Excel	SAS [1]
	Linear	IDE		142.1564	141.2901
		IQE 10	337.63	337.515	337.755
	Constant	IDE		169.406	169.136
		IQE 10		377.295	379.229
Styrene (524.2)	Hybrid	IDE	0.0175	-5.70E-08	0.0174
		IQE 10	0.04	-8.40E-07	0.041
	Exponential	IDE	0.1407	0.1423	0.1405
		IQE 10		not calc [3]	0.318
	Linear	IDE		-0.6099	-0.6000
		IQE 10	not calc [3]	not calc [3]	-2.180
	Constant	IDE		10.555	10.516
		IQE 10		23.301	23.420
Vanadium (1620)	Hybrid	IDE	10.6227	11.4032	10.6931
		IQE 10	24.33	25.889	24.338
	Exponential	IDE	10.5597	10.7036	10.6304
		IQE 10		25.094	25.112
	Linear	IDE		10.0290	9.9671
		IQE 10	26.04	26.029	26.049
	Constant	IDE		22.757	22.721
		IQE 10		50.684	50.943
Vinyl Chloride (524.2)	Hybrid	IDE	0.0840	-2.30E-07	0.0834
		IQE 10	0.22	-9.78E-07	0.219
	Exponential	IDE	0.3671	0.3701	0.3649
		IQE 10		not calc [3]	0.837
	Linear	IDE		-3.4286	-3.3451
		IQE 10	49.30	not calc [3]	49.158
	Constant	IDE		22.474	22.292
		IQE 10		49.394	49.647
Yttrium (1620)	Hybrid	IDE	3.2571	3.6382	3.2787
		IQE 10	7.51	8.305	7.516
	Exponential	IDE	3.2251	3.2726	3.2468
		IQE 10		7.833	7.839
	Linear	IDE		3.5420	3.5202
		IQE 10	8.96	8.955	8.962
	Constant	IDE		4.576	4.569
		IQE 10		10.192	10.244

[1] Calculated using SAS programs written by EPA to run IDE and IQE calculations. Results are the same as those presented in Tables 2 and 4.

[2] Limits in bold indicate the calculated IDE or IQE based on the model suggested as most appropriate based on the given software.

[3] No value could be calculated due to model not converging.

[4] Based on statistical tests, QCalc determined that the constant model should be used to calculate the IDE and IQE. However, determination of the IDE and IQE using the constant model is not run by this program.

Table 16. Summary Statistics of Ratios Comparing IDEs/IQEs using different Software Packages

Comparison Ratio	Model Type	Limit	Minimum	25th Percentile	Median	75th Percentile	Maximum
QC_{calc}/ SAS	Hybrid	IDE	-0.17	0.99	0.99	1.00	1.03
		IQE 10	-1.00	0.99	1.00	1.00	1.07
	Exponential	IDE	0.98	0.99	0.99	1.00	1.03
	Linear	IQE 10	0.97	0.99	1.00	1.00	1.00
Excel/ SAS	Hybrid	IDE	-0.11	-0.000003	1.10	1.32	3.22
		IQE 10	-0.65	-0.000009	1.06	1.35	3.27
	Exponential	IDE	1.00	1.01	1.01	1.02	1.05
		IQE 10	0.99	1.00	1.00	1.00	1.00
	Linear	IDE	1.00	1.01	1.01	1.02	1.06
		IQE 10	0.97	1.00	1.00	1.00	1.00
	Constant	IDE	0.99	1.00	1.00	1.01	1.02
		IQE 10	0.98	0.99	0.99	1.00	1.00
QC_{calc}/ Excel	Hybrid	IDE	-365,000	-12.85	0.54	0.93	1.01
		IQE 10	-225,000	-2.07	0.52	0.91	1.01
	Exponential	IDE	0.96	0.98	0.99	0.99	0.99
	Linear	IQE 10	0.99	1.00	1.00	1.00	1.00

Table 17. Comparison of Simulated 7-replicate ACIL CRVs to Overall CRV, ACIL Blanks

Analyte	# Blanks *	Overall CRV	# simulated 7-replicate CRVs	Mean of Simulated 7-replicate CRVs	Range of Simulated 7-replicate CRVs	Range of Days between 1st and Last of 7 consecutive replicates	% short-term CRVs exceeding Overall CRV
Barium	26	0.0039	20	0.0039	0.0011 to 0.0083	7 to 26	30
Cadmium	33	0.0012	27	0.0014	0.00044 to 0.0019	11 to 24	67
Chromium	55	0.0048	49	0.0051	0.0014 to 0.0117	7 to 20	29
Copper	52	0.0035	46	0.0039	0.0010 to 0.0059	7 to 20	78
Silver	45	0.0105	39	0.0100	0.0019 to 0.0326	7 to 20	28

* Analyzed over a period of 3 months

Table 18. Comparison of Simulated 7-replicate ACIL CRVs to Overall CRV, ACIL Blanks After Outlier Removal

Analyte	# Blanks *	Overall CRV	# simulated 7-replicate CRVs	Mean of Simulated 7-replicate CRVs	Range of Simulated 7-replicate CRVs	Range of Days between 1st and Last of 7 consecutive replicates	% short-term CRVs exceeding Overall CRV
Barium	25	0.0020	19	0.0021	0.0011 to 0.0029	11 to 26	74
Chromium	54	0.0040	48	0.0044	0.0014 to 0.0080	7 to 20	56
Silver	42	0.0031	36	0.0038	0.0019 to 0.0058	8 to 21	72

* Analyzed over a period of 3 months

This Appendix is included to support Appendices B of this Assessment Document, by providing example calculations of the single-laboratory variants of the Interlaboratory Detection Estimate (SL-IDE) and Interlaboratory Quantitation Estimate (SL-IQE) as described in ASTM D6091 and ASTM D6512, respectively. Example calculations of the method detection limit (MDL) and minimum level of quantitation (ML) also are included. The example calculations provided in this Appendix were used in the data analyses presented in Appendix B.

All abbreviations and symbols used in the SL-IDE and SL-IQE calculations match those given in the ASTM procedures. The linear and exponential standard deviation models and all recovery models were fit using the PROC REG procedure in SAS Version 8.1. The hybrid standard deviation model was fit using Newton's Non-Linear Least Squares procedure as described in ASTM D6512, programmed using SAS Version 8.1. The dataset used in these examples is that included for 1,1,1,2- tetrachloroethane in EPA's Episode 6000 (see Chapter 1 and Appendix B of this document for descriptions of datasets).

Single-Laboratory IDE (SL-IDE)

The procedure for calculating the IDE that is described in ASTM D6091 stipulates use of data from multiple laboratories. However, because analytes in the Episode 6000 dataset were only measured by a single laboratory, EPA calculated a variant of the IDE which was called the single-laboratory IDE (SL-IDE). The SL-IDE and the analyses performed using the SL-IDE are described in greater detail in Appendix B of this Assessment document.

In order to calculate the SL-IDE, means and standard deviations are needed for each spike level. The means and standard deviations for 1,1,1,2-tetrachloroethane are listed in Table 1.

Table 1. Mean and Standard Deviation Calculated at each Spike Level

Spike (ug/L)	N	Mean (ug/L)	SD (ug/L)
0.01	7	0.0016	0.0018
0.015	7	0.001	0.0017
0.02	7	0.0007	0.0010
0.035	7	0.0057	0.0036
0.05	7	0.0081	0.0024
0.075	7	0.0263	0.0202
0.1	6	0.0295	0.0039
0.15	7	0.0536	0.0046
0.20	7	0.0991	0.0158

Spike (ug/L)	N	Mean (ug/L)	SD (ug/L)
0.35	7	0.235	0.0078
0.50	7	0.3744	0.0257
0.75	6	0.6193	0.0262
1.0	8	0.8368	0.0814
2.0	7	1.9560	0.0980
5.0	8	5.0994	0.2382
10.0	7	10.4453	0.5469

In order to choose the appropriate model to calculate the IDE, significance tests were used.

The fitted unweighted linear model was:

S = 0.000039515 + 0.05326 * T, where T corresponds to spike concentration.

The slope of this model was significantly greater than 0, and therefore the constant model was rejected.

The fitted unweighted exponential model (fit by natural log-transforming standard deviations) was:

Log(S)= -5.02407 + 0.54851 * T

The slope of this model was significantly greater than 0, thus, the linear model was rejected.

Based on this assessment, the exponential model was used in Appendix B to calculate the IDE for this analyte. While the exponential model was chosen as the most appropriate model for this analyte, the calculation of the SL-IDE using all four model types is presented in this Appendix. This was done to provide a step-by-step example for the calculation of the SL-IDE using all of the different model types.

Constant model: The pooled within-spike variance was first calculated using the equation below:

$$g^2 = \frac{\sum_{i=1}^{16}[(n_i - 1) * s_i^2]}{\sum_{i=1}^{16} n_i - 16}$$

where: s_i is the standard deviation of the results for spike level i, and
n_i is the number of replicates for spike level i.

The calculated pooled within-spike variance (g^2) is 0.024, and the square root of this value, g, equals 0.155.

A linear regression model was then fit for the mean results for the 16 spike levels. The estimates of slope and intercept for this model are: a = -0.089 and b=1.0478, respectively.

Based on these results:

$YC = (k1 * g) + a = (0.155 * k1) - 0.089 = (0.155 * 2.6) - 0.089 = 0.3137$

where: YC = the recovery critical value as defined in ASTM D6091, and
k1 = 2.6 (a conservative number based on the total n of 112)

$LC = (YC - a)/b = (0.3137 + 0.089) / 1.0478 = 0.3848$

where: LC = the true concentration critical value as defined in ASTM D6091.

$IDE = LC + (k2 * g)/b = 0.3848 + (1.86 * 0.155)/1.0478 = 0.660$

where: k2 = 1.86 (a conservative number based on the total n of 112).

Linear Model:

An unweighted linear regression model was fit, predicting standard deviation based on concentration, using PROC REG in SAS Version 8.1. The estimated parameters are: g = 0.0000392 and h = 0.05326. Based on these parameters, weights for the recovery model were calculated for each spike value. For each concentration, the weight was calculated as:

$$weight = \frac{1}{\hat{s}_i^2} = \frac{1}{(g + h * T_i)^2}$$, for each true concentration T_i.

The calculated weights are given in Table 2.

Table 2. Calculated Weights based on Linear Model

Spike (ug/L)	Est. SD (ug/L)	Weight
0.01	0.00057	3,058,709
0.015	0.00084	1,423,673
0.02	0.00110	819,854
0.035	0.00190	276,031
0.05	0.00270	136,940
0.075	0.00403	61,454
0.1	0.00537	34,736
0.15	0.00803	15,514

Spike (ug/L)	Est. SD (ug/L)	Weight
0.20	0.01069	8,748
0.35	0.01868	2,865
0.50	0.02667	1,406
0.75	0.03999	625.4
1.0	0.05330	352.0
2.0	0.10657	88.1
5.0	0.26635	14.1
10.0	0.53267	3.52

Using these weights, the fitted recovery model estimates were a = -0.00898 and b = 0.6860. Based on these results:

YC = (k1 * g) + a = (0.0000392 * 2.6) - 0.00898 = -0.00888, and

LC = (YC - a)/b = (-0.00888 + 0.00898) / 0.6860 = 0.00015

For the linear model, the SL-IDE must be calculated recursively. The initial estimate of the SL-IDE, LD_0, was:

$LD_0 = LC + (k_2 * s(0)) / b = 0.00025$.

Each following estimate was calculated using the recursive formula:

$$LD_{i+1} = [k_1 * \hat{s}(0) + k_2 * (g + h * LD_i)] / b$$

Results of the recursive LD calculations are given in Table 3.

Table 3. Recursive SL-IDE Calculations, Linear Model

LD estimate run	LD estimate
0	0.000255
1	0.000291
2	0.000297
3	0.000297

The recursive estimates of LD converge to 6 decimal places by the third iteration. Therefore, the linear model estimate of the IDE = 0.000297 ug/L.

Exponential Model:

An unweighted linear regression model was fit, predicting natural log-transformed standard deviation based on concentration. The estimated parameters are: g = 0.00658 and h = 0.54851. Based on these parameters, weights for the recovery model were calculated for each spike value. For each concentration, the weight was calculated as:

$$weight = \frac{1}{\hat{s}_i^2} = \frac{1}{(g * e^{h*T_i})^2}$$, for each true concentration T_i.

The calculated weights are given in Table 4.

Table 4. Calculated Weights based on Exponential Model

Spike (ug/L)	Est. SD (ug/L)	Weight
0.01	0.00661	22,861
0.015	0.00663	22,736
0.02	0.00665	22,611
0.035	0.00671	22,242
0.05	0.00676	21,879
0.075	0.00685	21,287
0.1	0.00695	20,711
0.15	0.00714	19,606
0.20	0.00734	18,560
0.35	0.00797	15,744
0.50	0.00865	13,355
0.75	0.00993	10,152
1.0	0.01138	7,717
2.0	0.01970	2,576
5.0	0.10213	96
10.0	1.58566	0.40

Using these weights, the fitted recovery model estimates were a = -0.04585, and b = 0.91696. Based on these results:

YC = (k1 * g) + a = (0.00658 * 2.6) - 0.04585 = -0.0287, and

LC = (YC - a)/b = (-0.0287 + 0.04585) / 0.91696 = 0.0187

For the Exponential model, the SL-IDE must be calculated recursively. The initial estimate of the SL-IDE, LD_0, was:

$LD_0 = LC + (k_2 * s(0)) / b = 0.03199.$

Each following estimate was calculate using the recursive formula:

$$LD_{i+1} = [k_1 * \hat{s}(0) + k_2 * (g * e^{h*LD_i})] / b$$

Results of the recursive LD calculation are given in Table 5, below.

Table 5. Recursive SL-IDE Calculations, Exponential Model

LD estimate run	LD estimate
0	0.031993
1	0.032229
2	0.032231

The recursive estimates of LD converge to 6 decimal places by the second iteration. Therefore, the exponential model estimate of the IDE = 0.032231 ug/L.

Hybrid Model:

The Hybrid model was fit using Newton's Method for Non-linear Least Squares. Summary statistics from this fit of the hybrid model are presented in Table 6, using the same notation as shown in ASTM D6512-00.

Table 6. Summary Statistics from Newton's Non-Linear Least Squares

Run	g	h	u	v	c	d	p	q	Δg	Δh	dg%	dh%
0	0.00095	0.05465	1,254330	4,285	19,889	2×10^{-10}	555.95	-0.592	0.00048	-0.00237	50.5	43.4
1	0.00143	0.05228	981,892	4,275	15,368	3×10^{-10}	41.83	-1.132	0.00005	-0.00044	3.45	8.5
2	0.00148	0.05184	958,193	4,309	15,092	3×10^{-10}	4.47	-0.123	5×10^{-6}	-0.00005	0.37	0.9

Because dg% (the percent difference between the last 2 estimates of g) and dh% (the percent difference between the last 2 estimates of h) were both less than 1% in run 2, the model converged, and the estimated parameters of the hybrid model were:

$$g = g_{run\,2} + \Delta g_{run\,2} = 0.00148 + 0.000005 = 0.00149$$
$$h = h_{run\,2} + \Delta h_{run\,2} = 0.05184 - 0.00005 = 0.05179$$

Using these fitted parameters, the weights for the recovery model were calculated as shown in Table 7.

Table 7. Calculated Weights, Hybrid Model

Spike (ug/L)	Est. SD (ug/L)	Weight
0.01	0.00158	403,037
0.015	0.00168	355,066
0.02	0.00181	304,351
0.035	0.00234	181,881
0.05	0.00299	112,141
0.075	0.00416	57,811
0.1	0.00539	34,447
0.15	0.00791	15,987
0.20	0.01046	9,134
0.35	0.01819	3,024
0.50	0.02594	1,487
0.75	0.03887	662
1.0	0.05181	373
2.0	0.10358	93.2
5.0	0.25893	14.9
10.0	0.51786	3.73

Using these weights, the fitted recovery model estimates were a = -0.01471, and b = 0.74338. Based on these results:

YC = (k1 * g) + a = (0.00149 * 2.6) - 0.01471 = -0.01085, and

LC = (YC - a)/b = (-0.01085 + 0.01471) / 0.74338 = 0.00520

LD had to be calculated recursively. The initial estimate of LD was:

LD_0 = LC + (k_2*s(0)) / b = 0.00893.

Each following estimate was calculated using the recursive formula:

$$LD_{i+1} = [k_1 * \hat{s}(0) + k_2 * (g * e^{h*LD_i})] / b$$

Results of the recursive LD calculation are given in Table 8.

Table 8. Recursive SL-IDE Calculations, Hybrid model

LD estimate run	LD estimate
0	0.008925
1	0.009101
2	0.009108
3	0.009108

The recursive estimates of LD converge to 6 decimal places by the third iteration. Therefore, the hybrid model estimate of the IDE = 0.009108 ug/L.

Single-Laboratory IQE (SL-IQE)

The procedure for the IQE described in ASTM D6512 stipulates use of data from multiple laboratories. However, because analytes in the Episode 6000 dataset were only measured by a single laboratory, EPA calculated a variant of the IQE which was called the single-laboratory IDE (SL-IQE). The SL-IQE and the analyses performed using the SL-IQE are described in greater detail in Appendix B of this Assessment document.

Fitting and selection of models in the IQE calculation process are identical to the IDE calculation process except:

- The Hybrid model was considered in model selection instead of the Exponential model, based on significance tests for curvature as described in 6.3.3.2 (g) - (i) of ASTM D6512.
- A bias-correction adjustment factor is applied to calculated standard deviations prior to modeling as described in 6.3.3.2 (b) of ASTM D6512.

Therefore, the example calculation begins with the fitted model parameters for each model type, and demonstrates the calculation of each IQE value.

Constant model:

Using the same steps for fitting the constant model as described in the SL-IDE example, the fitted precision and recovery model parameters are determined to be:

g = 0.1615
a = -0.0894, and b = 1.0478.

The IQE (10%) was calculated as: IQE (10%) = (g/b)*(100/10) = 1.541

The IQE (20%) was calculated as: IQE (20%) = (g/b)*(100/20) = 0.770

The IQE (30%) was calculated as: IQE (30%) = (g/b)*(100/30) = 0.514

Linear model:

Using the same steps for fitting the linear model as described in the SL-IDE example, the fitted precision and recovery model parameters are determined to be:

$g = 4.2 \times 10^{-7}$, h = 0.0555
a = -0.0087, b = 0.6810

The IQE (10%) was calculated as: IQE (10%) = g/(b*(10/100)-h) = 3.3×10^{-5}

The IQE (20%) was calculated as: IQE (20%) = g/(b*(20/100)-h) = 5.2×10^{-6}

The IQE (30%) was calculated as: IQE (30%) = g/(b*(30/100)-h) = 2.8×10^{-6}

Hybrid model:

Using the same steps for fitting the hybrid model as described in the SL-IDE example, the fitted precision and recovery model parameters are determined to be:

g = 0.00155, h = 0.0540
a = -0.0147, b = 0.7434

The IQE (10%) was calculated as:

$$IQE(10\%) = \frac{g}{\sqrt{\left(\frac{10*b}{100}\right)^2 - h^2}} = 0.0304$$

The IQE (20%) was calculated as:

$$IQE(20\%) = \frac{g}{\sqrt{\left(\frac{20 * b}{100}\right)^2 - h^2}} = 0.0112$$

The IQE (30%) was calculated as:

$$IQE(30\%) = \frac{g}{\sqrt{\left(\frac{30 * b}{100}\right)^2 - h^2}} = 0.0072$$

Exponential model:

Using the same steps for fitting the constant model as described in the SL-IDE example, the fitted precision and recovery model parameters are determined to be:

g = 0.0069, h = 0.5482
a = -0.0459, b = 0.9170

For the Exponential model, the IQE must be solved recursively. The initial estimate of the IQE was set to the IDE (re-calculated using bias-corrected standard deviations, and therefore not matching the IDE presented in the example above). The IQE was then re-calculated using the estimate from the prior round, based on the equation below:

$$IQE(Z)_{i+1} = \frac{100g * e^{h * IQE(Z)_i}}{Zb},$$

where: Z i = 10, 20 or 30, depending on the IQE being calculated.

Results of the recursive calculations for the IQEs are given in Table 9.

Table 9. Recursive SL-IDE Calculations, Exponential model

Run	IQE (10%)	IQE (20%)	IQE (30%)
0	0.0355	0.0355	0.0355
1	0.0763	0.0381	0.0254
2	0.0780	0.0382	0.0253
3	0.0781	0.0382	0.0253
4	0.0781	0.0382	0.0253

MDL/ML

This section gives an example calculation of the MDL and ML determined using the Episode 6000 data, and presented in Appendix B. Due to the nature of the study design, MDLs could not be determined following the MDL procedure directly. Therefore, the MDL was calculated based on the results of the two lowest spike levels with all positive results for which the standard deviations were not significantly different.

The lowest two spike levels with all positive, non-zero results are 0.050 µg/L and 0.075 µg/L. From Table 1, the standard deviations at these concentrations are 0.0024 µg/L and 0.0202 µg/L, respectively. The F test was then run on the variances at these two spike levels:

$$F = \frac{(0.0202)^2}{(0.0024)^2} = \frac{0.0004}{0.000006} = 70.385$$

The critical value for the F test at α=0.10, where both variances are based on 7 results, is 3.05. Because 70.385 > 3.05, the variance at the higher concentration is significantly greater than the variance at the lower concentration, and these two concentrations cannot be used to calculate the MDL.

The next lowest spike level (0.10 µg/L) has only 6 results, but all results are greater than 0. Therefore, an F test was run comparing variances at 0.075 µg/L and 0.10 µg/L. From Table 1, the standard deviation at 0.10 µg/L is 0.0039 µg/L. The results of the F test are:

$$F = \frac{(0.0039)^2}{(0.0202)^2} = \frac{0.00002}{0.0004} = 0.037$$

The critical value for this F test is 3.11, slightly higher than for the prior comparison due to the fewer number of results at the higher spike level. Because $0.037 < 3.11$, the variance at the higher spike level is not significantly greater than the variance at the lower spike level. Therefore, the MDL is calculated based on these two spike levels:

$$MDL = \sqrt{\frac{(6-1)*(0.0039)^2+(7-1)*(0.0202)^2}{(6-1)+(7-1)}} * t_{(0.99,7+6-2)}$$

$$= 0.015 * 2.71$$

$$= 0.041$$

The ML is determined by first multiplying the pooled standard deviation (0.015 µg/L from the calculation above) by 10. This yields a result of 0.15 µg/L. Based on the ML rounding scheme, this becomes 0.2 µg/L.

DECLARATION OF JAMES DeWOLF

DECLARATION OF JAMES DeWOLFE

I, James DeWolfe, declare:

1. I am employed by Arcadis U.S., Inc. ("Arcadis"), as a Principal Environmental Engineer. My resume is attached to this Declaration as Exhibit A. Pacific Gas and Electric Company ("PG&E") has engaged Arcadis to assist with issues surrounding the chromium plume in Hinkley, California. I have been working on chromium treatment-related issues for PG&E since October 2009. I was asked to lead a team tasked with analyzing the feasibility of Draft Cleanup and Abatement Order No. R6V-2011-0005A1 (the "Draft CAO") insofar as it requires whole-house water replacement to residents and businesses whose well water supplies have potentially been impacted by the Hinkley chromium plume. The team consisted of Arcadis' Dennis Reid, Scott Seyfried, Katie Porter, Nicole Blute, Edward Means, Sunil Kommineni, Jenifer Beatty and me. The opinions I express in this Declaration are a result of our collective analysis.

2. My opinion is that:

(a) The Draft CAO's replacement water requirements are not feasible because there is no known technology or combination of technologies that can reliably achieve hexavalent chromium levels of 0.02 ppb or less; and

(b) Even if the appropriate technologies were available, the deadlines set forth in the Draft CAO cannot be met.

3. For purposes of our analysis, we made the following assumptions:

(a) Because the declared background Cr6 concentrations in the Hinkley area average 1.2 ppb and have a declared maximum value of 3.1 ppb, we assumed all wells in the "affected area," as defined in the Draft CAO, will have Cr6 levels above the Public Health Goal

("PHG") of 0.02 ppb (*i.e.*, two orders-of-magnitude below the declared average and declared maximum background Cr6 levels). The deadlines contained in the Draft CAO would not provide sufficient time for testing and analysis to determine the exact number of "impacted wells," as defined in the Draft CAO. Based on this assumption and a review of the number of homes within the "affected area," we predict that the Draft CAO, if adopted, would require that interim replacement water be provided to between 250 and 300 homes.

(b) Cr6 concentrations in the Hinkley area wells are known to fluctuate over time in a nearly random pattern. Due to the natural variability in Cr6 detection at any given well, we assumed that at any given point in time one-third of the wells in the "affected area" will have decreasing Cr6 concentrations, one-third will have stable concentrations and one-third will have increasing concentrations. Consequently, we estimate that 100 homes (one third of all wells in the "affected area") will require permanent replacement water.

(c) According to the Draft CAO, the interim replacement water supply must, "at a minimum," provide enough water for "drinking, cooking, and swamp cooler needs." Assuming an average of three occupants per household, we estimate that the average household will consume (via ingestion) 33 gallons per day ("gpd") for drinking and cooking based on the estimates for daily per capita faucet use found in the 1999 study *Residential End Uses of Water* by the American Water Works Association. Swamp coolers can add 40 gpd per household per day during warm months. In that regard, see http://www.consumerenergycenter.org/home/heating_cooling/evaporative.html.) These values do not include water used for other purposes, such as washing, showering, and irrigation – water use in these categories can vary widely. Thus, in order to comply with the interim replacement water requirements of the Draft CAO, PG&E would need to provide 73 gpd to the average household during the warm months. In light of our estimate that between 250 and 300 locations

would qualify, each month during the warm seasons PG&E would need to provide between 547,500 and 657,000 gallons of interim replacement water with Cr6 levels at or below 0.02 ppb.

(d) In light of our estimate that 100 locations would qualify for permanent replacement water, based on *Residential End Uses of Water* estimate of approximately 60 gpd per capita of indoor water use and the above described estimates for swamp cooler use, during each month of the warm seasons, PG&E would need to provide 660,000 gallons of permanent replacement water with Cr6 levels at or below 0.02 ppb.

4. Most chromium treatment studies that have focused on hexavalent chromium treatment have had target effluent concentrations of 1 to 5 ppb. Those studies include Brandhuber, *et al.*, *Low-Level Hexavalent Chromium Treatment Options: Bench-Scale Evaluation*, Project 2814, Water Research Foundation, Denver, Colorado, 2005; and McGuire, *et al.*, *Hexavalent Chromium Removal Using Anion Exchange and Reduction with Coagulation and Filtration*, Project 3167, Water Research Foundation, Denver, Colorado, 2007. Those targets are the manifestation of a scientific consensus that trying to achieve hexavalent chromium concentrations below 1 ppb is unrealistic at this time.

5. Outside of the laboratory, experiments with treatment technologies target much higher hexavalent chromium concentrations than what would be called for by the Draft CAO. For example, at West County Road 112 in Midland, Texas, the Texas Commission on Environmental Quality has installed whole-house, ion exchange treatment systems in forty-five homes. But those systems are targeting total chromium concentrations of 100 ppb. The Midland, Texas project is described at http://www.tceq.texas.gov/remediation/sites/cr112.html.

6. My team analyzed the available technologies and mechanisms for achieving the results that would be required by the Draft CAO. My conclusions are set forth in the following paragraphs. My overall conclusion is that reliably providing replacement water meeting the PHG of 0.02 ppb hexavalent chromium on the timeline set forth in the Draft CAO is technically infeasible.

7. Currently, there is no drinking water standard specific to hexavalent chromium in bottled water. Total chromium, which includes hexavalent chromium, in bottled water is regulated by the 100 ppb EPA standard for total chromium.

8. We considered the possibility of using bottled water to satisfy the requirements of the Draft CAO. Providing between 547,500 and 657,000 gallons of bottled water each month to between 250 and 300 locations throughout Hinkley poses logistical obstacles that could not be overcome in two weeks. In my opiuion, the distribution of bottled water is the best alternative available, but would not satisfy the Draft CAO's requirements for the following reasons:

(a) The treated bottled water concentrations for hexavalent chromium are typically significantly greater than 0.02 ppb. In that regard, see Krachler, M. and Shotyk, W. (2008), *Trace and Ultratrace Metals in Bottled Waters: Survey of Sources Worldwide and Comparison With Refillable Metal Bottles,*" Science of the Total Environment, 407:1089-1096 (132 brands surveyed with Cr6 concentrations ranging from 0.06 to 172 ppb and a median of 8.2 ppb).

(b) PG&E could not monitor at the source the extent to which bottled water distributed to the Hinkley community met the 0.02 ppb standard because bottled water providers (i) are not required to report or declare the hexavalent chromium concentrations to the consumers or regulators and (ii) often use water from different plants and employ different treatment processes.

(c) Theoretically, PG&E could test the bottles for Cr6 after they leave the plant. But in doing so, PG&E would be confronted with an almost impossible testing protocol. Because bottled water under one label often comes from multiple sources and has undergone different treatment processes, PG&E would have to test all of the bottles. In doing so, PG&E would necessarily have to break the seals, thereby exposing the water to microbial activity. And if a shipment of bottled water failed to meet the 0.02 ppb standard, PG&E would be forced to switch suppliers. But the new supplier is likely to use multiple sources and treatment processes, thereby creating the same problems associated with the original supplier.

9. We considered the use of bulk water delivery to homes and business in Hinkley by tanker trucks to satisfy the requirements of the Draft CAO and, for the following reasons, I concluded that it is not a viable option:

(a) Depending on the source of the water, the hexavalent chromium concentrations will likely be significantly greater than 0.02 ppb, the exact concentration depending on the source of the water.

(b) The bulk water delivery strategy would create ancillary problems. Bulk water will age in the storage tanks, and its quality will deteriorate over time. This could be partially mitigated by the addition of disinfectants to maintain microbiological quality, but that can create other risks.

10. I concluded that using water from Golden State Water Company would not satisfy the requirements of the Draft CAO for the following reasons:

(a) The design, planning, permitting and construction of transmission mains and a new distribution system would take at least a year and probably more than two years.

(b) Golden State Water Company's groundwater likely contains hexavalent chromium concentrations in excess of 0.02 ppb. Thus, treatment would be required via ion exchange, reverse osmosis, or reduction, clarification, and filtration technologies, or some combination of these technologies. All of the obstacles and limitations of those technologies, which I address in the following paragraphs, would have to be overcome.

11. We considered whole-house treatment using ion exchange to satisfy the requirements of the Draft CAO. But this technology is still unproven to treat to the 0.02 ppb level and, in any event, would create other significant environmental, logistical, health and safety issues. Therefore, I have concluded that it is not a viable option:

(a) Multi-stage ion exchange system with pH adjustment capability using acid and caustic feed systems are likely needed to meet the 0.02 ppb standard, but extensive and lengthy testing would be needed to demonstrate this technology.

(b) Incorporating acid and caustic feed systems for the whole-house treatment poses health, safety and operational concerns. Ion exchange treatment would generate a liquid residual stream, either brine or caustic, that would contain elevated concentrations of hexavalent chrominm and other constituents that could be classified as hazardous waste under federal law. There is also the practical problem that septic systems may not have the capacity to handle the flow from the ion exchange regeneration process, and the biological processes within the septic system would likely be negatively affected. Furthermore, effluent from the septic tanks entering a drain field would then likely reintroduce chromium to the environment.

(c) The ion exchange process can result in "chromatographic peaking" of other constituents, such as nitrate and sulfate. Chromatographic peaking is a phenomenon in which less preferentially absorbed ions appear in the effluent at higher concentrations than they appear in the influent as they are released from ion exchange resin when more strongly held ions

(in this case, chromium) are adsorbed. Multiple ion exchange units in either series or parallel operation and frequent monitoring can help minimize chromatographic peaking occurrence, bnt this adds substantial levels of operational complexity that are beyond the capabilities of most homeowners.

(d) Additional engineering studies would be necessary to achieve low-level hexavalent chromium targets, because the systems currently on the market are not designed to achieve 0.02 ppb levels. I predict that such studies will reveal that other constituents – iron, manganese and arsenic – would require removal prior to chromium treatment. This would be particularly problematic in home-based units because of added operational complexity and the generation of waste streams that require special handling.

(e) The California Department of Pnblic Health ("CDPH") allows the use of whole-house treatment systems for specific contaminants removal only on a limited basis, and there must be fewer than 200 connections. PG&E would need to apply to CDPH for a permit, and CDPH would not issue it until a pilot project was designed and completed. That process would take two to six months. Even then, CDPH typically only allows the use of whole-house treatment systems as an interim measure, perhaps for three years or less, until an alternative source is in place. CDPH would likely conclude that potential unforeseen risks of a new or untested technology would outweigh any public health benefit achieved by lowering Cr6 concentrations below natural background levels.

12. We considered whole-house treatment using reverse osmosis (RO) membranes to satisfy the requirements of the Draft CAO and, for the following reasons, I concluded that it is not a viable option:

(a) Assuming hexavalent chromium in the influent of 3.2 ppb and a treatment goal of 0.02 ppb, the RO membrane treatment needs to achieve 99.5 percent removal. A single-pass RO treatment system cannot likely remove the necessary quantities of hexavalent chromium to meet the 0.02 ppb goal. Consequently, a multi-pass RO system would be necessary.

(b) A multi-pass RO system will generate a significant quantity of rejected water that would require disposal. Approximately 50 to 75 percent of the feed flow will likely be rejected. Disposing of large volumes of RO reject to septic tanks is likely impossible, and would likely have deleterious impacts on the biological activities within the septic tanks. Furthermore, effluent from the septic tanks entering a drain field would then likely reintroduce chromium to the environment.

(c) The energy required to operate multi-pass RO systems will increase electrical power consumption and lead to higher electric utility bills. For example, a device utilizing 1,000 watts operating for twelve hours per day, with a $0.10/kilowatt-hour would cost $33.60 per month to operate. Were RO systems to be operating in multiple homes at the same time, there could be a significant load on the electrical power grid, depending on the number of homes utilizing a RO system. Also, separate breakers and adequate power services would be required to provide electricity for the operation of these RO systems. In older homes, this may require substantial upgrades to electrical services, which requires adequate time to plan, acquire and install the required components for an electrical service upgrade.

(d) The presence of other scale-forming compounds – such as silica, sulfate, barium and strontium – will limit the product water to feed water ratio.

(e) The RO systems currently on the market are not designed to achieve 0.02 ppb levels, so engineering advancements would likely be required to achieve 0.02 ppb levels.

(f) Pretreatment of waters prior to the use of RO may also be required to address the removal of performance-impacting constituents, which further complicates utilizing this technology for whole-house treatment to reliably meet the 0.02 ppb goal.

13. We considered the implementation of reduction, clarification and filtration (RCF) technologies via centralized treatment to satisfy the requirements of the Draft CAO, and primarily because the technology has not been demonstrated to produce effluent with a level of 0.02 ppb Cr6 or less, I concluded that they are not a viable option:

(a) The RCF process has been used only on a pilot-project scale, and those projects have demonstrated substantial logistical and process control issues.

(b) Separate unit processes are required to convert hexavalent chromium to the trivalent form (reduction), followed by oxidation to form large particles for settling (clarification), and also granular media extracted by low-pressure membrane filters (filtration). Extensive pilot testing would be required and could take a year or more to demonstrate. Furthermore, system operators would require advanced skills and extensive certifications that would require substantial training and CDPH approval, further lengthening the approval process for such a technology.

(c) Given RCF's present limitations, the effluent from this process would likely require RO treatment to achieve the 0.02 ppb goal for hexavalent chromium, which further complicates treatment. Those considerations, as well as RO's associated design and operational complications, are described above.

14. We considered the implementation of a central treatment and distribution system to satisfy the requirements of the Draft CAO, but a centralized treatment scheme itself would not achieve the 0.02 ppb goal. Central treatment would likely employ one or more of the technologies analyzed above: Ion exchange, multiple stage RO and/or RCF. The technologies

have simply not been proven to be able to achieve 0.02 ppb Cr6 concentrations. Furthermore, implementing a central treatment system would take far longer than the Draft CAO would allow because of the need to test, plan, obtain permits, design, obtain operator certification, and construct a central water supply, treatment and distribution system.

15. In summary, I have concluded that it is not feasible to install and operate a replacement water system for the Hinkley area to treat to the 0.02 ppb Cr6 level, and in the time frame required by the Draft CAO:

(a) Bottled water would be the best option in the short term, but even then the logistical, analytical and treatment requirements – including the inevitable negotiations and certifications with bottled water vendors and the process of demonstrating the capability to consistently achieve the 0.02 ppb goal – would take considerably longer than the deadlines established in the Draft CAO.

(b) The bulk delivery option would require at least six months to analyze the treatment technologies proposed by the vendors, implement those technologies and verify the quality of the water delivered.

16. I estimate that it would take approximately two and a half years before a central treatment and distribution system could be fully functional. The requisite pilot testing to demonstrate the feasibility of achieving 0.02 ppb Cr6 concentrations would consume six to

twelve months depending on the scalability of the facilities. An environmental impact report would likely be required for a centralized system, and that process alone could take a year, assuming no litigation-related delays. The design and construction of a small-scale system would take another six months. A system large enough to comply with the Draft CAO would likely take a year to design and construct.

(a) I reviewed the June 24, 2011 letter from David Loveday and Pauli Undesser of the Water Quality Association (the "WQA") to Harold Singer commenting on the Draft CAO. According to the letter, the WQA promotes sales of water treatment devices. I have several comments about the WQA letter: According to the letter, the technologies "readily available" to address Cr6 reduction include "reverse osmosis (using TFC or CTA membranes), distillation, strong base anion resin, and weak base anion resin." But the assertion that these technologies are "readily available" is contradicted by the next sentence of the WQA letter, which states: "However, California requires testing of such technologies to validate performance according to national standards and at this time, none of the best available technologies in a whole house format are [*sic.*] is tested and certified." Thus, none of the technologies can be considered "readily available."

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct, and that this Declaration was executed on July 8, 2011, at State College, PA.

James DeWolfe 7/8/11

EXHIBIT A

Declaration by:

James R. DeWolfe, PE, BCEE, CWO
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Water Planning Group
Malcolm Pirnie, the Water Division of ARCADIS
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State College, PA 16801
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Education:

United States Navy Nuclear Propulsion Program, Submarine Service, 1976-1982
BS, The Pennsylvania State University – Environmental Engineering, 1987
MS, The Pennsylvania State University – Environmental Engineering, 1990

Professional Affiliations:

American Water Works Association (AWWA)

- Member, AWWA Water Resources and Source Water Protection Technical Advisory Workgroup of the Water Utility Council (WUC)
    - **Mission:** To monitor and interact with USEPA, USDA and other federal agency activities on regulations that affect source water protection to protect drinking water supplies; compile, develop and analyze date related to source water protection; and develop draft official comments and testimony on source water protection regulatory activities and proposals.
- Member, Coagulation and Filtration Committee, Water Quality and Technology Division of the Technical and Educational Council (TEC)
    - **Mission:** To advance and disseminate knowledge which promotes the effective and economical application of coagulation and filtration in water treatment.
- Member, B100 Standard Committee for Granular Filter Media of the Standards Council
    - **Mission:** To develop and maintain standards and related manuals, reports, etc., for filtering materials for water treatment. Specific media covered include: silica sand, support gravel, anthracite coal, high density media, and granular activated carbon.
- Past Chair, Pennsylvania Section AWWA Research Committee
- Past Trustee, Pennsylvania Section AWWA North Central District

Professional Summary

- Senior member of Water Planning Division staff, providing services internationally to municipal and private sector clients.
- 22 years of experience in drinking water, wastewater, and industrial water planning, design, research and operations, gained through work in engineering consulting and private industry.
- Senior technical advisor to PG&E on ex-situ chromium remediation project in Topock, CA utilizing the reduction, clarification and filtration (RCF) process
- Leader of Water Planning Group's Operations and Process Specialist (OPS) team

DECLARATION OF JOSHUA HAMILTON

DECLARATION OF JOSHUA W. HAMILTON

I, Joshua W. Hamilton, declare:

1. I serve as the Chief Academic and Scientific Officer at the Marine Biological Laboratory ("MBL") in Woods Hole, Massachusetts and as Senior Scientist at the MBL's Bay Paul Center for Comparative Molecular Biology & Evolution, and also hold an appointment as a professor in the Department of Pathology and Laboratory Medicine at Brown University. Prior to joining the MBL in 2008, I held concurrent appointments in the Department of Pharmacology & Toxicology at the Dartmouth Medical School and Dartmouth's Department of Chemistry, as well as serving as an Associate Director and Senior Researcher at Dartmouth's Norris Cotton Cancer Center.

2. In 2000, I founded Dartmouth's Center for Environmental Health Sciences, a multi-disciplinary research, education and outreach program bringing together over thirty members of the faculty and their laboratories from fourteen Dartmouth departments to focus on the human health effects of environmental chemicals. I served as the Center's Director until 2008. I was also the former Director and Principal Investigator of the largest of the Center's research programs, the Superfund Research Program Project on Toxic Metals, sponsored by the National Institute of Environmental Health Sciences of the National Institutes of Health and by the U.S. Environmental Protection Agency to investigate the human health effects of chemicals in the environment. I am still affiliated with the program where I direct one of its five research projects. It is considered one of the scientific world's pre-eminent research programs on toxic metals. The principal focus is on the effects of chromium, arsenic and other metals on human health, which has been the primary focus of my own laboratory's research for the past two-plus decades. I have been continuously funded by NIH and other federal and non-federal agencies for the past twenty-six years, and have published numerous articles on these topics.

3. I am considered one of the leading experts on the toxicology of chromium. As such, I recently served as an External Reviewer for U.S. EPA's draft update of its Toxicological Profile for Hexavalent Chromium [1]. I have served on numerous other state and national

scientific committees as a toxicology expert, and regularly consult with local, state and federal agencies on issues related to toxic metals exposure and health effects. Attached to this Declaration as Exhibit A is a copy of my curriculum vitae.

4. I was asked by PG&E to consult on toxicology issues related to the chromium plume at Hinkley, California. I have reviewed the draft Cleanup and Abatement Order No. R6V-2011-0005A1 (the "Draft CAO") under consideration by the Lahontan Board [2].

5. The Draft CAO demonstrates a significant misunderstanding of the draft California EPA Office of Environmental Health Hazard Assessment (OEHHA) Public Health Goal ("PHG") [3] and the PHG process. Two passages in the Draft CAO are indicative of the Lahontan Board staff's misunderstanding of what is known as a Reference Exposure Level ("REL"), a PHG, and other public health and regulatory guidelines, how they relate to background levels of Cr(VI), and how they should be interpreted and applied. The first refers to OEHHA's establishment of a chronic inhalation REL: "[The REL][1] is important because it *demonstrates established science* that inhaled hexavalent chromium has *adverse impacts on human health at extremely low levels.*" ([2] § 15, p. 4; emphasis added) The second passage reads: "Based on the draft 2010 PHG, the Water Board has determined that hexavalent chromium in domestic wells above 0.02 µg/L *poses an immediate health risk to Hinkley residents* through continued household use of *contaminated water*, including drinking, preparing foods and beverages, bathing or showering, flushing toilets, and other household uses resulting in potential dermal and inhalation exposures." ([2] § 26, p. 7; emphasis added) These statements by the Lahontan Board suggest a fundamental misunderstanding about the difference between conservative public policy practices such as the setting of RELs and PHGs and the scientific information on which they are based.

6. The scientific community's foundational information about the relationship of Cr(VI) to potential adverse human health effects comes from two principal sources that bear little

[1] The Draft CAO also confuses Cr(VI) with chromic acid. The OEHHA REL for soluble Cr(VI) compounds is 0.2 $\mu g/m^3$ and is based on an animal exposure study in which rats were exposed to Cr(VI) for eighteen hours per day at concentrations ≥ 50 $\mu g/m^3$. The REL for chromic acid is 0.002 $\mu g/m^3$, and is based on human exposures to chromic acid in a chromium plating plant. The form of Cr(VI) in Hinkley is *not* chromic acid and, therefore, the chromic acid REL is irrelevant. In this regard, see http://oehha.ca.gov/air/chronic_rels/pdf/hexChroms.pdf.

to no resemblance to Cr(VI) concentrations to which Hinkley residents have been and are being exposed:

(a) Epidemiology studies of workers in occupational settings who were exposed to high concentrations of airborne Cr(VI) in chemical and physical forms that are *not* representative of exposures to Cr(VI) in Hinkley groundwater; and

(b) Studies of laboratory animals exposed to extremely high levels of Cr(VI) – in most cases at or near the maximum tolerated dose, and at thousands to tens of thousands of times higher levels than Hinkley well concentrations – over the practical lifetime of the animals.

7. The current California and Federal Maximum Contaminant Levels ("MCLs") for total chromium, which can include up to 100% Cr(VI), are 50 ppb and 100 ppb, respectively. The background concentrations in Hinkley are between 1 and 3 ppb, and the draft California PHG [3] seemingly embraced by the Draft CAO as a regulatory guideline is 0.02 ppb. Despite over eighty years of intense study reported in tens of thousands of scientific papers, the only demonstrated adverse health effects of chromium occurred at levels of exposure that are more than a thousand times higher than those that would be encountered in environmental and household settings, including those in Hinkley. Conversely, there are no studies showiug any adverse effects of Cr(VI) at levels anywhere near the current MCLs, let alone the background concentrations at Hinkley or the level proposed for the draft PHG.

8. The statements in the Draft CAO also indicate a fundamental misunderstanding about risk assessment methodology. For regulatory and public health purposes, risk assessors start with the scientific data from the high-dose studies, and then apply conservative assumptions using mathematical modeling to predict health risks at exposures that are tens of thousands to millions of times lower. For example, the lowest Cr(VI) concentration that caused tumors in animals in the National Toxicology Program study [4] which was the foundation for the draft PHG, was 20,000 µg/L. Notwithstanding, OEHHA proposed a PHG of 0.02 µg/L, *one million times lower* than the concentration that caused cancer in mice from a lifetime of drinking water exposure. The calculations embodied in the draft PHG do not represent "established science." And even if the *draft* PHG is adopted, regulators should not assume that exposures of the type

and duration that would be experienced by Hinkley residents will result in any adverse health impacts. In fact, there is no way to confirm any of the risk assessors' assumptions in constructing the models that ostensibly support the draft PHG, or to determine whether there are any measurable health effects as a result of exposures at 0.02 µg/L. They reflect a highly conservative, overly-protective regulatory limit that assumes a lifetime of exposure, but they do not represent levels that suggest a significant or immediate health threat.

9. EPA and OEHHA both understand and clearly articulate the limitations of PHGs and their equivalents. For example, in commenting on its Toxicological Profiles, including the profile for Cr(VI), EPA notes: "It should be emphasized that [the regulatory risk assessment methodology] leads to a plausible upper limit to the risk....Such an estimate, however, does not necessarily give a realistic prediction of the risk. *The true value of the risk is unknown, and may be as low as zero.*" ([1] emphasis added) EPA also noted in its 1996 Carcinogen Risk Assessment Guidelines: "Use of health protective risk assessment procedures as described in these cancer guidelines means that estimates, while uncertain, are more likely to overstate than understate hazard and/or risk." [5] Similarly, OEHHA is explicit that the draft Cr(VI) PHG is not and should not be used as a regulatory or cleanup standard: "PHGs are not regulatory requirements, but instead represent non-mandatory goals....PHGs are not developed as target levels for cleanup of ground or ambient surface water contamination, and may not be applicable for such purposes, given the regulatory mandates of other environmental programs." ([3] p. iii.) In sum, the draft Cr(VI) PHG, as its name implies, is at most a goal, not a regulatory level, and in no way should exposures to concentrations above 0.02 µg/L be interpreted as an immediate health risk to Hinkley residents nor should this proposed goal be used to set action or cleanup levels.

10. The Lahontan Board has also previously contended that the draft Cr(VI) PHG represents the best and most recent science. An objective assessment indicates otherwise:

(a) The initial draft Cr(VI) PHG drew on two principal studies: The 1968 Borneff, *et al.*, animal study [6], and the 1987 Zhang and Li epidemiology study. [7] Both are outdated and flawed, and they have been rejected by EPA and mainstream toxicology experts as

a foundation for Cr(VI) toxicology risk assessment. The Borneff study in particular is so profoundly flawed that it is unlikely it would be published if submitted today for peer review. One expert for the plaintiffs in a personal injury lawsuit alleging health effects from Cr(VI) exposure was quoted as saying it would be "totally stupid and scary" to base the OEHHA risk assessment on the Borneff study.[2] Likewise, the Zhang study is little more than a report, and lacks the necessary data to permit epidemiologists to evaluate Cr(VI) hazards and calculate risks. As a result, the Zhang study is not an appropriate foundation for assessing potential risk. Based on these and other criticisms [8], California withdrew its initial draft Cr(VI) PHG, and generated a revised draft PHG when the National Toxicology Program's studies of lifetime cancer risk in rodents were published. [4,9,10] Although OEHHA based the revised calculation of the current draft PHG principally on those NTP studies, the Borneff and Zhang studies are still cited as justification for the 0.02 μg/L.

(b) EPA is currently updating its Toxicological Profile for Cr(VI), which will form the basis for a possible federal MCL for Cr(VI) and/or total chromium in drinking water. The revised draft Profile [1] has been released for public comment, and an expert panel recently reviewed it in a public session.[3] I served on that panel, which presented and discussed its review of the draft Profile and listened to public comments from stakeholders. EPA's draft Profile appropriately omits any reference to the Bornoff study in its review of key animal studies. While the draft Profile discusses the Zhang study and three follow-up analyses, it correctly states that it should not be used for risk assessment purposes.[4] The panel agreed with these assessments. Thus, there is already significant disagreement between the draft PHG and EPA's draft Cr(VI) Toxicology Profile.

(c) During the Public Comment period the US EPA panel was given an overview of nearly-completed ninety-day toxicity studies that will soon be published in the peer-

[2] Max Costa, Los Angeles Times, Nov. 11, 2000, "Mice and Scientific Unknowns At Heart of Chromium Debate."
[3] U.S. Environmental Protection Agency, Notice of Peer Review Workshop, May 12, 2011. Federal Register, Volume 76, No. 70 (April 12, 2011), Pg. 20349-20350. See also U.S. EPA web site: http://cfpub.epa.gov/ncea/iris_drafts/recordisplay.cfm?deid=221433.
[4] U.S. EPA, referring to the Zhang study: "The epidemiology data are not sufficient to establish a causal association between exposure to hexavalent chromium by ingestion and cancer." ([1] p. 201, Lines 20-23).

reviewed literature (see for example [11,12] as emerging publications from these studies). Based on the results presented to date, these studies will unequivocally support a threshold mechanism as the Mode of Action ("MOA") for Cr(VI) in vivo via ingestion and inhalation exposure. In fact, these studies were specifically designed to investigate the MOA and to complement the 2008 NTP studies in all respects, including study design. The pending studies are even being conducted by the same scientists that conducted the 2008 NTP studies. The panel's consensus was that the pending studies provided important new information that was critical to an overall understanding of Cr(VI), and should be incorporated into the EPA's Profile. Thus, the panel urged EPA to wait for these studies to be published so that they may be taken into account in their assessment. The panel also called for other substantive changes to the draft Profile based on its view that EPA's Cr(VI) risk assessment model was flawed and should be revised based on a likely threshold MOA.

11. Once EPA's Cr(VI) Toxicological Profile is finalized, EPA will undertake to promulgate a federal MCL for Cr(VI). It would be prudent for OEHHA to wait to finalize the PHG for Cr(VI) until such time as the federal MCL for Cr(VI) is finalized. Again, it is worth noting that the current MCL for chromium (total chromium, up to 100% Cr(VI)) is 100 ppb, which was actually raised from 50 ppb several years ago in recognition that the scientific literature indicated a threshold mechanism for toxic and carcinogenic effects. Some have urged OEHHA to quickly finalize the draft PHG. However, as the US EPA Administrator stated at a public meeting in May 2011 in response to comments urging EPA to move quickly in finalizing the Toxicological Profile for Cr(VI): "We want it to be based on the best science....we want to get it right." [Personal Communication]

12. The Draft CAO expresses concern about potential exposure to Cr(VI) from evaporative coolers and other household appliances. OEHHA concluded in its draft Cr(VI) PHG that the principal exposure pathway of concern for chromium in drinking water is ingestion [2]. OEHHA also studied exposure to chromium via showering, which is generally assumed to be the principal inhalation pathway of concern for households with contaminants in drinking water supplies. However, OEHHA did not include dermal contact, having determined that such

exposures were insignificant. In addition, OEHHA concluded that exposure by inhalation during showering did not contribute significantly to the overall risk. And even with conservative assumptions regarding exposure during showering, the contribution to risk from inhalation was 180 times lower than that from drinking water exposure.[5]

13. I have further investigated exposure via inhalation from the use of swamp coolers and have concluded that exposure to airborne Cr(VI) from swamp coolers is not a pathway of concern for households in Hinkley or elsewhere:

(a) The scientific and regulatory literature confirms that inorganic constituents, including chromium, that may be present in the water used in swamp coolers are not volatile and do not evaporate with the water. Instead, the inorganic constituents remain behind on the filter or, for those units with recirculation versus a drip line and drain, in the sump. Moreover, a 1996 scientific publication by Finley et al. [13] examined Cr(VI)-contaminated water in an evaporative cooler, in a trial experiment in a Hinkley-area house with a typical evaporative cooler. They demonstrated that even using a concentration of Cr(VI) of 20,000 ppb in a unit running for twenty-four hours, there was no increase in the airborne Cr(VI) concentration above the natural outside and indoor backgrounds. Thus, there is no basis for any concerns regarding inhalation exposure risk from evaporative coolers, particularly at the concentrations in any impacted Hinkley households, which are more than 4,000 times lower than the levels examined in these experiments.

(b) To further evaluate the potential, if any, for exposure to Cr(VI) from the nse of swamp coolers, I did a comprehensive search for studies in peer-reviewed scientific literature. Only two relevant studies were located, Finley et al. 1996, and Paschold et al. 2003. [13,14] The Paschold findings supported the Finley results discussed above. Paschold studied airborne particulate matter, PM10 and PM2.5, and cooling water in ten residences in El Paso, Texas. [14] The homes were monitored for concurrent indoor and outdoor PM2.5 and PM10 with the use of swamp coolers. More than thirty elements in the PM fractions – including lead,

[5] The PHG associated with inhalation exposure may be readily calculated from the information in the draft PHG assessment by removing the contribution from oral exposures. The PHG associated with inhalation exposure is 3.6 µg/L.

manganese, copper, barium and chromium – were evaluated. Comparisons of the elemental concentrations of the evaporative cooler supply water and indoor PM demonstrated little or no correlation in all ten houses, including those with disabled bleed-lines.[6] From this, Paschold concluded that evaporative coolers were not introducing dissolved solids from the supply water into indoor air.

(c) To summarize, swamp coolers work by evaporating water into warmer air drawn in from the outdoors. The evaporation process cools the air, which is then blown into the house. Minerals that are non-volatile, including Cr(VI), are not transferred from the feed water into the cooled air, but remain in the system or are eliminated through the bleed-line. For these reasons, swamp coolers are not expected to be a source of Cr(VI) or other non-volatile constituents in indoor air, and the published studies of swamp coolers support this conclusion.

14. Like swamp coolers, other similar appliances (such as humidifiers and hot water vaporizers) that act by volatilizing heated water or by evaporating water from a filter will not be a potential source of Cr(VI) into indoor air because Cr(VI) will not be volatilized with the water.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct, and that this Declaration was executed on July 9, 2011, at Falmouth, Massachusetts.

Joshua W. Hamilton Ph.D.

[6] A bleed-line is a drainage tube with an external discharge inserted into the pad water supply hose for continuous removal of particle-laden cooler pan water.

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EXHIBIT A

Curriculum Vitae

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PERSONAL:

Born: July 31, 1956, Salem MA
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EDUCATION:

Cornell University, Ithaca, NY 14853. 1982 to 1985. Ph.D., Genetic Toxicology, 1985. Thesis: *Correlation Between Mixed-Function Oxidase Enzyme Induction and the Genotoxicity of Chemical Mutagen-Carcinogens in the Chick Embryo In Vivo.* (Stephen Bloom, Christopher Wilkinson, advisors)

Cornell University, Ithaca, NY 14853. 1980 to 1982. M.S., Genetics, 1982. Thesis: *Development of Basal and Induced Aryl Hydrocarbon (Benzo[a]pyrene) Hydroxylase Activity in the Chick Embryo In Ovo.* (Stephen Bloom, Christopher Wilkinson, advisors)

Bridgewater College, Bridgewater, MA 02324. 1976 to 1980. B.S., Biology, 1980 (*cum laude*).

POSTDOCTORAL TRAINING:

Postdoctoral Research Fellow (NIEHS, Norris Cotton Cancer Center and Department of Chemistry), Department of Chemistry (Karen E. Wetterhahn, advisor), Dartmouth College, 1985 to 1988.

ACADEMIC APPOINTMENTS:

Professor (MBL), Pathology and Laboratory Medicine, Brown University, 2010 to present.
Senior Scientist, Bay Paul Center, Marine Biological Laboratory (MBL), 2008 to present.
Professor (with tenure) of Pharmacology & Toxicology, Department of Pharmacology & Toxicology, Dartmouth Medical School, 2003 to 2008.

Adjunct Professor of Chemistry, Department of Chemistry, Dartmouth College, 2003 to 2008.
Adjunct Senior Scientist, Center for Integrated and Applied Toxicology, Bioscience Research Institute, University of Southern Maine, 2003 to present.
Associate Professor of Pharmacology & Toxicology, Department of Pharmacology & Toxicology, Dartmouth Medical School, 1994 to 2003.
Adjunct Associate Professor of Chemistry, Department of Chemistry, Dartmouth College, 1994 to 2003.
Adjunct Assistant Professor of Biology, Department of Biology, Dartmouth College, 1992 to 1993.
Assistant Professor of Pharmacology & Toxicology, Department of Pharmacology & Toxicology, Dartmouth Medical School, 1990 to 1994.
Adjunct Assistant Professor of Chemistry, Department of Chemistry, Dartmouth College, 1990 to 1994.
Member, Norris Cotton Cancer Center, Dartmouth-Hitchcock Medical Center, 1988 to present.
Research Assistant Professor of Chemistry, Department of Chemistry, Dartmouth College, 1988 to 1990.

OTHER PROFESSIONAL POSITIONS:

Acting Director, Cellular Dynamics Program, Marine Biological Laboratory, 2010-present.
Chief Academic and Scientific Officer, Marine Biological Laboratory, 2008 to present.
Associate Director, Norris Cotton Cancer Center at Dartmouth, 2006 to 2008.
Visiting Scientist, Harvard School of Public Health, September 2005 to June 2006.
Associate Director, Dartmouth College Center of Biomedical Research Excellence (COBRE) Program Project on Lung Biology, 2003 to 2008.
Director, Center for Environmental Health Sciences at Dartmouth, Dartmouth College / Dartmouth Medical School, 2000 to 2008.
Director / Principal Investigator, Dartmouth College Superfund Basic Research Program Project on Toxic Metals, Dartmouth College / Dartmouth Medical School, 1997 to 2008.
Director, Molecular Biology & Proteomics Core Facility (macromolecular synthesis and sequencing), Dartmouth College, 1995 to 2008.
Co-Director, Dartmouth College Superfund Basic Research Program Project on Toxic Metals, Dartmouth College / Dartmouth Medical School, 1995 to 1997.

AWARDS AND HONORS:

Teaching Assistantship, Department of Poultry and Avian Sciences, Cornell University, 1980.
Graduate Research Assistantship, National Institutes of Health (CA28953, Stephen E. Bloom, advisor), 1981.
Outstanding Teaching Assistant Award, Cornell University, 1983.
Jacob H. Bruckner Memorial Award for Excellence in Graduate Study, Cornell University, 1983.
Graduate Research Fellowship, National Institutes of Health (Environmental Toxicology Training Grant 08 T2 ES07052, Institute of Comparative and Environmental Toxicology, Cornell University), 1984.
Individual National Research Service Award (Postdoctoral Fellowship), National Institutes of Health (F32 ES05399, Molecular Biology, Karen E. Wetterhahn, advisor), 1987.

Junior Faculty Research Award, American Cancer Society (JFRA-323), 1991-1993.
Bohan Visiting Lecturer, University of Kansas Medical Center, May 1998.
Master of Arts (Honorary), Dartmouth College, May 2004.

PROFESSIONAL SERVICE, MAJOR COMMITTEE ASSIGNMENTS AND CONSULTATIONS:

Program Reviews:

- Member, External Advisory Committee, Massachusetts Institute of Technology Center for Environmental Health Sciences (NIEHS Center Grant) (1997 to 2003).
- External Advisor, Plymouth State University, Plymouth NH, Planning Group for creation of a new Center for the Environment at PSU, October 25-26, 2003.
- Member, External Advisory Committee, Dartmouth Medical School NIH-NCRR COBRE Lung Pathobiology Program, 2008 - present.
- External Advisor, Brown University NIH-NIEHS Superfund Research Program, 2008-present.
- Chair, External Advisory Committee, Brown University NIH-NIEHS Children's Environmental Health Sciences Center, 2010 - present.
- Member, External Advisory Committee, Rhode Island NSF EPSCoR Program, 2010 - present.

Scientific Report Reviews:

- External Reviewer, National Research Council Report, *Arsenic in Drinking Water, 2001 Update*, National Academy of Sciences, National Academy Press, 2001.
- Member, U.S. EPA Science Advisory Board (SAB) Review Committee, Framework for Metals Risk Assessment, 2004 - 2008.
- Member, U.S. EPA Science Advisory Board (SAB) External Review Committee, PAH Mixtures Risk Assessment, 2010 – present.
- Member, U.S. EPA Science Advisory Board (SAB) External Review Committee, Toxicological Profile for Hexavalent Chromium (September 2010 Draft), 2011 – present.

Grant Reviews:

- Ad Hoc Reviewer, Chemical Pathology A (CPA) Study Section, National Institutes of Health, June 1989, June 1993, June 1996.
- Ad Hoc Reviewer, Experimental Therapeutics A (ET1) Study Section, National Institutes of Health, June 1996.
- Chair, Special Emphasis Panel, Experimental Therapeutics A (ET1) Study Section, National Institutes of Health, December 1996.
- Ad Hoc Reviewer, Metabolic Pathology (MEP) Study Section, National Institutes of Health, December 1997.
- Ad Hoc Reviewer, Alcohol and Toxicology I (ATI) Study Section, National Institutes of Health, December 1998, February 1999.
- Ad Hoc Reviewer, W.M. Keck Foundation Faculty Fellowship Program, February 1999.
- Ad Hoc Reviewer, Center for Research on Environmental Disease Grant Program, M.D. Anderson / University of Texas, April 1999.
- Ad Hoc Reviewer, NSF SBIR / STTR Grant Program, April 2003.
- Ad Hoc Reviewer, NSF Civilian Research & Development Foundation (CRDF) Grant Program, May 2003.

Ad Hoc Reviewer, Kentucky Science & Engineering Foundation Grant Program, November 2001; September 2005.

Member, Special Review Committee, Environmental Sciences / Developmental Toxicology Grant Program, National Institutes of Health, December 2001.

Member, Review Panel, Beckman Foundation Scholars Program, 2001 - present.

Chair, Special Review Committee, NIH-NIEHS / Superfund Basic Research Program Small Business Innovative Research (SBIR) Grants, National Institutes of Health, March 2002.

Ad Hoc Reviewer, University of Arizona Center for Toxicology Pilot Projects Program, June 2002.

Ad Hoc Reviewer, United Kingdom National Environmental Research Council Environmental Genomics Research Grants Programme, June 2002.

Member, External Advisory Committee, Dartmouth NIH-NCRR COBRE Immunology Program Project (W. Green P.I.), 2003 - present.

Ad Hoc Reviewer, University of Wisconsin – Milwaukee WATER Institute Pilot Grant Program, 2004-2005.

Ad Hoc Reviewer, North Carolina Biotechnology Center, Science & Technology Development Program, January 2004.

Ad Hoc Reviewer, Woods Hole Oceanographic Institute Sea Grant Program, June 2005.

Ad Hoc Reviewer, University of Wisconsin – Milwaukee Research Growth Initiative, April 2006.

Ad Hoc Reviewer, NIH-NIEHS Special Emphasis Grant Review Panel, Environmental Influences on Epigenetic Regulation, April – May 2006.

Member, Review Committee, NIH-NIEHS P50 DISCOVER (Disease Investigation through Specialized Clinically-Oriented Ventures in Environmental Research) Program Project Grant Review (RFA-ES-06-001), National Institutes of Health, March 2007.

Member, Special Emphasis Panel Review Committee, NIH-NIEHS ONES (Outstanding New Environmental Scientist) Grant Review (ZES1 JAB-C-R2), National Institutes of Health, March 2008.

Member, Systemic Injury by Environmental Exposure (SIEE) Special Emphasis Panel (ZRG1 DKUS-C 90S), National Institutes of Health, 2008 – 2010.

Manuscript Reviews:

Ad Hoc (1988 to present): *Archives of Biochemistry and Biophysics, Aquatic Toxicology, Biochemica Biophysica Acta, Biochemical Journal, Biochemical Pharmacology, Cancer Research, Carcinogenesis, Cell Growth & Differentiation, Chemical Research in Toxicology, Chemico-Biological Interactions; Comparative Biochemistry & Physiology, Environmental & Molecular Mutagenesis, Environmental Health Perspectives, Hepatology, Journal of Biological Chemistry, Journal of Inorganic Biochemistry, Journal of Pharmacology & Experimental Therapeutics, Journal of Toxicology & Environmental Health, Molecular Carcinogenesis, Molecular Pharmacology, Pharmacology and Experimental Therapeutics, Toxicological Sciences, Toxicology and Applied Pharmacology, Xenobiotica.*

Editorial Board: *Toxicology and Applied Pharmacology* (1997 to 1998), *Chemico-Biological Interactions* (1998 to 2008).

National Committees:

Member, Directors Association, NIEHS Superfund Basic Research Program, 1997 to 2008; President, 2002 to 2004.

Co-Organizer, Karen E. Wetterhahn Memorial Symposium, American Chemical Society Meeting, Boston MA, August 23-27, 1998.

Organizer and Chair, Society of Toxicology Continuing Education Course, "Methods in Cell Signaling," SOT Meeting, Seattle WA, March 1998.

Member, Society of Toxicology Program Committee, 1998 to 2000.

Organizer and Chair, NIH-NIEHS-sponsored Scientific Conference on "Arsenic in New England," Manchester NH, May 29-31, 2002 (Organized and hosted by the Dartmouth Superfund Basic Research Program).

Member, Expert Panel on Biomonitoring, Research Foundation for Health and Environmental Effects (RFHEE), Herndon VA, November 12-13, 2004.

Member, U.S. EPA Science Advisory Board, Risk Assessment Framework Review Panel, 2004 to 2006.

Member, Human Health Risk Assessment Committee, Chesapeake Bay Research Consortium, Spring 2005.

Member and Presenter, Fundulus Genomics Strategy Workshop, Charleston SC, May 4-5, 2006. Organized by the Hollings Marine Laboratory, College of Charleston, Charleston SC.

Co-Organizer and Host, NIH-NIEHS-sponsored New England Workshop on "Arsenic in Landfills," Bostou MA, Oct. 2-4, 2006 (Second of two workshops co-organized by the Arizona and Dartmouth Superfund Basic Research Programs).

Member, U.S. EPA Science Advisory Board, Polycyclic Aromatic Hydrocarbon (PAH) Mixtures External Review Panel, 2010 to present.

Member, U.S EPA Science Advisory Board, Toxicological Profile for Hexavalent Chromium (September 2010 Draft) External Review Panel, 2011 to present.

Regional Committees:

Organizer, Ninth Annual New England Membrane Enzyme Group (Nutmeg) Conference, Center Harbor NH, November 10-12, 1991.

Organizer, Tenth Annual New England Membrane Enzyme Group (Nutmeg) Conference, Center Harbor NH, November 8-10, 1992.

Member, New Hampshire Healthy NH 2010 Committee, NH Department of Health and Human Services, Concord NH, May - September 2000.

Member, Montshire Museum of Science Corporation, 2000 to present.

Member, New Hampshire Arsenic Consortium (Dartmouth Toxic Metals Program, NH Dept. Health & Human Services, NH Dept. Environmental Services, U.S. Geological Survey, U.S. EPA region I, Agency for Toxic Substances & Disease Registry), 2000 - present.

Member, New Hampshire Public Health Biomonitoring Committee, NH Dept. Health & Human Services, 2002 - 2008.

Member, Montshire Museum of Science Board of Trustees, 2002 to present.

Member, New Hampshire Health Tracking Program Advisory Committee, NH Dept. Health & Human Services, 2004 – 2008.

Co-Organizer, Fourteenth Annual MDIBL / NIEHS Center Environmental Health Sciences Symposium, "Human Health and the Environment: Arsenic and Mercury, A Public Health Crisis?" Mt. Desert Island Biological Laboratory, Salsbury Cove ME, July 18-19, 2007.

Member, Independent Technical Review Team, Sediment in Baltimore Harbor: Quality and Suitability for Innovative Reuse, sponsored by Maryland Sea Grant and Maryland Department of Environmental Service, 2008-2009.

Co-Organizer, Twenty-first Annual Nutmeg Conference, Woods Hole MA, October 4-6, 2009.

Co-Organizer, Twenty-second Annual Nutmeg Conference, Woods Hole MA, October 7-9, 2010.

Co-Organizer, 2011 Northeast Regional SRP Meeting, Woods Hole MA, April 24, 2011

University / Program Committees:

Dartmouth College Radiation Safety Sub-Committee (of Biosafety), 1989 to 1991.
Dartmouth College Biosafety Committee, 1989 to 1992.
Co-organizer, Dartmouth College Structural Biology Seminar Series, 1990 to 2005.
Hughes Undergraduate Research Initiative Grant Review Committee, 1990 to 2005.
Dartmouth College Radiation Safety Committee, 1991 to 1996; Chair, 1991 to 1996.
Mary Hitchcock Memorial Hospital Radiation Safety Committee (*ex officio*), 1991 to 1996.
Dartmouth College Environmental Health and Safety Policy Advisory Committee, 1992 to 1996; Chair, 1994 to 1995.
Dartmouth College Search Committee, Environmental Health and Safety Specialist, Spring-Summer 1992.
Dartmouth College Women in Science Program (WISP) Advisory Committee, 1992 to 2008.
Dartmouth College Task Force on the Library of the 21st Century, 1993 to 1998.
Dartmouth College Task Force on Information Technology, 1995 to 1998.
Dartmouth College Computer Technology Venture Capital Fund Advisory Committee, 1995 to 2008.
Dartmouth College Search Committee, Director of Environmental Health and Safety, Spring-Summer 1995.
Dartmouth College / Norris Cotton Cancer Center Molecular Biology Core Facility Advisory Committee, Chair, 1995 to 2008.
Dartmouth College / Norris Cotton Cancer Center's Center for Biological and Biomedical Computing Core Facility Advisory Committee, 1995 to 2008.
Norris Cotton Cancer Center Scientific Advisory Committee, 1995 to 2001.
Dartmouth Superfund Basic Research Program Project Executive Committee, 1995 to 2008 (Chair, 1997 to 2008).
Dartmouth College Search Committee, University Radiation Safety Officer, Spring-Fall 1996.
Dartmouth College Women in Science Program (WISP) Task Force, 1996 to 1997.
Norris Cotton Cancer Center, Committee to Review Clinical Protocol Office, 1996 to 1997.
Dartmouth Medical School Search Committee, Facilities Director, Fall 1996.
Dartmouth Cystic Fibrosis Program Project Executive Committee, 1996 to 2008.
Norris Cotton Cancer Center, American Cancer Society Scientific Advisory Committee, 1997 to 2008.
Dartmouth College Re-Accreditation Internal Evaluation Committee, Undergraduate Research Opportunities Sub-Committee, 1998 to 1999.
Center for Environmental Health Sciences Executive Committee (Chair), 2000 to 2008.
Dartmouth-Hitchcock Medical Center / Norris Cotton Cancer Center Committee for Expansion of Rubin Cancer Center Building, 2001 to 2005.
Dartmouth Medical School Research Resources Advisory Committee, 2001.
Dartmouth COBRE Lung Pathobiology Research Program Executive Committee, 2003 to 2008.
Dartmouth College Women in Science Program (WISP) External Review Committee, May 2003.
Dartmouth Medical School / Norris Cotton Cancer Center Faculty Search Committee (Asst. / Assoc. Prof. - Proteomics position), 2004 to 2006.
Dartmouth College Women in Science Program (WISP) Faculty Advisory Committee, 2005 to 2008.
Dartmouth Medical School / Dartmouth-Hitchcock Medical Center Planning Committees for Koop Medical Research and Education Complex, 2006 to 2008; Chair, Core Committee.
Norris Cotton Cancer Center at Dartmouth Executive Committee, 2006 to 2008.
Norris Cotton Cancer Center at Dartmouth Cancer Research Committee, 2006 to 2008.

Dartmouth Medical School Graduate Program in Experimental and Molecular Medicine (PEMM) Program Committee, 2006 to 2008.
Dartmouth Medical School Appointments, Promotions and Titles Committee, 2007 to 2008.
Brown University Pathobiology Graduate Program Admissions Committee, 2008-2009.

Departmental Committees:
Dartmouth Medical School, Pharmacology & Toxicology Faculty Search Committee (Assistant Professor), Fall 1990 to Winter 1991.
Dartmouth College, Chemistry Faculty Search Committee (Assistant Professor - Structural Biology), Fall 1990 to Winter 1991.
Dartmouth Medical School, Pharmacology & Toxicology United Way Campaign Coordinator, 1991 to 2005.
Dartmouth Medical School, Pharmacology & Toxicology Graduate Pharmacology Course Committee, 1993 to 1995.
Dartmouth Medical School, Pharmacology & Toxicology Graduate Program Committee, 1994 to 2001.
Dartmouth Medical School, Microbiology Faculty Search Committee (Assistant / Associate Professor – Immunology), Winter / Spring 2003.

MEMBERSHIPS IN PROFESSIONAL SOCIETIES:

American Association for the Advancement of Science (AAAS), 1981 to present.
Environmental Mutagen Society (EMS), 1981 to 2008.
American Association for Cancer Research (AACR), 1988 to 2008.
Society of Toxicology (SOT), 1990 to present.
American Chemical Society (ACS), 1998 to 2008.
Society of Enviromental Toxicology and Chemistry (SETAC), 2008 to present.

TEACHING EXPERIENCE / RESPONSIBILITIES:

Courses:
Biology Tutor (undergraduate), Bridgewater State College, 1978 to 1980.
Lecturer, *Animal Cytogenetics* (undergraduate/graduate), Cornell University, 1981 to 1985.
Laboratory Instructor, *Animal Cytogenetics* (undergraduate/graduate), Cornell University, 1981 to 1984.
Lecturer, Pharmacology 123, *Topics in Toxicology: Mechanisms of Chemical Carcinogenesis* (graduate), Dartmouth Medical School, Winter 1989.
Co-organizer and Lecturer, Biochemistry 134 (co-listed as Chemistry 134), *Biochemistry of Nucleic Acids* (graduate), Dartmouth Medical School, Fall 1990, Winter 1993. Course revised 1995: Organizer and Lecturer, Pharmacology 134 (co-listed as Chemistry 134 and Biochemistry 134), *Nucleic Acids: Chemistry, Biochemistry and Pharmacology* (graduate), Dartmouth Medical School, Winter 1995, Winter 1997.
Lecturer, Pharmacology 122, *Topics in Pharmacology: Cancer Biology* (graduate), Dartmouth Medical School, Winter 1991.

Coordinator, *Pharmacology & Toxicology Workshop* (graduate), Dartmouth Medical School, Fall 1991, Fall 1996.

Organizer and Lecturer, Medical Pharmacology PharmFlex Unit, *Introductory Toxicology* (medical/gradnate), Dartmouth Medical School, Fall 1991, 1992, 1993.

Lecturer, Pharmacology 123, *Principles of Toxicology* (graduate), Dartmouth Medical School, Fall 1992.

Organizer and Principal Lecturer, Pharmacology 123 (revised), *Graduate Toxicology* (graduate and undergraduate), Dartmouth Medical School, Fall 1995, Spring 1998, Spring 2001, Spring 2003, Spring 2005, Winter 2008.

Co-organizer and Lecturer, Biology 77/78, *Introductory Biochemistry* (undergraduate), Dartmouth College, Fall 1992/Winter 1993.

Lecturer, *Environmental Pathology* (graduate), University of Vermont, Spring 1994.

Lecturer, Pharmacology 215, *Medical Pharmacology* (medical), Dartmouth Medical School, Fall 1994, 1995, 1996, 1997, 1998, 1999, 2000, 2001, 2002, 2003, 2004, 2005.

Lecturer, Pharmacology 129, *Principles of Receptor Action* (graduate and undergraduate), Dartmouth Medical School, Spring 1994, 1996; Fall 1997; Winter 2000, Spring 2002, Winter 2004.

Lecturer, Pharmacology 130, *Graduate Pharmacology* (graduate and undergraduate), Dartmouth Medical School, Spring 1995, 1997, 2008.

Faculty Facilitator, *Nature Medicine Course* (first year medical), Dartmouth Medical School, Spring 1997.

Lecturer, Pharmacology 133, *Heavy Metals II: Chemistry, Biochemistry and Pharmacology* (graduate and undergraduate), Dartmouth Medical School, Winter 1998.

Lecturer, Hematology & Oncology Fellows Continuing Education Lecture Series, Summer 1996, 1997, 1998, 1999, 2000.

Lecturer, Chemistry 67, *Biophysical Chemistry* (undergraduate and graduate), Dartmouth College, Winter 1999.

Lecturer, Chemistry 63, *Environmental Chemistry* (undergraduate), Dartmouth College, Summer 2000, 2001, 2002, 2003, 2004, 2005.

Lecturer, Immunology 142, *Advanced Immunology* (graduate), Dartmouth Medical School, Fall 2001.

Lecturer, Pharmacology 122, *Modern Approaches in Experimental Therapeutics* (graduate), Dartmouth Medical School, Winter 2003.

Lecturer, Evaluative and Clinical Sciences 151, *Environmental and Occupational Health* (graduate), Dartmouth Medical School, Winter 2003, 2004, 2005, 2008.

Undergraduate Research Advising:

Sally Lim (Dartmouth '94) 1/91 - 4/91. WISP fellow.

Nicole Baptiste (Dartmouth '92, Biochemistry) 3/91 - 9/92. Hughes fellow, Honors thesis.

Steven Hunt (Dartmouth '92, Biology) 6/91 - 6/92. Waterhouse fellow, Honors thesis.

Kristen Doherty (Regis College, '93, Chemistry) 6/91 - 9/91. Dartmouth REU fellow.

Michael Reed (Dartmouth '92, Biology) 9/91 - 6/92. Honors thesis.

Nandini Joseph (Dartmouth '93, Biochemistry) 1/92 - 9/92. Hughes fellow.

Rukmini Sichitiu (Dartmouth '95) 1/92 - 2/94. WISP fellow.

Kamala Dansinghani (Dartmouth '94, Biology) 8/92 to 8/93. Hughes, Waterhouse, Presidential Scholars fellow.

Patsa Hungspreugs (Dartmouth '96) 12/92 to 6/93. WISP fellow.

Vijay Shankaran (Dartmouth '94, Chemistry) 12/92 to 6/94. Waterhouse fellow, Honors thesis.

Carrie Pesce (Dartmouth '97, Biology) 1/94 to 6/97. WISP, Presidential Scholars, Hughes, Waterhouse fellow.
Nicole LaRonde (Rivier College, '95, Chemistry) Dartmouth REU fellow, 6/94 - 9/94.
Anne Stone (Dartmouth '96, Psychology) 9/94 to 12/94.
Bruce Turpie (Dartmouth '96, Biology) 9/94 to 6/96.
Johanna Blaxall (Dartmouth '98) 1/95 to 6/95. WISP fellow.
Erin Rowell (Dartmouth '96, Art History/Chemistry) 3/95 to 6/96. Waterhouse fellow, Honors thesis.
Sara Ogdon (Dartmouth '96, Chemistry) 6/95 to 6/96. Waterhouse fellow, Honors thesis.
Elaine Gilmore (Providence College '96, Chemistry / Biology) 6/95 to 8/95, Dartmouth REU fellow.
Karana Pierre (Xavier College '96, Biology) 6/95 to 8/95, Leadership Alliance fellow.
Susan Darling (Amherst College, '97, Biology) 6/96 to 8/96, Dartmouth REU fellow.
Nadiue Burnett (Dartmouth '98, Biology), 9/96 to 6/97, E.E. Just Fellow.
Jannet Oh (Dartmouth '98, Biology), 9/96 to 6/98.
Joie Jager-Hyman (Dartmouth '00, Biology), 12/96 to 6/97. WISP Fellow.
Amy Feldmann (Dartmouth '98, Chemistry), 9/97 to 6/98.
Kaili Temple (Dartmouth '01, Biology), 12/97 to 6/01. WISP Fellow, Presidential Scholar
Stacey Davis (Dartmouth '99, Chemistry), 1/98 to 6/99.
Alisa Davis (Dartmouth '01, Chemistry), 6/98 to 6/01. Goldwater Fellow, Hughes Fellow, Waterhouse Fellow, Beckman Scholar, Presidential Scholar.
Daniel Paik (Dartmouth '00, Biology), 9/98 to 6/00. Hughes Fellow.
Emily Feingold (Dartmouth '02, Biology), 12/98 to 6/99. WISP Fellow, Presidential Scholar.
Rahshaana Green (Dartmouth '00, Biology), 3/99 to 6/00. E.E. Just Fellow, NIEHS Minority Fellow.
Lauren Kingsley (Dartmouth '04, Chemistry), 11/00 to 6/04. WISP Fellow, B.E. Krute Memorial Fellow, Presidential Scholar, Beckman Scholar, Richter Scholar, Honors thesis.
Caryn Barnet (Dartmouth '03, Chemistry), 12/01 to 6/03.
Rebecca Wang (Dartmouth '05), 12/01 to 6/02. WISP Fellow.
Katherine Harrison (Dartmouth '06), 12/02 to 9/04. WISP Fellow.
Caitlin Stanton (Brown U. '06), 6/03 to 8/06. MDIBL Fellow.
Manida Wungjiranirun (Dartmouth '07), 12/03 to 6/07. WISP Fellow, Presidential Scholar.
Jenna Sherman (Dartmouth '08), 12/04 to 6/07. WISP Fellow.
Angela Wang (Dartmouth '10), 12/06 to 8/07. WISP Fellow.
Anais Carnescu (Dartmouth '11), 12/07 to 6/08. WISP Fellow.
Chelsea Connolly (Valdosta State University '12), 6-8/10. NSF REU Fellow.
Morgan Kelly (Harvard '14), 6-8/11. NSF REU Fellow.

Post-Baccalaureate Training:
Cavus Batki (B.S., U. Bristol, UK '02), 9/02 – 8/03. Council Exchange Internship USA graduate internship.
Liam Ingram (B.S., U.Bristol, UK '03), 10/03 – present. Council Exchange Internship USA graduate internship.

Graduate Research Advising:
Major Advisor:

Jennifer McCaffrey (Dartmouth Medical School, Pharmacology & Toxicology) Ph.D. 1/94. Thesis: *The Effects of Chemical Carcinogens on Hormone-Inducible Gene Expression.* Strohbehn Award 1994.

Rosemary Caron (Dartmouth Medical School, Pharmacology & Toxicology) Ph.D. 10/95. Thesis: *Differential Effects of Mitomycin C on Constitutive and Inducible Gene Expression in the Chicken Embryo Liver In Vivo: Correlation with Developmental Age and Chromatin Structure.* Borison Fellowship 1994. Strohbehn Award 1996.

Amy Warren (Dartmouth College, Chemistry) Ph.D. 6/96. Thesis: *Characterization of the Interaction of the Chemotherapeutic Drug Mitomycin C with DNA In Vitro and In Vivo and Effects on Specific DNA-Protein Interactions.* Wolfenden Teaching Prize 1995. Croasdale Award 1996.

Michael Ihnat (Dartmouth Medical School, Pharmacology & Toxicology) Ph.D. 3/97. Thesis: *Effects of Mitomycin C and Other DNA Crosslinking Agents on Gene Expression: Modulation of Cancer Cell Multidrug Resistance in Cell Culture and In Vivo.* Ryan Fellow 1994-1996. AACR Travel Award 1996.

Jeu-Ming Yuann (with Karen Wetterhahn) (Dartmouth College, Chemistry) Ph.D. 6/97. Thesis: *The Roles of Glutathione and Ascorbate in Chromium(VI)-Induced Carcinogenesis In Vivo.*

Ronald Kaltreider (Dartmouth Medical School, Pharmacology & Toxicology) Ph.D. 6/00. Thesis: *Characterization of the Molecular Mechanism by which Arsenic and Chromium alter Inducible Gene Expression.* Ryan Fellow 1998-2000. SOT Travel Award 2000. SOT Metals Specialty Section Award 2000. Strohbehn Award 2000.

David Mustra (Dartmouth Medical School, Pharmacology & Toxicology) Ph.D. 6/01. Thesis: *The Biophysical Characterization of the Interaction of Xeroderma Pigmentosum A Protein with a Mitomycin C-DNA Complex.*

Rangan Maitra (Dartmouth Medical School, Pharmacology & Toxicology) Ph.D. 6/01. Thesis: *Regulation of the Cystic Fibrosis Transmembrane Conductance Regulator by P-Glycoprotein Modulators.*

Athena Nomikos (Dartmouth Medical School, Pharmacology & Toxicology) M.S. 12/07. Thesis: *Physiological consequences of low dose arsenic exposure in culture and in whole mouse liver.* SOT Travel Award 2007.

Courtney Kozul (Dartmouth Medical School, Program in Experimental & Molecular Medicine) Ph.D. 4/10. Thesis: *Immunomodulatory effects of chronic low dose arsenic exposure.* SOT Travel Award 2007, 2009. NIEHS-SBRP Best Student Poster Award 2007, 2008. Nutmeg Wetterhahn Student Poster Award 2007. SOT MBSS Student Research Award 2008, 2009. NIH-NIEHS International Conference Invitation and Travel Award, 2008. NIH-NIEHS Wetterhahn Award, 2010.

Committee Member:

Licheng Xu (Dartmouth Medical School, Pharmacology & Toxicology, E. Bresnick advisor) Ph.D. 6/91.

William Berndt (Dartmouth Medical School, Pharmacology & Toxicology, T. Ciardelli advisor) Ph.D. 6/93.

Injae Chung (Dartmouth Medical School, Pharmacology & Toxicology, E. Bresnick advisor) Ph.D. 6/94.

Bruce Sneddon (Dartmouth Medical School, Pharmacology & Toxicology, P. Friedman advisor) Ph.D. 10/94.

Claudine Louis (Dartmouth Medical School, Pharmacology & Toxicology, J. Sinclair advisor) Ph.D. 2/95.

Melinda Treadwell (Dartmouth Medical School, Pharmacology & Toxicology, A. Barchowsky advisor) Ph.D. 1/96.

Flora Ciampolillo (Dartmouth Medical School, Physiology, B. Stanton advisor) M.S. 6/96.

Pamela Buchli (Dartmouth Medical School, Pharmacology & Toxicology, T. Ciardelli advisor) Ph.D. 12/96.

Salvatore Morana (Dartmouth Medical School, Pharmacology & Toxicology, A. Eastman, advisor) Ph.D. 6/98.

Elizabeth Cox (Dartmouth College, Chemistry, D. Wilcox advisor) Ph.D. 8/98.

Jason Nawrocki (Dartmouth Medical School, Pharmacology & Toxicology, C. Lowrey, advisor) M.S. 11/98.

Jennifer Shumilla (Dartmouth College, Chemistry, A. Barchowsky / K. Wetterhahn, advisors) Ph.D. 4/99.

Stefano Liparoto (Dartmouth Medical School, Pharmacology & Toxicology, T. Ciardelli, advisor) Ph.D. 9/00.

Michael Nemeth (Dartmouth Medical School, Pharmacology & Toxicology, C. Lowrey, advisor) Ph.D. 6/01.

Keith DePetrillo (Dartmouth Medical School, Pharmacology & Toxicology, F. Gesek, advisor) Ph.D. 5/02.

Michael Layon (Dartmouth Medical School, Pharmacology & Toxicology, C. Lowrey, advisor) Ph.D. 6/04.

Kyle MacLea (Dartmouth Medical School, Pharmacology & Toxicology, A. Eastman, advisor) Ph.D. 12/02.

Ethan Kohn (Dartmouth Medical School, Pharmacology & Toxicology, A. Eastman, advisor) Ph.D. 9/03.

Scott Gleim (Dartmouth Medical School, Pharmacology & Toxicology, advisor) Ph.D. 8/09.

External Committee Member:

Edward Cable (Biochemistry, University of Massachusetts (Worcester), Herbert Bonkovsky advisor) Ph.D. 6/93.

Joseph Lynch (Toxicology, University of Southern Maine, John Wise advisor) 2/04 to 4/06.

Beth Peterson-Roth (Biochemistry, Brown University, Anatoly Zhitkovich advisor) Ph.D., 4/06.

Post-doctoral Research Training:

Carolyn Bentivegna (Ph.D. 1991, Environmental Toxicology, Rutgers) 6/91 to 8/94. Post-doctoral Fellow.

Stephen Anthony (D.O. 1988, Philadelphia College of Osteopathic Medicine) 10/94 to 6/97. Hematology / Oncology Fellow.

Janet Jeyapaul (Ph.D. 1991, Toxicology, Cancer Research Institute, Bombay India) 8/95 to 10/95. Post-doctoral Fellow.

Olga Bajenova (Ph.D. 1987, Molecular Biology, St. Petersburg Academy of Sciences USSR) 12/95 to 11/97. Post-doctoral Fellow.

Angela Nervi (M.D. 1993, Stanford) 1/97 to 6/99. Hematology / Oncology Fellow. 7/99 to present, Post-doctoral Research Associate.

Veronika Dubrovskya (Ph.D. Chemistry, Institute for Bioorganic Chemistry, Novosibirsk USSR) (with Karen Wetterhahn) 1/97 to 11/97. Post-doctoral Fellow.

Edward Dudek (Ph.D. Toxicology, Illinois Institute of Technology) (with Karen Wetterhahn) 1/97 to 12/97. Post-doctoral Fellow.

Bogdan Gulanowski (Ph.D. Chemistry, Wroclaw Medical University, Wroclaw Poland) (with Karen Wetterhahn) 1/97 to 6/98. Post-doctoral Fellow.

Diane Stearns (Ph.D. Chemistry, UC Berkeley) (with Karen Wetterhahn) 1/97 to 6/97. Research Assistant Professor.

Kent Sugden (Ph.D. Chemistry, Montana State University, Bozeman) (with Karen Wetterhahn) 1/97 to 12/98. Post-doctoral Fellow / Research Assistant Professor.

Amy Warren (Ph.D. 1996, Chemistry, Dartmouth) 8/97 to 3/01. Postdoctoral Fellow.

Joseph Shaw (Ph.D., 2001, Toxicology, Kentucky) 3/01 - present. Postdoctoral Fellow.

Angeline Andrew (Ph.D., 2001, Pharmacology & Toxicology, Dartmouth) 9/01 – 6/04. Postdoctoral Fellow / Research Assistant Professor.

Julie Gosse (Ph.D., Chemistry, Cornell) 3/05 – 12/07. Postdoctoral Fellow. SOT Travel Award 2007. Women in Toxicology Award 2007.

Fokko Zandbergen (Ph.D., Nutrition, Metabolism and Genomics, Wageningen Netherlands) 11/08 – present. Postdoctoral Fellow.

RESEARCH INTERESTS:

Dr. Hamilton's principal research interests are in the areas of molecular toxicology, metals toxicology, developmental toxicology, gene regulation, pathophysiology associated with toxicant exposures, and the use of –omics technologies to understand the environmental etiology of human disease. The primary focus of his research over the past decade has been on the molecular toxicology of arsenic and other toxic metals. The current focus of the laboratory is on three principal research directions related to this interest.

The first area is focused on understanding the molecular and mechanistic basis for the effects of arsenic as an endocrine disruptor, which was first discovered and reported by Dr. Hamilton's lab. They have demonstrated in a series of studies that arsenic is a very potent endocrine disruptor at extremely low concentrations at or below the current U.S. drinking water standard, i.e., 10 ppb. This was first demonstrated with the steroid hormone receptor for glucocorticoids, but has since been shown to also occur with the steroid receptors for estrogen, progesterone, androgen and mineralocorticoids, i.e., all five steroid receptor classes. Similar effects have also been seen with other non-steroid nuclear hormone receptors, i.e., those for thyroid hormone and retinoic acid. Interestingly, the mechanism for this appears to be unique since arsenic does not act as a ligand for these receptors, i.e., it is neither an agonist or competitive antagonist, nor does arsenic appear to interfere with normal hormone binding, activation of the receptor, translocation to nuclear chromatin, or binding to hormone-responsive DNA elements that regulate hormone-responsive genes. However, in the presence of arsenic these hormone-activated, chromatin-bound receptors function abnormally as transcription factors, with either greatly enhanced gene signaling at very low doses or greatly suppressed signaling at slightly higher doses. The shared effects of arsenic on all these different receptors that represent two entirely different classes of nuclear hormone receptors, despite their lack of absolute shared sequence or structure, suggests that there is a common regulatory component or other shared machinery which is the actual molecular target(s) for arsenic. Current research in this area is focused on precisely how arsenic is able to elicit these effects on receptor-mediated gene expression at the cell and molecular level.

The broad effects of arsenic on this suite of important hormone pathways also suggests an important role of arsenic-mediated endocrine disruption on arsenic's ability to increase the risk of various cancers, type 2 diabetes, reproductive and developmental effects, vascular and cardiovascular disease, neurological and cognitive disorders, and the growing list of other known pathophysiological consequences on humans and on natural populations that are exposed chronically to arsenic environmentally in food or water. Thus, a second major focus of the lab is to investigate these pathophysiological consequences of such endocrine disruption using model whole animal systems, and also in collaboration with epidemiologists and ecologists studying human or natural populations, respectively. Recent work from the lab has shown that arsenic can profoundly disrupt certain developmental or physiological programs that are critically dependent on hormone receptors that have been shown to be disrupted by low dose arsenic. For example, arsenic at very low doses, equivalent to human drinking water levels of concern, blocks thyroid hormone-dependent tadpole metamorphosis in the frog, *Xenopus*. Likewise, arsenic at similar levels disrupts the ability of the euryhaline fish, *Fundulus*, to adapt to changes in water salinity equivalent to the changing salt marsh tides, a process which is regulated hy the glucocorticoid hormone, cortisol, and its control of a key salt regulatory protein, CFTR (the same protein which, when mutated, causes the human disease, cystic fibrosis). Current research is extending these studies to other systems to determine what other

effects, at what levels, and the extent to which such endocrine disruption can explain the myriad adverse effects of arsenic observed in exposed populations.

The third area focuses on using genomics and proteomics tools to investigate more broadly the effects of arsenic, chromium and other toxicants on gene and protein expression in model systems in order to understand their overall biological effects. These experiments are useful both to test hypotheses and to generate new avenues of research based on biological discovery. Previous work in the lab has shown, using whole genome microarrays, that arsenic broadly affects hormone regulation of gene expression at low doses. For example, the lab demonstrated that the synthetic glucocorticoid hormone, dexamethasone, significantly alters expression of over a thousand genes in mouse liver, and that low doses of arsenic affect the hormone regulation of virtually all of these genes. Conversely, in the lungs of the mice in these same experiments, it was observed that the dominant effect of arsenic at low doses is to profoundly alter immune response, and this is now a new avenue of research in the lab based on this discovery. The lab has also pioneered the use of microarrays in environmentally relevant species, particularly the aquatic freshwater zooplankton, *Daphnia*, and the marine fish, *Fundulus*. These two species are ideal because they can be used both in controlled laboratory experiments and also in the environment as sentinel species for natural populations. The lab is continuing to develop and apply genomics tools in these species in collaboration with other laboratories in order to establish them as model organisms for use in their own studies but also broadly shared within a larger research community. Related to this genomics research, the lab has been pioneering the development and application of new analytical tools and methods for obtaining richer and more accurate biological information from the large data sets that are generated in a typical whole genome microarray, which allows comparisons among different treatments and different experimental species.

RESEARCH FUNDING:

As Principal Investigator:

Previous:

6/87 - 11/88. NIH Individual NRSA Postdoctoral Research Fellowship F32 ES05399 (Molecular Biology, Karen E. Wetterhahn, advisor).

10/87 - 9/88. American Cancer Society Institutional Research (Seed) Grant IN-157D, total direct costs $10,000.

12/88 - 6/94. NIH FIRST Grant R29 CA49002, "Effect of carcinogens on gene expression *in vivo*," total direct costs $348,062.

1/91 - 12/93. American Cancer Society Junior Faculty Research Award (JFRA) JFRA-323, "Effect of carcinogens on gene expression *in vivo*," total direct costs $90,500.

7/91 - 6/94. International Life Sciences Institute Research Foundation Research Award, "Targeting of DNA damage *in vivo*," total direct costs $100,000.

11/92 - 6/94. Hitchcock Foundation, "Antibodies to MMC-DNA adducts," total direct costs $6,500.

7/94 - 3/99. NIH Research Grant R01 CA49002, "Effect of carcinogens on gene expression," total direct costs $658,404.

1/95 - 6/96. Norris Cotton Cancer Center Interactive Program Project, "Suppression of p-glycoprotein expression by mitomycin C," total direct costs $25,000.

4/96 - 3/00. NIH / NIEHS Program Project P42 ES07373, Project Director of "Toxic Metals in the Northeast: from Biological to Environmental Implications," total direct costs $4,410,619. As Principal Investigator: Project 2, "Molecular basis for effects of carcinogenic metals on inducible gene expression," total direct costs $479,808. Core 1, "Adminstrative Core," total direct costs, $264,600. Core 2, "Molecular Biology Core Facility," total direct costs $408,058. Core 4, "Education and Training Core,"total direct costs $513,665.

12/96 - 5/97. Bristol-Myers Squibb, "Modulation of multidrug resistance by mitomycin C," total direct costs $50,000.

1/97 - 12/98. Cystic Fibrosis Foundation Pilot Project, "Modulation of CFTR expression by mitomycin C," total direct costs $69,100.

1/97 - 12/98. Immunex, "A pilot clinical trial of mitomycin C modulation of multidrug resistance proteins," total direct costs $20,000.

3/97 - 7/99. NIH Research Grant R01 CA45735, "Chromium effect on gene expression," total direct costs $684,170 (Dr. Hamilton assumed responsibility for this grant for the late Dr. Karen Wetterhahn and is managing it for her laboratory through its completion date).

3/97 - 6/99. NIH Research Grant R01 ES07167, "Mechanism of chromium carcinogenicity," total direct costs $1,212,100 (Dr. Hamilton assumed responsibility for this grant for the late Dr. Karen Wetterhahn and is managing it for her laboratory through its completion date).

6/98 - 5/01. Bristol-Myers Squibb, "Modulation of multidrug resistance by DNA crosslinking agents," total direct costs $320,000.

4/00 - 3/05. NIH / NIEHS Program Project P42 ES07373, Program Director of "Toxic Metals in the Northeast: from Biological to Environmental Implications," total direct costs (5 years) $10,457,254. As Principal Investigator: Project 2, "Effects of carcinogenic metals on gene expression," total direct costs $975,301; "Administrative Core," total direct costs, $917,864; "Molecular Biology Core Facility," total direct costs $841,837; "Education and Training Core,"total direct costs $562,002.

6/01 - 5/02. NIH National Council for Research Resources (NCRR) Grant S10 RR14644, "Purchase of LCQ Mass Spectrometer System," total direct costs $220,950.

9/01 - 8/02. NSF Major Research Instrumentation (MRI) Grant 0116413, "Acquisition of a MALDI-TOF Mass Spectrometer," total direct costs $217,176.

4/01 - 3/03. Cystic Fibrosis Foundation Grant HAMILT01GO, "Anthracyclines for treatment of CF," total direct costs $129,600.

4/02 - 4/03. NIH-NCI Contract 263-MQ-209007, "NCI Contract to measure arsenic in water samples," total direct costs $7,620.

5/02 - 12/03. BioReliance Contract BCR-1108-28, "Selenium determination in association with selective tumors," total direct costs $28,050.

4/05 - 3/08. NIH-NIEHS SBRP Program Project P42 ES07373, Program Director of "Toxic Metals in the Northeast: from Biological to Environmental Implications," total direct costs (3 years) $5,765,083. As Principal Investigator: Project 2, "Arsenic as an endocrine disruptor," total direct costs $656,186; "Administrative Core," total direct costs, $299,016; "Molecular Biology & Proteomics Core Facility," total direct costs $313,094.

9/02 - 8/08. NSF BE/GEN-EN Research Grant DEB-0221837, "Development of methods linking genomic and ecological responses in a freshwater sentinel species," total direct costs $2,000,000.

4/06 – 12/08. Cystic Fibrosis Foundation Pilot & Feasibility Grant HAMILT0610, "Anthraquinones for treatment of CF," total direct costs $86,400.

Current:

4/08 - 3/13. NIH-NIEHS Program Project P42 ES07373, "Toxic Metals in the Northeast: from Biological to Environmental Implications" (PI Bruce A. Stanton), total direct costs (5 years) $9,551,339. As Principal Investigator: Project 2, "Arsenic as an endocrine disruptor," total direct costs $1,165,149.

9/09 – 8/11. NIH-NCRR Program Project Supplement to P41 RR001395-27S1, "Biocurrents Research Center: Physiological Factors Affecting Ovarian Cancer," total direct costs $895,215.

Pending:

None.

As Co-investigator:

Previous:

7/87 - 6/90. NIH Research Grant R01 CA45735, "Effect of chromium on gene expression *in vivo*," (P.I. Karen E. Wetterhahn), total direct costs $411,687.

6/89 - 5/94. NIH Research Grant R01 CA34869, "Mechanism of chromium carcinogenicity," (P.I. Karen E. Wetterhahn), total direct costs $909,186.

9/91 - 7/94. NIH Research Grant R01 CA45735, "Effect of chromium on gene expression *in vivo*," (P.I. Karen E. Wetterhahn), total direct costs $324,818.

3/97 - 7/99. NIH Research Grant R01 CA45735, "Chromium effect on gene expression," (P.I. Karen E. Wetterhahn), total direct costs $684,170.

3/97 - 6/99. NIH Research Grant R01 ES07167, "Mechanism of chromium carcinogenicity," (P.I. Karen E. Wetterhahn), total direct costs $1,212,100.

7/03 – 6/06. NIH Research Grant R01 R01 CA098889, "DNA repair gene polymorphisms and pancreatic cancer," (P.I. Eric J. Duell), total direct costs $600,000.

9/02 - 6/08. NIH Research Grant R01 R01 ES11819, "Arsenic effects on glucocorticoid receptor action," (P.I. Jack E. Bodwell), total direct costs $900,000.

7/03 – 6/08. NIH-NCRR COBRE Program Project Grant P20 RR018787, "Cellular and Molecular Mechanisms of Lung Disease," (P.I. Bruce A. Stanton), total direct costs $8,000,000. Co-Director of program project, Director of Proteomics Core, Senior Mentor on Project 4, "Respiratory effects of air pollution in New Hampshire" (P.I. Melinda Treadwell), Advisor on Project 5, "Environmental epidemiology of lung cancer in New Hampshire: a multilevel approach using GIS and case-control methods."

4/05 – 3/10. NIH Research Grant R01 ES013168, "Arsenic, Histone Modifications, and Transcription" (P.I. Lynn Sheldon), total direct costs $1,125,000.

Current:
None.

Pending:
None.

CLINICAL RESEARCH TRIALS (TRANSLATIONAL)

Active / Completed Clinical Protocols:

DMS 9503: A pilot clinical trial of mitomycin C modulation of P-glycoprotein and a Phase I evaluation of mitomycin C and paclitaxel in patients with advanced carcinoma and lymphoma. P.A. Kaufman (PI), J.W. Hamilton, S.P. Anthony, A.M. Nervi, M.S. Ernstoff, L.D. Lewis, R.J. Barth, and V.A. Memoli.

DMS 9614: A pilot clinical trial of mitomycin C modulation of multidrug resistance proteins and a Phase I evaluation of mitomycin C and mitoxantrone in patients with acute myelogenous leukemia. C.H. Lowrey (PI), J.W. Hamilton, S.P. Anthony, A.M. Nervi, M.S. Emstoff, L.D. Lewis, and N.B. Levy.

DMS 9704: A study of carboplatin as a modulator of the multidrug resistance phenotype followed by concurrent chemo/radiotherapy utilizing paclitaxel in head and neck cancer. T.H. Davis (PI), J.W. Hamilton, S.P. Anthony, A.M. Nervi, M.S. Ernstoff, L.D. Lewis, J.J.B. Gosselin, R.J. Amdur, and A. Siegel.

DMS 9715: A Phase I study of carboplatin and paclitaxel used post bone marrow transplantation for women with Stage IV breast cancer. L.E. Mills (PI), J.W. Hamilton, S.P. Anthony, A.M. Nervi, M.S. Ernstoff, L.D. Lewis, R.J. Barth and V.A. Memoli.

DMS 9816: A pilot clinical trial of carboplatin modulation of P-glycoprotein and a Phase I evaluation of carboplatin and paclitaxel in patients with advanced carcinoma and lymphoma. M.S. Ernstoff (PI), J.W. Hamilton, A.M. Nervi, S.P. Anthony, L.D. Lewis, R.J. Barth, and V.A. Memoli.

PATENTS

Pending:

Three patents have been filed based on discovery of novel application of chemotherapy drugs for treatment of deltaF508 CFTR CF patients.

One patent has been filed based on discovery of a novel application of chemotherapy drugs for treatment of multidrug resistant human solid and hematological malignancies.

Intl. Appl. No. PCT/US00/27443. J.W. Hamilton and B.A. Stanton. Compositions and methods for modulating ATP-binding cassette transmembrane reporter protein expression. Priority Date Oct. 6, 1999; Intl. Filing Date Oct. 4, 2000; Intl. Publ. Date Apr. 12, 2001.

INVITED PRESENTATIONS

Scientific Presentations (selected 2000 - present):

University of California at Davis, Environmental Toxicology Seminar Series, Davis CA, January 31, 2000, "Arsenic as an essential element, cancer chemotherapy drug and human carcinogen."

Society of Toxicology 39th Annual Meeting, Philadelphia PA, March 21, 2000, Poster Discussion Session (Organizer and Chair): Mechanisms of Arsenic Carcinogenesis.

Dartmouth Community Medical School 2000: Environmental Toxins: Are Our Public Policies Rational?, Dartmouth College, April 17-18, 2000, "An introduction to toxicology: environmental carcinogens as a paradigm."

NIOSH Molecular Mechanisms of Metal Toxicity Meeting, National Institute of Occupational Safety and Health, Morgantown WV, September 12, 2000, " Mechanistic basis for arsenic and chromium carcinogenicity: insights from gene expression studies."

Dartmouth Community Medical School 2000: Environmental Toxins: Are Our Public Policies Rational?, Manchester NH, October 26, 2000, "An introduction to toxicology: environmental carcinogens as a paradigm."

NIEHS Conference, Superfund Basic Research Program: Oxidative Processes: Stress to Remediation, Chapel Hill NC, December 13, 2000, "The New Hampshire Arsenic Coalition: A partnership of university, state and federal agencies."

Dartmouth Community Medical School 2001: Heal Thyself?, Dartmouth College, April 10, 2001, "Foreign Invasion: How Our Bodies Deal With Vitamins, Drugs, Toxins And Dietary Supplements."

Dartmouth Community Medical School 2001: Heal Thyself?, Manchester NH, October 3, 2001, "Foreign Invasion: How Our Bodies Deal With Vitamins, Drugs, Toxins And Dietary Supplements."

North American Cystic Fibrosis Conference 15th Annual Meeting, Orlando FL, October 26, 2001, CFTR New Therapeutic Strategies session, "The model anthracycline, doxorubicin, increases functional cell surface expression of □F508-CFTR protein by altering its structure and biogenesis."

Northeast Society of Toxicology 2001 Annual Meeting, Cambridge MA, November 16, 2001, "Toxic metal-induced alterations in patterns of gene expression."

NIEHS Conference, Superfund Basic Research Program: Assessing Risks of Hormonally Active Agents, Gainesville FL, December 11, 2001, "Arsenic as an endocrine disruptor."

University of Arizona, Southwest Environmental Health Science Center, Tucson AZ, May 16, 2002, "Arsenic as an endocrine disruptor."

University of Oklahoma Health Sciences Center, Oklahoma Center for Toxicology Interdisciplinary Seminar Program, Oklahoma City OK, May 17, 2002, "Arsenic as an endocrine disruptor: possible role in carcinogenesis, vascular disease and diabetes."

Tufts University Medical School, Pharmacology and Toxicology Seminar Series, Boston MA, June 12, 2002, "Arsenic is an endocrine disruptor: role in carcinogenesis, vascular disease and diabetes."

NIEHS / Center for Environmental Health Sciences at Dartmouth Scientific Conference: Arsenic in New England: A Multidisciplinary Scientific Conference, Manchester NH, May 30, 2002, "Arsenic as an endocrine disruptor: role in cancer, vascular disease, and diabetes."

First Annual Daphnia Genome Consortium Meeting, Indiana University, Bloomington IN, October 3, 2002, "Differential display and microarray: linking genomic responses to metal toxicity."

New England Society of Toxicology Annual Meeting, Phfizer Inc., Groton CN, November 8, 2002, K-12 Educational Program on Introduction to Toxicology, "Arsenic: Poison of Kings and king of poisons."

NIH-NIEHS Division of Extramural Research and Training (DERT) Leadership Annual Retreat, Wilmington NC, November 21-22, 2002, "Molecular mechanisms of arsenic toxicity."

Society of Toxicology 42nd Annual Meeting, Salt Lake City UT, March 10, 2003, Symposium on Health Risk Assessment of Hexavalent Chromium in Drinking Water: Carcinogenicity, Research and Regulation, "Mechanism of Hexavalent Chromium [Cr(VI)] Toxicity and Carcinogenicity."

Boston University, Boston MA, Biomolecular Seminar Series, March 31, 2003, "Arsenic as an Endocrine Disruptor: Role in Cancer, Diabetes and Vascular Disease."

Second Annual Daphnia Genome Consortium Meeting, University of New Hampshire / Dartmouth College, at Center of New Hampshire, Manchester NH, September 9-11, 2003, "Development of methods linking genomic and ecological responses in a freshwater sentinel species."

University of Southern Maine, Bioscience Research Institute, Applied Medical Sciences Seminar Series, Portland ME, January 22, 2004, "Arsenic as an endocrine disruptor."

University of Vermont Medical School, Pathobiology Seminar Series, Burlington VT, March 15, 2004, "Arsenic is a potent endocrine disruptor at very low levels: implications for cancer, diabetes and other arsenic associated diseases."

York College of Pennsylvania, Biology Department, Richard Clark Lecture Series, York PA, March 22, 2004, "Arsenic: It's not just for breakfast anymore."

Stony Brook University, Marine Sciences Research Program Seminar Series, Stony Brook NY, May 7, 2004, "Arsenic and old mines – or – don't take it for granite."

3rd International Conference on Non-Linear Dose-Response Relationships in Biology, Toxicology and Medicine, U. Massachusetts – Amherst, Amherst MA, June 9, 2004, "Arsenic as an endocrine disruptor: Complex dose dependent effects of arsenic on steroid receptor signaling."

New England England Water Environment Association (NEWEA) Arsenic Symposium, University of New Hampshire, Durham NH, October 14, 2004, "Arsenic: Human health effects."

U.S. EPA Research Seminar Series, Region I U.S. EPA, "Arsenic: Health Effects and Public Policy," Boston MA, December 15, 2004, "Arsenic and health effects: mechanisms of action."

Upper Valley Chapter, New Hampshire League of Women Voters, Hanover NH, February 15, 2005, "Environmental Chemicals and Human Health Risks."

Dartmouth-Montshire Institute, Hanover NH, NYC high school student summer workshop, July 6, 2005, "An introduction to toxicology and environmental health."

8th Annual John B. Little Symposium, J.B. Little Center for Radiation Sciences and Environmental Health, Harvard School of Public Health, Boston MA, October 28, 2005, "Use of genomics to examine low level effects of environmental agents."

SETAC North America 26th Annual Meeting, Baltimore MD, November 15, 2005, Symposium on Omics Technologies – Current and Future Applications to Ecotoxicology, "Differences in microarray gene expression profiles of *Daphnia pulex* exposed to metals."

Third International Daphnia Genome Consortium Meeting, Indiana University, Bloomington IN, January 17, 2006, Keynote Address, "Daphnia as a model for toxicogenomics."

2006 Toxicology and Risk Assessment Conference, Cincinnati OH, April 26, 2006, Symposium on Heavy Metals of Emerging Toxicological Concern, "Toxicogenomics as a tool for identifying biomarkers and assessing mechanisms of action of toxic metals."

Fundulus Genomics Strategy Workshop II, Hollings Marine Laboratory, Charleston SC, May 5, 2006, "Killifish as a toxicogenomics model to investigate effects of arsenic as an endocrine disruptor."

New England Society of Environmental Toxicology and Chemistry (SETAC) Annual Meeting, Portland ME, June 9, 2006, "Toxicogenomics as a tool for identifying biomarkers and assessing mechanisms of action of toxic metals in the environment."

Mt. Desert Island Biological Laboratory, Mt. Desert Island ME, August 27, 2006, "Use of toxicogenomics to investigate the mechanism of action of arsenic as an endocrine disruptor."

Columbia University, New York City NY, September 18, 2006, "Toxicogenomics of arsenic."

CIESM - the Mediterranean Science Commission, Research Workshop No. 31, "Marine Sciences and Public Health – Some Major Issues," Geneva Switzerland, September 27-30, 2006, "Use of toxicogenomics to investigate the effects of toxicants in aquatic systems."

NIH-NIEHS SBRP / U.S. EPA / ATSDR Workshop on Arsenic, "Arsenic and Landfills: Protecting Water Quality," Boston MA, October 3-4, 2006, "Recent Advances in understanding health effects of arsenic: molecular and cellular mechanisms."

Third Annual Great Issues in Medicine and Global Health Symposium on Cancer, "Cancer, Nutrition and the Environment," Dartmouth-Hitchcock Medical Center, Hanover NH, November 16, 2006, "Environmental toxins: how much cause for concern?"

Dartmouth Medical School, Pharmacology and Toxicology Seminar Series, June 6, 2007, "Use of genomics to understand the biology of low dose arsenic."

Mt. Desert Island Biological Laboratory / NIEHS Center 14th Annual Environmental Health Sciences Symposium, "Human Health and the Environment," Salsbury Cove ME, July 19, 2007, "Arsenic and endocrine disruption."

U.S. Environmental Protection Agency, Research Triangle Park NC, January 17, 2008, "The biology and toxicology of low dose arsenic."

Duke University, NIEHS Environmental Health Sciences Center Interdisciplinary Seminar Series, Durham NC, January 18, 2008, "The biology and toxicology of low dose arsenic."

University of Vermont, Lung Pathology Program, May 5, 2008, "The biology and toxicology of low dose arsenic: effects on lung biology and pathophysiology."

Brown University, Pathobiology Graduate Program Retreat, August 26, 2008, "A biologically based approach to genomics analysis: insights from studies of low dose arsenic."

Marine Biological Laboratory, Bay Paul Center, September 19, 2008, "Use of genomics tools to understand the biology and toxicology of low dose arsenic."

Nutmeg Conference, Woods Hole MA, October 7, 2008, "Arsenic as an endocrine disruptor."

Tufts University, Biology Department (student invited speaker), October 10, 2008, "Arsenic: King of poisons, poison of kings."

Superfund Basic Research Program Annual Meeting, Asilomar CA, December 9, 2008, "Arsenic as an endocrine disruptor."

Workshop on Mercury Exposure and Public Health, New York NY, May 20, 2009, "Current issues in mercury exposure, effects and risk analysis."

Third Congress of the International Society of Nutritigenetics and Nutrigenomics, NIH, Bethesda MD, October 22, 2009, "Laboratory diet profoundly alters gene expression and confounds genomic analysis."

Bridgewater State College, Bridgewater MA, Department of Biology FISH Seminar Series, February 26, 2010, "Arsenic: it's not just for breakfast anymore."

National Institute of Environmental Health Sciences, Research Triangle Park NC, Toxicology and Pharmacology Seminar Series, April 8, 2010, "The biology and toxicology of low dose arsenic."

NIH-NIEHS Workshop, Phenotypic Anchoring of Arsenic Dose-Response in Experimental Models of Human Disease, October 21, 2010, "Phenotypic anchoring of low-dose arsenic effects in the C57BL6 mouse."

Bridgewater State College, Bridgewater MA, Department of Biology FISH Seminar Series, April 8, 2011, "MBL Stew: Arsenic, glowing frogs, limping lampreys and other fun projects."

Woods Hole Oceanographic Institution, Woods Hole MA, Department of Biology, April 28, 2011, "Arsenic: number one environmental health threat."

Harvard School of Public Health, Boston MA, Superfund Research Program Seminar Series, May 5, 2011, "Arsenic as an endocrine disruptor and immune modulator."

Community Service / Public Communication:

WNTK radio station (Lebanon NH), March 4, 1992, "Viewpoint" call-in/discussion show: "Chemicals and Health - Part I."

WNTK radio station (Lebanon NH), April 22, 1992, "Viewpoint" call-in/discussion show: "Chemicals and Health - Part II."

Norris Cotton Cancer Center, Fourth Annual Symposium on Breast Cancer, October 6, 1997, "Lab to bedside: drug resistance."

Dartmouth Community Medical School, Spring / Fall 2000 Curriculum (April 17-18, October 26, 2000 lectures), "Environmental Toxins: Are Our Public Policies Rational?"

Newton Middle School, South Strafford VT, 7th and 8th grade science classes, November 20, 2000, "An Introduction to Toxicology."

"Living on Earth" National Public Radio program interview, "Arsenic as an endocrine disruptor," March, 2001.

Ad Hoc Toxicology Consultant, Elizabeth Mines Community Advisory Group, South Strafford VT, April 2000 to present.

Dartmouth Community Medical School, Spring / Fall 2001 Curriculum (April 10, 2001 and October 3. 2001 lectures), "Foreign Invasion: How Our Bodies Deal With Vitamins, Drugs, Toxins And Dietary Supplements."

New England Society of Toxicology Annual Meeting, Phfizer Inc., Groton CN, November 8, 2002, K-12 Educational Program on Introduction to Toxicology, "Arsenic: Poison of Kings and King of Poisons."

Thetford Academy Middle School, Thetford VT, 7th and 8th grade science classes, February 11, 2003, "An Introduction to Toxicology."

Barre Middle School, Barre VT, 7th and 8th grade science classes, October 30, 2003, "An Introduction to Environmental Toxicology."

Rivendell Middle School, Orford NH, 7th and 8th grade science classes, November 20, 2003, "An Introduction to Environmental Toxicology."

Lebanon High School, Lebanon NH, 11th and 12th grade Advanced Biology class, May 21, 2004, "Introductory Toxicology and the Problem with Arsenic."

New England England Water Environment Association (NEWEA) Arsenic Symposium, University of New Hampshire, Durham NH, October 14, 2004, "Arsenic: Human health effects."

Upper Valley Chapter, New Hampshire League of Women Voters, Hanover NH, February 15, 2005, "Environmental Chemicals and Human Health Risks."

Dartmouth-Montshire Institute, Hanover NH, NYC high school student summer workshop, July 6, 2005, "An introduction to toxicology and environmental health."

Phillips Exeter Academy (grade 9-12 private school), June 1, 2006, lecture in environmental chemistry course on "An introduction to toxicology and environmental health."

Third Annual Great Issues in Medicine and Global Health Symposium on Cancer, "Cancer, Nutrition and the Environment," Dartmouth-Hitchcock Medical Center, Hanover NH, November 16, 2006, "Environmental toxins: how much cause for concern?"

"Greener Living with Dr. G" radio show, WTIC AM 1080, June 6, 2009, "Arsenic effects on immunity and H1N1 flu exposure."

"The Point with Mindy Todd" radio show, WCAI FM 90.1, February 24, 2011, "Environmental chemicals and human health."

"What's Falmouth Reading 2011?" and Falmouth Hospital Cancer Center Winter 2011 joint public seminar series, February 26, 2011, "Environmental chemicals and cancer."

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Research Articles:

1. Hamilton JW, Bloom SE. Developmental differences in basal and induced aryl hydrocarbon (benzo[a]pyrene) hydroxylase activity in chick embryo liver and lung, *in ovo*. *Biochem Pharmacol* 32:2986-2988, 1983.
2. Hamilton JW, Denison MS, Bloom SE. Development of basal and induced aryl hydrocarbon (benzo[a]pyrene) hydroxylase activity in the chicken embryo, *in ovo*. *Proc Natl Acad Sci USA* 80:3372-3376, 1983.
3. Hamilton JW, Bloom SE. Correlation between mixed-function oxidase enzyme induction and aflatoxin B_1-induced unscheduled DNA synthesis in the chick embryo, *in vivo*. *Environ Mutagen* 6:41-48, 1984.
4. Denison MS, Hamilton JW, Wilkinson CF. Comparative studies of aryl hydrocarbon hydroxylase and the *Ah* receptor in nonmammalian species. *Comp Biochem Physiol* 80c:319-324, 1985.
5. Denison MS, Okey AB, Hamilton JW, Bloom SE, Wilkinson CF. *Ah* receptor for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin: Ontogeny in chick embryo liver. *J Biochem Toxicol* 1:39-49, 1986.
6. Hamilton JW, Bloom SE. Correlation between induction of xenobiotic metabolism and DNA damage from chemical carcinogens in the chick embryo *in vivo*. *Carcinogenesis* 7:1101-1106, 1986.
7. Hamilton JW, Wetterhahn KE. Chromium(VI)-induced DNA damage in chick embryo liver and blood cells *in vivo*. *Carcinogenesis* 7:2085-2088, 1986.
8. Faribault G, Weibkin P, Hamilton JW, Longnecker DS, Curphy TJ. γ-Glutamyl transferase activity in atypical acinar cell nodules of rat pancreas. *Toxicol Appl Pharmacol* 88:338-345, 1987.
9. Hamilton JW, Bement WJ, Sinclair PR, Sinclair JF, Wetterhahn KE. Expression of 5-aminolaevulinate synthase and cytochrome P-450 in chicken embryo hepatocytes *in vivo* and in cell culture: Effect of porphyrinogenic drugs and haem. *Biochem J* 255:267-275, 1988.
10. Hamilton JW, Wetterhahn KE. Differential effects of chromium(VI) on constitutive and inducible gene expression *in vivo* and correlation with chromium(VI)-induced DNA damage. *Mol Carcinog* 2:274-286, 1989.
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12. Wetterhahn KE, Hamilton JW. Molecular basis of hexavalent chromium carcinogenicity: Effect on gene expression. *Sci Total Environ* 86:113-129, 1989.
13. Wetterhahn KE, Hamilton JW, Aiyar J, Borges KM, Floyd R. Mechanism of chromium(VI) carcinogenesis: Reactive intermediates and effect on gene expression. *Biol Trace Element Res* 21:405-411, 1989.
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15. Mackie JE, Back DW, Hamilton JW, Marks GS. Elevation of □-aminolevulinic acid synthase and cytochrome PB_1 P-450 messenger RNA levels by dihydropyridines, dyhidroquinolines, sydnoues, and N-ethylprotoporphyrin IX. *Biochem Pharmacol* 42:475-483, 1991.

16. Hamilton JW, Bement WJ, Sinclair PR, Sinclair JF, Alcedo JA, Wetterhahn KE. Inhibition of protein synthesis increases the transcription of the phenobarbital-inducible *CYP2H1* and *CYP2H2* genes in chick embryo hepatocytes. *Arch Biochem Biophys* 298:96-104, 1992.
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20. McCaffrey J, Hamilton JW. Comparison of effects of direct-acting DNA methylating and ethylating agents on inducible gene expression *in vivo*. *Environ Mol Mutagen* 23:164-170, 1994.
21. McCaffrey J, Hamilton JW. Developmental regulation of basal and hormone-inducible phosphoenolpyruvate carboxykinase gene expression in chick embryo liver *in vivo*. *Arch. Biochem Biophys* 309:10-17, 1994.
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DECLARATION OF BRIAN SCHROTH

DECLARATION OF BRIAN SCHROTH

I, Brian Schroth, declare:

1. I am employed by CH2M HILL, Inc., as a Senior Technologist. My resume is attached to this Declaration as Exhibit A. Pacific Gas and Electric Company engaged CH2M HILL to assist it in connection with issues surrounding the chromium plume in Hinkley, California. I was asked to analyze the presence of naturally-occurring hexavalent chromium in California's Mojave Desert.

2. I have been working on these issues since 2007. I am currently registered in California as a Professional Geologist and Certified Hydrogeologist. I attended the University of California at Berkeley, receiving a Ph.D. in soil science with an emphasis in environmental geochemistry. This was preceded by a masters of science degree in hydrology/hydrogeology from the University of Nevada at Reno, and a bachelors of science degree in geology from San Diego State University. I have over nineteen years of experience in consulting and applied academic work focusing on groundwater and geochemistry, including eight years assessing the geochemistry and hydrogeology of sites in the Mojave Desert and the surrounding area.

3. My opinions are that:

(a) Naturally-occurring hexavalent chromium is ubiquitous in groundwater systems throughout the Mojave Desert and globally, with naturally-occurring concentrations sometimes exceeding 50 µg/L in alluvial aquifers in the western Mojave Desert[1] and elsewhere in central and southern Arizona,[2] and western New Mexico.[3] The ability of manganese dioxides,

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[1] Izbicki, James A., Ball, James W., Bullen, Thomas, D., Sutley, Stephen J., 2008, "*Chromium, Chromium Isotopes, And Selected Trace Elements, Western Mojave Desert, USA.*," Applied Geochemistry 23: pages 1325-1352. http://ca.water.usgs.gov/news/Chromium-report.pdf; Izbicki, J.A., 2008, "*Chromium Concentrations, Chromium Isotopes, And Nitrate In The Unsaturated Zone And At The Water-Table Interface, El Mirage, California*," Cooperative Water Resources Study submitted to Lahontan Regional Water Quality Control Board, December, 2008.

[2] Robertson, F.N., 1975, "Hexavalent Chromium In The Ground Water,Iin Paradise Valley, Arizona," Ground Water 13, 516–527.; Robertson, F.N., 1991, "*Geochemistry Of Ground Water In Alluvial Basins Of Arizona And Adjacent Parts Of Nevada, New Mexico, And California*," U.S. Geol. Surv. Prof. Paper 1406-C.

common in desert environments, to oxidize Cr(III) to Cr(VI) is well established.[4] Thus, both the mechanism of natural production of Cr(VI) and the widespread presence of naturally-occurring Cr(VI) in groundwater is well documented.

(b) Concentrations of naturally-occurring Cr(VI) vary significantly geographically, vertically and laterally in aquifer systems due to many factors, including the geochemical conditions present[5] and the composition of earth material sources.[6]

(c) Concentrations of Cr(VI) detected in wells are naturally variable over time at any given well. As a result, increases or decreases in the concentration of Cr(VI) at a given well do not necessarily signify the arrival or departure of a particular source or plume of Cr(VI).

4. My opinions are supported by the following information from published studies by the United States Geological Survey ("USGS"), data from the California Department of Public Health ("CDPH") and California Department of Health Services ("CA DHS"), the Califomia State Water Resources Control Board ("SWRCB"), and consumer confidence reports

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[3] Robertson, F.N., 1991, "*Geochemistry Of Ground Water In Alluvial Basins Of Arizona And Adjacent Parts Of Nevada, New Mexico, And California*," U.S. Geol. Surv. Prof. Paper 1406-C.

[4] Bartlett, R. and James, B., 1979, "*Behavior Of Chromium In Soils: III Oxidation*," J. Environ Qual., 8, 31–35; Eary, L.E., and Rai, D., 1986, "*The Kinetics Of Cr(VI) Reduction To Cr(III) By Ferrous Iron-Containing Solids*," Geol. Soc. Am. Abstr. Programs, 18, 6, 591; Fendorf, S.E., and Zasoski, R.J., 1992, "*Chromium (III) Oxidation By δ-MnO2. 1. Characterization*," Environ. Sci. & Technol., 26, 1, 79–83.

[5] Ball, J.W., and Izbicki, J.A., 2004, "*Occurrence Of Hexavalent Chromium In Ground Water In The Western Mojave Desert, California*," Applied Geochemistry, Vol. 19, pp. 1123-1135; Izbicki, J.A., 2008, "*Chromium Concentrations, Chromium Isotopes, And Nitrate In The Unsaturated Zone And At The Water-Table Interface, El Mirage, California*," Cooperative Water Resources Study submitted to Lahontan Regional Water Quality Control Board, December, 2008; Izbicki, James A., Ball, James W., Bullen, Thomas, D., Sutley, Stephen J., 2008, "*Chromium, Chromium Isotopes, And Selected Trace Elements, Western Mojave Desert, USA.*," Applied Geochemistry 23: pages 1325-1352. http://ca.water.usgs.gov/news/Chromium-report.pdf.

[6] Chromium occurs naturally in the earth's crust, with an average concentration of 100 mg/kg, and has been found in rock-forming minerals of the San Gabriel Mountains at concentrations up over 1,000mg/kg. (Izbicki, *et al.*, 2008.) Detectable concentrations of Cr(VI) occur naturally in alkaline groundwater (pH greater than 7.5) with dissolved oxygen greater than 0.5 milligrams per liter in alluvial aquifers in the western Mojave Desert. (Izbicki, *et al.*, 2008.) Cr(III) oxide is among the ten most abundant elements compounds in the earth's crust. Crustal rock on earth contains an average of 140 parts per million of chromium; seawater contains 0.6 µg/L and stream water contains 1.0 µg/L. (Guertin, *et al.*, 2004.)

for numerous water supply companies in the Mojave Desert. See the Table 1 attached to this Declaration as Exhibit B.

5. Drinking water quality data collected by the CDPH and the USGS and others confirm that Cr(VI) is present in groundwater throughout California, including the Mojave Desert area. Table 1 summarizes numerous published studies and drinking water supply reports for the Mojave Basin evaluating Cr(VI) and/or total chromium conceutrations in groundwater. These studies were reviewed to assess the range and average concentrations of naturally-occurring chromium in groundwater.

6. In typical groundwater systems nearly all of the dissolved chromium present is in the Cr(VI) form, with a much smaller fraction in the trivalent form of chromium.[7] Cr(III) is the most common form of chromium found in rocks and soil and is highly insoluble and, thus, not generally present in the dissolved phase in groundwater. Therefore, although some of the studies reviewed only analyzed for Cr(T), it can be inferred that dissolved Cr(T) in most groundwater systems primarily consists of Cr(VI).

7. Results of the drinking water supply reports and others referred to below are consistent with scientific studies conducted by the USGS that have identified the presence of naturally-occurring Cr(VI).[8] The frequency of reports of naturally-occurring Cr(VI) has risen over recent years. This is primarily the result of the CA DHS mandating the use of lower analytical detection limits.

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[7] Ball, J.W., and Izbicki, J.A., 2004, "*Occurrence Of Hexavalent Chromium In Ground Water In The Western Mojave Desert, California*," Applied Geochemistry, Vol. 19, pp. 1123-1135.

[8] Izbicki, James A., Ball, James W., Bullen, Thomas, D., Sutley, Stephen J., 2008, "*Chromium, Chromium Isotopes, And Selected Trace Elements, Western Mojave Desert, USA.*," Applied Geochemistry 23: pages 1325-1352. http://ca.water.usgs.gov/news/Chromium-report.pdf; Izbicki, J.A., 2008, "*Chromium Concentrations, Chromium Isotopes, And Nitrate In The Unsaturated Zone And At The Water-Table Interface, El Mirage, California*," Cooperative Water Resources Study submitted to Lahontan Regional Water Quality Control Board, December, 2008; Robertson, F.N., 1991, "*Geochemistry Of Ground Water In Alluvial Basins Of Arizona And Adjacent Parts Of Nevada, New Mexico, And California*," U.S. Geol. Surv. Prof. Paper 1406-C; Schmitt, S.J., Milby Dawson, B.J., and Belitz, K., 2008, "*Groundwater-Quality Data In The Antelope Valley Study Unit, 2008: Results From The California GAMA Program*," United States Geological Survey. Data Series 479.

8. Notable findings of the literature review showing site-specific chromium levels throughout California are summarized below (see Exhibit B for additional details and references):

(a) The CDPH produced a plot of Cr(VI) detections in groundwater, attached to this Declaration as Exhibit C, that confirms and illustrates that Cr(VI) is ubiquitous in California groundwater, including the Mojave Desert area. Data compiled by the CDPH shows that Cr(VI) was reported greater than the 1 µg/L detection limit in over half of the groundwater supply wells that were tested (3,156 out of 5,943 between 1997 and 2008).[9] The three counties in California with the greatest number of wells containing Cr(VI) concentrations exceeding 1 µg/L were Fresno, Los Angeles, and San Bernardino.

(b) The printout of data from the SWRCB Geotracker attached to this Declaration as Exhibit D provides a printout of data from the SWRCB Geotracker database that shows many water supply wells in the Mojave Desert area with concentrations of Cr(VI) greater than 1 µg/L.[10]

(c) A study of groundwater conducted by the USGS and SWRCB in the Mojave area in 2008 also confirmed that Cr(VI) is present in groundwater at concentrations up to 16 µg/L.[11] Consistent with the SWRCB data, the USGS reported Cr(VI) concentrations ranging from 1 to 16 µg/L in 15 out of 22 well samples analyzed. Exhibit E to this Declaration shows the distribution of Cr(VI) detected throughout the Mojave Area.

(d) Annual water quality reports for drinking water supply companies were also reviewed. In reports where Cr(VI) was reported, municipal supply wells extracting water

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[9] State Water Resources Control Board Division of Water Quality GAMA Program, 2009, Groundwater Information Sheet Chromium VI. September. http://www.swrcb.ca.gov/water_issues/programs/gama/docs/coc_hexchromcr6.pdf

[10] State Water Resources Control Board Division of Water Quality GAMA Program, 2011, Groundwater Ambient Monitoring & Assessment Program, accessed on July 6, 2001. http://www.swrcb.ca.gov/water_issues/programs/gama/geotracker_gama.shtml.

[11] Mathany, Timothy M., and Belitz, K., 2008, "*Groundwater Quality Data In The Mojave Study Unit, 2008: Results From The California GAMA Program,*" http://pubs.usgs.gov/ds/440/.

from the Alto and Este sub-basins of the Mojave River Basin show the presence of naturally-occurring Cr(VI).

(i) In the Victorville area, thirty-five miles southeast of Hinkley, reports for drinking water supply wells extracted from the Alto and Este sub-basins of the Mojave River Basin indicated detectable Cr(VI) in three areas.[12] The average Cr(VI) concentrations were: 5.1 µg/L (range 5 to 5.1 µg/L) in the Desert View System, 2.5 µg/L (range non-detect ("ND") to 6.3 µg/L) in Apple Valley South, and 2.7 µg/L (range ND to 4.6 µg/L) in Lucerne.

(ii) The Twentynine Palms Water District (located approximately 100 miles southeast of Hinkley) extracts groundwater from four sub-basins. In 2009, an average Cr(VI) concentration of 6 µg/L was detected with a range from ND to 29 µg/L.[13]

(e) A USGS groundwater investigation of the Joshua Tree and Copper Mountain sub-basins reported a median naturally-occurring Cr(VI) concentration of 13 µg/L.[14]

(f) Groundwater investigation of the Cadiz and Fenner Valleys reported naturally-occurring Cr(VI) concentrations ranging from 15 to 26 µg/L.[15]

(g) A study of naturally-occurring Cr(VI) concentrations in groundwater from approximately 200 public supply, irrigation, and observation wells in the western Mojave Desert indicated a median Cr(VI) concentration of 7 µg/L, with a range of 0.2 to 60 µg/L.[16]

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[12] Golden State Water Company, 2010a, "*Water Quality Report: Apple Valley South Water System,*" http://www.gswater.com/csa_homepages/documents/AppleValleySouth061110.pdf; Golden State Water Company, 2010a, "*Water Quality Report: Barstow Water System,*" http://www.gswater.com/csa_homepages/documents/Barstow061110.pdf; Golden State Water Company, 2010b, "*Water Quality Report: Desert View Water System,*"http://www.gswater.com/csa_homepages/documents/DesertView061110.pdf; Golden State Water Company, 2010c, "*Water Quality Report: Lucerne Water System.*"

[13] Twentynine Palms Water District, June 2010, "*2009 Consumer Confidence Report,*" http://www.29palmswater.org/pdf/Consumer_Confidence_Report_2009.pdf.

[14] Nishikawa, Tracy, Izbiki, John A., Hevesi, Joesph A., Stamos, Christina L., and Martin, Peter, 2004, "*Evaluation Of Geohydraulic Framework, Recharge Estimates, And Ground-Water Flow Of The Joshua Tree Area, San Bernardino County, California.*"

[15] Metropolitan Water District of Southern California and Bureau of Land Management. September 13, 2001, "*Final Environmental Impact Report Final Environmental Impact Statement Cadiz Groundwater Storage And Dry-Year Supply Program, San Bernardino County, California.*"

9. Groundwater quality records collected by the CDPH show that concentrations of Cr(VI) detected in water supply wells vary considerably over time at any given well.[17] As a result, increases or decreases in the concentration of Cr(VI) at a given well do not always signify the arrival or departure of a particular source or plume of Cr(VI). Rather, these changes may be expected as a result of other factors, including sample collection procedures, seasonal changes, changes in well operation, laboratory analysis, variations in annual precipitation, and other factors.

10. Groundwater data collected by the CDPH in the Mojave area show that the concentrations of Cr(VI) at these wells typically fluctuate over time.[18] Exhibits F and G to this Declaration illustrate changes in Cr(VI) concentrations measured over time in several wells in the Mojave area. On these figures, the highest concentration of Cr(VI) detected at each water supply well (or well cluster) is shown. In addition, plots of concentrations of Cr(VI) over time for select wells within a well cluster are shown. As shown on these charts, it is common for the concentration of Cr(VI) to vary in a random pattern around a naturally-occurring background value.

11. Other water quality records compiled by the CDPH corroborate the variability in the concentrations of Cr(VI) detected at individual water supply wells in the Mojave area over time.[19] A review of results for hundreds of water supply wells in San Bernardino County indicates that chromium is often present above the laboratory reporting limit of 1 µg/L, and that Cr(VI) concentrations are often variable. For example, concentrations of Cr(VI) detected in Hesperia Water District well 15-A have ranged from 2.6 to 7.93 µg/L. Similar concentration ranges were reported for Victor Valley Water District well 208 (Cr(VI) ranging between 4.2 and

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[16] Ball, J.W., and Izbicki, J.A., 2004, "*Occurrence Of Hexavalent Chromium In Ground Water In The Western Mojave Desert, California*," Applied Geochemistry, Vol. 19, pp. 1123-1135.
[17] California Department of Public Health, 2011, "*Chromium-6 in Drinking Water Sources: Sampling Results*," Web page accessed on 7/6/2011. http://www.cdph.ca.gov/certlic/drinkingwater/Pages/Chromium6sampling.aspx
[18] *Id.*
[19] *Id.*

9.5 µg/L). The Loma Linda University Anderson Well 2 reported a Cr(VI) range of 1.3 to 5.4 µg/L, while Anderson Well 3 has a reported Cr(VI) range from 2.0 to 4.5 µg/L.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct and that this Declaration was executed on July 12, 2011, at Sacramento, California.

Brian Schroth

Brian Schroth

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EXHIBIT A

Brian K Schroth, Ph.D., P.G., C.Hg.

Senior Geochemist/Hydrogeologist

Education

Ph.D., Soil Science, University of California, Berkeley
Emphasis: Environmental Geochemistry
M.S., Hydrogeology, University of Nevada Reno
B.S., Geology, San Diego State University

Professional Registrations

Professional Geologist, California, No. 7423
Certified Hydrogeologist, California, No. HG 793

Distinguishing Qualifications

Dr. Schroth is a senior geochemist with over 19 years of experience in consulting and applied academic work. His expertise is centered on trace metal geochemistry, and has also strong knowledge of geochemical reaction path modeling, fate and transport of organic chemicals, and stable isotope geochemistry. His published research has focused on the potential effects of organic compounds present in landfill waste on the fate and mobility of trace metals in groundwater. He combines geochemistry with his strong background in hydrogeology, groundwater modeling, and soil science to help define fate and transport pathways in the environment. Dr. Schroth has emloyed the use of geochemical data on several projects with the goals of identifying different sources of contaminants, performing water balances, and defining and monitoring contaninant flowpaths. In water supply and subsurface water storage applications, Dr. Schroth has used geochemical modeling software to predict potentially harmful reactions (such as well clogging or the release of undesireable metals to groundwater), as well as to propose treatment options to prevent such reactions.

Relevant Experience

U.S. Department of Energy, Hanford Facility, Richland, WA, 2011
Dr. Schroth was the lead author for the Remedial Investigation report that focused on uranium-impacted soil and groundwater. He summarized a complex body of research and interpreted recently-collected data to describe the mechanisms of uranium leaching, vadose-zone transport, and groundwater mobility in a near-river environment. The fluctuating river level creates changes in geochemical conditions, which in turn affect the mobility of uranium. Dr. Schroth used his knowledge of hydrogeology and trace metal geochemistry to identify the key properties and assumptions involved in predicting mobility in this complex environment.

Shell Canada Scotford Facility, Alberta, Canada, 2010

Dr. Schroth combined data from several different waste streams at a water quality upgrading facility and modeled the potential precipitation reactions that could occur both on the surface and during deep well injection. He used the USGS geochemical modeling software PHREEQC to predict reactions under different mixing scenarios and at elevated temperature and pressure in a deep wastewater injection well. Dr Schroth's model interpretations will be used to identify water treatment methods to minimize injection well clogging by precipitated mineral phases.

Confidential Client, Lansing, Illinois, 2010
Dr. Schroth has used the USGS geochemical modeling software PHREEQC and PHAST to simulate the geochemical fate and transport of trace metals at a chemical processing site. The groundwater contains significant concentrations of organic waste chemicals and their breakdown products, and Dr. Schroth has utilized his research experience in mixed organic-metal waste to produce a more accurate simulation of metal transport in this regime. His work shows that metal mobility will be more limited than conservative models would predict, and when approved will allow the client to avoid costly and unnecessary remediation.

EPA Tar Creek Site, Northeastern Oklahoma, 2009-2011
Dr. Schroth was the lead geochemist for a large-scale lead/zinc mining site where EPA is proposing injection of fine to medium-grained tailings ("chat") into former mine workings. Dr. Schroth evaluated the geochemical data and used the geochemical modeling software PHREEQC and PHAST to simulate the reactions and transport of trace metals (cadmium, lead, zinc, and arsenic) in this environment. He combined hydraulic and geochemical skills to demonstrate that the injection of chat fines would have a temporary and minimal impact on the groundwater environment.

EPA Former Zinc Ore Processing Sites, Illinois, 2010-present
Dr. Schroth is currently the lead geochemist for three former zinc ore processing sites in which substantial amounts of process waste (slag) have been deposited as fill or in waste piles in the past. The slag has the potential to leach trace metals (cadmium, zinc, lead, nickel, arsenic) into the soil and groundwater, and Dr. Schroth is helping the team decide on well locations and constituents to be analyzed in the surface and groundwater samples. The goal of each project is to accurately assess the scale and impact of the problem and to produce innovative, cost-effective solutions for site cleanup.

Phosphate Mine Sites, eastern Idaho (EPA and USFS review), 2004-present
Dr. Schroth evaluated the fate and mobility of selenium in several phosphate mining sites in which natural selenium was mobilized by exposure to the atmosphere. He identified the key reactions that would enhance or limit mobility using geochemical analysis tools and modeling software. Dr. Schroth also reviewed the hydrogeologic analysis of the fractured bedrock aquifer and provided comments for EPA to help better evaluate the migration of selenium and other trace elements through this complex medium.

Confidential Client, Needles, CA, 2003-present
Dr. Schroth was the task manager for both geochemical evaluation and groundwater flow model development at this site where groundwater is contaminated with hexavalent chromium. He has determined the applicable geochemical and biogeochemical reactions at the site that limit chromium mobility in soil and groundwater and has presented geochemical analyses

numerous times to both technical and non-technical groups, including government agencies, tribal representatives, and consultants for a large municipal water district. Dr. Schroth wrote the background trace metals study for groundwater in the region, and was one of the main authors of the remedial investigation report, which included geochemical interpretation of site groundwater and surface water. He has employed the use of stable isotopes, ^{18}O and ^{2}H as well as ^{53}Cr, to further distinguish different water sources, chemical evolution, and mixing in the surface and subsurface. Dr. Schroth is also providing input to another consultant on the subject of potential migration of the *in situ* treatment byproducts manganese and arsenic, which are released from the soil under more chemically reducing conditions.

Rosevill Municipal Landfill, Roseville, CA, 2005-2011
Dr. Schroth is the senior technical reviewer for an ongoing monitoring program at a retired municipal landfill facility. In addition to interpreting data and reviewing reports, he is responsible for utilizing forensic geochemical techniques to identify potential sources of contaminants that are not believed to be associated with the facility. Dr. Schroth is currently reviewing data from offsite facilities and suggesting sampling and analysis methods that will better identify original sources of contamination.

EPA Lava Cap (Former Mine Site), Nevada City, CA, 2000-2007
Dr. Schroth provided geochemical analysis of groundwater and surface water data for this arsenic-contaminated site. A creek was inundated with mine tailings when a dam failed during a winter storm. The tailings were from a former gold mine and are rich in sulfide, iron, and arsenic. Dr. Schroth reviewed monitoring well, private well, and creek water analyses to assess the fate and mobility of arsenic in surface and groundwater. He has employed the use of stable isotopes, ^{18}O and ^{2}H, along with arsenic speciation data to determine that tailings likely have limited impact to groundwater outside of the area surrounding the creek.

West Basin Municipal Water District (WBMWD), Los Angeles County, CA, 2001
Dr. Schroth made use of natural tracers to estimate mixing and travel time of injected water from the West Coast Basin Barrier Project. Injection of imported and treated water is implemented parallel to the coast to prevent seawater intrusion from degrading water quality in municipal wells located further inland. WBMWD eventualy plans on injecting 100% treated water at the barrier, and Dr. Schroth's work helped to allieviate agency concerns regarding sufficient residence time of injected water. In addition, Dr. Schroth employed geochemical modeling to examine potential water quality effects that would come with switching to 100% treated water injection. Through this work, a revised monitoring plan is being developed with key monitoring points and analytes for verifying the model predictions.

Project Geochemist, City of Green Bay, Wisconsin, 2002
Dr. Schroth provided data analysis and geochemical modeling to address the unintended release of arsenic to groundwater during aquifer storage and recovery (ASR). He identified quantities of sulfide minerals present in the subsurface in larger quantities than anticipated by previous workers, and used his modeling skills to identify likely mechanisms for release and persistance of arsenic in groundwater. He is currently advising a Ph.D. study at the University of California at Berkeley that is using core samples from this study to identify more precisely

the key geochemical reactions that release and later control arsenic concentrations in groundwater.

Confidential Client, Richmond, CA, 1999-2002
Dr. Schroth was task manager in charge of data assessment and site conceptual model development for a former waste/stormwater retention facility. He combined historical boring logs, chemical data, and hydraulic information to create a holistic conceptual model. Dr. Schroth led a team to develop a finite element numeric model that brought complex hydraulic information together and accounted for subsurface drainage and saltwater intrusion along San Francisco Bay. The model was used to review site closure options and predict contaminant concentrations in an ecological receptor area.

Dr. Schroth was also the senior geochemist on this project. He identified groundwater zones of dissolved chlorinated solvent degradation and used this information to help delineate groundwater flowpaths. Dr. Schroth's geochemical analysis proved essential in showing that a site previously believed to be contaminated by chemical spills was in fact contaminated by rising groundwater carrying contaminants from another site.

Project Geochemist, Calleguas Municipal Water District, California, 2000
Dr. Schroth used geochemical modeling to assess the likelihood of chemical precipitation surrounding injection wells during aquifer storage and recovery (ASR). The success of ASR is largely dependent on avoiding clogging during injection from processes such as precipitation, biofouling, and clay destabilization. Dr. Schroth evaluated these factors in his evaluation.

Project Geochemist, INEEL CERCLA Disposal Facility, Idaho, 2001
Dr. Schroth predicted leachate concentrations of radionuclides in a proposed low-level waste landfill using geochemical modeling. The landfill was modeled for potential leachate impacts on deep groundwater. He selected key mineral phases of rare-earth elements for model input, and also evaluated mobility of both inorganic and organic compounds for vertical transport modeling.

Academic Experience

Assistant Professor, San Francisco State University, California (1997 – 2000)
Responsible for teaching majors courses in Hydrogeology and Groundwater Contamination at the undergraduate and graduate levels. Built a laboratory for use in hydrogeochemical research and established an agreement with local agencies to provide internship and access for the first graduate hydrogeology student at the university, whose thesis work involved basin boundary definitions and hydrologic budget for San Francisco and the Northern San Francisco Peninsula. Mentored several students to produce undergraduate thesis projects in hydrogeology and geochemistry. Taught other graduate courses in research methods and quantitative methods in Applied Geosciences. Also taught general education courses, including Environmental Geology and The Violent Earth, and computer applications for geologists.

Publications

Schroth, B.K. and G. Sposito. 1998. Effect of Landfill Leachate Organic Acids on Trace Metal Adsorption by Kaolinite, *Environmental Science & Technology* **32**: 1404-1408.

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EXHIBIT B

TABLE 1 - Hinkley Background Results Compared to Published Studies within Region
Declaration of Dr. Brian Scroth - Naturally Occurring Chromium in Groundwater

Study/Report	Naturally Occurring Total Chromium (µg/L)	Naturally Occurring Hexavalent Chromium (µg/L)	Hydrogeologic Setting	Description
[a]Hinkley Background Study, Mean	1.54	1.26	Wells are completed primarily in unconsolidated alluvial and floodplain sediments, composed of sand, gravel, silt and clay. Iron Mountain to the west is comprised primarily of granitic, metavolcanic, and metasedimentary rocks. Mount General to the east is comprised primarily of granitic, igneous and metamorphic rocks of felsic to intermediate composition, volcanic and sedimentary rocks. Two aquifers which are hydraulically connected are present, the Floodplain (Mojave River) and Regional Aquifers. The Floodplain and Regional Aquifers are further divided into Upper and Lower Aquifers which have limited hydraulic connection.	48 supply wells were sampled from over a 1-year period (2-4 times each). Well construction information available for only 20 of the 48 wells. Most wells with logs were screened across both the Upper and Lower Aquifers with 55 to 115 foot long screens. This indicates that blending of reducing water (with low to non-detect chromium) with oxic water from the Upper Aquifer may have occurred. It is probable that most wells without logs are also screened across both aquifers, or primarily the Lower Aquifer. The calculated UTL may be more representative of the Lower than Upper Aquifer.
[a]Hinkley Background Study, UTL	3.23	3.09		
[b]Topock Background Study, Mean	9.37	7.80	Groundwater samples were collected from alluvial and/or fluvial materials typically overlaying a consolidated Miocene conglomerate layer, underlain by metamorphic bedrock.	Six sampling events (25 wells) were used to develop background concentrations from mostly long screened supply wells in the greater Topock area, near Needles California. Fluvial materials were commonly associated with reducing conditions and low to non-detect chromium concentrations, therefore the UTLs may be conservatively low for wells screened in the alluvial aquifer under oxic conditions.
[b]Topock Background Study, UTL	34.1	31.8		
[c]USGS Western Mojave Desert, Range	not calculated	Concentration range: ND to 60	Groundwater samples collected from supply, irrigation and observation wells completed in alluvium derived from San Gabriel Mountains (eroded from mafic, granitic, metamorphic and volcanic rocks).	Approximately 200 wells were sampled. In addition, depth discrete samples were collected, which indicated that Cr(VI) concentrations could vary from <0.1 to 36 µg/L in a single well due to variable redox conditions. Cr(VI) concentrations were low near mountain recharge areas where pH values were neutral and low in discharge areas where there was low dissolved oxygen. The highest Cr(VI) concentrations (up to 61 µg/L) were reported for wells completed within alluvium derived from mafic rocks, with lower concentrations (up to 36 µg/L) reported for alluvium derived from less mafic granitic, volcanic and metamorphic rocks.
[d]USGS Western Mojave Desert, Sheep Creek fan, and Surprise Springs area, Median	NM	7.0	Follow on USGS paper presenting additional results from study listed above. The highest Cr(VI) concentrations were observed in alluvial aquifers eroded from mafic rock. Cr(VI) as high as 27 µg/L was also observed in aquifers eroded from granitic rock. The presence of Cr(VI) in granitic aquifers may be partially attributed to oxidation of Cr(III) to Cr(VI) by manganese oxides.	Results for 157 public supply, irrigation and observation wells in the Western part of the Mojave desert were included. Cr(VI) did not exceed 5 µg/L at pH < 7.5 in any geologic conditions. Range in Cr(VI) = 0.2 - 60 µg/L. Study indicated that majority of chromium detected was in the form of Cr(VI). Cr(VI) distribution in soil samples was found to be greatest above and near the water table, and concentrations rapidly decreased with depth. This observation was supported with chromium groundwater sample results.
[e]ADEQ Sacramento Valley Arizona Study, Mean	42	NM	Wells primarily sampled from alluvial aquifer materials. Mountain ranges forming basin boundaries consist predominantly of granitic, volcanic, and metamorphic rocks, with limited sedimentary outcrops.	Regional Arizona Department of Environmental Quality (ADEQ) groundwater study of basin in NW Arizona (immediately east of the Mojave Basin) comprising 1,500 square miles east of the Colorado River. The upper 95% confidence interval for Cr(T) was 83 µg/L, and the lower 95% confidence interval for Cr(T) was 1 µg/L.

TABLE 1 - Hinkley Background Results Compared to Published Studies within Region
Declaration of Dr. Brian Scroth - Naturally Occurring Chromium in Groundwater

Study/Report	Naturally Occurring Total Chromium (µg/L)	Naturally Occurring Hexavalent Chromium (µg/L)	Hydrogeologic Setting	Description
[f]USGS Regional Aquifer System Analysis Program, Mean	10.3	NM	Variable, multiple basin study. Maximum Cr(T) concentrations were observed in basins bounded by intermediate volcanic rocks, with lesser concentrations associated with basins bounded by intrusive rocks.	436 samples were collected from 72 basins in central and southern Arizona, southeastern California and Nevada, and western New Mexico. Results for 5 percent of samples collected were greater than 50 µg/L. Range in Cr(T) concentrations was 0 to 300 µg/L, standard deviation = 30.7 µg/L.
[g]CA State Water Resources Control Board, GAMA Program	NM	53% of wells > then 1.0	Variable. State Wide Study.	California Department of Health Services data for 1997-2008 were evaluated. 3,156 out of 5,943 tested public water wells (active and standby) throughout CA have detected Cr(VI) at concentrations greater than the laboratory reporting limit of 1 µg/L. Los Angeles, San Bernardino, and Fresno counties had the highest number of detections greater than 1 µg/L.
[h]Joshua Tree and Copper Mountain groundwater sub-basins, San Bernardino County, Median	NM	13.1	Alluvial deposits, overlying sedimentary and volcanic deposits, which overly granitic and metamorphic basement rocks.	Cr(VI) concentrations for 6 wells (23 samples total) ranged from 0.6 to 36.6 µg/L, with a median of 13.1 µg/L.
[i]Cadiz and Fenner Valleys, Mojave Desert (south eastern CA), Range	NM	15-26	Alluvial basin, bounded by basin and range rocky mountain ranges	Chromium concentrations were generally uniform throughout study area, indicating that Cr(VI) was naturally occurring.
[j]Twentynine Palms Water District, Mean	NM	6.0	Water is supplied from four different alluvial aquifers, the Fortynine Palms Groundwater Basin, the Indian Cove Groundwater Basin, the Eastern Groundwater Basin, and the Mesquite Springs Groundwater Basin.	Public water supply system. Range in Cr(VI) concentrations was ND to 29 µg/L.
[k]Golden State Water Company, Barstow, Range	NM	ND to 1.1	Groundwater is supplied from the Mojave River Basin-Centro sub-basin which is the center of the Mojave Basin extending northwesterly and southeasterly from the Mojave River.	Public water supply system. Range in concentrations was ND to 1.1 µg/L in 2006 samples reported in 2008. 2010 report did not include data for Cr(VI) or Cr(T).
[l]Golden State Water Company, Victorville Desert View Water System, Mean	NM	5.1	Groundwater is supplied from the Mojave River Basin-Este sub-basin which is located in the Lucerne Valley area east of the Mojave River.	Public water supply system. Range in concentrations was 5.0 to 5.1 µg/L.
[m]Golden State Water Company, Victorville Apple Valley South Water System, Mean	NM	2.5	Groundwater is supplied from the Mojave River Basin-Alto sub-basin which is the upper portion of the Mojave River.	Public water supply system. Range in concentrations ND to 6.3 µg/L.

TABLE 1 - Hinkley Background Results Compared to Published Studies within Region
Declaration of Dr. Brian Scroth - Naturally Occurring Chromium in Groundwater

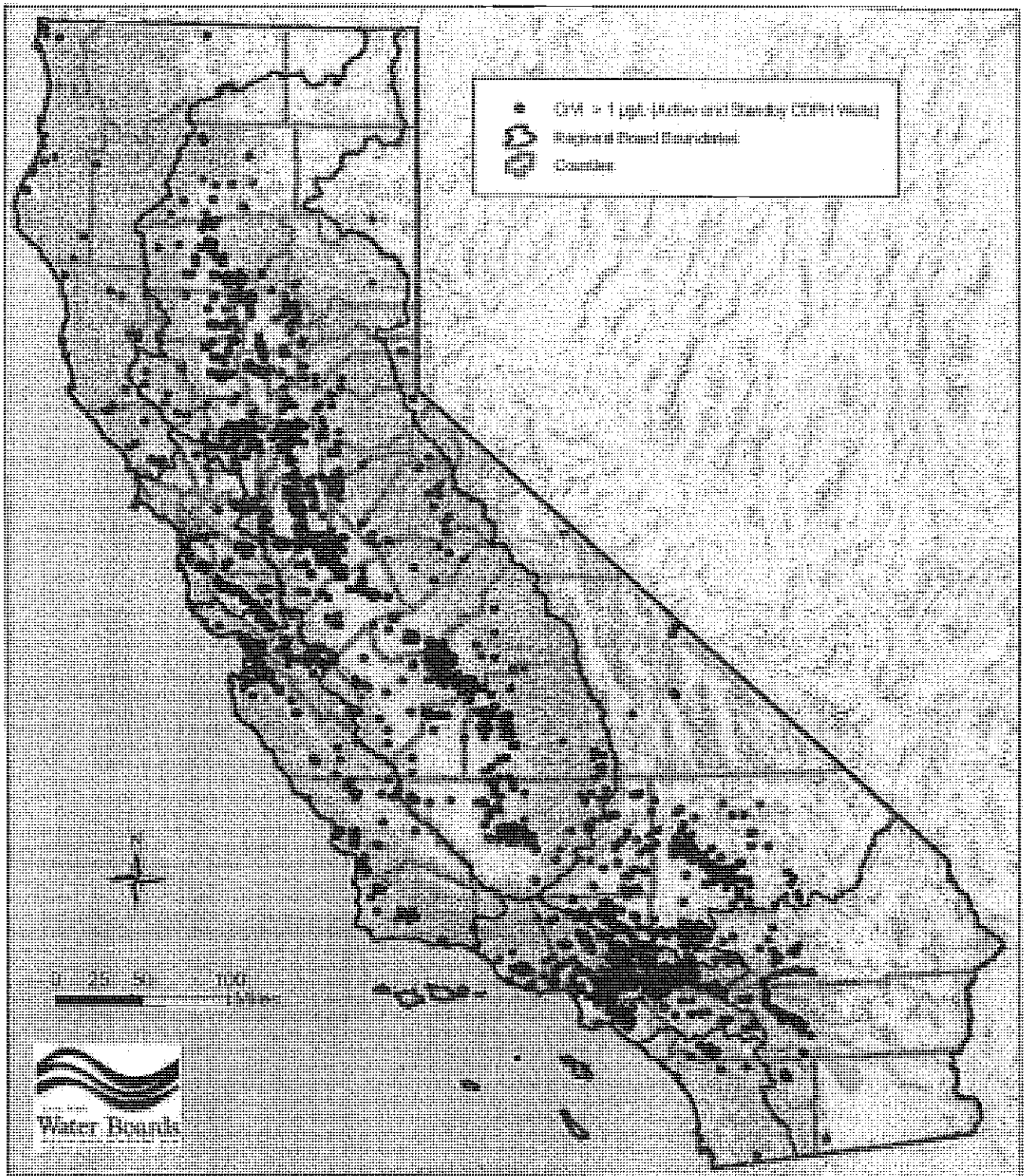
Study/Report	Naturally Occurring Total Chromium (µg/L)	Naturally Occurring Hexavalent Chromium (µg/L)	Hydrogeologic Setting	Description
[n]Golden State Water Company, Lucerne Water System, Mean	NM	2.7	Groundwater is supplied from the Mojave River Basin-Este sub-basin which is located in the Lucerne Valley area east of the Mojave River.	Public water supply system. Range in concentrations ND to 4.6 µg/L.

References:

[a]CH2M HILL. 2007. Groundwater Background Study Report, Hinkley Compressor Station, Hinkley, California. February 7.
[b] CH2M HILL, 2007. Groundwater Background Study, Steps 3 and 4: Final Report of Results PG&E Topock Compressor Station, Needles, California
[c] Ball James W., and Izbicki, J.A., 2004. Occurrence of Hexavalent Chromium in Groundwater in the Western Mojave Desert, California
[d] Izbicki, James A., Ball, James W., Bullen, Thomas, D., Sutley, Stephen J. Sutley. 2008. Chromium, Chromium Isotopes, and Selected Trace Elements, Western Mojave Desert, USA
[e] Arizona Department of Environmental Quality (ADEQ) Open File Report June 2001. Ambient Groundwater Quality of the Sacramento Valley Basin: A 1999 Baseline Study
[f] Robertson, Frederick N. 1991. Geochemistry of Ground Water in Alluvial Basins of Arizona, and Adjacent Parts of Nevada, New Mexico, and California. U.S. Geological Survey Professional Paper 1406-C.
[g] State Water Resources Control Board Division of Water Quality GAMA Program. September 2009. Groundwater Information Sheet Chromium VI.
[h] Evaluation of Geohydraulic Framework, Recharge Estimates, and Ground-Water Flow of the Joshua Tree Area, San Bernardino County, California. 2004. Nishikawa, Tracy., Izbicki, John A., Hevesi, Joesph A., Stamos, Christina L., and Martin, Peter.
[i] Metropolitan Water District of Southern California (MWD) and Bureau of Land Management. 2001. Cadiz Groundwater Storage and Dry-Year Supply Program, Final EIR/EIS response to Comments.
[j] Twentynine Palms Water District. 2010. June 2009 Consumer Confidence Report. June
[k]Golden State Water Company. 2008 and 2010. Water Quality Report, Barstow Water System.
[l]Golden State Water Company. 2010. Water Quality Report, Desert View Water System.
[m]Golden State Water Company. 2010. Water Quality Report. Apple Valley South Water System.
[n]Golden State Water Company. 2010. Water Quality Report. Lucerne Water System.

Abbreviations:
µg/L = micro-grams per liter
Cr(T) = total chromium, dissolved
Cr(VI) = hexavalent chromium, dissolved
ND = not detected at laboratory reporting limits
NM = not measured
UTL= upper tolerance limit
USGS = United States Geological Survey

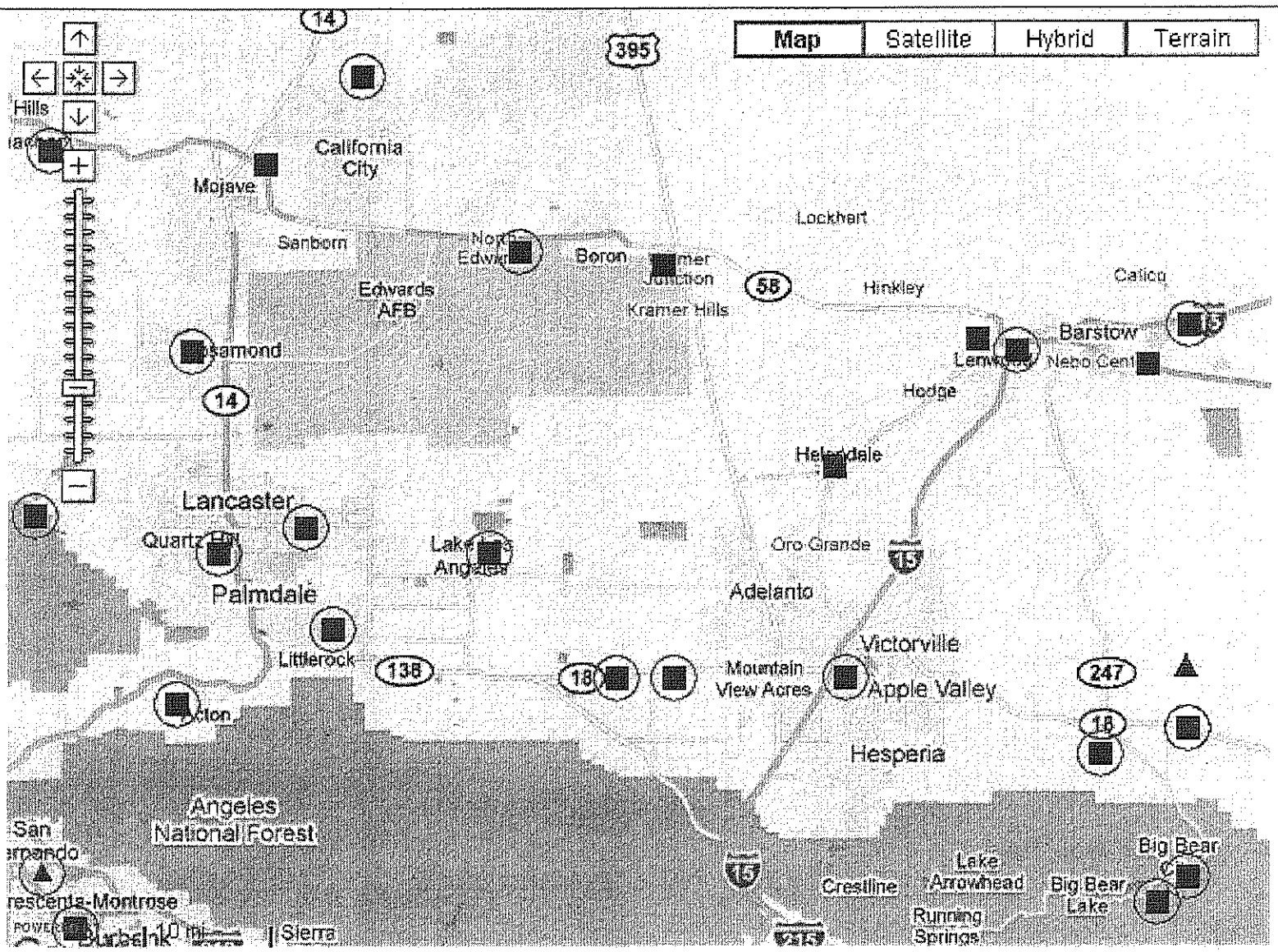
EXHIBIT C



Source: Figure adopted from State Water Resources Control Board Division of Water Quality GAMA Program. 2009. Groundwater Information Sheet Chromium VI. September.

FIGURE 1
Concentration of Hexavalent Chromium Detected at Active and Standby CDPH Wells
Pacific Gas and Electric Company
Hinkley, California

EXHIBIT D



Legend

- Supply Wells - CDPH (within one mile of actual location)
- Cluster of Supply Wells

Source: Figure based off of State Water Resources Control Board GEOTRACKER GAMA (http://geotracker.waterboards.ca.gov/gama/)

FIGURE 2
Mojave Area Showing Water Supply Wells with Cr(6) Greater than 1 Part Per Billion
Pacific Gas and Electric Company
Hinkley, California

CH2MHILL.

EXHIBIT E

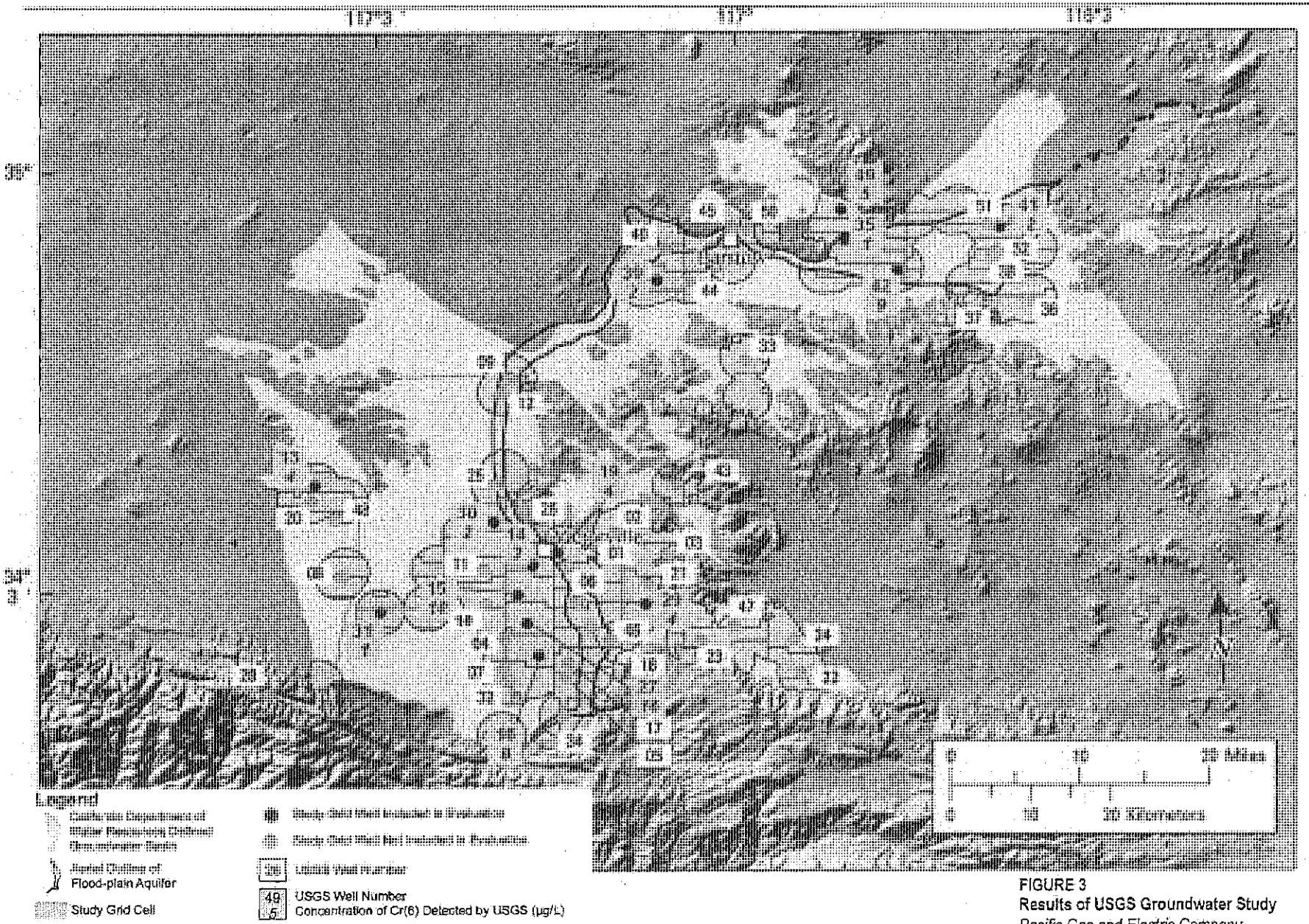


FIGURE 3
Results of USGS Groundwater Study
Pacific Gas and Electric Company
Hinkley, California

Source: Figure based off of USGS report titled "Groundwater Quality Data in the Mojave Study Unit, 2008: Results from the California GAMA Program".

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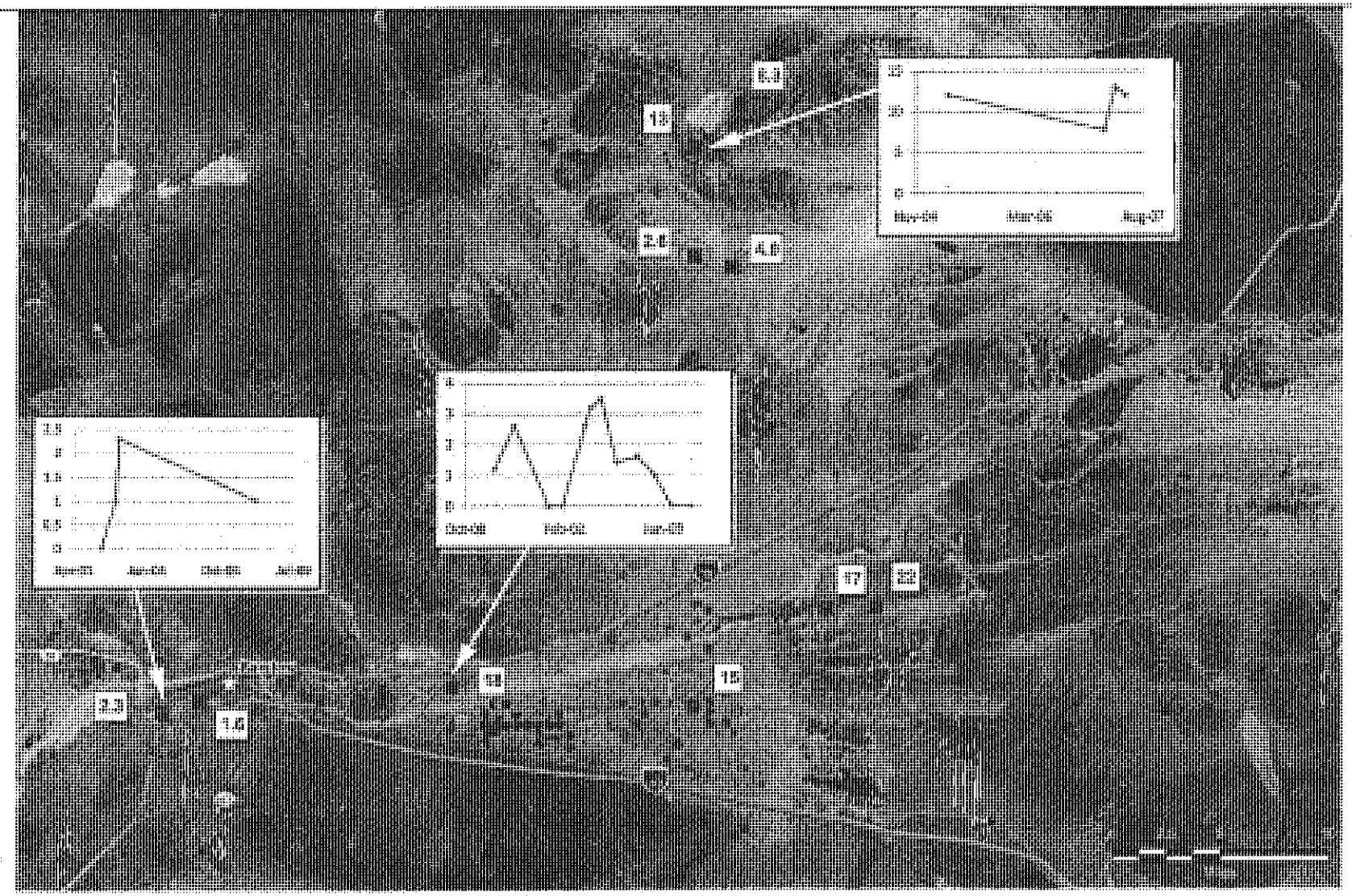
ES062311133809BAO Fig3_Results_USGS_GW_Study.ai 070711 ez

EXHIBIT F



Legend

- Water Supply Wells - CDPH
- Cluster of Water Supply Wells
- 26 — Highest Concentration of Cr(6) Detected at Well or Well Cluster
- Graph Represents Cr(6) Detections from the Well(s) Over Time

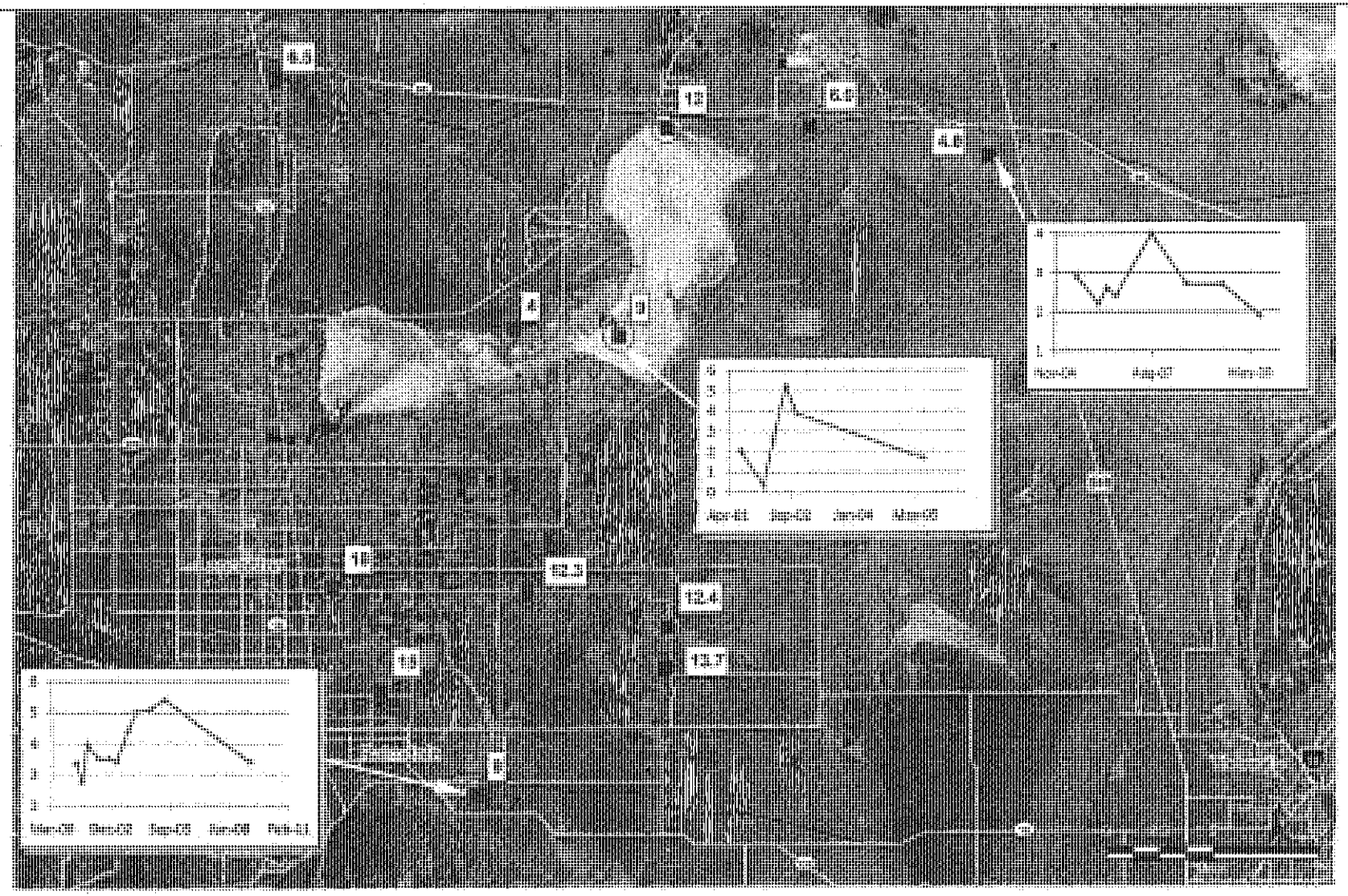
Note: results are in µg/L

Source: http://geotracker.waterboards.ca.gov/gama/qpublicmap/?CMD=runreport&myaddress=Hinkley+California

FIGURE 4a
Detected Cr(6) Results in the Mojave Area Water Supply Wells
Pacific Gas and Electric Company
Hinkley, California

CH2MHILL

EXHIBIT G



Legend

- Water Supply Wells - CDPH
- Cluster of Water Supply Wells
- 6.5 Highest Concentration of Cr(6) Detected at Well or Well Cluster

Graph Represents Cr(6) Detections from the Well(s) Over Time

Note: results are in µg/L

Source: http://geotracker.waterboards.ca.gov/gama/pubmap/?CMD=runreport&myaddress=Hinkley+California

FIGURE 4b
Detected Cr(6) Results in the Mojave Area Water Supply Wells
Pacific Gas and Electric Company
Hinkley, California

CH2MHILL

Attachment III:
Declaration of Thomas C. Wilson,
dated October 24, 2011

(*Request for Immediate and Emergency Stay; Petition for Review; and Memorandum of Points and Authorities in Support Thereof*)

DECLARATION OF THOMAS C. WILSON

I, Thomas C. Wilson, declare:

1. I am employed by PG&E. I started with the company in 1975. My first position was working as a biologist on the company's environmental programs. My current position is Director of Environmental Remediation. My responsibilities include overseeing PG&E's efforts in connection with the Hinkley community.

2. PG&E has for many years acknowledged with genuine regret its responsibility for the chromium contamination in the Hinkley community. PG&E is committed to continuing to work cooperatively with the Lahontan Board, interested agencies and Hinkley residents to address the environmental impacts and community concerns stemming from PG&E's past operations at its Hinkley Compressor Station.

3. As part of PG&E's responsibility for remediation, PG&E currently operates what I understand to be the largest in-situ barrier chromium remediation system in the world, as well as several large land treatment units, including one at the Desert View Dairy. PG&E has also been controlling a portion of the plume with a large fresh water injection system, which PG&E expanded earlier this year. In addition, PG&E recently expanded agricultural pumping to further control plume migration that will result in more than a 300% increase in plume control pumping. PG&E is also actively pursuing additional remedial options as part of what is being called the "final remedy."

4. In addition to these extensive remedial activities, PG&E has been actively working to reduce the Hinkley residents' ongoing concerns. At this time, less than ten domestic wells in the project area are known to contain chromium levels above identified natural background levels and no domestic well in the project area is known to have chromium levels above the state drinking water standard. Nevertheless, PG&E has undertaken a number of voluntary actions to address and respond to these concerns, including:

a. Beginning in the Fall of 2010, offering to test for chromium concentrations in any domestic well within one mile of the plume.

b. Beginning in the Fall of 2010, purchasing properties near the plume with domestic wells that have tested above background levels for hexavalent chromium at prices significantly above the properties' appraised values.

c. Since the Fall of 2010, providing bottled water to landowners whose domestic well water contains hexavalent chromium concentrations above natural background levels, and to all domestic well owners within approximately a half mile of the plume regardless of chromium concentrations in the wells, as well as to the Hinkley School and the Hinkley Senior Center.

d. Since the Fall of 2011, offering to supply bottled drinking water to any resident within one mile of the chromium plume, regardless of whether their domestic well water exceeds background levels.

5. PG&E's voluntary program to supply bottled water to Hinkley residents fully satisfies the first prong of the Lahontan Board's recent Cleanup and Abatement Order (the "CAO"). If the State Water Resources Control Board were to stay of the CAO, PG&E would continue its voluntary program.

6. While PG&E's voluntary efforts are consistent with key aspects of the CAO, PG&E is concerned about the far- reaching implications of certain provisions. For example, the CAO:

a. Sets a standard for hexavalent chromium concentrations that is more than one hundred times lower than the naturally occurring background concentrations in Hinkley, as well as hundreds of times lower than levels experienced in the drinking water supplies of some other communities around the state.

b. Requires replacement water for domestic wells containing concentrations well below natural background levels, a requirement that is inconsistent with California law and may be impossible to achieve.

c. Establishes criteria for bottled water so low that the commercially available bottled water provided as a part of PG&E's program, which is consumed by people across North America, may not meet the standards set in the CAO.

7. If PG&E's Petition is not resolved in the near future, PG&E would be required to begin significant activities that may ultimately be determined unnecessary or unsupported by law.

8. Unless the CAO is stayed, PG&E may also be penalized for non-compliance, even if the State Board ultimately rejects the CAO. As has consistently been the case, PG&E will make all reasonable efforts to comply with the CAO. Nonetheless, the risk that the Lahontan Board will view PG&E's efforts differently is quite real.

9. PG&E may also sustain intangible harms unless a stay is ordered. Even if PG&E's Petition is ultimately successful, the Lahontan Board may impose penalties for noncompliance with the provisions of the CAO. Penalties have not only financial, but also reputational consequences for any discharger, including PG&E. Furthermore, the CAO may have consequences far beyond Hinkley. For example, the CAO may serve as precedent for requirements elsewhere in California that a discharger would have to provide water that is better than applicable federal or state drinking water standards. Thus, even if the State Board ultimately grants PG&E's Petition, in the meantime PG&E, and potentially other dischargers, may face the "consequences" of the CAO.

10. The irreparable harm to PG&E (as described above in paragraphs 7, 8 and 9) might not be persuasive if public safety were at issue. But PG&E will continue to take the same steps as it has in the past to protect the Hinkley community while its Petition is pending.

I declare under penalty of perjury that the foregoing is true and correct, and that I executed this Declaration on October 14, 2011, in San Francisco, California.

By: _____
Tom Wilson

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Attachment IV:
Declaration of Anita Broughton,
dated October 17, 2011

(*Request for Immediate and Emergency Stay; Petition for Review; and Memorandum of Points and Authorities in Support Thereof*)

DECLARATION OF ANITA BROUGHTON

I, Anita Broughton, declare:

1. If called as a witness, I would and could competently testify thereto to all facts within my personal knowledge except where stated upon information and belief.

2. I am employed as a Lead Risk Assessor by Haley & Aldrich, a Consulting firm that specializes in underground engineering, environmental science and management consulting. Pacific Gas and Electric Company ("PG&E") has engaged Haley & Aldrich to assist with issues that have arisen in connection with the chromium plume in Hinkley, California. I have been specifically asked to state my professional opinion of Order No. 3 on Page 12 of CAO No. R6V-2011-0005A1 ("CAO"), entitled "Determination of Impacted Wells."

3. I declare under penalty of perjury under the laws of the State of California that the following statements represent my professional conclusions:

(a) Order No. 3(a) of the CAO to "perform an initial and quarterly evaluation of every domestic or community well in the affected area to determine if detectable levels of hexavalent chromium between the maximum background level and the PHG represent background conditions" is not supported by standard operating practices for remediation of groundwater contamination.

(b) The stated belief of the Lahontan Regional Water Board ("Regional Board") in Paragraph 29 of the CAO that background contaminant levels should be determined on a well-by-well basis, without regard to a single standard customary maximum background level is not supported.

4. A search of available information reveals that no facilities in California require the assessment of individual wells on a site for the determination of multiple background concentrations of a particular contaminant.

5. In my personal experiences as an environmental consultant and human health risk assessor for Haley & Aldrich with greater than 29 years as an environmental consultant, 25 years experience conducting multi-media human health risk assessments, and 22 years working with regulatory agencies in California, I have never seen an order to require background assessments on a well-by-well basis and have always understood an appropriate published concentration or statistically derived site-specific maximum background threshold to be the proper background concentration used for site data comparison purposes. Based on my experience, the latter approach has become the preferred approach by regulatory agencies as documented in several guidance documents, included those identified below:

- California Environmental Protection Agency, 1997. Selecting Inorganic Constituents as Chemicals of Potential Concern at Risk Assessments at Hazardous Waste Sites and Permitted Facilities, Final Policy. February.

- United States Environmental Protection Agency (USEPA), 2010. ProUCL Version 4.1.00, Technical Guidance (Draft), Statistical Software for Environmental Applications for Data Sets with and without Nondetect Observations. May.

- United States Environmental Protection Agency, 2009. Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities, Unified Guidance. March.

For other project sites that I have been involved within in California, regulatory-approved background approaches have generally included 1) the comparison of published statistically derived regional background threshold concentrations (e.g., arsenic concentrations in the Los Angeles area) to site data; or 2) the comparison of statistically-derived site-specific maximum background threshold concentrations. For a given constituent, these site-specific threshold concentrations are developed either using a set of regulatory agency agreed upon on-site or off-site background sample locations, or using other statistical techniques using a broader data set.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct and that this Declaration was executed this 17 day of October, 2011, at San Diego, California.

Anita Broughton, CIH

Attachment V:
Memorandum by OEHHA to Harold Singer, dated August 17, 2011, regarding proposed PHG for hexavalent chromium

(*Request for Immediate and Emergency Stay; Petition for Review; and Memorandum of Points and Authorities in Support Thereof*)

Office of Environmental Health Hazard Assessment

George V. Alexeeff, Ph.D., D.A.B.T., Acting Director
Headquarters • 1001 I Street • Sacramento, California 95814
Mailing Address: P.O. Box 4010 • Sacramento, California 95812-4010
Oakland Office • Mailing Address: 1515 Clay Street, 16th Floor • Oakland, California 94612

Matthew Rodriquez
Secretary for
Environmental Protection

Edmund G. Brown Jr.
Governor

MEMORANDUM

TO: Harold J. Singer, Executive Officer
Lahontan Regional Water Quality Control Board
2501 Lake Tahoe Boulevard
South Lake Tahoe, California 96150

FROM: George V. Alexeeff, Ph.D., D.A.B.T.
Acting Director

DATE: August 17, 2011

SUBJECT: PROPOSED PUBLIC HEALTH GOAL FOR HEXAVALENT CHROMIUM

Thank you for your inquiry of July 19, 2011 requesting guidance on the use of the new Public Health Goal (PHG) for hexavalent chromium (Cr VI) as a possible replacement standard for drinking water in Hinkley, California. On July 27, 2011, the Office of Environmental Health Hazard Assessment (OEHHA) published its PHG for Cr VI. Consequently, this PHG is no longer proposed but has been officially established by OEHHA at 0.02 parts per billion (ppb). This puts California in the position of having in place a non-mandatory goal for Cr VI without a corresponding state or federal regulatory standard. We appreciate that this may create challenges for regional water boards. The current situation in Hinkley described in your letter is one such example.

You have posed five specific questions to OEHHA covering three different aspects of the newly finalized PHG for Cr VI:

1. Whether the PHG is appropriate for use as a drinking water replacement standard?
2. Whether the PHG is scientifically justified given the comments of Dr. Joshua W. Hamilton, Ph.D.?
3. Whether evaporative coolers (a.k.a., swamp coolers) pose an inhalation risk by increasing the concentration of airborne Cr VI?

Responses to these questions have been prepared by OEHHA staff and are attached. Feel free to contact me at (916) 322-6235 if you require further information on how California's PHG for Cr VI was developed.

The energy challenge facing California is real. Every Californian needs to take immediate action to reduce energy consumption.

Printed on Recycled Paper

Attachment

Question 1. When is OEHHA scheduled to adopt the proposed PHG for hexavalent chromium?

Answer 1. The PHG for hexavalent chromium is now final and was posted on our Web site on July 27, 2011. It can be accessed at http://oehha.ca.gov/water/phg/072911Cr6PHG.html.

Question 2. What is OEHHA's position on the applicability of the proposed PHG as a value that would be protective of public health related to potential exposure of residents in Hinkley? If OEHHA's response is that use of the PHG is not applicable, please indicate if the current CA MCL is protective of public health and should be the standard that is used as the basis for providing replacement water. If neither the proposed PHG nor the CA MCL are the appropriate values to use, what would be an appropriate value that would be protective of public health?

Answer 2. By law, PHGs are determined by OEHHA's scientific assessments of the health risks posed by drinking water contaminants. In the case of hexavalent chromium, the PHG identifies a level of the metal in drinking water (0.02 ppb) that would pose no more than a one-in-one million cancer risk to individuals consuming water with that level of the contaminant daily over a 70-year lifetime. The PHG is a non-regulatory guideline that does not define an acceptable level of a contaminant in drinking water. State law requires the California Department of Public Health (CDPH) to set state Maximum Contaminant Levels for contaminants as close to the corresponding PHGs as is economically and technically feasible. In setting MCLs, CDPH considers important information (i.e., economic costs, technical feasibility, detection limits and water-supply issues) that by law OEHHA cannot consider when it develops PHGs.

Question 3. What is OEHHA's position on the comments by Dr. Joshua W. Hamilton Ph.D. (Attachment 3) on the scientific basis for the development of the PHG by OEHHA, specifically points 8-10 and 12?

Answer 3.

Comment 8-1: "For example, the lowest Cr(VI) concentration that caused tumors in animals in the National Toxicology Program study [4] which was the foundation for the draft PHG, was 20,000 µg/L. Notwithstanding, OEHHA proposed a PHG of 0.02 µg/L, *one million times lower* than the concentration that caused cancer in mice from a lifetime of drinking water exposure."

Response 8-1. The lowest Cr VI concentration causing a statistically significant increase in tumors compared to controls was 30,000 µg/L for adenomas and carcinomas of the small intestines of male mice (NTP, 2008). While the second sentence of this comment is literally true, it misses a critical point. Due to the limited number of mice used in the two-year bioassay (NTP, 2008), the absence of tumors at the lower Cr VI drinking water concentrations should not be interpreted as a threshold for tumor induction. Indeed, the genotoxic mechanism of action of Cr VI discussed in

the PHG document suggests that tumors would have been increased at dose levels well below those tested in the bioassay if more animals had been used in the experiment.

Comment 8-2: "The calculations embodied in the draft PHG do not represent 'established science.'"

Response 8-2. This statement is contradicted by the following:

1. Standard methodology was followed to model the rodent tumor data (U.S. EPA, 2005; OEHHA, 2009).
2. Professors from both the University of California and other universities reviewed the draft PHG documents. While there was not unanimity regarding the choice of method for modeling the rodent tumor data, the consensus opinion was that OEHHA had modeled the data according to the best current practices (see Responses to Comments document, available at http://oehha.ca.gov/water/phg/072911Cr6PHG.html).
3. Both the U.S. EPA (2010) and the New Jersey Department of Environmental Protection (2009) chose the same methodology as OEHHA for calculating the cancer potency of Cr VI. All three organizations derived the identical cancer potency value, suggesting that "established science" had been followed.

Comment 8-3: "And even if the draft PHG is adopted, regulators should not assume that exposures of the type and duration that would be experienced by Hinkley residents will result in any adverse health impacts. In fact, there is no way to confirm any of the risk assessors' assumptions in constructing the models that ostensibly support the draft PHG, or to determine whether there are any measurable health effects as a result of exposures at 0.02 µg/L. They reflect a highly conservative, overly-protective regulatory limit that assumes a lifetime of exposure, but they do not represent levels that suggest a significant or immediate health threat."

Response 8-3. It is not possible to measure tumor incidence in rodents at low Cr VI concentrations in drinking water because too many animals would be needed (U.S. EPA, 2005). Thus, the commenter is correct in suggesting that tumor induction cannot be measured in rodents exposed to Cr VI in the parts per billion (ppb) and parts per trillion (ppt) ranges. However, the best carcinogenicity data we have for exposures at low dose levels come from the human A-bomb survivors. Those data indicate a linear relationship between dose and cancer incidence that extends to the lowest dose levels analyzed for any carcinogen (Brenner *et al.*, 2003). Therefore, linear extrapolation is indicated for genotoxic carcinogens (U.S. EPA, 2005; OEHHA, 2009). This methodology was used in the PHG document to quantify the cancer risks posed by concentrations of Cr VI in the ppb and ppt ranges.

Comment 9-1: "Similarly, OEHHA is explicit that the draft Cr(VI) PHG is not and should not be used as a regulatory or cleanup standard: 'PHGs are not regulatory requirements, but instead represent non-mandatory goals....PHGs are not developed as target levels for cleanup of ground or ambient surface water contamination, and may

not be applicable for such purposes, given the regulatory mandates of other environmental programs.' ([3] p. iii.)"

Response 9-1. The commenter is correct in stating that PHGs are not developed as groundwater cleanup standards. Rather, PHGs are used by the California Department of Public Health (DPH) in establishing primary drinking water standards (State Maximum Contaminant Levels or MCLs).

Comment 9-2: "In sum, the draft Cr(VI) PHG as its name implies, is at most a goal, not a regulatory level, and in no way should exposures to concentrations above 0.02 µg/L be interpreted as an immediate health risk to Hinkley residents nor should this proposed goal be used to set action or cleanup levels."

Response 9-2. The value 0.02 µg/L is the 70-year exposure level estimated to be associated with a one in one million increased risk of cancer. In other words, one extra case of cancer would be expected in a population of one million persons consuming drinking water for seventy years at this concentration. A drinking water concentration ten times higher would yield a ten-fold higher risk (for example).

Comment 10-1: "The initial draft Cr(VI) PHG drew on two principal studies: The 1968 Borneff, et al., animal study [6], and the 1987 Zhang and Li epidemiology study. [7] Both are outdated and flawed, and they have been rejected by EPA and mainstream toxicology experts as a foundation for toxicology risk assessment."

Response 10-1. U.S. EPA's current Draft Toxicological Review of Hexavalent Chromium (2010) contains an extensive discussion of the epidemiology study by Zhang and Li (1987). This study is an important part of that document's discussion of the human relevance of the rodent tumor data. The final PHG document does the same. It should be noted that the U.S. EPA document specifically supports the re-analysis of the original Zhang and Li (1987) study conducted by Beaumont *et al.* (2008). Dr. Beaumont is one of the authors of the final PHG document. With regard to Borneff *et al.* (1968), discussion of this study was moved to the Appendix of the PHG document on the advice of peer reviewers. The study was included in the Appendix so as to generate a PHG document that cites all significant studies that tested Cr VI carcinogenicity via the oral route. Neither Borneff *et al.* (1968) nor Zhang and Li (1987) is used to calculate the PHG of 0.02 µg/L. That calculation is based on rodent tumor data from NTP (2008).

Comment 10-2: "EPA's draft Profile appropriately omits any reference to the Borneff study in its review of key animal studies. While the draft profile discusses the Zhang study and three follow-up analyses, it correctly states that it should not be used for risk assessment purposes. The panel agreed with these assessments. Thus, there is already significant disagreement between the draft PHG and EPA's draft Cr(VI) Toxicology Profile."

Response 10-2. Borneff *et al.* (1968) is reviewed in the Draft U.S. EPA Toxicology Review of Hexavalent Chromium (2010). As mentioned above in Response 10-1, Zhang and Li (1987) is thoroughly evaluated in the U.S. EPA document, where it is an important part of the discussion concerning the human relevance of the rodent data.

Also as noted above, U.S. EPA selected the re-analysis of Zhang and Li (1987) by Beaumont *et al.* (2008) over Kerger *et al.* (2009) as representing the most useful re-analysis of the original data. Dr. Beaumont is one of the authors of the PHG document. Lastly, the OEHHA PHG document and the U.S. EPA document develop identical cancer potencies for Cr VI via the oral route. This does not support the claim in Comment 10-2 that "there is already significant disagreement between the draft PHG and EPA's draft Cr(VI) Toxicology Profile."

Comment 10-3: "The panel's consensus was that the pending studies provided important new information that was critical to an overall understanding of Cr(VI), and should be incorporated into the EPA's Profile. Thus, the panel urged EPA to wait for these studies to be published so that they may be taken into account in their assessment."

Response 10-3. OEHHA will review papers and materials relating to the American Chemistry Council study of Cr VI toxicology when they are published. If the study produces compelling information that should be reflected in the PHG document, OEHHA will take appropriate action.

Comment 12-1: "In addition, OEHHA concluded that exposure by inhalation during showering did not contribute significantly to the overall risk. And even with conservative assumptions regarding exposure during showering, the contribution to risk from inhalation was 180 times lower than that from drinking water exposure."

Response 12-1. This is correct. Less than one percent of the cancer risk due to Cr VI in drinking water was due to inhalation during showering compared to over 99 percent due to ingestion.

Question 4. What is OEHHA's position on the validity of footnote No. 5 in Attachment 3?

Answer 4.

Footnote 5: "The PHG associated with inhalation exposure may be readily calculated from the information in the draft PHG assessment by removing the contribution from oral exposures. The PHG associated with inhalation exposure is 3.6 µg/L."

Response to Footnote 5. It is not clear what Dr. Hamilton was trying to say in footnote 5. A PHG for a carcinogen is determined to be the drinking water concentration associated with a 10^{-6} cancer risk due to all applicable routes of exposure. The PHG for Cr VI in drinking water is 0.02 µg/L. This is based on exposure via ingestion and via inhalation during showering. Since so little Cr VI is inhaled during showering, a PHG based only on ingestion is identical (after rounding) to that based on ingestion plus inhalation during showering: 0.02 µg/L. The correct and useful interpretation is that the fractional cancer risk due to inhalation of Cr VI is very small, and that inhalation exposure cannot be used as a basis for establishing the PHG.

Question 5. What is OEHHA's position on Dr. Hamilton's conclusion that swamp coolers do not pose an inhalation risk? If OEHHA believes that Dr. Hamilton's

conclusions are not supported by the available information (including but not necessarily limited to the references cited), does OEHHA believe that swamp coolers could pose a risk, and if so, at what hexavalent level? If OEHHA believes that the available information is insufficient to reach a conclusion, would OEHHA be willing to perform an evaluation of a typical residence in Hinkley to determine if the use of swamp coolers with water which contains low levels of hexavalent chromium poses a health risk to the residents? This evaluation could be in collaboration with the Agency for Toxic Substances Disease Registry which has done similar studies on other constituents.

Answer 5. We agree with Dr. Hamilton's conclusion that swamp coolers do not increase the concentration of airborne Cr VI. Thus, with regards to Cr VI, swamp coolers do not constitute an inhalation health risk. This is based on the following studies located in the scientific literature:

1. Finley *et al.* (1996) demonstrated that swamp coolers operating with water containing concentrations of Cr VI up to 20 mg/L did not increase the concentration of Cr VI in indoor air. The American Society for Testing and Materials (ASTM) Method D5281 was used. This allowed measurement of total Cr VI in the air, whether in the form of fumes, aerosols or particulates.
2. Paschold *et al.* (2003a) determined that indoor swamp coolers lowered rather than raised the levels of airborne particulate matter ($PM_{2.5}$ and PM_{10}) potentially harboring Cr VI.
3. Paschold *et al.* (2003b) extended their previous study (Paschold *et al.*, 2003a) by analyzing the elements comprising airborne particulate matter ($PM_{2.5}$ and PM_{10}) collected in the presence of swamp coolers. They found no evidence that swamp coolers introduced metals from the cooling water into the indoor air, whether in the form of particulates or aerosols.

These studies appear to have been well-conducted and the conclusions are warranted by the data. Therefore, the data on hand support Dr. Hamilton's conclusion that swamp coolers do not increase the concentration of airborne chromium.

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