

Contaminants in tilapia (*Oreochromis mossambicus*) from the Salton Sea, California, in relation to human health, piscivorous birds and fish meal production

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Abstract A summary of all existing information collected since 1980 on contaminants in tilapia from the Salton Sea is presented and risks to humans and fish-eating birds assessed. Of the 17 trace elements, 42 organic pesticides and 48 polychlorinated biphenyls (PCBs) analyzed in tilapia whole body and fillet samples, only selenium (Se), arsenic (As) and possibly dichlorodiphenyltrichloroethane (DDE) were found at levels high enough to be of concern for fish,

birds or humans. Average current concentration of arsenic (As) was $0.7 \mu\text{g g}^{-1}$ wet weight (ww) in whole body samples and $1.2 \mu\text{g g}^{-1}$ ww in fillet samples, or 2.8 and $5.7 \mu\text{g g}^{-1}$ dry weight (dw), respectively. Inorganic As averaged $0.006 \mu\text{g g}^{-1}$ ww ($0.03 \mu\text{g g}^{-1}$ dw) in fillet samples, which represented 0.3% of total As. By U.S. Environmental Protection Agency (U.S.EPA) guidelines, As levels in tilapia pose no threat of non-cancerous adverse health effects in children and adults. As is a known human carcinogen, however, and U.S.EPA cancer risk assessment procedures indicate that a weekly consumption of 540 g (19 oz) or more for 70 years would increase the upper bound cancer risk by 1 in 100,000 consumers exposed. Average current Se concentration was $2.2 \mu\text{g g}^{-1}$ ww in tilapia whole body samples and $1.9 \mu\text{g g}^{-1}$ ww in fillet (8.3 and $9 \mu\text{g g}^{-1}$ dw, respectively). Consumption of Se-contaminated tilapia was found to present no unacceptable risk for adverse health effects for adults consuming up to 1000 g (35 oz) of fillet per week even when additional intakes of Se from other food items were taken into account. Similarly, children weighing 30 kg or more could safely eat up to 430 g (15 oz) of tilapia fillet on a weekly basis. A health advisory issued by the State of California in 1986 recommended, on the basis of Se levels, that consumption of any fish from the Salton Sea be limited to 114 g (4 oz) every 2 weeks, but the rationale and calculations

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Saline Waters and their Biota

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on which that advisory was based are unavailable. We suggest that the existing health advisory for Salton Sea tilapia be revised by the state in light of this new information and updated risk parameters for As and Se. Dichlorodiphenyltrichloroethane (DDE) was detected in all samples of tilapia, with current levels averaging $0.085 \mu\text{g g}^{-1}$ in whole tilapia and $0.032 \mu\text{g g}^{-1}$ in fillet ww. Compared to screening values proposed by the U.S. EPA, these concentrations are unlikely to cause non-cancerous health effects in anglers, but one might exceed a 1×10^{-5} increase in cancer risk by consuming more than 4 meals of tilapia per week. Similarly, polychlorinated biphenyls (PCBs) were detected in tilapia fillets at levels that may increase the cancer risk for those anglers also eating more than 4 meals of tilapia per week. These determinations are based, however, on DDE concentrations reported from a small sample size ($n = 4$), and on screening values recommended by U.S.EPA. The paucity of DDE and total DDT analyses carried out in recent times on the edible portion of Salton Sea tilapia warrants additional analyses in order to evaluate the need for issuance of a fish consumption advisory with regards to long term exposure to total DDT via consumption of Salton Sea fish. With regards to the potential impact on fish and piscivorous birds, we cannot conclude whether concentrations of As in tilapia could pose a threat to the fish and the birds feeding on them. Se concentrations, however, may be elevated enough to negatively affect fish health, and reproduction and immune systems of piscivorous birds, but definitive studies are lacking. Total DDT and PCB concentrations in whole tilapia are not elevated enough to have adverse effects on fish and piscivorous birds. Fish harvesting for fish meal production has been proposed for the Salton Sea. Based on whole fish dry weight values of 61% protein and 21% ash, and the determined contaminant levels, tilapia could yield a meal of reasonable quality for use in formulating poultry, livestock and aquaculture feeds.

Keywords Saline lake · Selenium · Arsenic · Human health · Piscivorous birds · Fish meal · DDE · DDT

Introduction

The Salton Sea has been for a century the ultimate repository of pollutants released from the intensifying agricultural activities, urbanization, and industrial development in its watershed. This includes the Coachella and Imperial valleys in California, the northern part of the Mexicali Valley, Mexico, and the flanks of the bounding mountain ranges. Once these pollutants reach the Sea, they can bioaccumulate, be sequestered in sediments or be degraded at varying rates in different abiotic compartments of the ecosystem. Bioconcentration and bioaccumulation in fish present in the Sea could potentially create risks for human consumers and piscivorous birds.

Human health studies conducted thus far have considered the inorganic pollutants selenium (Se) and arsenic (As). A Se health advisory was issued by the State of California in 1986 (Office of Environmental Health Hazard Assessment, OEHHA), advising that no more than 114 g (4 oz) of fish caught from the Salton Sea be consumed over a 2-week period. In addition to Se, As in tilapia may impact human health. Although most of the As present in fish is of the organic type (Edmonds & Francesconi, 1993), which does not appear to interact with the human system (ATSDR, 2000), it is usually the smaller inorganic fraction of As, notably As (III) and As (V), that poses the most serious threat to human health (Braman, 1983, ATSDR, 2000). Long term ingestion of inorganic As may lead to adverse health effects including neurotoxicity of both peripheral and central nervous systems, and liver damage.

There has been little study of the potential impact of these contaminants on piscivorous birds at the Salton Sea. The Salton Sea is also experiencing rising salinity and nutrient loading, which further impacts the biota and could be exacerbated by the presence of contaminants. Although bird and fish die-offs have occurred since the creation of the Sea in 1907, their frequency and intensity increased in the 1990s, as did the diversity of disease pathogens causing avian epizootic outbreaks (Friend, 2002). Although a clear correlation cannot be presently established, contaminant levels in fish-eating birds may impair the

bird's immune system (Bobker, 1993; Bruehler & de Peyster, 1999; Friend, 2002). Anoxic and sulfide events, parasites, high salinity and low water temperature probably contribute to the irregular recruitment of the tilapia population and the massive die-offs. In addition, mortality of young-of-the-year tilapia during winter months may be due to decreased resistance to prolonged low water temperatures because of Se, a condition known as Winter Stress Syndrome (Lemly, 1996b).

Fish harvesting from the Salton Sea to produce fish meal, pet food or fish-based fertilizers has been proposed. If sustainable, large scale harvesting of fish could potentially help alleviate the eutrophic state of the lake by allowing substantial amounts of phosphorus to be removed from the ecosystem (González et al., 1998; Costa-Pierce & Riedel, 2000). Benefits in the short term could be decreased bird and fish die-offs, increased production of zooplanktonic and zoobenthic populations utilized as food by these vertebrate populations (Detwiler et al., 2002; Tiffany et al., 2001; Watts et al., 2001), reduction in odors produced by the Sea, and increased value of the Sea for sport fishing and other recreational activities. Such benefits were among the goals described in the Salton Sea Reclamation Act of 1998 (Public Law 105–372).

Although four species constitute the sport fishery at the Sea, the species most worth considering for a commercial enterprise is tilapia (*Oreochromis mossambicus* Peters; Cichlidae). This exotic species originating from Africa, escaped from a private pond in Niland, California, and accidentally reached the Sea in the mid 1960s. It became the dominant fish in the lake from the mid 1970s through the late 1990s, when it supported one of the most productive salt lake fisheries in the world. It is now very scarce (Caskey et al., 2007). Microsatellite DNA analyses suggest that the Salton Sea tilapia is a hybrid of *O. mossambicus* and *O. urolepis hornorum*, two tilapia species originally from Africa (Costa-Pierce & Doyle, 1997; Costa-Pierce & Riedel, 2000). The scant DNA samples of *O. u. hornorum* used for the microsatellite DNA comparison preclude, however, a definitive conclusion. Costa-Pierce & Doyle (1997) also suggested that the

high level of heterozygosity of the Salton Sea tilapia compared to that of other species of tilapia found in California may make it a unique strain.

If the productivity of the tilapia population were high enough to support a fish meal manufacturing plant, important factors in the feasibility and viability of this economic enterprise would be nutritional and other characteristics of the final products, including organic and inorganic contaminant concentrations. Because the Salton Sea has no outlet, natural as well as anthropogenic contaminants have had the opportunity to accumulate in the water and sediments and to be taken up and sequestered by the biota. They can also be transformed via biotic and abiotic processes and their metabolites bioaccumulated.

The objectives of this study were three-fold: to summarize all existing information on contaminants in Salton Sea tilapia, most of which is unpublished; to re-assess the implications of this information for human health and wildlife protection; and to assess its implications for the commercialization of potential tilapia products such as fish meal.

Methods

Results of several studies conducted by six agencies and institutions are reported. Four of these data sets have not been previously published. The largest data set is from the Toxic Substance Monitoring Program (TSMP) of the California State Water Resources Control Board. This program, initiated in 1976, annually analyzes biotic samples collected from more than 100 waterbodies in California that are believed to have water quality problems (Rasmussen & Blethrow, 1990). Two U. S. Geological Survey reports on the potential impact of irrigation drainage on fish and avian populations of the Salton Sea (Setmire et al., 1990, 1993; Schroeder et al., 1993) present information on 17 trace elements and pesticides in water, bottom sediments and biota, including tilapia, collected in the Salton Sea and its vicinity. A U.S. Fish and Wildlife Services study identified 14 trace elements in the four most abundant fish species in the Salton Sea, including tilapia (Saiki, 1990).

Two masters thesis projects from the Graduate School of Public Health (GSPH), San Diego State University (Surico-Bennet, 1999; Vicario-Fisher, 1999) determined the concentrations of selenium (Se) and arsenic (As), respectively, in tilapia and two other fish species and assessed the human health risk associated with their consumption. The SDSU Salton Sea Ecosystem Research Group (SSERG) assessed contaminants in four sets of tilapia collected in 2000–2002. We collected 5 tilapia from fishermen in April 2000 for a preliminary assessment of As, Se, DDD, DDE and DDT concentrations. For more definitive information, tilapia were collected from 5 locations in the Sea in both December 2000 and in May 2001 and analyzed for 7 trace elements, 42 synthetic organics and 48 PCB congeners. In Fall 2002 As speciation analyses were carried out on skinned fillet from 8 tilapia. All results from SDSU studies are presented here for the first time.

Riedel et al. (2002a) collected 14 specimens of three fish species, including 5 tilapia and analyzed them for 9 trace metals, 31 pesticides and 31 PCB congeners.

Methodologies for the different studies are given below. Authors of studies were contacted to obtain missing data or clarifications of methodology when necessary, and this information is incorporated here.

Toxic Substances Monitoring Program,
1980–1996

Toxicant concentrations in tilapia, either whole or fillet, were analyzed for 30 composite samples collected at the Salton Sea from 1980 to 1996 (Rasmussen & Blethrow, 1990, 1991; Rasmussen, 1993, 1995, 1997). Whenever possible, a minimum of six tilapia were collected for each composite sample using various fishing methods (electrofishing, gillnetting) at different sampling stations. Number of fish in each composite, fork lengths and station locations are given in Table 1. Upon collection, samples were placed in clean stainless steel buckets until they were double-wrapped in extra-heavy duty aluminum foil, labeled and placed on dry ice. Methods in the FDA Pesticide Analytical Manual (U.S. Food and Drug

Administration, 1970) were followed for tissue sample extraction. Weight and length of fish and moisture and lipid content were usually determined. Fillet and liver concentrations were determined for selected trace elements (As, Ag, Cd, Cr, Cu, Hg, Ni, Pb, Se, Zn) and organic compounds (chlordanes isomers, chlorpyrifos, DCPA (dimethyl tetrachloroterephthalate, Dacthal®), diazinon, DDT isomers, dieldrin, dicofol, endosulfan I and II, endosulfan sulfate, endrin, ethion, heptachlor, heptachlor benzene, heptachlor epoxide, hexachlorocyclohexane isomers (HCH), methoxychlor, nonachlor isomers, oxadiazon, ethyl and methyl parathion, PCBs 1248, 1254, and 1260, and toxaphene). Of all these, only Se, DCPA and DDT isomers (namely DDE) were detected in tilapia samples.

National Fisheries Contaminant Research
Center, 1985

Saiki (1990) analyzed a total of 14 tilapia (*O. mossambicus*) captured by hook and line in the vicinity of the New River Delta in August 1985. Once captured, the live specimens were cleaned under running water to remove excess debris, wrapped, bagged and kept cold in ice until they could be frozen at -10°C . Fish were then thawed, measured (total length), weighed and the moisture content determined. Composite samples (2 samples of 5 fish each, 1 with 4) were homogenized and then analyzed for whole body concentrations of As, B, Cd, Co, Cu, Fe, Hg, Mo, Ni, Pb, Tl, V and Zn. Concentrations of As, Hg and Se were determined by atomic absorption spectrophotometry. Concentrations of the other trace elements were determined by spectrometry. Elemental concentrations were reported in $\mu\text{g g}^{-1}$ dw and converted to a wet weight basis using the percent moisture content reported by the laboratory that did the analyses.

U.S. Geological Survey, 1986

Tilapia (*O. mossambicus* and *O. zilli*) were collected with dipnets or small seines from various stations at the Sea during August 1986, were wrapped in aluminum foil and frozen in polyethylene bags (Setmire et al., 1990). Sample collection and

Table 1 Provenance and tissue characteristics of tilapia samples collected from the Salton Sea during 1980–2001 by the respective studies

Program ^a (date)	Sample number		Species ^b	Location ^c	Collection date	Sample size ^d – Tissue (W.F.L) ^e	Weight ^f (g)	Length ^f (mm)	%Water	%Lipid
	New	Original								
TSMP (1980–96)	1	40.1.S180	TLM	SS/S	21-May-80	7-F	227	216	na	0.5
	2	40.1.F.F.84	TL	SS/W/S	20-Jun-84	3-F	287	234	78	1.2
	3	40.2.F.85	TL	SS/S	7-Aug-85	5-F/L	1017	368	77/74	3.9/na
	4	119.01.W.87	T/LZ	SC/M	7-Jul-87	4-na	8	69	na	na
	5	40.4.F.87	TL	SS/S	7-Oct-87	6-F	805	360	79	na
	6	040.002.F.95.FR	T/LZ	SS/S	27-Oct-95	5-F	333	266	88	0.4
	7	040.001.F.95	T/LZ	SS/S	27-Oct-95	6-F	343	272	79	0.2
	8	728.00.92	TL	SS/N	30-Oct-96	6-F	101	162	78	0.4
	9	040.003.F.96.FR	TL	SS/N	30-Oct-96	5-F	99	171	77	0.8
	10	040.003.F.97	TL	SS/S	20-Nov-97	6-F/L	466	293	79/76	0.9
	11	040.003.F.98	TL	SS/N	10-Nov-98	6-F/L	470	305	77/69	60
	12	040.001.F.98	TL	SS/S	11-Nov-98	6-F/L	647	323	77.2/69	1.4
	13	040.001.F.00	TL	SS/S	09-Nov-00	5-F	665	334	77	0.7
	14	040.003.F.00FR	TL	SS/S	09-Nov-00	4-F	770	349	76	1.0
NFCRC (1985)	15	R1-SS-1	TLM	NRD	7-Aug-85	5-W	624	303	69	na
	16	R1-SS-2	TLM	NRD	7-Aug-85	5-W	613	317	68	na
	17	R1-SS-3	TLM	NRD	7-Aug-85	4-W	722	338	68	na
USGS (1986)	18	LNSS86-018	TLM	SSNR	Aug-86	20g-W	na	na	76	1.5
	19	LNSS86-05B	TLM	SSNR	Aug-86	20g-W	na	na	74	1.0
	20	LNSS86-40	TLM	SSNR	Aug-86	20g-W	na	na	72	6.9
	21	LNSS86-52B	TLM	NRD	Aug-86	20g-W	na	na	72	1.2
	22	LNSS86-48B	TLM	WRD	Aug-86	20g-W	na	na	76	1.3
	23	LNSS86-49B	TLM	WRD	Aug-86	20g-W	na	na	74	1.5
	24	none	TLM	WRD	Aug-86	20g-W	na	na	na	na
	25	none	TLM	WRD	Aug-86	20g-W	na	na	na	na
	26	none	TLM	NRD	Aug-86	20g-W	na	na	na	na
	27	none	TLM	NRD	Aug-86	20g-W	na	na	na	na
	28	none	TLM	ARD	Aug-86	20g-W	na	na	na	na
	29	none	TLM	ARD	Aug-86	20g-W	na	na	na	na
30	none	TLM	ARD	Aug-86	20g-W	na	na	na	na	
GSPH (1998–99)	31	T1	TLM	SSRA	Jun-Jul 98	1-F	612	289	78	na
	32	T2	TLM	SSRA	Jun-Jul 98	1-F	482	198	79	na
	33	T3	TLM	SSRA	Jun-Jul 98	1-F	564	272	80	na
	34	T4	TLM	SSRA	Jun-Jul 98	1-F	561	256	76	na
	35	T5	TLM	SSRA	Jun-Jul 98	1-F	510	211	77	na
	36	T6	TLM	SSRA	Jun-Jul 98	1-F	559	256	79	na

Table 1 continued

Program ^a (date)	Sample number		Species ^b	Location ^c	Collection date	Sample size ^d – Tissue (W,F,L) ^e	Weight ^f (g)	Length ^f (mm)	% Water	% Lipid
	New	Original								
	37	T7	TLM	SSRA	Jun–Jul 98	1-F	570	259	80	na
	38	T8	TLM	RH	Jun–Jul 98	1-F	644	401	78	na
	39	T9	TLM	RH	Jun–Jul 98	1-F	683	430	80	na
	40	T10	TLM	RH	Jun–Jul 98	1-F	595	360	79	na
	41	T11	TLM	BB	Jun–Jul 98	1-F	723	460	78	na
	42	T12	TLM	BB	Jun–Jul 98	1-F	709	410	80	na
	43	T13	TLM	SSRA	Jun–Jul 98	1-F	584	260	78	na
	44	T14	TLM	SSRA	Jun–Jul 98	1-F	607	240	80	na
	45	T15	TLM	SSRA	Jun–Jul 98	1-F	972	390	79	na
	46	T16	TLM	SSRA	Jun–Jul 98	1-F	581	350	78	na
	47	T17	TLM	SSRA	Jun–Jul 98	1-F	482	196	78	na
	48	T18	TLM	SSRA	Jun–Jul 98	1-F	595	236	78	na
	49	T19	TLM	SSRA	Jun–Jul 98	1-F	624	290	79	na
	50	T20	TLM	SSRA	Jun–Jul 98	1-F	553	250	79	na
	51	T21	TLM	SSRA	Jun–Jul 98	1-F	780	495	78	na
	52	T22	TLM	SSRA	Jun–Jul 98	1-F	695	410	78	na
	53	T23	TLM	SSRA	Jun–Jul 98	1-F	624	400	78	na
	54	T24	TLM	SSRA	Jun–Jul 98	1-F	680	405	78	na
GSPH (1999)	55	GS723-TIL1	TLM	NRD	26-May-99	1-F	480	306	na	na
	56	GS723-TIL2	TLM	NRD	26-May-99	1-F	474	299	na	na
	57	GS723-TIL4	TLM	NRD	26-May-99	1-F	426	298	na	na
	58	GS723-TIL5	TLM	NRD	26-May-99	1-F	575	319	na	na
	59	GS723-TIL6	TLM	NRD	26-May-99	1-F	463	294	na	na
	60	GS723-TIL7	TLM	NRD	26-May-99	1-F	441	298	na	na
	61	GS723-TIL9	TLM	NRD	26-May-99	1-F	427	302	na	na
	62	GS723-TIL10	TLM	NRD	26-May-99	1-F	433	301	na	na
	63	GS723-TIL12	TLM	NRD	26-May-99	1-F	409	302	na	na
	64	GS723-TIL13	TLM	NRD	26-May-99	1-F	430	310	na	na
	65	GS723-TIL15	TLM	NRD	26-May-99	1-F	450	303	na	na
	66	GS723-TIL16	TLM	NRD	26-May-99	1-F	430	304	na	na
	67	GS313-TIL9	TLM	SS-C	14-Apr-99	1-F	417	295	na	na
	68	GS313-TIL10	TLM	SS-C	14-Apr-99	1-F	498	312	na	na
	69	GS313-TIL11	TLM	SS-C	14-Apr-99	1-F	327	262	na	na
	70	GS313-TIL12	TLM	SS-C	14-Apr-99	1-F	375	279	na	na
	71	GS313-TIL13	TLM	SS-C	14-Apr-99	1-F	520	315	na	na
	72	GS313-TIL14	TLM	SS/C	14-Apr-99	1-F	398	284	na	na
	73	GS313-TIL15	TLM	SS/C	14-Apr-99	1-F	440	307	na	na
	74	GS223-TIL2	TLM	SS/N	12-Apr-99	1-F	387	283	na	na

Table 1 continued

Program ^a (date)	Sample number		Species ^b	Location ^c	Collection date	Sample size ^d – Tissue (W,F,L) ^e	Weight ^f (g)	Length ^f (mm)	%Water	%Lipid
	New	Original								
SSERG ^h (2000)	75	GS223-TIL4	TLM	SS/N	12-Apr-99	1-F	348	271	na	na
	76	GS223-TIL5	TLM	SS/N	12-Apr-99	1-F	319	268	na	na
	77	GS223-TIL6	TLM	SS/N	12-Apr-99	1-F	367	286	na	na
	78	GS223-TIL7	TLM	SS/N	12-Apr-99	1-F	324	255	na	na
	79	GS223-TIL8	TLM	SS/N	12-Apr-99	1-F	400	292	na	na
	80	GS223-TIL9	TLM	SS/N	12-Apr-99	1-F	390	279	na	na
	81	GS223-TIL10	TLM	SS/N	12-Apr-99	1-F	358	277	na	na
	82	GS223-TIL11	TLM	SS/N	12-Apr-99	1-F	447	290	na	na
	83	GS223-TIL12	TLM	SS/N	12-Apr-99	1-F	418	295	na	na
	84	580221-01	TLM	SSRA	21-Jul-00	1-W	500	na	78	2.4
SSERG (2000–01)	85	580221-02	TLM	SSRA	21-Jul-00	1-W	662	na	76	4.6
	86	580221-03	TLM	SSRA	21-Jul-00	1-W	550	na	80	1.7
	87	580221-04	TLM	SSRA	21-Jul-00	1-W	585	na	77	3.1
	88	580221-05	TLM	SSRA	21-Jul-00	1-W	740	na	72	5.0
	89	01-0632-FC1	TLM	WRD	6-Dec-00	8-W	731	349	71	6.2
	90	01-0633-FC2	TLM	ARD	6-Dec-00	8-W	708	345	70	6.2
	91	01-0634-FC3	TLM	SC/M	6-Dec-00	8-W	661	345	70	6.3
	92	01-0635-FC4	TLM	SSB	6-Dec-00	4-W	541	346	73	5.0
MASGC (2000)	93	01-0636-FC5	TLM	SS/C	6-Dec-00	7-W	674	329	72	5.1
	94	01-1373-FC2	TLM	ARD	13-May-01	8-W	649	336	73	5.0
	95	01-1374-FC2'	TLM	ARD	13-May-01	8-W	631	325	73	4.2
	96	01-1375-FC3	TLM	SC/M	13-May-01	8-W	511	324	71	3.6
	97	01-1376-FC4	TLM	SSB	13-May-01	8-W	754	328	75	5.7
	98	01-1377-FC5	TLM	SS/C	13-May-01	8-W	581	323	74	4.1
	99	None	TLM	SS/S	1-Apr-00	3-F	515 ^g	312 ^g	na	1.4
	100	None	TLM	S/RD	1-Apr-00	2-F	688	335	na	2.0
	101	TIL-AR-1	TLM	ARD	1-Sep-02	1-F	739	332	na	na
	102	TIL-AR-2	TLM	ARD	4-Oct-02	1-F	760	330	na	na
SSERG (2002)	103	TIL-AR-3	TLM	ARD	4-Oct-02	1-F	1052	370	na	na
	104	TIL-AR-4	TLM	ARD	4-Oct-02	1-F	832	350	na	na
	105	TIL-AR-5	TLM	ARD	4-Oct-02	1-F	610	315	na	na
	106	TIL-AR-6	TLM	ARD	4-Oct-02	1-F	636	315	na	na
	107	TIL-NR-1	TLM	NRD	21-Oct-02	1-F	1272	380	na	na
	108	TIL-LR-1	TLM	LR	22-Nov-02	1-F	988	350	na	na

Table 1 continued

na, not analyzed/not determined

^a Programs: TSMP = Toxic Substances Monitoring Program; USGS = U.S. Geological Survey; NFCRC = National Fisheries Contaminant Research Center; GSPH = Graduate School of Public Health, SDSU; SSERG = Salton Sea Ecosystem Research Group, SDSU; MASGC = Mississippi-Alabama Sea Grant Consortium

^b Species code: TL = Tilapia, species unspecified; TLM = Mozambique tilapia (*Oreochromis mossambicus*); TLZ = Redbelly tilapia (*O. zillii*)

^c Locations: ARD = Alamo River Delta; BB = Bombay Beach; LR = Lack Road; NRD = New River Delta; RH = Red Hill Marina; SC/M = Salt Creek Mouth; SS/C = Salton Sea Center; SS/N = Salton Sea North; SS/S = Salton Sea South; SS/WS = Salton Sea West Shores; S/RD = South/River Delta (both Alamo and New Rivers); SSB = Salton Sea Beach; SSRA = Salton Sea Recreation Area Headquarters; SSNR = Salton Sea National Refuge; WRD = Whitewater River Delta

^d Sample size: number of fish analyzed in composite or weight of composite

^e Tissue code: W = whole fish; F = fillet; L = liver

^f Average weight and length reported when sample sizes greater than 1. All length are total length except for the ones reported by TSMP which are fork lengths

^g Average weight and length determined for only 2 tilapia, but analyses done on samples composited from 3 fish

^h Erroneous lab results not reported here but submitted in a report to the Salton Sea Authority (Contract #CA 98099-01)

handling were in accordance with procedures described in the “Field Operations Manual for Resource Contaminant Assessment” of the U.S. Fish and Wildlife Services (Hickey et al., 1984). 20 g of homogenized whole fish (number of fish per composite sample not recorded) were analyzed for Ag, Ba, B, Cd, Cr, Cu, Hg, Mn, Mo, Ni, Pb, Tl, U, Vn and Zn using inductively coupled plasma-emission spectroscopy following a preconcentration treatment. Determinations of As and Se concentrations were made using hydride-generation atomic-absorption spectroscopic methods. Organochlorine pesticide residues were measured using gas/liquid chromatography. Samples were analyzed and quality control procedures were performed at contracted laboratories under the supervision of the Patuxent Analytical Control Facility, U.S. Fish and Wildlife Service, Maryland.

SDSU Graduate School of Public Health (GSPH), 1998–1999

Selenium analysis

For Se analysis, a total of 24 tilapia were collected at Red Hill Marina, Bombay Beach and at the State Recreational Area Headquarters during June and July 1998 (Surico-Bennett, 1999). Of the collected tilapia, five freshly dead fish were collected on the shores at the State Recreational Area (samples 31–35), three were caught with hook and line (38–40), and the others (36, 37, 41–54) were donated by fishermen (Table 1). The fish were weighed, measured (total length) and immediately placed on ice in a cooler and transported to the laboratory, and stored at -11°C until analysis. Fillet sample collected from each fish was analyzed individually. Approximately 500 g of muscle samples were filleted off each fish, and all scales, bones and skin were removed. Samples were then weighed, placed in a drying oven at 65°C overnight, weighed again and dry weight determined. After nitric acid digestion of the dried samples, Se concentrations were determined using a Perkin Elmer SIMAA 6000 Graphite Furnace Atomic Absorption Spectrometer with Zeeman background correction and an AS-72 furnace auto sampler.

Arsenic analysis

For determination of total As concentration, 29 tilapia were caught using gillnets at 3 different stations in the Salton Sea during April and May 1999 (Vicario-Fisher, 1999) (Table 1). The first station was located in the northern part of the lake ($33^{\circ}26' \text{N}$; $115^{\circ}56' \text{W}$; samples 74–83, Table 1), the second in the center of the lake ($33^{\circ}20' \text{N}$; $115^{\circ}50' \text{W}$; samples 67–73, Table 1), and the third near the outlet of the New River ($33^{\circ}08' \text{N}$; $115^{\circ}41.6' \text{W}$; samples 55–66, Table 1). Once caught, the fish were weighed, measured (total length), filleted and the middle portions of the dorsal fillet were placed in labeled Zip-Lock[®] one-gallon freezer storage bag and kept on ice until their arrival at San Diego State University where they were placed in a freezer at -11°C until analysis. Fresh tissue samples weighing 1.50 g were placed in labeled acid-washed petri dishes, and dried for 48 h at 118°C . Once dried, 0.20 g of each sample was transferred to a teflon bomb liner in a Parr Microwave Acid Digestion Bomb, and 5 ml of 35% nitric acid were added to the sample. The liner was then placed in the bomb shell, sealed, and microwaved for 30 sec. The samples were then allowed to cool before being transferred to coded polyethylene bottles for temporary storage. Total As concentrations were determined using a Perkin-Elmer SIMMA 6000 Graphite Furnace Atomic Absorption Spectrometer, with a detection limit of 5 ppb or ng g^{-1} dw.

Salton Sea Ecosystem Research Group (SSERG), 2000–2001

A total of 74 tilapia were collected from the Salton Sea using gillnets at 5 fixed stations in early December 2000 and late May 2001. These dates corresponded to the post- and pre-spawning periods, i.e. presumed low and high points of their lipid content. Coordinates of and water depth at the five stations sampled in December were: Whitewater delta (WRD), $\text{N } 33^{\circ}30.13' / \text{W } 116^{\circ}03.00'$, 5 m; Alamo delta (ARD), $\text{N } 33^{\circ}12.9' / \text{W } 115^{\circ}37.40'$; 6 m; Salt Creek mouth (SCM), $\text{N } 33^{\circ}27.13' / \text{W } 115^{\circ}51.35'$, 5 m; Salton Sea Beach (SSB), $\text{N } 33^{\circ}19.5' / \text{W } 115^{\circ}56.00'$, 4 m; center of lake (SS/C), $\text{N } 33^{\circ}18.01' / \text{W } 115^{\circ}47.9'$, 12 m.

Coordinates and depth of the stations sampled in May were: Whitewater delta, N 33°27.4'/W 116°02.7', 3.5 m; Alamo delta, N 33°16.7'/W 115°36.60'; 3 m; Salt Creek mouth, N 33°26.3'/W 115°50.9', 4 m; Salton Sea Beach N 33°19.5'/W 115°56.1', 3.5 m; center of lake N 33°15.7'/W 115°50.9', 12 m. The stations were geographically dispersed to represent the northern, southern, eastern, western and central parts of the lake. Fish were removed from nets, weighed, measured (total length), placed on ice in labeled coolers, returned to San Diego State University and kept at -15°C until being shipped on dry ice to the California Department of Fish and Game, Fish and Wildlife Water Pollution Control Laboratory (WPCL) in Rancho Cordova, California.

Composite samples consisted of six males and two females collected from the same location, with length of the smallest fish being at least 75% that of the longest. The December composite sample for SSB consisted of four fish only, three males and one female. Fish in each composite sample were homogenized together using a commercial meat grinder and Büchi homogenizer. Homogenates were then prepared for extraction (organic contaminants) or digestion (metals) as appropriate.

Subsamples for organic pesticides and herbicides were extracted and analyzed using gas chromatography utilizing an electron capture or other appropriate detector. Extraction methods employed were developed and validated by the Water Pollution Control Laboratory (WPCL, 1999). Extract cleanup and partitioning methods are modifications of the multi-residue methods for fatty and non-fatty foods provided by the U. S. FDA (1994).

WPCL sent frozen subsamples of the fish homogenates to the Marine Pollution Studies Laboratory (MPSL), Moss Landing Laboratory, California, for the elemental analyses. Subsamples for metal analyses were digested using a 4:1 mixture of nitric and perchloric acids. Analyses of Se, As, and Pb were done using ICP-MS, while Hg analysis was conducted using FIMS (Field Ionization Mass Spectrometry).

Nutritional properties of the fish homogenates were determined by Michelson Laboratories following official methods of analysis of the Asso-

ciation of Official Analytical Chemists (AOAC). The following characteristics were assessed: Total protein (Kjeldahl procedure; method AOAC 928.08); fat content (method AOAC 963.15); crude fiber (method AOCS Ba 6–84); ash (method AOAC 923.03); calcium, potassium and sodium (method EPA 200.7), phosphorus (method AOAC 962.02), sodium chloride (Volhard procedure, method AOAC 935.47).

Differences in contaminant levels and nutritive properties between the pre-spawning and post-spawning samples were assessed using paired *t*-tests, with samples paired by station.

Of the 8 tilapia collected for As speciation analyses, six were caught by the Alamo River delta on September 9 (one specimen) and October 4, 2002 (five specimens), one specimen was collected October 21, 2002 by the New River delta and the last one was collected by a fisherman off Lack Road on the southern end of the Sea on November 22, 2002. These fish were kept frozen at -15°C until preparation for analyses. The sex, total length and weight of each fish were determined. Approximately 100 g of skinned fillet tissue from each fish was removed, wrapped in aluminum foil, and placed in labeled double bags on dry ice. Samples were then shipped overnight to Frontier Geosciences Inc., Seattle, Washington. Each fish sample was then homogenized prior to total As and inorganic As analyses. Total As concentration was determined using ICP-MS (Inductively Coupled Plasma-Mass Spectrophotometry) after complete digestion of approximately 0.5 g of sample by concentrated nitric acid. Inorganic As concentration in each sample was determined using HG-CT-GC-AAS (Hydride Generation Cryogenic Trapping Gas Chromatography Atomic Absorption Spectrophotometry) after leaching the trace element from each sample with hydrogen chloride.

Mississippi–Alabama Sea Grant Consortium (MASGC), 2000

A total of five tilapia were caught with gillnets in the southern end of the lake: three from near-shore stations and one at each of the Alamo River and New River mouth. Trace elements (Ag, Al, As, Cd, Cr, Cu, Fe, Hg, Mn, Ni, Pb, Se, Sn, Zn)

and organic contaminants (aldrin, chlordane, chlorpyrifos, DDT isomers, endosulfan I, II and endosulfan sulfate, dieldrin, endrin, HCH isomers, heptachlor, heptachlor-epoxide, hexachlorobenzene, methoxychlor, mirex, *trans*- and *cis*-nonachlor, oxychlordane, pentachloroanisole, pentachlorobenzene, and tetrachlorobenzene isomers) as well as 31 PCBs congener (Table 3 in Riedel et al., 2002a) concentrations in three species of fish (tilapia, bairdiella, *Bairdiella icistia* Jordan and Gilbert, and orangemouth corvina, *Cynoscion xanthulus*, Jordan and Gilbert) found at the Salton Sea were analyzed at B&B Laboratories, College Station, Texas. Refer to Riedel et al. (2002a) for detailed field and laboratory analytical protocols.

Risk assessment methodologies

Several approaches were employed to assess the risks to humans and wildlife posed by contaminants in Salton Sea tilapia.

Arsenic and selenium

Due to the importance of the sportfishery at the Salton Sea, and the presence of contaminants in tilapia, human consumers may ingest levels of contaminant that cause adverse health effects. Therefore, consumption limits expressed in weekly intake rates and number of Salton Sea tilapia meals that could be safely consumed per month were determined for the two contaminants of potential concern for human health, Se and As.

We estimated risk-based consumption limits using recent data on Se (samples 31–54, Table 3) and As (total As: samples 55–83; total and inorganic As: samples 101–108; Table 2) concentrations in tilapia fillet and the most current parameters and guidelines suggested by the U.S. EPA in “Guidance for Assessing Chemical Contaminant Data for use in Fish Advisory” (2000a, 2000b). The chronic toxicity criteria, the reference dose (RfD) and, for As, cancer slope factor (CSF) were obtained from U.S. EPA Integrated Risk Information System (U.S.EPA, 2004). The risk-based consumption limits for Se and inorganic As were determined using the average and maximum concentrations estimated for tilapia fillets.

In accordance with the U.S.EPA (2000b), these risk-based consumption limits are estimates of the maximum daily consumption rates of contaminated fish that would not be expected to cause any adverse health effects in human consumers.

On the assumption that no other source of Se and As exists in the diet of consumers, the allowable daily consumption limits for which no adverse health effects are expected are determined using:

$$CR_{lim} = (RfD \times BW) \times C_m^{-1},$$

where CR_{lim} = maximum safe daily consumption rate of tilapia ($kg\ day^{-1}$), RfD = Reference dose for each contaminant ($mg\ kg^{-1}\ day^{-1}$), BW = average human body weight (kg), C_m = concentration of the contaminant in the edible portion of fish (in $mg\ kg^{-1}\ ww$). The RfD, is determined by the U.S.EPA, and is an estimate of a daily intake of a contaminant over a lifetime that would not be expected to cause adverse health effect (U.S.EPA, 2000b).

Human dietary exposure to Se and inorganic As is also determined by their concentrations in other foods, as well as drinking water in the case of inorganic As. Not taking into account additional intake of these trace elements from sources other than Salton Sea tilapia, would lead to acceptable consumption rates that would exceed the protective limits determined by the U.S.EPA. Therefore, estimates of daily intake of Se and As from the diet and tapwater were subtracted from the RfD in order to determine the remaining daily intake of Se and As that could be ingested through consumption of tilapia fillet. Accounting for additional sources of Se and As in the diet, generated a second, more conservative set of safe consumption rates for consumers of Salton Sea tilapia (Table 6). The procedures and values used for this were as follows:

Selenium: Estimates of daily Se intake for the U.S population range from 0.071 to 0.152 mg per day (ATSDR, 2001). We took the midpoint daily intake of 0.111 mg Se, converted it to $0.0016\ mg\ kg^{-1}\ day^{-1}$ for a 70 kg adult, and subtracted this from the RfD ($0.005\ mg\ kg^{-1}\ d^{-1}$) to obtain an estimate of the daily intake of Se from Salton Sea tilapia that would still be safe. This

Table 2 Total and inorganic arsenic concentrations in Salton Sea tilapia. Concentrations and detection limits (DL) are reported in μg (ww: wet weight; dw: dry weight;

F: fillet; L: liver; W: whole fish). GM = geometric mean; – = not analyzed; pre = pre-spawning; post = post-spawning. See Table 1 for provenance of samples

Program, date, sample number and tissue type	Total As		Inorganic As		Program, date, sample number and tissue type	Total As		Inorganic As	
	ww	Dw	ww	dw		ww	dw	ww	dw
TSMP (1980–2000)					78-F	0.75	3.50	0.002	0.011
DL	0.05	–	–	–	79-F	0.51	2.39	0.002	0.007
10-F	0.64	3.06	–	–	80-F	0.80	3.70	0.002	0.011
10-L	1.03	4.29	–	–	81-F	1.90	8.82	0.006	0.027
11-F	1.90	9.50	–	–	82-F	0.73	3.41	0.002	0.011
11-L	0.85	4.01	–	–	83-F	0.68	3.15	0.002	0.010
12-F	1.31	5.75	–	–	GM	1.04	4.83	0.003	0.015
12-L	0.70	2.26	–	–					
13-F	1.13	4.93	–	–	SSERG /WPCL (2000–01) ^d				
14-F	1.42	5.84	–	–	DL	0.01	–	–	–
GM-L	0.85	3.39	–	–	89-W	1.41	4.88	–	–
GM-F	1.21	5.45	–	–	90-W	0.91	3.06	–	–
			–	–	91-W	1.29	4.25	–	–
NFRC (1985)					92-W	1.42	5.16	–	–
DL	0.05	–	–	–	93-W	1.52	5.49	–	–
15-W	0.83	2.64	–	–	GM (post)	1.29	4.48	–	–
16-W	0.99	3.06	–	–					
17-W	0.84	2.62	–	–	94-W	0.93	3.43	–	–
GM	0.88	2.77	–	–	95-W	1.26	4.72	–	–
			–	–	96-W	1.25	4.34	–	–
USGS (1986) ^a					97-W	1.46	5.72	–	–
DL	–	0.5	–	–	98-W	1.29	5.01	–	–
24-W	0.57	2.60	–	–	GM (pre)	1.23	4.58	–	–
25-W	0.24	1.10	–	–	P-values^e	0.69	0.87	–	–
26-W	0.26	1.20	–	–					
27-W	0.18	0.84	–	–	SSERG /FG (2000–01) ^e				
28-W	0.29	1.30	–	–	DL	0.5	–	0.002	–
29-W	0.29	1.30	–	–	90A-W	1.00	4.65	0.033	0.15
30-W	0.57	2.60	–	–	91A-W	1.15	5.35	0.038	0.18
GM	0.31	1.43	–	–	92A-W	1.23	5.72	0.038	0.18
			–	–	93A-W	1.21	5.63	0.047	0.22
GSPH (1999) ^{a,b}					GM	1.14	5.32	0.039	0.18
DL	–	0.005	–	–					
55-F	2.73	12.70	0.008	0.039	94A-W	0.8	3.72	0.039	0.18
56-F	1.24	5.78	0.004	0.018	96A-W	1.11	5.16	0.029	0.13
57-F	1.67	7.76	0.005	0.024	97A-W	1.4	6.51	0.034	0.16
58-F	1.08	5.03	0.003	0.016	98A-W	1.17	5.44	0.034	0.16
59-F	1.55	7.21	0.005	0.022	GM	1.10	5.11	0.034	0.16
60-F	1.27	5.91	0.004	0.018					
61-F	1.31	6.07	0.004	0.019	MASGS (2000) ^a				
62-F	1.16	5.38	0.004	0.017	DL	0.15	–	–	–
63-F	0.69	3.19	0.002	0.010	99-F	1.19	5.4	–	–
64-F	1.05	4.88	0.003	0.015	100-F	1.14	5.2	–	–
65-F	1.43	6.67	0.004	0.021	GM	1.16	5.29	–	–
66-F	1.72	8.01	0.005	0.025					
67-F	0.58	2.69	0.002	0.008	SSERG (2002) ^a				
68-F	0.46	2.14	0.001	0.007	DL	0.5	–	0.002	–
69-F	0.83	3.85	0.003	0.012	101-F	2.6	12.09	0.002	0.009
70-F	1.45	6.73	0.004	0.021	102-F	2.28	10.60	0.004	0.019
71-F	0.97	4.49	0.003	0.014	103-F	4.61	21.44	0.003	0.014
72-F	1.32	6.13	0.004	0.019	104-F	1.06	4.93	0.009	0.042
73-F	0.66	3.08	0.002	0.010	105-F	1.81	8.42	0.008	0.037
74-F	0.65	3.03	0.002	0.009	106-F	3.98	18.51	0.008	0.037

Table 2 continued

Program, date, sample number and tissue type	Total As		Inorganic As		Program, date, sample number and tissue type	Total As		Inorganic As	
	ww	Dw	ww	dw		ww	dw	ww	dw
75-F	0.78	3.65	0.002	0.011	107-F	2.24	10.42	0.011	0.051
76-F	1.55	7.23	0.005	0.022	108-F	1.33	6.19	0.009	0.042
77-F	1.50	6.98	0.005	0.022	GM	2.23	10.39	0.006	0.031

^a Concentration converted to wet or dry weight assuming a moisture content of 78.5% (average of 24 fillet samples) or 72.7% (average of 18 whole fish samples)

^b Values given here for inorganic As are calculated values, not measured ones. They are based on the assumption that inorganic As comprised 0.31% of total As, as determined directly to be the case for samples 101–108

^c *P*-values are for paired *t*-test comparing GM for pre-spawning (samples 94–98) and post-spawning (samples 89–93) seasons

^d Analyses performed by WPCL = Water Pollution Control Laboratories

^e Analyses performed by FG = Frontier Geosciences of most of same samples (89–98) analyzed earlier by WPCL

estimate was then used to determine the safe weekly consumption rate (Table 6).

Arsenic: MacIntosh et al. (1997) estimated that the mean daily intake of inorganic As for the U.S. population was 10.22 µg per day. Subsequent estimates of daily dietary intake of inorganic As have ranged from 1 to 20 µg per day (Schoof et al., 1999a, b). Therefore, we used a midpoint daily intake of 10 µg, converted it to 0.00014 mg kg⁻¹ day⁻¹ for a 70 kg adult, and used this as an estimate of the daily dietary intake of inorganic As. We also assumed that the vast majority of recreational anglers fishing at the Salton Sea live in southern California. Therefore concentrations of inorganic As present in tap water delivered to residents of the Coachella Valley, Imperial Valley, San Diego, and Los Angeles areas were obtained from the most recent water quality monitoring reports generated by water treatment facilities. Levels of As in tap water were at or below 2 µg l⁻¹ for the cities of San Diego (CSDWD, 2002) and El Centro (CEC, 2002), and averaged 3.5 µg l⁻¹ for the Los Angeles area (LADWP, 2002). The average for the Coachella Valley (CVWD, 2002) was 2.2 µg l⁻¹. Highest concentrations were for water supplies of a few communities along the eastern side of the Salton Sea, averaging 18 µg l⁻¹ for the communities of Mecca, Bombay Beach, North Shores and Hot Mineral Spa. We assumed a mean As level in tap water of 4 µg l⁻¹. Tap water intake rates were assumed to be 1.4 l d⁻¹ for adults and 0.74 l d⁻¹ for children (U.S.EPA, 1997). Consequently, the estimated inorganic As intake from tap water is

0.08 µg kg⁻¹ d⁻¹ for adults and 0.1 µg kg⁻¹ d⁻¹ for children, assuming body weights of 70 and 30 kg, respectively.

Estimates of intake through food items and tap water were then subtracted from the U.S.EPA RfD (0.0003 mg kg⁻¹ d⁻¹) to obtain an estimate of the safe daily intake of inorganic As from Salton Sea tilapia. This daily intake was then converted to a weekly intake as above.

Inorganic As is also classified as a known human carcinogen (U.S.EPA, 2000b). The maximum safe daily consumption rate for a carcinogenic contaminant is given by:

$$CR_{lim} = (ARL \times BW) \times (CSF \times C_m)^{-1},$$

where CR_{lim} = maximum safe consumption rate (kg fish d⁻¹), ARL = maximum acceptable individual lifetime risk level (set at 10⁻⁵) (unitless), BW = consumer body weight (set at 70 kg), CSF = cancer slope factor for inorganic As (1.5 mg kg⁻¹ d⁻¹), C_m = concentration of inorganic As measured in Salton Sea tilapia. The ARL represents an arbitrary risk level corresponding to one additional case of cancer per 100,000 individuals over a 70-year lifetime. The consumption limit, CR_{lim}, is a rough estimate of the amount of Salton Sea tilapia that would have to be consumed daily for 70 years in order to increase one's cancer risk by 1 chance in 100,000.

For each contaminant, two concentrations values were used when computing the safe consumption rates: the geometric mean and the

Table 3 Selenium concentrations in Salton Sea tilapia. Concentrations and detection limits (DL) are reported in $\mu\text{g g}^{-1}$ (ww: wet weight; dw: dry weight). GM = geometric mean; – = not analyzed; F = fillet; L = liver; pre = pre-spawning; post = post-spawning. See Table 1 for provenance of samples

	Program, date, sample number and tissue type	ww	dw	Program, date, sample number and tissue type	ww	dw
	TSMP (1980–2000)			GSPH (1998)		
	DL	0.2–0.05	–	35-F	1.71	7.42
	3-F	1.70	7.26	36-F	1.80	8.45
	3-L	3.90	15.00	37-F	1.66	7.79
	4-F	2.00	9.10 ^a	38-F	1.58	7.20
	5-F	3.00	14.56	39-F	1.43	7.26
	6-F	3.20	26.02	40-F	2.06	9.86
	7-F	2.90	14.01	41-F	1.93	8.87
	8-F	3.60	16.51	42-F	1.45	7.15
	9-F	3.20	14.10	43-F	1.67	7.74
	10-F	1.31	6.20	44-F	1.71	8.36
	10-L	6.65	27.90	45-F	1.96	9.13
	11-F	2.73	11.87	46-F	1.90	8.71
	11-L	6.27	20.23	47-F	1.87	8.65
	12-F	2.20	9.65	48-F	1.56	7.16
	12-L	4.15	13.39	49-F	1.01	4.8
	13-F	2.60	11.30	50-F	1.27	6.05
	14-F	2.70	11.00	51-F	1.73	8.05
	GM-F	2.50	11.79	52-F	1.90	8.70
	GM-L	5.10	18.35	53-F	1.72	7.90
				54-F	1.43	6.71
	NFRC (1985)			GM	1.67	7.8
	DL	0.05	–			
	15-W	2.10	6.7	SSERG/WPCL (2000-01)		
	16-W	4.50	13.9	DL	0.02	–
	17-W	2.30	7.3	89-W	2.31	7.99
	GM	2.80	8.8	90-W	1.90	6.40
				91-W	2.05	6.77
	USGS (1986) ^a			92-W	2.44	8.89
	DL	–	0.50	93-W	2.17	7.83
	24-W	1.39	6.3	GM (post)	2.17	7.52
	25-W	0.77	3.5			
	26-W	3.74	17.0	94-W	1.91	7.05
	27-W	0.95	4.3	95-W	2.15	8.05
	28-W	3.08	14.0	96-W	2.14	7.43
	29-W	2.64	12.0	97-W	2.13	8.35
	30-W	3.74	17.0	98-W	2.27	8.83
	GM	1.97	9.0	GM (pre)	2.12	7.92
				P-values^c	0.72	0.47
	GSPH (1998) ^a					
	DL	0.001	–	MASGS (2000) ^a		
	31-F	1.84	8.19	DL	0.5	–
	32-F	1.72	8.14	99-F	2.39	10.9
	33-F	1.69	8.55	100-F	1.89	8.6
	34-F	1.81	8.24	GM	2.12	9.7

maximum concentration observed. The values used for Se were 1.67 and 2.06 $\mu\text{g g}^{-1}$ ww respectively (samples 31–54; Table 3), and for inorganic As were 0.006 and 0.011 $\mu\text{g g}^{-1}$, respectively (samples 101–108; Table 2). These daily consumption limits were multiplied by 7 to obtain a safe weekly intake (CR^*_{lim}), and then converted to the number of monthly fish meals using:

$$\text{CR}_{\text{mm}} = (\text{CR}^*_{\text{lim}} \times T_{\text{ap}}) \times \text{MS}^{-1},$$

where CR_{mm} = maximum allowable tilapia consumption rate (meals month⁻¹), CR^*_{lim} = maximum weekly consumption rate of tilapia (kg week⁻¹), T_{ap} = time averaging period (4.3 week month⁻¹), MS = meal size, 227 g (8 oz) for adults and 114 g (4 oz) for children (U.S.EPA, 2000b).

Other contaminants

Of the 25 contaminants the U.S.EPA recommends be tested for in fish fillet when carrying a fish health advisory, Cr, Cd, Zn, and total DDT (tDDT) were detected in tilapia samples (Tables 4, 5). Mean concentrations were compared to screening values (SV) proposed by the U.S.EPA (2000a), to assess whether their levels were high enough to generate risk based consumption limits. The purpose of the SVs is to give state and local agencies reference contaminant concentrations against which concentrations in locally caught fish can be compared during the initial phase of a fish and shellfish monitoring program. SVs are concentrations of contaminants in edible tissues of fish or shellfish for which there could be public health concern (U.S.EPA, 2000a). For contaminant concentrations analyzed in fish collected from the field not in exceedance of SVs, then no additional monitoring or human health risk assessment needs to be undertaken until a later screening study is carried out (U.S.EPA, 2000a). If a target contaminant level is, however, in exceedance of the proposed SV, then more intensive sampling needs to be done in order to assess the magnitude of the contamination problem and potential ramifications for human health. SVs are computed by the U.S.EPA for recreational and subsistence anglers assuming daily consumption rates of 17.5 g and 142.2 g of fish, respectively. Based on those consumption rates, the U.S.EPA recommends, for example, a SV for Cd of 4.0 mg kg^{-1} for recreational anglers and 0.50 mg kg^{-1} for subsistence fishers. If Cd concentrations in tilapia fillet are lower than the SVs, then adverse health effects are unlikely to be observed.

Wildlife risk assessment

Trace element and organic contaminant concentrations in whole tilapia were compared to maximum level guidelines for wildlife protection, and to information on effects on fish and birds reported in the ecotoxicological literature.

Results

Elemental and pesticide concentrations, as well as moisture and lipid content when available, are reported for 108 samples of tilapia (Tables 1–5). Se, As and DDT congeners were detected in all of the samples analyzed. Results for individual elements and organochlorine residues are discussed below. All means reported in our tables and referred to in the text are geometric means. Values below the detection limit in datasets were replaced by the detection limit prior to log transformation, and resulting means for such datasets are reported as “less than”.

Arsenic

Whole body and fillet analyses

As was found to be a trace element of concern in the tilapia from the Salton Sea. Average whole body As concentrations ranged from 0.84 to $5.49 \text{ } \mu\text{g g}^{-1} \text{ dw}$ (Table 2, samples 15–30, 89–98). Three samples analyzed by NFRC obtained by the mouth of the New River had a mean of $2.77 \text{ } \mu\text{g g}^{-1} \text{ dw}$ (Table 2, samples 15–17). Samples collected by USGS near the mouth of the Whitewater River (samples 24,25), New River (samples 26,27) and Alamo River (28–30) had means of 1.68, 1.00 and $1.64 \text{ } \mu\text{g g}^{-1} \text{ dw}$, respectively (Table 2).

Higher As concentrations are expected in whole fish homogenates than in fillets, as As was found to be sequestered mostly in various organs such as the brain, ovaries, intestine, gill and liver in fish such as tilapia, green sunfish (*Lepomis cyanellus*, Rafinesque), and lake whitefish (*Coregonus clupeaformis*, Mitchill) (Sorensen et al., 1979; Suhendrayatna et al., 2001; 2002; Pedlar & Klaverkamp, 2002). Yet mean fillet concentration (samples 55–83, 99–108, Table 2) was $5.68 \text{ } \mu\text{g g}^{-1} \text{ dw}$ while mean body concentration (samples 15–17, 24–30, 89–98, 90A–98A, Table 2) was $2.90 \text{ } \mu\text{g g}^{-1} \text{ dw}$. Comparing fillet and whole body As concentrations obtained by the same analytical laboratory confirms higher As concentrations in fillet with a mean of $10.39 \text{ } \mu\text{g g}^{-1} \text{ dw}$ (samples 101–108; Table 2) than in whole body

Table 4 Trace element concentrations (in $\mu\text{g g}^{-1}$ ww) in Salton Sea tilapia. Detection limits (DL) are reported when known. F = fillet; L = liver; GM = geometric mean; na = not analyzed; pre = pre-spawning; post = post-spawning. For data set containing non-detected values,

the GM was computed by replacing the non-detected values by the detection limit and reporting the GM as below the computed mean. See Table 1 for provenance of samples

Program, date, sample number and tissue type	B	Cd	Cr	Cu	Fe	Hg	Mg	Mn	Mo	NI	Pb	V	Zn
TSMP (1997–2000) DL	na	0.002	0.001	0.02	na	0.001	na	na	na	0.001	0.0003	na	0.10
10-F	na	<0.002	<0.001	0.13	na	0.003	na	na	na	0.003	<0.0003	na	2.30
10-L	na	0.032	0.302	1.26	na	na	na	na	na	0.305	0.032	na	25.2
12-F	na	<0.002	0.084	0.28	na	<0.001	na	na	na	0.007	<0.0003	na	4.70
12-L	na	0.012	0.158	0.974	na	na	na	na	na	0.148	0.015	na	17.3
13-F	na	<0.002	na	na	na	<0.001	na	na	na	0.011	na	na	na
14-F	na	<0.002	na	na	na	<0.001	na	na	na	0.017	na	na	na
GM-F	na	<0.002	<0.01	0.19	na	<0.001	na	na	na	0.008	<0.0003	na	3.29
GM-L	na	0.022	0.22	1.11	na	na	na	na	na	0.21	0.02	na	21.0
NFCRC (1985) DL	1.0	0.06	na	1.00	2.00	0.02	na	na	0.20	0.06	0.20	0.04	1.00
15-W	6.60	<0.06	na	5.10	161	<0.02	na	na	<.20	0.25	2.50	1.10	15.1
16-W	4.30	<0.06	na	0.75	159	<0.02	na	na	<.20	0.32	<0.20	0.80	18.7
17-W	4.70	<0.06	na	4.10	83.2	<0.02	na	na	0.66	0.20	0.20	<0.04	18.0
GM	5.11	<0.06	na	2.50	129	<0.02	na	na	<0.30	0.25	<0.46	<0.33	17.2
USG ^a (1986) DL	4.40	0.09	1.76	0.44	na	na	na	na	0.9	0.7	1.76	0.88	na
24-W	<4.40	0.11	<1.76	1.25	156	0.05	1100	26.40	<0.90	1.03	<1.76	3.52	11.0
25-W	<4.40	<0.09	<1.76	<0.44	123	0.04	68.2	2.64	<0.90	<0.70	1.76	<0.88	1.91
26-W	4.40	<0.09	<1.76	<0.44	174	0.03	440	15.84	<0.90	<0.70	na	1.45	9.90
27-W	4.84	0.10	<1.76	1.72	77.0	0.04	264	8.58	<0.90	<0.70	na	<0.88	5.50
28-W	4.62	0.09	<1.76	<0.44	46.2	0.04	308	3.74	<0.90	<0.70	<1.76	<0.88	18.3
29-W	<4.40	0.09	<1.76	<0.44	57.2	0.05	396	2.86	<0.90	<0.70	<1.76	1.32	16.9
30-W	9.24	<0.09	<1.76	0.55	41.8	0.04	308	3.08	<0.90	<0.70	<1.76	<0.88	9.02
GM	<4.99	<0.09	<1.76	<0.64	161.3	0.04	318	6.10	<0.90	<0.70	<1.76	<1.22	8.50
SSERG (2000–01) DL	na	0.001	0.01	0.005	na	0.02	na	na	na	0.003	0.005	na	0.01
89-W	na	0.003	0.47	0.40	na	<0.02	na	na	na	<0.003	0.04	na	19.1
90-W	na	0.003	0.38	0.38	na	<0.02	na	na	na	<0.003	0.03	na	20.5
91-W	na	0.003	0.41	0.39	na	<0.02	na	na	na	<0.003	0.07	na	21.9
92-W	na	0.003	0.73	0.47	na	<0.02	na	na	na	<0.003	0.09	na	21.9
93-W	na	0.002	0.42	0.38	na	<0.02	na	na	na	<0.003	0.08	na	22.0
GM (post)	na	0.003	0.47	0.40	na	<0.02	na	na	na	<0.003	0.05	na	21.1
94-W	na	0.003	0.60	0.43	na	<0.02	na	na	na	<0.003	0.09	na	20.4
95-W	na	0.003	0.43	0.42	na	<0.02	na	na	na	<0.003	0.07	na	20.4
96-W	na	0.003	0.43	0.41	na	<0.02	na	na	na	<0.003	0.07	na	19.2
97-W	na	0.003	0.40	0.45	na	<0.02	na	na	na	<0.003	0.04	na	21.6
98-W	na	0.004	0.62	0.94	na	<0.02	na	na	na	<0.003	0.06	na	20.3
GM (pre)	na	0.003	0.49	0.50	na	<0.02	na	na	na	<0.003	0.06	na	20.4
P-values^b	na	0.37	0.89	0.32	na	na	na	na	na	na	0.83	na	0.37
MASGC (2000) DL	na	0.10	0.2	0.09	na	0.002	na	na	na	0.10	0.11	na	0.20
99-F	na	<0.10	0.03	0.22	6.17	<0.002	na	1.77	na	0.06	0.05	na	13.0
100-F	na	0.18	0.21	0.64	22.7	<0.002	na	1.13	na	0.95	0.03	na	12.2
GM	na	<0.13	0.08	0.38	11.8	<0.002	na	1.41	na	0.24	0.04	na	12.6

^a Concentrations were originally reported on a dry weight basis and have been converted to wet weight by assuming a moisture content on 78.5% (average of 24 fillet samples)

^b *P*-values are for paired *t*-test comparing GM for pre-spawning (samples 94–98) and post-spawning (samples 89–93) seasons

homogenates with a mean of $5.21 \mu\text{g g}^{-1}$ dw (samples 90A–98A; Table 2, *t*-test, *P* = 0.01).

Although total As was found to be substantially higher in tilapia fillet than in whole fish

samples, the fraction of inorganic As was 10 times higher in whole tilapia samples (2.9%) than in fillet (0.31%) (samples 90A–98A and 101–108, respectively, Table 2). These results suggest that

Table 5 Concentrations of DDT residues (in $\mu\text{g g}^{-1}$ ww) in Salton Sea tilapia. Detection limits (DL) are reported when known. GM = geometric mean; na = not analyzed/

not available; pre = pre-spawning; post = post-spawning. See Table 1 for provenance of samples

Program, date, sample number and tissue type		p,p' -DDE	p,p' -DDD	Total DDT	Program, date, sample number and tissue type		p,p' -DDE	p,p' -DDD	Total DDT
TSMP (1980–00)	DL	0.005	0.01	na	SSERG (2000–01)	DL	0.002	0.002	na
	1-F	0.028	<0.01	0.028		89-W	0.09	0.003	0.09
	2-F	0.020	<0.01	0.020		90-W	0.11	0.003	0.11
	3-F	0.077	<0.01	0.077		91-W	0.10	0.003	0.11
	6-F	0.005	<0.01	0.005		92-W	0.10	0.003	0.10
	7-F	0.006	<0.01	0.006		93-W	0.06	<0.002	0.06
	8-F	0.018	<0.01	0.018		GM (post)	0.09	<0.003	0.09
	9-F	0.012	<0.01	0.012		94-W	0.11	0.003	0.12
	10-F	0.031	<0.01	0.031		95-W	0.08	0.002	0.08
	11-F	0.007	<0.01	0.007		96-W	0.06	<0.002	0.06
	12-F	0.036	<0.01	0.036		97-W	0.09	0.003	0.09
	13-F	0.012	<0.01	0.012		98-W	0.08	0.002	0.08
	14-F	0.018	<0.01	0.018		GM (pre)	0.08	<0.002	0.08
	GM	0.017	<0.01	0.017		P-values^a	0.60	0.70	0.62
USGS(1986)	DL	0.01	0.01	na	MASGC (2000)	DL	0.0001	0.0001	-
	18-W	0.35	<0.01	0.35		99-F	0.04	0.001	0.04
	19-W	0.20	<0.01	0.20		100-F	0.13	0.005	0.14
	20-W	0.37	0.36	0.76		GM	0.07	0.003	0.07
	21-W	0.23	0.08	0.34					
	GM	0.28	<0.04	0.37					

^a *P*-values are for paired *t*-test comparing GM for pre-spawning (samples 94–98) and post-spawning (samples 89–93) seasons

organs play an important role in sequestering inorganic As and that the majority of the As present in tilapia fillet is organic.

Spatial and temporal variations

A conclusive interpretation of spatio-temporal trends of As and Se levels in Salton Sea tilapia is hindered by several factors. These include information that was not recorded by all investigators (i.e. age and reproductive stage of the tilapia collected, exact sampling location), difference in sample processing protocol, improvements over time in analytical methods and unknown movement patterns of the tilapia in the Salton Sea. However, general speculations can be brought forward.

Overall, As concentrations in whole tilapia were lower in 1985–1986 than in 2001. The average in the mid-1980s was $1.75 \mu\text{g g}^{-1}$ dw (samples 15–17, 24–30) compared to $4.53 \mu\text{g g}^{-1}$ dw in 2001 (samples 89–98) (Table 2). This could possibly indicate that As levels are increasing in Salton

Sea fish. Great caution is advisable, however, when comparing data obtained by different laboratories at different times.

In 1999, mean fillet As concentration of tilapia collected from the New River delta (samples 55–66, Table 2) was $6.21 \mu\text{g g}^{-1}$ dw, almost twice as high as the As levels for fish collected from the center of the lake (samples 67–73; mean: $3.84 \mu\text{g g}^{-1}$ dw) or from the northern station (samples 74–83; mean: $4.18 \mu\text{g g}^{-1}$ dw). This increase is possibly due to the geothermal activity at the southern end or to higher levels of As being present in the rivers that flow into the southern end basin of the Salton Sea. Analyses of water samples collected between 1986 and 1989 reported levels of total As averaging $5.2 \mu\text{g l}^{-1}$ by the Alamo River outlet and $8.04 \mu\text{g l}^{-1}$ by the New River outlet (Setmire et al., 1990; Schroeder et al., 1993). In 2001, slightly higher concentrations were detected in tilapia collected from Salton Sea Beach ($5.43 \mu\text{g g}^{-1}$ dw, samples 92 and 97) and the center of the lake (samples 93 and 98; mean: $5.24 \mu\text{g g}^{-1}$ dw) than in fish collected near

the Alamo River mouth (samples 90,94 and 95; mean: $3.67 \mu\text{g g}^{-1} \text{dw}$).

EDLs

Levels of As in Salton Sea tilapia are high relative to those in fish collected from other bodies of water in California. Elevated Data Levels (EDLs) were introduced in 1983 by the TSMP as arbitrary comparative standards for contaminant concentrations in fish collected from polluted waters of California. Cumulative frequency distributions and percentiles are obtained for specific contaminants once all measurements of their individual concentrations in specific fish and tissue types are ranked from the highest to not detected (Rasmussen & Blethrow, 1990). EDL 85 and EDL 95 are the concentrations of a contaminant below which are 85% and 95% of all TSMP records of that contaminant in similar fish and tissue types for a specific period of time. These values do not imply that toxic effects occur at those levels but only that toxicant levels in the Salton Sea are relatively high compared to those in other waterbodies in California. Compared to the EDLs reported for freshwater fish fillet (computed from 133 samples) and freshwater whole fish (computed from 170 samples) for the 1978–1995 TSMP data sets, all measured As concentrations in Salton Sea tilapia were higher than both the EDL 85 value of $0.14 \mu\text{g g}^{-1} \text{ww}$ for fillet and $0.41 \mu\text{g g}^{-1} \text{ww}$ for whole fish and the EDL 95 value of $0.43 \mu\text{g g}^{-1} \text{ww}$ for fillet and $0.88 \mu\text{g g}^{-1} \text{ww}$ for whole fish (Table 2).

Salton Sea tilapia concentrations were also elevated compared to the As levels reported by the National Contaminant Biomonitoring Program (Schmitt & Brumbaugh, 1990). Average As concentrations based on 3,249 fish samples collected from 117 rivers and lakes throughout the United States were 0.27, 0.16, 0.15, and $0.14 \mu\text{g g}^{-1} \text{ww}$ in 1976–77, 1978–79, 1980–81 and 1984, respectively (May & McKinney, 1981; Lowe et al., 1985; Schmitt & Brumbaugh, 1990). Furthermore, Schmitt & Brumbaugh (1990) reported elevated As concentrations (geometric mean of 3 composite samples: $0.93 \mu\text{g g}^{-1} \text{ww}$) in fish collected in 1984 from the Colorado River near Yuma, Arizona, presumably due to the application of

arsenical insecticides in the intensively cultivated regions of the lower Colorado River watershed.

Selenium

Whole body and fillet analyses

Whenever analyzed for, Se was always detected in Salton Sea tilapia. The average whole body concentrations ranged from 7.7 to $8.9 \mu\text{g g}^{-1} \text{dw}$ (samples 89–98 and 15–17 plus 24–41 respectively; Table 3), with a mean for all whole fish samples of $8.3 \mu\text{g g}^{-1} \text{dw}$. Average fillet concentrations ranged from 7.9 (samples 31–54, 99, 100) to $13.5 \mu\text{g g}^{-1} \text{dw}$ (samples 3–9), giving a mean of $9.0 \mu\text{g g}^{-1} \text{dw}$ for all fillet samples analyzed (Table 3).

Spatial and temporal variations

Average Se concentrations in whole tilapia were slightly lower in 2001 (samples 89–98; mean: $7.7 \mu\text{g g}^{-1} \text{dw}$) compared to the mid-1980s (samples 15–17, 24–30; mean: $8.9 \mu\text{g g}^{-1} \text{dw}$, Table 3).

Setmire et al. (1990) found that the highest Se concentrations in Salton Sea fish, including tilapia, were obtained from samples collected by the three river mouths (samples 24–30), which are directly affected by agricultural drain waters. In contrast, we detected highest Se levels (samples 89–98) in fish collected near Salton Sea Beach (samples 92, 97; mean: $8.6 \mu\text{g g}^{-1} \text{dw}$) and the center of the lake (samples 93, 98; mean: $8.3 \mu\text{g g}^{-1} \text{dw}$). Fish collected close to the mouth of the Alamo River had an average Se level of $7.1 \mu\text{g g}^{-1} \text{dw}$ (samples 90, 94, 95; Table 3), half that of samples collected by USGS at the Alamo River outlet (mean = $14.2 \mu\text{g g}^{-1} \text{dw}$, samples 28–30; Table 3). It is possible that tilapia samples analyzed by USGS were collected closer to the Alamo River mouth than were ours, or that Se levels in the rivers have decreased since the late 1980s. USGS reported much higher Se concentrations in Alamo River and New River water samples ($8 \mu\text{g l}^{-1}$ and $4 \mu\text{g l}^{-1}$, respectively) than in Salton Sea composite water samples ($1 \mu\text{g l}^{-1}$) (Setmire et al., 1990; Schroeder et al., 1993). Se levels in

river inflows were slightly lower in samples analyzed in 1999, when concentrations averaged $5.8 \mu\text{g l}^{-1}$ and $3.8 \mu\text{g l}^{-1}$ or the Alamo and New rivers, respectively (Huston et al., 2000).

EDLs

Following observation of high embryonic deformity and mortality rates of waterfowl found at Kesterson National Wildlife Refuge catastrophe in 1983 due to Se toxicosis, the TSMP has routinely analyzed Se concentrations in all fish fillet samples collected in California (Rasmussen & Blethrow, 1990). Compared to the concentrations reported for fillet (566 samples) and whole body (194 samples) samples of fresh water fish collected between 1978 and 1995, all mean values for Salton Sea tilapia fillet and whole body Se concentrations are higher than the EDL 95 of $1.80 \mu\text{g g}^{-1}$ ww (fillet) and $1.90 \mu\text{g g}^{-1}$ ww (whole) except for the concentrations measured by GSPH (1999) (samples 31–54; mean: $1.7 \mu\text{g g}^{-1}$ ww; Table 3).

Metals

Other trace elements were analyzed in tilapia whole body samples but none were found at elevated levels (Table 4). When detected, B, Cd, Cr, Cu, Fe, Hg, Mg, Mn, Ni, Pb, Vn, and Zn were at levels below those likely to have adverse impact on aquatic wildlife (Setmire et al., 1990). Despite a thorough literature search, no direct evidence of chronic poisoning of wildlife exposed to the metals analyzed was found. Only two fillet samples were analyzed for metals (samples 99–100, Table 4), and the levels were generally lower than those obtained for whole body samples.

Only for Cr did Salton Sea tilapia concentrations (samples 89–98; mean: $0.48 \mu\text{g g}^{-1}$ ww) exceed the EDL 85 value, which for Cr was determined as $0.23 \mu\text{g g}^{-1}$ ww from 167–174 whole fish samples collected between 1978 and 1995 in California (Rasmussen, 1997).

Furthermore, Saiki (1990) found that B, Cd, Cu, Hg, Fe, Mo, Vn and Zn levels in Salton Sea fish, including tilapia (samples 15–17), were similar to or slightly lower than levels measured in

whole body samples of common carp (*Cyprinus carpio*, Linnaeus), largemouth bass (*Micropterus salmoides*, Lacepède) and striped mullet (*Mugil cephalus*, Linnaeus) collected from the lower Colorado River (Radtke et al., 1988; Schmitt & Brumbaugh, 1990).

Pesticides and polychlorinated biphenyls (PCBs)

Elevated concentrations of pesticides were expected to be present in tilapia as the lake receives its water from drains and rivers impacted by agricultural drainwaters. However, besides DDT and its congeners, none of the pesticides analyzed for were detected at elevated concentrations in tilapia samples from the Salton Sea. Additional organic compounds that were detected included the herbicide DCPA, PCBs and a few organochlorine pesticides detected at low concentrations (Rasmussen & Blethrow, 1990, 1991; Rasmussen, 1993, 1997; Riedel et al., 2002a).

DCPA (dimethyl tetrachloroterephthalate, Dacthal®) is a chlorinated benzoic acid compound used as a general herbicide. It was detected above detection levels in 4 of the 14 samples analyzed by TSMP, with a mean of $1.44 \mu\text{g g}^{-1}$ ww (samples 1,6,7,12). DCPA was also detected in one other sample in 2000 at a level just above the detection limit (sample 90; $0.0022 \mu\text{g g}^{-1}$ ww).

Riedel et al. (Table 2; 2002a) detected 18 and 22 organochlorine pesticides in fillets of tilapia collected at two stations near the southwestern and southeastern shores and at two off the Alamo and New river mouths. Means for the two nearshore and for the two river mouth stations were reported. Excluding dieldrin and DDT congeners, concentrations of organochlorine pesticides reported above detection limit ranged from 0.0001 to $0.0027 \mu\text{g g}^{-1}$ ww ($n = 17$ compounds) in nearshore samples, and 0.0001 to $0.0025 \mu\text{g g}^{-1}$ ww ($n = 21$ compounds) for river mouth samples. Mean dieldrin concentration was $0.0012 \mu\text{g g}^{-1}$ ww for nearshore samples and $0.0035 \mu\text{g g}^{-1}$ ww for river mouth samples (samples 99–100; Table 5), approximately 10 times higher than the mean concentration of the other organochlorine pesticides.

Several PCB congeners were also detected in samples collected in 2000–2001, with total concen-

trations of $0.004 \mu\text{g g}^{-1}$ ww and $0.009 \mu\text{g g}^{-1}$ ww for nearshore and river mouth samples, respectively. Riedel et al., (2002a) concluded that levels were not elevated enough to be of concern for wildlife, but that additional analyses were recommended. PCBs were also analyzed in samples collected by SSERG and TSMP in recent years (samples 89–98 and 13,14, respectively), but in all these samples, concentrations were below the detection limits of $0.05 \mu\text{g g}^{-1}$ ww.

The most persistent DDT metabolite, DDE, was detected in all samples collected, while DDD was detected in only 52% of them (samples 20–21, 89–92, 94, 95, 97, 98; Table 5). Mean concentration of DDE for fillet homogenates was $0.02 \mu\text{g g}^{-1}$ ww (samples 1–14, 99–100; Table 5), while mean values for whole fish homogenates were higher, ranging from $0.02 \mu\text{g g}^{-1}$ ww to $0.34 \mu\text{g g}^{-1}$ ww (samples 18–21; Table 5). Total DDT concentrations may have decreased in the Salton Sea tilapia population, paralleling an overall downward trend observed since 1976 in fish collected from 20 rivers and lakes in the U.S. (Schmitt et al., 1985, 1990). Elevated concentrations are expected in whole fish homogenates as organochlorine pesticides are preferentially sequestered in fatty tissues and organs that are not included in fish fillets. Total DDT concentrations in whole tilapia averaged $0.37 \mu\text{g g}^{-1}$ ww in 1986, but only $0.08 \mu\text{g g}^{-1}$ ww in 2000, a decline of 78% (samples 18–21 and 89–98; Table 5). For the tilapia collected in 1986, DDE constituted the major fraction of DDT isomers detected, the DDD fraction never exceeding 23.5% of total DDT.

Compared to the EDLs reported for freshwater fish fillets (202 samples) collected between 1978 and 1995, the concentrations of DDT isomers in tilapia were lower than the EDL 85 of 1.57 and $2.39 \mu\text{g g}^{-1}$ ww for DDE and total DDT, respectively (Rasmussen, 1997).

Human consumption limits

For each contaminant and for potential cancer and non-cancer effects, the amount of tilapia that can be safely consumed will be different. Safe consumption rates specified in health advisories for the general public will be based on the contaminant posing greatest risk. We present safe consumption rates estimated using the two most

extensive data sets for fillet concentrations of As and Se (samples 31–83; Table 6). Estimates are given using both the mean and maximum observed concentrations of each.

Arsenic

Because only inorganic As is toxic and potentially carcinogenic to human consumers (U.S.EPA, 2000b), risk-based consumption limits of Salton Sea tilapia were determined using recent As speciation analyses carried on skinned fillet collected from the Salton Sea, where inorganic As averaged 0.31% of the total As concentration (Table 2, samples 101–108, footnote b). This is lower than the inorganic As content of edible marine fish, which averages 2% of total As (Edmonds & Francesconi, 1993).

If we assume no additional intake of inorganic As through other foods or drinking water, even effectively unlimited consumption of tilapia would be unlikely to cause non-cancer adverse health effects in adults and children (Table 6). Taking into account additional intakes of inorganic As at a total rate of $0.08 \mu\text{g kg}^{-1} \text{d}^{-1}$ for adults and $0.1 \mu\text{g kg}^{-1} \text{d}^{-1}$ for children based on a dietary intake of $10 \mu\text{g d}^{-1}$ and inorganic As concentration in tap water of $4 \mu\text{g l}^{-1}$ decreases the safe weekly consumption rate of tilapia 5- and 6-fold for children and adults, respectively. Nonetheless, the maximum consumption rate of tilapia unlikely to cause non-cancer adverse health effects in adult and child consumers is still more than one meal of tilapia per day (Table 6).

With regards to carcinogenic effects, however, one additional cancer case in 100,000 people exposed would be expected with a consumption rate during a 70 year period of 540 g (19 oz) or 300 g (10.6 oz) of tilapia per week, based on the average and maximum concentrations of inorganic As, respectively (Table 6). In other words, eating for 70 years 10 meals per month of tilapia fillet with an average inorganic As concentration of $0.006 \mu\text{g g}^{-1}$ ww could result in one additional person diagnosed with cancer out of 100,000. The same risk would be incurred by consumption of 6 meals per month if the inorganic As concentration in tilapia fillet was $0.011 \mu\text{g g}^{-1}$ ww (sample

Table 6 Maximum dietary intake of Se and As on a weekly basis and safe consumption rates for children and adults consuming Salton Sea tilapia

Contaminant	Se				Inorganic As					
	Health effects				Non-cancer				Cancer	
	Mean		Maximum		Mean		Maximum		Mean	Maximum
Tilapia fillet concentration ($\mu\text{g g}^{-1}$)(ww) ^a	1.67		2.06		0.006		0.011		0.006	0.011
Daily intake unit per body weight from sources other than tilapia ($\mu\text{g g}^{-1} \text{d}^{-1}$) ^b	0	0.0016	0	0.0016	0	0.00008 ^c	0	0.00008 ^c	–	–
						0.000024 ^d		0.00024 ^d		
Safe weekly consumption rate of tilapia (g week^{-1}) ^e					10500	2100	5730	1150	–	–
Consumer					24500	6530	13360	3560	540	300
Child – 30 kg	630	430	510	350						
Adult – 70 kg	1470	1000	1190	810						
Safe number of meals of tilapia per month ^e					401	80	218	43	–	–
Consumer					469	121	256	68	10	6
Child – 30 kg ^f	24	16	19	13						
Adult – 70 kg ^g	28	19	23	15						

^a Concentrations for Se from samples 31–54 (Table 3), and for As, from samples 55–83 and 101–108 (Table 2)

^b Additional intake due to presence of inorganic As in food items other than tilapia and in drinking tap water

^c Daily intake for adults assuming a water drinking rate of 1.4 l day⁻¹ (EPA, 1997)

^d Daily intake for children assuming a water drinking rate of 0.74 l day⁻¹ (EPA, 1997)

^e Safe levels are those that would not cause adverse health effects of a non-cancerous nature or that would not cause more than one additional case of cancer per 100,000 persons exposed population for 70 years (see text)

^f Meal size for children is 114 g (4 oz)

^g Meal size for adults is 227 g (8 oz)

107; Table 2), the maximum observed in the SSERG data set (Table 6).

Selenium

Consideration only of health risks posed by selenium leads to higher estimates of safe tilapia consumption rates. With an average Se concentration in tilapia fillet of 1.67 $\mu\text{g g}^{-1}$ ww and assuming a background daily Se intake per unit body weight from other food sources of 0.0016 $\mu\text{g g}^{-1}$, adults consuming 1000 g or less of tilapia per week, the equivalent of 19 meals (each 227 g or 8 oz) per month, are unlikely to experience adverse health effects. A decrease in maximum safe consumption rate of only 15%, to 15 meals per month, is obtained when the maximum Se concentration observed in fillet (sample 40; Table 3) is used instead of the average concentration.

If the same daily intake of Se from other food sources can be assumed for children, then a weekly consumption of 510 g (18 oz) or less of tilapia fillet is unlikely to cause adverse health

effects. Therefore, children could eat up to 19 meals (each 114 g or 4 oz) per month of tilapia fillet without exceeding the reference dose set by the U.S.EPA, based on the average Se concentration of the fish edible tissue, or up to 13 meals based on the maximum detected concentration.

DDT and its metabolites, DDE and DDD

The SVs for tDDT for non-cancer health effects are 2.00 and 0.25 $\mu\text{g g}^{-1}$ for recreational and subsistence anglers, respectively, based on a daily consumption rate of 17.5 g of fish for recreational anglers and 142.2 g for subsistence anglers (U.S.EPA, 2000a). Levels of tDDT in 2000–2001 averaged 0.032 $\mu\text{g g}^{-1}$ ww in tilapia filets (samples 13–14, 99–100; Table 5). These levels do not present a risk of non-cancer effects for either type of consumer, although the small sample size precludes a reliable conclusion.

DDT and its metabolites are classified as “probable human carcinogen”, based on animal carcinogenicity data (U.S.EPA, 1985a). The SVs for tDDT for cancer health effects are 0.117 and

0.0144 $\mu\text{g g}^{-1}$ for recreational and subsistence anglers respectively (U.S.EPA, 2000a). Based on U.S.EPA risk assessment guidelines, an increase in cancer risk from tDDT exposure of 1 in 100,000 might be incurred by anglers consuming 450 g (16 oz) of tilapia per week, the equivalent of 8 meals per month, for 70 years. Additional analyses of tDDT in tilapia fillet are necessary as the contaminant concentration in this assessment was determined from a small sample size ($n = 4$).

Nutritive properties

Whole tilapia are comprised of approximately 722 $\mu\text{g g}^{-1}$ (72.2%) moisture and have a lipid content of 51 $\mu\text{g g}^{-1}$ (5.1%) on a wet weight basis (average of samples 84–93, Tables 1, 7). Of dry weight, ash constitutes 21.3% and organic matter for the remaining 78.7%. The protein content of whole body tilapia was approximately 61% dw, based on 9.8% nitrogen dw (Table 7), and did not show any change with the reproductive status of the fish. Pre-spawning tilapia had a lipid content 25% lower than that of post-spawning fish, possibly reflecting the lipid depletion in pre-spawning fish that were allocating energy to gamete formation. Crude fiber was also lower in pre-spawning fish by 28.2%. Moisture, sodium (Na) and potassium (K) were 2.8, 34.3 and 60.4% higher, respectively, in pre-spawning fish than in post-spawning fish. Ash, Ca, phosphorus and NaCl showed no clear change with the reproductive status of the fish.

Discussion

Because the Sea has been used as an agricultural wastewater depository for almost a century, concerns have been expressed regarding the impact of contaminants on human consumers, fish and birds. Our results suggest that tilapia from the Salton Sea are, in general, safe for humans to eat, that As levels are of more concern than Se levels, and that further evaluation of risks posed by tDDT and PCBs is desirable. These conclusions are, however, dependent upon the parameters chosen in our assessment procedures [*Human risk*

assessment assumptions and uncertainties below]. For the tilapia population and piscivorous birds utilizing the Salton Sea, Se is a greater threat than As, other trace elements or pesticides. Discussion of these findings follows.

Arsenic concentrations set limits to human consumption

Arsenic levels in tilapia do not pose a risk of non-cancer health effects to human consumers based on the RfD determined by the U.S.EPA, and the levels of inorganic As present in tilapia fillets. Adults and children should be able to eat more than one meal of tilapia per day without risking adverse non-cancer health effects, even when considering additional intakes of inorganic As from other foods and tap water, assuming an average inorganic As concentration in water of 0.004 mg l^{-1} (Table 6). However, analyses of tap water delivered to residents of Mecca, Bombay Beach, North Shore and Hot Mineral Spa averaged 0.018 mg l^{-1} and ranged from 0.014 to 0.027 mg l^{-1} (CVWD, 2002). Assuming a daily intake of tap water of 1.4 l and an average adult body weight of 70 kg, the average intake of inorganic As from tap water is approximately 0.0004 $\mu\text{g g}^{-1} \text{d}^{-1}$ for residents of the aforementioned areas. This intake alone exceeds the daily intake considered safe of 0.0003 $\mu\text{g g}^{-1} \text{d}^{-1}$ (U.S.EPA, 2002). When the tilapia fishery is productive, subsistence fishing may be common among the residents of Mecca, Bombay Beach and North Shores.

Consideration of the carcinogenic potential of inorganic As leads to lower estimates of safe tilapia consumption rates, 6 to 10 meals per month, based on the maximum and average concentrations, respectively (Table 6). An adult can consume 10 meals of tilapia per month without incurring an increased risk of cancer greater than 1 in 100,000 (Table 6). Nevertheless, this is 10 times the maximum consumption rate recommended in the 1986 health advisory based on Se levels. Additional inorganic As analyses of tilapia might be carried out by the State to verify the consumption rates estimated here. These risk assessments would benefit from more accurate estimates of daily inorganic As

Table 7 Nutritional characteristics of whole body tilapia homogenates. See Table 1 for sample provenance. All values are reported as percent (g 100g⁻¹) dry weight

Date and sample number	Moisture ^a	Lipid ^b	Protein	Crude fiber	Ash	Ca	N	P	Na	K	NaCl	
Post-spawning (Dec-00)	89	71.1	20.1	58.3	3.8	19.1	5.9	9.3	3.5	0.7	0.6	2.2
	90	70.3	20.6	60.7	3.7	20.4	6.4	9.7	4.1	0.7	0.5	2.3
	91	69.7	19.2	57.1	2.3	17.9	6.9	9.2	2.8	0.6	0.5	1.6
	92	72.5	19.0	64.4	4.9	26.0	9.0	10.3	5.0	0.6	0.5	2.4
	93	72.3	21.4	65.3	3.6	27.1	8.2	10.5	3.2	0.8	0.6	2.5
	GM	71.2	20.0	61.2	3.6	21.8	5.9	9.8	3.6	0.7	0.5	2.2
Pre-spawning (May-01)	94	72.9	16.6	60.5	3.4	22.9	6.6	9.7	3.0	0.9	0.8	2.0
	95	73.3	13.8	60.2	2.7	16.1	6.7	9.6	3.3	0.9	0.8	2.3
	96	71.2	10.9	56.4	1.7	19.9	6.0	9.2	3.0	1.0	1.0	2.3
	97	74.5	21.2	66.4	3.5	24.9	6.4	10.5	2.5	0.9	0.9	3.1
	98	74.3	14.0	62.1	2.2	21.5	7.1	9.9	2.7	0.9	0.9	2.1
	GM	73.2	14.9	61.1	2.6	20.9	6.6	9.8	2.9	0.9	0.9	2.4
Percent change^c		+2.8	-25.5	-0.1	-28.2	-4.3	+11.9	-0.1	-20.4	+34.3	+60.4	+7.8
P values^d		0.001	0.07	0.97	0.009	0.59	0.46	0.91	0.14	0.01	0.002	0.49

^a Moisture content reported by WPCL

^b Mean of lipid values independently reported by WPCL and Michelson laboratories

^c Percent change (of geometric means) from post-spawning period (December) to pre-spawning period (May)

^d *P*-values are for paired *t*-test comparing GMs for pre-spawning (samples 94–98) and post-spawning (samples 89–93) seasons

intakes from other foods and tap water by the subpopulations consuming Salton Sea fish in largest quantities.

Selenium not a problem for human consumers

The present advisory (http://www.oehha.org/fish/so_cal/saltonsea.html) regarding consumption of fish caught from the Salton Sea was first issued in the mid-1980s and has not been revised since. The data and calculations on which it was based are no longer available (M. Gassel, OEHHA, pers. comm.), although a newspaper report stated that it was based on analyses done for the California Water Resources Control Board that showed that “selenium levels of 3.8 ppm in seven gulf croaker, 3.6 in six orangemouth corvina, 2.1 ppm in six sargo and 1.7 ppm in five tilapia” (Swanson, 1986). The State still recommends that no more than 114 g (4 oz) of any fish caught from the Salton Sea be consumed over a 2 week period, which translates to 227 g (8 oz) per month. Our re-evaluation suggests that adults could safely consume tilapia at approximately 10 times that rate. Furthermore, adults would have to consume approximately 6.5 times this amount before exceeding the lowest dose for which signs

of Se toxicity would be observed. This dose, the lowest observable adverse effect level (LOAEL), has been set at 0.023 μg g⁻¹ d⁻¹ (U.S.EPA, 2002) based on a human epidemiological study (Yang et al., 1989).

The assumptions used in the original state advisory recommendation were based on scientific knowledge of human Se dietary requirements, acceptable daily intake and toxicity threshold available 20 years ago. Selection of additional variables used in the advisory, such as risk values, additional sources of exposure, if considered, average consumer body weight and meal size, was also contingent upon decisions made by the U.S.EPA and state agencies. The sampling and analytical protocols used, along with the number of fish analyzed, were also important factors in the determination of the advisory.

However, assumptions in the risk assessment of the original advisory have changed since the mid 1980s, and, in light of new information, a revision should be considered. The informal advice we offer here can hopefully prompt the California Office of Environmental Health Hazard Assessment to carry out further sampling and assessments, bringing about the

development of a more accurate advisory based on As, Se, tDDT and PCB values, for all sport fish species present in the Salton Sea. Risk-based consumption limits for Se-contaminated tilapia were not determined from a cancer risk assessment, as evidence of carcinogenic effects is lacking (U.S.EPA, 1985b).

Other trace elements not a concern for human consumers

Based on the RfDs used by the U.S.EPA, maximum concentrations of Cd, Cr, and Zn concentrations measured in Salton Sea tilapia samples were 63, 99 and 85%, respectively, lower than the SVs proposed by the U.S.EPA for a consumer eating daily 142 g (5 oz) of recreationally caught fish. This suggests that from the public health perspective, these trace elements are not a concern. Lead was detected in Salton Sea tilapia samples at concentrations of 0.03 to 0.09 $\mu\text{g g}^{-1}$ (samples 89–98, Table 4). Because of the lack of a dose-response threshold below which health effects are not experienced, the U.S.EPA (2002b) has not determined a RfD or SV to which Pb concentrations in Salton Sea tilapia could be compared. However, all measured Pb concentrations in Salton Sea tilapia were lower than the EDL 85 value of 1.4 $\mu\text{g g}^{-1}$ ww for freshwater whole fish (174 samples) in the 1978–1995 TSMF data sets.

Additional analyses of tDDT and PCBs are needed

Due to the paucity of recent tDDT ($n = 4$, samples 13–14, 99–100, Table 5) and PCBs ($n = 5$; Table 4 in Riedel et al., 2002a) analyses of Salton Sea tilapia fillets, the health risk potentially incurred by anglers from tDDT and PCBs cannot be reliably ascertained. Total DDT levels in tilapia do not exceed the SVs of 2.0 and 0.245 $\mu\text{g g}^{-1}$ issued by the U.S.EPA (2000a) as guidelines for non-carcinogenic effects for recreational and subsistence anglers consuming daily 17.1 g (0.6 oz) and 142 g (5 oz) of caught fish, respectively. They do exceed the SV of 0.014 $\mu\text{g g}^{-1}$ recommended for protection against carcinogenic health effects for those anglers consuming more

than 142 g of tilapia daily or four 8-oz (227 g) meals per week during their lifetime.

PCBs may also be of concern for human health (Riedel et al., 2002a), with levels of PCBs ranging from 0.003 to 0.011 $\mu\text{g g}^{-1}$ ww, with a mean of means of 0.008 $\mu\text{g g}^{-1}$ (Table 4 in Riedel et al., 2002a). These PCBs levels exceed the SVs of 0.0098 and 0.0025 $\mu\text{g g}^{-1}$ ww recommended by U.S. EPA (2000a) to protect anglers consuming 142 g (5 oz) daily of recreationally caught fish from adverse non-cancerous and cancerous health effects, respectively. Because of the potential human health concern, we recommend that additional PCBs analyses be done on tilapia and other fish present at the Salton Sea.

Although additional analyses of tDDT and PCBs are desirable to evaluate potential human health risks due to exposure to these contaminants, an advisory issued to protect anglers from cancer effects due to consumption of As-contaminated tilapia might be restrictive enough to protect anglers against adverse health effects due to exposure to tDDT and PCBs in Salton Sea tilapia.

Human risk assessment assumptions and uncertainties

The reliability and practical importance of these consumption limits are contingent upon several assumptions, parameters chosen, and other uncertainties associated with the risk assessment process. Some of the most crucial assumptions and uncertainties are those associated with the choice of parameter values that are used when computing risks associated with exposure to contaminants.

The choice of parameter values will influence estimates of safe consumption rates (Table 6). These include tap water drinking rate (1.4 l d⁻¹ for adults and 0.74 l d⁻¹ for children), weight (70 kg for adults and 30 kg for children), duration of exposure to contaminants with carcinogenic effects via consumption of Salton Sea tilapia (assumption of 70-year exposure), and the maximum acceptable cancer risk over a lifetime (1 in 100,000 over 70 years). These parameters were selected as they are utilized as default values by the U.S.EPA in their “Guidance for Assessing Chemical Contaminant Data for Use in Fish

Advisory” (U.S. EPA, 2002a, b), with the exception of water drinking rate, which was provided in the U.S.EPA “Exposure Factors Handbook” (1997). An underestimate of the risk incurred will be obtained for adults and children drinking more or weighing less, or both, than assumed by the parameters chosen. Other risk assessors may choose different parameters, resulting in different acceptable fish consumption rates. Furthermore, consumption limits are based on contaminant concentrations in skinned tilapia fillets. Analyses of contaminants in fillets with skin on may yield different acceptable consumption rates.

Sources of uncertainty in the determination of the present consumption limits include sampling and analytical variability, different protocols used by different laboratories over the years, and fish population heterogeneity. Other sources of uncertainty inherent in the risk assessment process are associated with exposure parameters and toxicity criteria factors. Uncertainty in exposure factors include the selection of the a daily intake of As and Se via water and food sources other than tilapia; these were averages for the U.S. population, except for the intake of inorganic As via drinking tap water, which was the average level present in tap water for these communities near the Salton Sea. Actual daily intake via these other routes by Salton Sea fish consumers may deviate considerably from the estimates used. Furthermore, it was assumed that both children and adults have the same daily intake of As and Se. An estimate of the daily dietary and water intakes of Se and As for the targeted adult and child subpopulations would provide a more accurate estimate of the safe intake from Salton Sea fish. In addition, it is assumed that no portion of the contaminant is lost or magnified during the preparation and cooking processes, and that all the contaminant present in tilapia fillet is absorbed by the consumer.

The choice of toxicity criteria also introduces uncertainty in the results. For both Se and As, the chronic toxicity criteria, reference dose (RfD) and cancer slope factor (CSF) were obtained from U.S. EPA Integrated Risk Information System (U.S.EPA, 2004). With regards to As, a RfD of $0.0003 \text{ mg kg}^{-1} \text{ d}^{-1}$ is utilized by default

for inorganic As. However, U.S.EPA scientists consider that RfD values might range from 0.0001 to $0.0008 \text{ mg kg}^{-1} \text{ d}^{-1}$ (U.S.EPA, 2004). Ignoring additional intake from other food sources and tap water, this range of RfD values would translate into a safe weekly intake of tilapia ranging from ca. 3 times lower to 3 times greater than the ones computed with a RfD of $0.0003 \text{ mg kg}^{-1} \text{ d}^{-1}$, for both adults and children. If additional dietary sources of inorganic As are taken into account, the most conservative RfD would result in no safe weekly intake of Salton Sea tilapia, as the estimate of weekly intake would already exceed the RfD. Determination of the As carcinogenic risk parameter also introduces uncertainty. The carcinogenic risk is computed from the CSF derived from a study by Tseng et al. (1968) correlating inorganic As in drinking water and skin cancer as an end-point. It is assumed, but not confirmed, that the CSF for other types of cancers due to ingestion of inorganic As is the same as the one computed from the skin cancer study. Furthermore, the CSF is based on an assumed consumption of tilapia over a lifetime, or 70 years (EPA, 2000b). Finally, there is also ambiguity due to possible synergistic effects of contaminants.

Advice to Salton Sea anglers

Fish advisories are issued by the State of California when levels of contaminants detected in fish might present a risk to human consumers. These advisories are presented as a recommended meal frequency, often on a monthly basis, as encouragement to anglers to minimize their exposure to contaminants that could have adverse health effects over time. For contaminants potentially having carcinogenic effects, the acceptable or safe meal frequency is that estimated to cause an additional risk of 1 in 100,000 of developing cancer over a lifetime of regularly eating fish at the advisory level. That is, this risk is in addition to the background cancer risk. Currently, the risk from all causes of developing cancer by age 70 is approximately 20,000 in 100,000, or 1 in five persons (U.S.CSWG, 2003). Primary factors are lifestyle (e.g. smoking, diet) and hereditary factors. Although the cancer risk

from eating As-contaminated tilapia from the Salton Sea cannot be predicted with certainty, a monthly consumption of 10 meals, each approximately 8 oz (227 g), may not increase one's lifetime cancer risk at all, or it may increase it from roughly 20,000 in 100,000 to roughly 20,001 in 100,000.

In general, fish are a good source of protein and healthful fat. When consumed in moderation, tilapia from the Salton Sea may be more beneficial than detrimental to consumers' health. However, Se has been found to be embryotoxic and teratogenic in animal experiments (Goyer, 1996). Therefore, women who are pregnant or planning on becoming pregnant soon should minimize their exposure to Se by not eating fish caught at the Salton Sea

Arsenic, fish and piscivorous birds

Studies documenting adverse effects on fish and piscivorous birds due to chronic exposure to As are scarce, and most knowledge in this area is derived from laboratory studies. It has been established, however, that, with respect to their toxic potential to wildlife and humans, As (III) is more toxic than As (IV), and both are more toxic than any of the organoarsenic species such as arsenobetaine and arsenocholine, which are relatively non-toxic. Also, As metabolism and toxic endpoints vary greatly among species, developmental stages and environmental conditions (Eisler, 1994; Stemberger & Chen, 1998; Chen & Folt, 2000; Storelli & Marcotrigiano, 2000).

Inorganic As species are the prevalent forms in sediments and water, and are converted into organoarsenic compounds by phytoplankton, and metabolized through the food chain to arsenobetaine, the major organic species present in higher trophic levels (Hanaoka et al., 1992; Suhendrayatna & Maeda, 2001; Kirby & Maher, 2002). Foley et al. (1978) and Lowe et al. (1985) determined that environmental factors besides As concentrations in water column and prey items are important in explaining arsenic concentrations in fish tissues. Salinity, temperature and habitat type were also found to be correlated with the accumulation of As in marine fish species

(Norin et al., 1985), while As accumulation in fish found in lakes was correlated with total nitrogen in the water, dissolved organic carbon, and percent of watershed dedicated to agriculture (Chen & Folt, 2000; Chen et al., 2000). While As is known to bioaccumulate, it was not found to biomagnify in upper trophic level organisms (Eisler, 1994; Chen et al., 2000; Chen & Folt, 2000; Kirby & Maher, 2002).

In marine ecosystems, Hanaoka et al. (1992) observed that arsenobetaine and other organosugars accumulated in the tissues of live organisms, and were degraded back to inorganic As when fish were killed by bacteria of the *Vibrio* and *Aeromonas* group. We note that Salton Sea tilapia have suffered mortality due to bacterial infections, with dead and moribund fish showing external signs of bacterial septicemia. Isolated bacterial agents from the fish organs included *Vibrio alginolyticus*, *V. vulnificus*, *V. damsela* and *Pseudomonas putrefaciens* (Winton, 2003). In addition to the bacterial infections, botulism spores were isolated from the intestinal tract of diseased fish, apparently associating epizootic tilapia die offs with outbreaks of avian botulism (type C) in fish eating birds, notably pelicans (Winton, 2003)

The reported elevated As levels of about $1 \mu\text{g g}^{-1}$ ww in Salton Sea tilapia do not imply high toxicity risk to the tilapia or organisms feeding on the fish, as far as can be inferred from the literature. Average As tissue concentrations ranged from 0.001 to $0.4 \mu\text{g g}^{-1}$ ww for fish collected from uncontaminated water bodies (Lacayo et al., 1992), and up to $220 \mu\text{g g}^{-1}$ ww in fish heavily contaminated environments (Moore et al., 1983). Tilapia (*O. mossambicus*) collected from aquaculture ponds in an area in Taiwan where human blackfoot disease was reported, had an average fillet As concentrations of $3.55 \mu\text{g g}^{-1}$ dw (Liao et al., 2003), compared to an average of $0.36 \mu\text{g g}^{-1}$ dw (range $0.13\text{--}1.45 \mu\text{g g}^{-1}$ dw) measured in tilapia fillets sold on the market in Taipei, Taiwan (Han et al., 1998). Blackfoot disease incidences were found to be correlated to long-term exposure to As in drinking water supplied from artesian wells in Taiwan (Chen et al., 2001). While artesian well water is no longer used for drinking water in those blackfoot disease areas,

well water is still used for aquaculture operations (Liao et al., 2003). Although As concentrations measured in Salton Sea water ($9 \mu\text{g l}^{-1}$; Schroeder et al, 1993; $1.55\text{--}9.95 \mu\text{g l}^{-1}$; Holdren & Montaña, 2002) are lower than those in the aquaculture ponds in Taiwan (18 to $49 \mu\text{g l}^{-1}$; Liao et al., 2003), As levels in fillet of Salton Sea tilapia ($5.7 \mu\text{g g}^{-1}$ dw; average for samples 55–83 and 101–108; Table 2) are elevated compared to those in tilapia fillet from aquaculture ponds in Taiwan. These results may relate to differences in age of fish, food type and abiotic factors driving the bioavailability of As in the Salton Sea and aquaculture ponds.

As concentrations in fish collected in brackish water polluted by effluents from a copper smelter, ranged from 0.42 to $2.6 \mu\text{g g}^{-1}$ ww, while fish from unpolluted areas in the same region had concentrations ranging from 0.14 to $1.2 \mu\text{g g}^{-1}$ ww (Norin et al., 1985). No field monitoring investigations were found relating high As levels in either water or organisms to adverse impacts on fish endpoints, i.e. mortality, growth or reproduction.

A few long-term investigations relating biological endpoints measured in fish to water or dietary As uptake have been conducted. McGeechay & Dixon (1990) investigated the effects of temperature on the chronic toxicity of sodium arsenate to fingerling rainbow trout (*Oncorhynchus mykiss* Gilberti). Whole-body residues in surviving juveniles exposed at 5°C for 77 days to sodium arsenate concentrations of 36mg l^{-1} (24mg As l^{-1}) in water were $2\text{--}3 \mu\text{g g}^{-1}$ ww, while dead fish had body burdens above $5 \mu\text{g g}^{-1}$ ww. In contrast, juveniles exposed at 15°C for the same duration and the same water concentrations had similar whole body residues but neither growth nor survivorship was affected, demonstrating the different toxicokinetics of As with varying water temperature regimes. Gilderhus (1966) exposed adults and juvenile green sunfish (*Lepomis cyanellus* Rafinesque) to As water concentrations of $2.31\text{--}11.4 \text{mg l}^{-1}$ for 112 days in large outdoor pools. Reduced survivorship and growth were observed in adults with whole body As concentrations of $11.6 \mu\text{g g}^{-1}$ ww, but no changes in either survivorship or growth were observed with a body concentration of $5.5 \mu\text{g g}^{-1}$ ww. Growth

and survivorship of juveniles were decreased at As body concentrations of 2.2 to $11.7 \mu\text{g g}^{-1}$ ww.

If low temperature increases As toxicity and given that Salton Sea water temperature during winter (average 13°C , Watts et al., 2001) is close to the lowest tolerable temperature for most tilapia species ($10\text{--}11^\circ\text{C}$, Popma & Masser, 1999), adverse effects in winter of As on Salton Sea tilapia with whole body concentrations of $1 \mu\text{g g}^{-1}$ ww might occur.

A relation between the concentration of As in tilapia and potential adverse effects this might cause to piscivorous birds cannot be established. We know of no field studies relating As concentration in food items and toxicological endpoints, including reproduction endpoints, observed in birds feeding on those food items. Toxicity criteria relevant to wildlife were reported by Sample et al. (1996). These wildlife criteria are based on contaminant concentrations in food items of wild birds extrapolated from experimentally derived concentrations for which no adverse effects (NOAEL) were observed. Also reported is the lowest dietary concentration for which adverse effects (LOAEL) were observed in experimental birds. Both NOAEL and LOAEL were determined from dietary As concentrations administered as sodium arsenite to mallard ducks (*Anas platyrhynchos* Linnaeus) (U.S. Fish and Wildlife Service, 1964). The toxicological endpoint assessed was mortality at 128 days of exposure. Using a factor to adjust for the difference in body size, NOAEL and LOAEL values for great blue heron (*Ardea herodias* Linnaeus) and osprey (*Pandion halietus*, Linnaeus) were estimated. These two piscivorous species are found at the Salton Sea. Food-based NOAELs for mallard, great blue heron and osprey were 51 , 29 and $26 \mu\text{g l}^{-1}$ dw, respectively, and LOAELs were 128 , 73 and $64 \mu\text{g l}^{-1}$ dw, respectively (Sample et al., 1996). These NOAELs and LOAELs are approximately 6 and 12 times the As concentration in whole tilapia homogenate samples (samples 89–98; Table 2). Based on these derived concentrations, mortality of great blue herons and ospreys present at the Salton Sea is unlikely to occur as a result of As exposure. However, differences in species sensitivity, and the potential interaction of As with other contaminants or

environmental stressors make the impact of dietary As on birds feeding on Salton Sea tilapia difficult to ascertain.

Selenium, fish and piscivorous birds

Since the collapse of the fishery at Belews Lake, North Carolina, in the mid 1970s and the high avian deformity and mortality rates observed at Kesterson National Wildlife Refuge (KNWR) in the San Joaquin Valley of Central California, between 1983 and 1985 due to Se toxicosis, Se has been recognized as a serious environmental pollutant (Cumbie & Van Horn, 1978; Lemly, 1985, 1993a, 2002; Ohlendorf, 1989). Similar problems of impaired reproduction, embryonic teratogenesis and reduced hatchability were also reported for birds utilizing evaporation ponds in the Tulare Lake Basin, California (Skorupa & Ohlendorf, 1991). Paradoxically, Se is also an essential trace element for aquatic organisms, but concentrations required are just about one order of magnitude lower than those associated with toxic effects in fish (Hilton et al., 1980; Hodson & Hilton, 1983; U.S.EPA, 1998). Given similarities between the Salton Sea and Kesterson ecosystems, including high abundance of resident and migratory birds, arid climate and dependence on agricultural wastewaters, Se has long been a contaminant of concern at the Salton Sea.

Based on the information obtained from the Se poisoning event of Belews Lake, the U.S.EPA (1987) lowered the maximum permissible concentrations of waterborne Se of from $35 \mu\text{g l}^{-1}$ to $5 \mu\text{g l}^{-1}$ for long-term exposure in order to increase protection of fish and other aquatic organisms (Skorupa, 1998; Hamilton & Lemly, 1999). Due to the propensity of Se to bioaccumulate through the food chain to toxic dietary concentrations, the criterion for chronic exposure of $5 \mu\text{g l}^{-1}$ of waterborne Se for the protection of freshwater organisms may be inadequate (Hamilton & Lemly, 1999; Hamilton, 2002). Waterborne concentrations of Se below the recommended $5 \mu\text{g l}^{-1}$ criterion were found to have adverse impacts on fish and bird communities subjected to agricultural runoffs in the western United States (Ohlendorf et al., 1986; Barnum & Gilmer, 1988; Saiki, 1990; Skorupa & Ohlendorf, 1991).

Se entering an aquatic system is taken up by algae and ultimately reaches the fish and avian species through the food chain (Lemly & Smith, 1987; Lemly et al., 1993; Luoma et al., 1992; Maier et al., 1993). Se uptake through the food chain is believed to have led to Se-induced reproductive failure of sensitive fish population present in reservoirs of the eastern United States, bringing about their decline or extinction (Cumbie & Van Horn, 1978; Lemly, 1985, 1993b; Gillespie & Baumann, 1986).

In sensitive fish species, such as salmonids, toxic effects were observed when whole body Se concentrations ranged from $2 \mu\text{g g}^{-1}$ dw (hematological changes, reduced growth) to $18 \mu\text{g g}^{-1}$ dw (impaired reproduction, mortality) (Hodson et al., 1980, 1984; Ogle & Knight, 1989; Hamilton et al., 1990; Saiki et al., 1992). Non-sensitive fish species chronically exposed to sublethal Se concentrations developed histopathological changes in liver, kidney, gill and ovarian tissues as well as decreased growth, edema, hematological anomalies, reproductive failure, embryonic deformities and mortality (Sorensen et al., 1983, 1984; Gillespie & Bauman, 1986; Sorensen, 1988; Ogle & Knight, 1989; Lemly, 1993b).

Lemly (1996a) reviewed the literature and concluded that Se levels higher than $4 \mu\text{g g}^{-1}$ dw for whole body and $8 \mu\text{g g}^{-1}$ dw for fillets may impair the health and reproductive success of freshwater and anadromous fish, and recommended that these levels be established as concentrations of concern. Levels in Salton Sea tilapia reported in the present study, $8 \mu\text{g g}^{-1}$ dw for whole body and $8\text{--}10 \mu\text{g g}^{-1}$ dw for fillet, are at or above these threshold values. The terata associated with Se contamination have not been observed, however, in the numerous collections of adult and juvenile tilapia made by our group at the Salton Sea. Nonetheless, the tilapia population at the Salton Sea has suffered heavy mortality since 2000 and was unable to successfully recruit most of the years since 1995 (Riedel et al., 2002b, Caskey et al., 2007). Other factors such as anoxia, rising salinity, hydrogen sulfide events and microbial parasites (Kuperman et al., 2001; Watts et al., 2001; Holdren & Montano, 2002) probably are greater threats to the health and survivorship of the Salton Sea tilapia population than is Se.

Se may increase the susceptibility of tilapia and other fish species to such stress factors, however. Substantial tilapia die-offs have often occurred in late winter at the Salton Sea, and low water temperature may be a potential factor. By mid-winter, water temperature drops to 13–14°C at mid lake (Watts et al., 2001) and occasionally lower in shallow nearshore areas.

Lemly (1993c; 1996b) introduced the term Winter Stress Syndrome (WSS) to describe a condition of metabolic stress experienced by warm water fish. This disorder results from prolonged exposure of fish to low temperatures in the presence of an additional external stressor that increases metabolic demand and oxygen consumption. During warmer months, fish compensate for this metabolic increase with increased feeding. However, prolonged exposure to cold temperature results in a decrease in feeding activity, resulting in an impaired capacity to counteract the impacts of the stressor.

WSS was first demonstrated in juvenile of warm-water bluegills (*Lepomis macrochirus* Rafinesque) exposed to dietary and waterborne Se (5.1 $\mu\text{g g}^{-1}$ in food and 2 $\mu\text{g l}^{-1}$ in water) (Lemly, 1993c). After 4 months, a cumulative mortality of 33% was observed for the juveniles exposed to Se and low temperature (4°C), compared to a cumulative mortality of 2.5% for the juveniles exposed to low temperature and no Se. Furthermore, total body lipids and body condition factors of juveniles in the low temperature-high Se treatment were decreased by 45 and 50%, respectively, compared to the other treatments. Hematological effects were also observed in juveniles exposed to Se. A similar synergism may affect Salton Sea juvenile tilapia.

Diseases have killed large numbers of birds connected with the Salton Sea and associated aquatic habitats since 1907, although the frequency and severity of epizootics, including avian cholera and salmonellosis, Type C botulism and Newcastle disease, have increased in the past decade (Friend, 2002). While causes of the outbreaks are mostly unknown, diseased tilapia were associated with a 1996 outbreak of Type C botulism that killed more than 15,000 pelicans and other fish-eating birds at the Salton Sea, including 15% of the population of white pelican (*Pelecanus*

erythrorhynchos Gmelin) and as many as 1,400 individuals of the endangered brown pelican (*Pelecanus occidentalis* Linnaeus) (Bruehler & de Peyster, 1999; Friend, 2002). Elevated tissue levels of Se and other trace elements might have compromised the immune system of the birds, making them less resistant to bacterial and viral agents (Bobker, 1993). Pelicans are the only bird species known to consume adult tilapia at the Salton Sea and for which recent Se levels have been determined. When comparing trace element concentrations in livers of both white and brown pelicans from the Salton Sea that died during the 1996 botulism outbreak and livers of healthy captive brown pelicans obtained from Sea World, San Diego, California, Bruehler & de Peyster (1999) found that only Se was elevated in the birds from the Salton Sea. Se levels averaged 16.9 and 19.3 $\mu\text{g g}^{-1}$ dw for brown and white pelicans from the Salton Sea, respectively, and 9.3 $\mu\text{g g}^{-1}$ dw for brown pelicans from Sea World. Although Se levels were below those leading to reproductive failure or death, they were elevated enough to potentially impair the birds' immune system. Cd, Cr, Cu, and Pb concentrations in liver were similar for the two locations. Lower liver concentrations of Fe and Zn, however, were measured in both pelican species from the Salton Sea compared to those from Sea World. Bruehler & de Peyster (1999) speculated that botulinum toxin may have caused liver damage in the Salton Sea pelicans, resulting in the observed lower Fe levels.

Potential adverse impacts on most other fish eating birds feeding at the Sea cannot be assessed as most of them prey upon juvenile tilapia, for which Se body burden is unknown. Nonetheless, Se concentrations in Salton Sea tilapia are above 3 $\mu\text{g g}^{-1}$ dw, the suggested risk threshold in aquatic food chain organisms for protection of wildlife (Lemly, 1996a). Impaired reproduction was observed in birds fed a diet containing 3–8 $\mu\text{g g}^{-1}$ dw in feeding trial with selenomethionine (Wilber, 1980; Heinz, 1996). Setmire et al. (1990, 1993) and Schroeder et al. (1993) reported that Se concentrations in liver of eared grebe (*Podiceps nigricollis* Brehm), double-crested cormorant (*Phalacrocorax auritus* Pearson), northern shoveler (*Anas clypeata* Linnaeus), and ruddy duck (*Oxyura jamaicensis* Gmelin) from

the Salton Sea, were likely to cause reproductive problems. Olhendorf & Marois (1990) concluded that the Se concentrations determined from black-crowned night heron (*Nycticorax nycticorax* Linnaeus) eggs collected at the Sea were elevated but would not affect reproductive success.

Sample et al. (1996) proposed food-based NOAELs and LOAELs for great blue herons, derived from experimental dietary exposure of mallard ducks and black-crowned night herons (*Nycticorax nycticorax* Gmelin), and for osprey, derived from dietary exposure of screech owl (*Otus asio* Linnaeus), to selenomethionine (Smith et al., 1988; Heinz et al., 1989; Wiemeyer & Hoffman, 1996). The duration of the studies ranged from 13 to 14.3 weeks, and the endpoints measured included egg production, hatchability, and nestling survivorship. The NOAELs reported for great blue herons differed substantially according to which test species they were extrapolated from: $2.3 \mu\text{g g}^{-1}$ dw derived from mallard ducks vs. $10.2 \mu\text{g g}^{-1}$ dw from black-crowned night heron (Sample et al., 1996). This demonstrates the level of uncertainty associated with such indirectly determined benchmark values. Only the LOAEL for great blue heron extrapolated from mallard data was reported ($4.55 \mu\text{g g}^{-1}$ dw). NOAEL and LOAEL for osprey, as extrapolated from screech owl data, were 2.2 and $7.5 \mu\text{g g}^{-1}$ dw, respectively (Sample et al., 1996). Compared to Se concentration in whole tilapia, these values indicate that reproductive impairment might occur in great blue herons and osprey feeding on Salton Sea tilapia with a Se concentration averaging $8 \mu\text{g g}^{-1}$ dw.

Se may play its most important role at the Sea by depressing immune system responses of birds to diseases such as avian cholera and botulism (Bobker, 1993; Bruehler & de Peyster, 1999), but the role of Se as a potential immunotoxic agent in birds has not been investigated sufficiently to derive threshold levels of toxicity (Olhendorf, 2003). An evaluation of the risk from Se exposure incurred by tilapia eating birds at the Salton Sea is needed.

Other trace elements and wildlife

None of the trace elements analyzed in tilapia from the Salton Sea were elevated (Table 4),

although Setmire et al. (1990) reported elevated concentrations of Cr, Ni, and Zn in Salton Sea sediments. Nickel was not detected in tilapia samples, and neither Cr nor Zn showed any unusual exposure or accumulation pattern. In addition, concentrations of Cr and Zn in tilapia samples (Table 4) were approximately 90 and 70% lower, respectively, than the food-based NOAELs derived for both great blue herons and ospreys (Sample et al., 1996). Zinc levels were lower than the average of $21.7 \mu\text{g g}^{-1}$ ww obtained from fish (315 whole body composite samples) collected for a nationwide survey of 109 bodies of water in 1984 (Schmitt & Brumbaugh, 1990). In addition, levels of Zn in livers of brown and white pelicans collected during the botulism die-off in 1996 at the Salton Sea were lower than those of pelicans from San Diego Bay, California (Roberts, 1997; Bruehler & de Peyster, 1999). From the above and a review of the literature, we conclude that none of the trace elements detected in tilapia were elevated enough to pose a risk to the fish and birds feeding on them.

Boron (B) has been mentioned as a contaminant of concern at the Salton Sea (Setmire et al., 1990, 1993). No study was found documenting either tissue-based toxicity or toxic dietary exposure levels of B for fish, but we speculate that the impact of the comparatively low concentrations of B on tilapia is negligible. For birds, no study was found reporting the toxic dietary intake for piscivorous birds.

Boron levels in liver samples of brown and white pelicans that died during the botulism outbreak in 1996 at the Salton Sea were found to be similar to those of pelicans from Sea World, San Diego, California, and below those known to cause reproductive problems in adult mallards (Smith & Anders, 1989; Roberts, 1997). Further investigations to assess the effect of dietary B intake on piscivorous birds and the toxic threshold level are needed but we speculate that the impacts of B concentrations on birds feeding on tilapia from the Salton Sea are minor.

Pesticides, PCBs and wildlife

None of the pesticides and PCBs analyzed for by different studies were found to be a concern for

fish and piscivorous birds. A DDT metabolite, DDE, however, has been considered a contaminant of concern for birds at the Salton Sea and in nearby aquatic habitats (Setmire et al., 1990, 1993; Bennett, 1998; Roberts, 2000). DDE concentrations in tilapia fillet ($0.032 \mu\text{g g}^{-1}$ ww; samples 13, 14, 99, 100; Table 5) and whole body samples ($0.085 \mu\text{g g}^{-1}$ ww samples 89–98; Table 5) in 2000–2001, are approximately 70% lower than those measured in whole carp (*Cyprinus carpio* Linnaeus) collected from the cotton farming regions of the lower Mississippi River basin in 1995 (mean: $0.29 \mu\text{g g}^{-1}$ ww; Schmitt, 2002), and in carp collected in the Lower Rio Grande Valley of Texas in 1997 (mean $0.25 \mu\text{g g}^{-1}$ ww; Wainwright et al., 2001). These tilapia concentrations are higher, however, than the average DDE concentrations reported by TSMP for whole fish collected California waterbodies during 2000–2001 ($0.058 \mu\text{g g}^{-1}$ ww), but lower than the EDL85 computed from the TSMP dataset ($0.432 \mu\text{g g}^{-1}$ ww) (data from TSMP, available at <http://www.swrcb.ca.gov/programs/smw/>). Determining the impacts of such DDE levels on tilapia is difficult as no field studies showing a correlation between pathological symptoms and DDE tissue residues are known for fish. Under laboratory settings, adverse effects were observed in freshwater fish when whole body concentrations of tDDT exceeded $0.5 \mu\text{g g}^{-1}$ ww with effects varying among species and exposure regimes (Jarvinen & Ankley, 1999). Based in these findings, we speculate that DDE levels do not represent a significant risk to the tilapia population at the Salton Sea.

Previous studies have found, however, elevated levels of tDDT in bird tissues and eggs collected from the Salton Sea area. Based on their findings of elevated DDE concentrations in tissues of birds collected from the Salton Sea and its tributaries, Schroeder et al. (1993) concluded that birds feeding in the Salton Sea were at high risk of DDE-induced reproductive failure. In addition, eggs of black-crowned night heron and great egret (*Ardea alba* Gmelin) collected from the Salton Sea in 1985 had high DDE concentrations (mean 8.60 and $24 \mu\text{g g}^{-1}$ ww, respectively; Ohlendorf & Marois, 1990). Shells of black-crowned night-heron eggs collected from

nests at the Sea in 1993 were 12 percent thinner than those of pre-DDT era eggs (Bennett, 1998). Total DDT levels were also elevated in muscle tissue of brown pelicans that died during the 1996 botulism event (mean: $2.60 \mu\text{g g}^{-1}$ ww; Roberts, 1997). Whether these elevated levels are due to dietary exposure from feeding on Salton Sea tilapia cannot be ascertained. For the protection of brown pelican reproduction, a dietary threshold concentration of $0.15 \mu\text{g g}^{-1}$ ww tDDT in fish was established by the U.S.EPA (1980). Brown pelicans are considered highly sensitive to tDDT (Blus, 2003). Newell et al. (1987) calculated the highest level of tDDT in whole fish that would have no detrimental impact on brown pelicans to be $0.20 \mu\text{g g}^{-1}$ ww. Using this criterion, and assuming that the brown pelican is the most sensitive of all piscivorous bird species found at the Salton Sea, we conclude that mean tDDT concentrations of $0.085 \mu\text{g g}^{-1}$ ww in whole tilapia do not likely present a significant risk to piscivorous bird populations.

Fish meal quality and production

Salton Sea tilapia would yield a fish meal of moderate nutritive quality. High quality fish meal, has a moisture content of about 5%, a protein content of approximately 68%, and a maximum ash content of 17% (B. Burris, pers. comm.). We found protein and ash contents (dw) of Salton Sea tilapia to be approximately 61 and 21%, respectively (Table 7). Ismond (2001) reported dw values of 71% protein and 20% ash for a compositional analysis of a single Salton Sea tilapia. He also obtained dw values of 64% protein and 26% ash for 18 kg of Salton Sea tilapia that were put through a benchtop simulated fish meal production process. For comparison, two other tilapia species, *Oreochromis niloticus* (Linnaeus) and *O. aureus* (Steindachner), raised in freshwater production systems had average dw protein and ash contents of 57% and 19%, respectively (Boyd & Green, 1998). Quality of fish meal made from Salton Sea tilapia would be improved if the ash content were reduced by a process that accomplished at least partial removal of bones and scales.

One benefit of large scale tilapia harvesting for fish meal production is that it would remove phosphorus from the lake in significant quantities. Phosphorus appears to be the limiting nutrient in this highly eutrophic lake (Bain et al., 1970; Holdren & Montaña, 2002). González et al. (1998) suggested that “massive, sustained yield harvesting of tilapia...might be not only a feasible way to remove nutrients (from the Salton Sea) in helpfully large quantities but also be an economically profitable venture,” since fish meal in recent years has sold for as much as \$600 per metric ton. In their microcosm experiment with Salton Sea tilapia they observed that tilapia reduced total phosphorus in the water column by 64% and calculated that removal of the one fish in each tank at the end of the experiment was equivalent to a sustained yield harvest of $>300 \text{ kg ha}^{-1} \text{ yr}^{-1}$. Sustained yields much higher than that are common in lake and reservoir tilapia fisheries in tropical regions (Fernando & Holcik, 1982; Fernando, 1984; Amarasinghe & Pritcher, 1986; Moreau & De Silva, 1991).

With an average phosphorus content of 3.25% dw (Table 7) or 0.91% ww (given an average moisture content of 72%: Table 1, samples 89–98) and a Salton Sea area of 980 km^2 , a harvest of 300 kg ha^{-1} would remove 29,400 t of fish and 267 t of phosphorus from the lake. That is a significant fraction of total annual external phosphorus loading to the Salton Sea, which has been estimated at 1,515 t for 1996–1997 and 1,385 t for 1999 (Holdren & Montaña, 2001). Combined with steps now underway to reduce silt and phosphorus loading to the Sea, massive fish harvesting clearly could assist remediation of the Sea’s eutrophic state, whether the fish were used for fish meal or some other purpose such as compost or fertilizer (Ismond, 2001).

Contaminants that would be of possible concern in fish meal made from Salton Sea tilapia are selenium, arsenic, and DDE. Although we do not know the concentration of contaminants that would remain in fish meal or other products obtained from tilapia harvested from the Salton Sea, the concentrations we have documented in Salton Sea tilapia can be compared to guidelines established for animal feed.

Se is a nutritionally required element, and livestock and poultry sometimes suffer from Se deficiency syndromes. Examples include myopathy or “white muscle” disease in cattle, swine and poultry, liver necrosis in swine and pancreatic necrosis in poultry (Oldfield, 1997). To prevent economic loss of livestock to selenium deficiency, animal commercial feeds are supplemented with sodium selenite and sodium selenate. The addition of selenium to animal feed is regulated by the Food and Drug Administration, and the maximum concentration allowed in livestock and poultry feed is $0.30 \mu\text{g g}^{-1}$ (NAS/NAE, 1973). If the Se concentration in pure undiluted fish meal made from Salton Sea tilapia were similar to that for whole tilapia ($8 \mu\text{g g}^{-1} \text{ dw}$), the Se concentration in that meal would be 24 times the maximum allowable concentration in livestock and poultry feed. This does not pose a problem, however, as fish meal normally constitutes only 3–10% of manufactured feeds for fish, poultry, swine and dairy cattle. Fish meal from other sources—or other non-fish protein—could be used as a diluent when necessary.

The concentration of Se in fish meal fed to aquaculture organisms may be subject to different regulations than those governing poultry and livestock feedstuffs. Aquaculture feeds often are formulated with artificial Se supplements. When given Se-deficient diets, aquaculture-raised fish such as rainbow trout (*O. mykiss*), Atlantic salmon (*Salmo salar* Linnaeus), and channel catfish (*Ictalurus punctatus* Rafinesque), show reduced weight gain, feed efficiency, and liver glutathione peroxidase activity. The latter is a selenoenzyme important in the defense mechanisms of organisms against toxicants and cell damage caused by lipid peroxides (Hilton et al., 1980; Bell et al., 1987; Wang & Lovell, 1997). Organic forms of Se were found to be more bioavailable than inorganic forms to channel catfish (Wang & Lovell, 1997) and Atlantic salmon (Lorentzen et al., 1994) in aquaculture as evidenced by enhanced absorption and muscle retention. While Wang & Lovell (1997) determined that the inorganic Se dietary requirement of catfish for weight gain was $0.28 \mu\text{g g}^{-1}$, this requirement was decreased to 0.09–0.11 $\mu\text{g g}^{-1}$ when Se was provided in organic form. If Se present in tilapia fish meal is mostly bound to amino acids, its nutritional

value would therefore be increased and its manufacturing cost decreased as sodium selenite would not have to be added. Furthermore, increased bioavailability of the organic form would lead to a reduction of total Se in fish feed, hence leading to a decrease of Se entering culture systems and ultimately a decrease in Se outputs from aquaculture operations into the environment.

Arsenic is also a desired element in animal feeds. Organic arsenicals are added to poultry and swine feed, primarily as pentavalent phenylarsonic acids and their salts, such as arsenilic acid and sodium arsenilate (Ledet & Buck, 1978). In swine, addition of either of these organic arsenicals increases weight gain rate and improves feed efficiency in growing young. Arsenic salts aid and control hemorrhagic enteritis and bloody dysentery. In poultry, As promotes growth and feed efficiency and improves pigmentation. Recommended arsenilic acid concentration in poultry and livestock feed is 45–90 g t⁻¹ ($\mu\text{g kg}^{-1}$) or 13–26 $\mu\text{g As g}^{-1}$ (NAS/NAE, 1973; Newell et al., 1987). Assuming that As concentration in fish meal made from Salton Sea tilapia would be similar to that for whole tilapia (3.60 $\mu\text{g g}^{-1}$ dw), feeds made from this meal would still need to be fortified with As. As with Se, it would be desirable to know to what degree As levels might be reduced during the fish meal production process. Unlike the case with selenium, however, the concern might be how to minimize that reduction.

Present tDDT concentrations in tilapia are approximately 90% lower than the action levels and tolerances established by the FDA for deleterious substances present in human food and animal feed (U.S. FDA, 1998). The action level for DDE in foodstuff is 0.50 $\mu\text{g g}^{-1}$ ww (Code of Federal Regulations, Title 21, Parts 109 and 509). Furthermore, the lipidic portion obtained during the fish meal manufacturing process can be discarded, producing final levels of hydrophobic compounds lower than those in whole fish homogenates.

The feasibility of a fish meal operation at the Salton Sea would also depend on a sustainable tilapia harvest. Under present conditions, a good sustainable harvest seems out of the question. A previously published estimate of sustainable yield for the nearshore region of 3600 kg ha⁻¹ y⁻¹ (Costa-Pierce & Riedel, 2000) is now recognized

to be too high by at least an order of magnitude (Caskey et al., 2007). Moreover, gillnet surveys conducted during 1999–2002 found that most tilapia caught were members of the 1995-year class, and that the 1996–2002 year classes either were very small or failed to survive their first winter (Riedel et al., 2002b; Caskey et al., 2007). Low recruitment and massive kills of adults resulted in tilapia catch per unit effort to drop from 15.9 to 0.025 fish net⁻¹ h⁻¹ at four monitoring stations between 1999 and 2002 (Caskey et al., 2007). There is no information on whether tilapia recruitment failures occurred prior to the mid-1990s. Large sustained tilapia harvests for any purpose clearly must await restoration of the Sea to a more benign environment for fish.

Conclusions

From this synthesis, we conclude that the existing official health advisory on consumption of fish from the Salton Sea should be revised.

Various large-scale projects are under consideration for restoring the Sea to a healthier state. These projects are aimed at stabilizing the water level, reducing and stabilizing salinity and perhaps ameliorating eutrophic conditions. Any such project would result in large changes in the ecology and biogeochemistry of the Sea, and have the potential for altering contaminant levels in fish and other ecosystem components. In evaluating these restoration options, two recommendations emerge from our analysis: (1) with respect to the sport fishery and anglers' exposure to As, project options that would result in lower As levels in fish are desirable, and (2) with respect to fish and piscivorous birds, as well as potential fish meal operations, options that would result in reduced input of Se into the system are desirable. Information regarding the sources of As in the lake as well as its movement and transformation within the lake is not available at this time. Project options will need to take into consideration their potential impacts on the movement of Se and As within the system and their ramifications for wildlife and human health. Furthermore, Se and As monitoring plans should be implemented following the

adoption of a restoration project. Despite some similarities with other ecosystems where Se-induced impacts were reported, the Salton Sea ecosystem is unique enough to warrant its own set of investigations and assessments.

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References

- Amarasinghe, U. & T. Pritcher, 1986. Assessment of fishing effort in Parakrama Samudra, an ancient man-made lake in Sri Lanka. *Fisheries Research* 4: 271–282.
- ATSDR, 2000. Toxicological Profile for Arsenic. Atlanta, GA: U.S. Agency for Toxic Substances and Disease Registry Department of Health and Human Services, Public Health Service.
- ATSDR, 2001. Draft - Toxicological Profile for Selenium. Atlanta, Georgia: U.S. Agency for Toxic Substances and Disease Registry Department of Health and Human Services, Public Health Service.
- Bain, R. C., A. M. Caldwell, R. H. Clawson, H. L. Scotten & R. G. Wills, 1970. Salton Sea California: Water quality and ecological management considerations. U.S. Dept. Interior, Federal Quality Administration, Pacific Southwest Region, 53 pp.
- Barnum, D. A. & D. S. Gilmer, 1988. Selenium levels in biota from irrigation drainwater impoundments in the San Joaquin valley, California. *Lake Reservoir Management* 4: 181–186.
- Bell, J. G., C. B. Cowey, J. W. Adron & B. J. Pirie, 1987. Some effects of selenium deficiency on enzyme activities and indices of tissue peroxidation in Atlantic salmon parr (*Salmo salar*). *Aquaculture* 65: 43–54.
- Bennett, J., 1998. Effects of selenium and other contaminants associated with irrigation drainage in the Salton Sea are, California, 1992–1994. Information Report No 4, Department of the Interior, National Irrigation Water Quality Program, Washington (D.C.): 35 pp.
- Blus, L. J., 2003. Organochlorine pesticides. In Hoffman D. J., B. A. Rattner, G. A. Burton Jr & J. Cairns Jr. (eds), *Handbook of Ecotoxicology*. CRC Press, Inc., Boca Raton (Florida): 313–340.
- Bobker, G., 1993. Death in the ponds: selenium-induced waterbird death and deformities at agricultural evaporation ponds. The Bay Institute of San Francisco, Sausalito (California): 52.
- Boyd, C. E. & B. W. Green, 1998. Dry matter, ash, and elemental composition of pond-cultured tilapia *Oreochromis aureus* and *O. niloticus*. *Journal of the World Aquaculture Society* 29: 125–128.
- Braman, R. S., 1983. Environmental reaction and analysis methods. In Fowler B. A. (ed.), *Biological and Environmental Effects of Arsenic*, Vol. 6. Elsevier, Amsterdam: 141–154.
- Bruehler, G. L. & A. de Peyster, 1999. Selenium and other trace metals in pelicans dying at the Salton Sea. *Bulletin of Environmental Contamination and Toxicology* 63: 590–597.
- Caskey, L. L., R. R. Riedel, B. Costa-Pierce, J. Butler & S. H. Hurlbert, 2007. Population dynamics, distribution, and growth rate of tilapia (*Oreochromis mossambicus*) in the Salton Sea, California, with notes on *Bairdiella (Bairdiella icistia)* and orangemouth corvina (*Cynoscion xanthulus*). *Hydrobiologia* 576: 185–203.
- CEC, 2002. Water Quality Report 2002. City of El Centro. Available <http://www.cityofelcentro.org/pworks/2003.ccr.pdf> (visited 2003, July 27).
- Chen, C. J., Y. M. Hsueh, M. P. Tseng, Y. C. Lin, L. I. Hsu, W. L. Chou, H. Y. Chiou, I. H. Wang, Y. L. Chou, C. H. Tseng & S. H. Liou, 2001. Individual susceptibility to arseniasis. In Chappell, W. R., C. O. Abernathy & R. L. Calderon (eds), *Arsenic Exposure and Health Effects: Proceedings of the Fourth International Conference on Arsenic Exposure and Health Effects*, Elsevier, Amsterdam, 135–143.
- Chen, C. Y., & C. L. Folt, 2000. Bioaccumulation and diminution of arsenic and lead in a freshwater food web. *Environmental Science and Technology* 34: 3878–3884.
- Chen, C. Y., R. S. Stemberger, B. Klaue, J. D. Blum, P. C. Pickhardt, & C. L. Folt, 2000. Accumulation of heavy metals in food web components across a gradient of lakes. *Limnology and Oceanography* 45: 1525–1536.
- CSDWD, 2002. Additional Physical, Mineral, and Metal Characteristics – Year 2002 by Water Treatment Plant. City of San Diego Water Department. Available: http://sandiego.gov/water/quality/additional_testccr02.pdf (visited 2003, July 27).
- CVWD, 2002. Coachella Valley Residents Tap High Quality, Healthful water. Coachella Valley Water District. Available <http://www.cvwd.org/Water%20Quality%20Report%202002.pdf> (visited 2003, July 27).
- Costa-Pierce, B. A. & R. W. Doyle, 1997. Genetic identification and status of tilapia regional strains in southern California. In Costa-Pierce B. A. & J. E. Rakocy (eds), *Tilapia Aquaculture in the Americas*, Vol. 1. The World Aquaculture Society, Baton Rouge (Louisiana): 1–17.

- Costa-Pierce, B. A. & R. Riedel, 2000. Fisheries ecology of the tilapias in subtropical lakes in the United States. In Costa-Pierce B. A. & J. E. Rakocy (eds), *Tilapia Aquaculture in the Americas*, Vol. 2. The World Aquaculture Society, Baton Rouge (Louisiana): 1–20.
- Cumbie, P. M. & S. L. Van Horn, 1978. Selenium accumulation associated with fish mortality and reproductive failure. Proceeding from the Annual Conference of the Southeastern Association of Fish and Wildlife Agencies 32: 612–624.
- Davis, J. M. & R. W. Elias, 1996. Risk Assessment of Metals. In Chang L. W. (ed.), *Toxicology of Metals*. CRC Lewis Publishers, Boca Raton (Florida): 55–79.
- Detwiler, P., M. F. Coe, & D. M. Dexter, 2002. The invertebrates of the Salton Sea: distribution and seasonal dynamics. *Hydrobiologia* 473: 139–160.
- Edmonds, J. & K. Francesconi, 1993. Arsenic in seafoods: Human health aspects and regulations. *Marine Pollution Bulletin* 26: 665–674.
- Eisler, R., 1994. A review of arsenic hazards to plants and animals with emphasis on fishery and wildlife resources. In Nriagu J. O. & M. S. Simmons (eds), *Toxic Contaminants in the Great Lakes*. John Wiley & Sons, New York (New York): 185–261.
- Fernando, C. H. 1984. Reservoirs and lakes of Southeast Asia (Oriental Region). In: Taub F. B. (ed.), *Lakes and Reservoirs. Ecosystems of the World* 23. Elsevier, Amsterdam: 411–446.
- Fernando, C. H. & J. Holcik, 1982. The nature of fish communities: a factor influencing the fishery potential of tropical lakes and reservoirs. *Hydrobiologia* 97: 127–140.
- Foley, R. E. J. R. Spotila, J. P. Giesy & C. H. Wall, 1978. Arsenic concentrations in water and fish from Chautauqua Lake, New York. *Analytical Chemistry* 48: 120–125.
- Friend, M., 2002. Avian disease at the Salton Sea. *Hydrobiologia* 473: 293–306.
- Gilderhus, P. A., 1966. Some effects of sublethal concentrations of sodium arsenite on bluegills and the aquatic environment. *Transactions of the American Fisheries Society* 95: 289–296.
- Gillespie, R. B. & P. C. Baumann, 1986. Effects of high tissue concentrations of selenium on reproduction by bluegills. *Transactions of the American Fisheries Society* 115: 208–215.
- González, M.R., C.M. Hart, J. Verfaille & S.H. Hurlbert, 1998. Salinity and fish effects on Salton Sea microecosystems: water chemistry and nutrient cycling. *Hydrobiologia* 381: 105–128.
- Goyer, R. A., 1996. Toxic effects of metals. In Klaassen C. D., M. A. Amdur & J. D. Doull (eds), *Casarett And Doull's Toxicology – The Basic Science of Poisons* Fifth Edition. McGraw-Hill, New York (New York): 691–736.
- Hamilton, S. J., 2002. Rationale for a tissue-based selenium criterion for aquatic life. *Aquatic Toxicology* 57: 85–100.
- Hamilton, S. J. & A. D. Lemly, 1999. Water-sediment controversy in setting environmental standards for selenium. *Ecotoxicology and Environmental Safety* 44: 227–235.
- Hamilton, S. J., K. J. Buhl, N. L. Faerber, R. H. Wiedmeyer, & F. A. Bullard, 1990. Toxicity of organic selenium in the diet to Chinook salmon. *Environmental Toxicology and Chemistry* 9: 347–358.
- Han, B. C., W. L. Jeng, R. Y. Chen, G. T. Fang, T. C. Hung & R. J. Tseng, 1998. Estimation of target hazard quotients and potential health risks for metals by consumption of seafood in Taiwan. *Archives of Environmental Contamination and Toxicology* 35: 711–720.
- Hanaoka, K., S. Tagawa, & T. Kaise, 1992. The fate of organoarsenic compounds in marine ecosystems. *Applied Organometallic Chemistry* 6: 139–146.
- Heinz, G. H., 1996. Selenium in birds. In Beyer W. N., G. H. Heinz & A. W. Redmon (eds), *Interpreting Environmental Contaminants in Animal Tissues*. CRC Lewis Publishers, Boca Raton (Florida): 453–464.
- Heinz, G. H. D. J. Hoffman & L. G. Gold, 1989. Impaired reproduction of mallards fed an organic form of selenium. *Journal of Wildlife Management* 53: 418–428.
- Hickey, J. T., L. A. Barclay, G. A. Hansen, J. J. Brabander, J. B. Elder & J. Jacknow, 1984. *Field Operation Manual for Resource Contaminant Assessment*: U.S. Fish and Wildlife Service, [looseleaf]: 495 pp.
- Hilton, J. W., P. V. Hodson & S. J. Slinger, 1980. The requirement and toxicity of selenium in rainbow trout (*Salmo gairdneri*). *Journal of Nutrition* 110: 2527–2535.
- Hodson, P. V. & J. W. Hilton, 1983. The nutritional requirements and toxicity to fish of dietary and waterborne selenium. *Ecological Bulletin* 35: 335–340.
- Hodson, P. V., D. J. Spry & B. R. Blunt, 1980. Effects on rainbow trout (*Salmo gairdneri*) of a chronic exposure to waterborne selenium. *Canadian Journal of Fisheries and Aquatic Sciences* 37: 233–240.
- Hodson, P. V., D. M. Whittle & D. J. Hallet, 1984. Selenium contamination of the Great Lakes and its potential effects on aquatic biota. In Nriagu J. O. & M. S. Simmons (eds), *Toxic Contaminants in the Great Lakes*. John Wiley & Sons, New York (New York), 371–394.
- Holdren, G. C. & A. Montaña, 2002. Chemical and physical characteristics of the Salton Sea, California. *Hydrobiologia* 473: 1–21.
- Huston, D. W., C. B. Cook & G. T. Orlob, 2000. New and Alamo rivers project – Preliminary data collection and analysis for development of hydrodynamic and water quality river models. Salton Sea Authority and State Water Resources Control Board Report 99-3: 82 pp.
- Ismond, A. 2001. Examination of the commercial uses for Salton Sea tilapia. Unpublished report submitted to the Salton Sea Authority, La Quinta, California. Aqua-Terra Consultants, Bellevue, Washington.
- Jarvinen, A. W. & G. T. Ankley, 1999. Linkage of Effects to Tissue Residues: Development of a Comprehensive Database for Aquatic Organisms Exposed to Inorganic and Organic Chemicals. SETAC Press, Pensacola (Florida): 358 pp.
- Kirby, J. & W. Maher, 2002. Tissue accumulation and distribution of arsenic compounds in three marine fish

- species: relationship to trophic position. *Applied Organometallic Chemistry* 16: 108–115.
- Kuperman, B. I., V. E. Matey & S. H. Hurlbert, 2001. Parasites of fish from the Salton Sea, California, U.S.A. *Hydrobiologia* 466: 195–208.
- Lacayo, M. L., A. Cruz, S. Calero, J. Lacayo & I. Fomsgaard, 1992. Total arsenic in water, fish, and sediments from Lake Xolotlan, Managua, Nicaragua. *Bulletin of Environmental Contamination and Toxicology* 49: 463–470.
- LADWP, 2002. City of Los Angeles Water Quality Report 2002. Los Angeles Department of Water and Power. Available: <http://www.ladwp.com/Water/Quality/Annual/AnnReport/index.htm> (visited 2002, Nov. 2).
- Ledet A. E. & W. B. Buck, 1978. Toxicity of organic arsenicals in feedstuffs. In Oehme F. W. (ed.), *Toxicity of Heavy Metals in the Environment*. Marcel Dekker Inc., New York (New York): 375–391.
- Lemly, A. D., 1985. Toxicology of selenium in freshwater reservoir: implications for environmental hazard evaluation and safety. *Ecotoxicology and Environmental Safety* 26: 181–204.
- Lemly, A. D., 1993a. Guidelines for evaluating selenium data from aquatic monitoring and assessment studies. *Environmental Monitoring and Assessment* 28: 83–100.
- Lemly, A. D., 1993b. Teratogenic effects of selenium in natural populations of freshwater fish. *Ecotoxicology and Environmental Safety* 26: 181–204.
- Lemly, A. D., 1993c. Metabolic stress during winter increases the toxicity of selenium to fish. *Aquatic Toxicology* 27: 133–158.
- Lemly, A. D., 1996a. Selenium in aquatic organisms. In: Beyer W. N., G. H. Heinz & A. W. Redon-Norwood (eds), *Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations*. CRC Lewis Publishers, New York (New York): 427–446.
- Lemly, A. D., 1996b. Winter Stress Syndrome: An important consideration for hazard assessment of aquatic pollutants. *Ecotoxicology and Environmental Safety* 34: 223–227.
- Lemly, A. D., 2002. Symptoms and implications of selenium toxicity in fish: the Belews Lake case example. *Aquatic Toxicology* 57: 39–49.
- Lemly, A. D. & G. J. Smith, 1987. Aquatic cycling of selenium: Implications for fish and wildlife. *Fish and Wildlife Leaflet* 12. U. S. Fish and Wildlife Services, Washington DC.
- Lemly, A. D., S. E. Finger & M. K. Nelson, 1993. Sources and impacts of irrigation drainwater contaminants in arid wetlands. *Environmental Toxicology and Chemistry* 12: 2265–2279.
- Liao, C. M., B. C. Chen, S. Singh, M. C. Lin, C. W. Liu & B. C. Han, 2003. Acute toxicity and bioaccumulation of arsenic in tilapia (*Oreochromis mossambicus*) from a blackfoot disease area in Taiwan. *Environmental Toxicology* 18: 252–259.
- Lorentzen, M., A. Maage & K. Julshamn, 1994. Effects of dietary selenite or selenomethionine on tissue selenium levels of Atlantic salmon (*Salmo salar*). *Aquaculture* 121: 359–367.
- Lowe, T. P., T. W. May, W. G. Brumbaugh & D. A. Kane, 1985. National Contaminant Biomonitoring Program: Concentrations of seven elements in freshwater fish, 1978–1981. *Archives of Environmental Contamination and Toxicology* 14: 363–388.
- Luoma, S. N., C. Johns, N. S. Fisher, N.A. Steinberg, R. S. Oremland & J. R. Reinfelder, 1992. Determination of selenium bioavailability to a benthic bivalve from particulate and solute pathways. *Environmental Science and Technology* 26: 485–491.
- MacIntosh, D. L., P. L. Williams, D. J. Hunter, L. A. Sampson, S. C. Morris, W. C. Willett & E. B. Rimm, 1997. Evaluation of a food frequency questionnaire-food composition approach for estimating dietary intake of inorganic arsenic and methylmercury. *Cancer Epidemiology, Biomarkers & Prevention* 6: 1043–1050.
- Maier, K. J., C. G. Foe & A. W. Knight, 1993. Comparative toxicity of selenate, selenite, seleno-DL-methionine, and seleno-DL-cystine to *Daphnia magna*. *Environmental Toxicology and Chemistry* 12: 755–763.
- May, T. W. & G. L. McKinney, 1981. Cadmium, mercury, arsenic, and selenium concentrations in freshwater fish, 1976–1977 – National Pesticide Monitoring Program. *Pesticide Monitoring Journal* 15: 14–38.
- McGeachy, S. M. & D. G. Dixon, 1990. Effect of temperature on the chronic toxicity of arsenate to rainbow trout (*Oncorhynchus mykiss*). *Canadian Journal of Fisheries and Aquatic Sciences* 47: 2228–2234.
- Moore J. W., J. Ramamoorthy & E. E. Ballantyne, 1983. *Heavy Metals in Natural Waters, Applied Monitoring and Impact Assessment*. Springer Publishers, New York (New York): 279 pp.
- Moreau, J. & S. DeSilva, 1991. Predicting yield models for lakes and reservoirs in the Philippines, Sri Lanka and Thailand. *FAO Fisheries Technical Paper* 319. FAO, Rome.
- NAS /NAE, 1973. *Water Quality Criteria*. National Academy of Science – National Academy of Engineering, U.S. Government Printing Office, Washington (D.C.): 594 pp.
- Newell, A. J., D. W. Johnson & L. K. Allen, 1987. Niagara River Biota Contamination Project – Fish flesh criteria for piscivorous wildlife, Technical Report 87-3. New York State Department of Environmental Conservation, Division of Fish and Wildlife, Bureau of Environmental Protection, Technical Report 87-3: 183 pp.
- Norin, H., M. Vahter, A. Christakopoulos & M. Sandström, 1985. Concentration of inorganic and total arsenic in fish from industrially polluted water. *Chemosphere* 14: 123–131.
- OEHHA, 1986. *Safe Water and Toxic Enforcement Act of 1986 – Chapter 6.6 added by Proposition 65 1986 General Election*. Office of Environmental Health Hazard Assessment Available: <http://www.oehha.ca.gov/prop65/law/p65.html> (visited 2003, November 2).
- Ogle, R. S. & A. W. Knight, 1989. Effects of elevated foodborne selenium on growth and reproduction of

- the fathead minnow (*Pimephales promelas*). Archives of Environmental Contamination and Toxicology 18: 795–803.
- Oldfield, J. E., 1997. Observations on the efficacy of various forms of selenium for livestock: A review. Biomedical and Environmental Sciences 10: 280–290.
- Ohlendorf, H. M., 1989. Bioaccumulation and effects of selenium in wildlife. In Jacobs, L. W. (ed.), Selenium in Agriculture and the Environment, Soil Science Society of America Special Publication Number 23, Soil Science Society of America, Madison (Wisconsin): 133–177.
- Ohlendorf, H. M., 2003. Ecotoxicology of selenium. In Hoffman D. J., B. A. Rattner, G. A. Burton, & J. Cairns (eds), Handbook of Ecotoxicology, 2nd edn. CRC Lewis Publishers, Boca Raton, (Florida): 465–500.
- Ohlendorf, H. M. & K. C. Marois, 1990. Organochlorines and selenium in California night heron and egret eggs. Environmental Monitoring and Assessment 15: 91–104.
- Ohlendorf, H. M., D. J. Hoffman, M. K. Saiki, T. W. Aldrich, 1986. Embryonic mortality and abnormalities of aquatic birds: apparent impacts of selenium from irrigation drainwater. Science of the Total Environment 52: 49–63.
- Pedlar, R. M. & J. F. Klaverkamp, 2002. Accumulation and distribution of dietary arsenic in lake whitefish (*Coregonus clupeaformis*). Aquatic Toxicology 57: 153–166.
- Popma, T. & M. Masser. 1999. Tilapia – Life history and biology. Southern Regional Aquaculture Center Publication No. 283.
- Radtke, D. B., W. G. Kepner & R. J. Effertz, 1988. Reconnaissance Investigation of Water Quality, Bottom Sediment, and Biota Associated with Irrigation Drainage in the Lower Colorado River Valley, Arizona, California, and Nevada, 1986–87. Water Resources Investigation Report 88-4002. U.S. Geological Survey, Tucson (Arizona): 77pp.
- Rasmussen, D. & H. Blethrow, 1990. Toxic Substances Monitoring Program – Ten Year Summary Report 1978–1987 No 90-1WQ. Water Resources Control Board, State of California, Sacramento (California): 133 pp + appendices.
- Rasmussen, D. & H. Blethrow, 1991. Toxic Substances Monitoring Program – 1988–1989 No 91-1WQ. State Water Resources Control Board, California Environmental Protection Agency, Sacramento (California): 104 pp + appendices.
- Rasmussen, D., 1993. Toxic Substances Monitoring Program – 1991 Data Report No 93-1WQ. State Water Resources Control Board, California Environmental Protection Agency, Sacramento (California): 27 pp + appendices.
- Rasmussen, D., 1995. Toxic Substances Monitoring Program – 1992–93 Data Report No 95-1WQ. State Water Resources Control Board, California Environmental Protection Agency, Sacramento (California): 33 pp + appendices.
- Rasmussen, D., 1997. Toxic Substances Monitoring Program – 1994–95 Data Report . California State Water Resources Control Board, California Environmental Protection Agency, Sacramento (California): 30 pp + appendices.
- Riedel, R., D. Schlenk, D. Frank & B. A. Costa-Pierce, 2002a. Analyses of organic and inorganic contaminants in Salton Sea fish. Marine Pollution Bulletin 44: 403–411.
- Riedel, R., L. Caskey & B. A. Costa-Pierce, 2002b. Fish biology and fisheries ecology of the Salton Sea, California. Hydrobiologia 161: 229–244.
- Roberts, C. A., 1997. Contaminants in pelicans collected during the avian botulism event at the Salton Sea in 1996. U.S. Fish and Wildlife Service and Bureau of Reclamation, Carlsbad (Ca): 20 pp + appendices.
- Roberts, C. A., 1997. Contaminants in pelicans collected during the avian botulism event at the Salton Sea in 1996. U.S. Fish and Wildlife Service and Bureau of Reclamation, Carlsbad (California): 20 pp + appendices.
- Saiki, M. K., 1990. Elemental concentrations in fishes from the Salton Sea, Southeastern California. Water, Air, and Soil Pollution 52: 41–56.
- Saiki, M. K., M. R. Jennings & R. H. Wiedmeyer, 1992. Toxicity of agricultural subsurface drainwater from the San Joaquin Valley, California, to juvenile Chinook and striped bass. Transactions of the American Fisheries Society 121: 78–93.
- Sample, B. E., D. M. Opresko, & G. W. Suter II, 1996. Toxicological Benchmarks for Wildlife: 1996 Revisions. ES/ER/TM-86/R3. Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- SCCWRP and MBCAES (Southern California Coastal Water Research Project and MBC Applied Environmental Sciences), 1994. Santa Monica Bay Seafood Consumption Study: Final Report. Available: http://ftp.sccwrp.org/pub/download/PDFs/273_seafood_consumption.pdf (visited 2003, March 15).
- SDCDHS, 1990. San Diego Bay Health Risk Study: an Evaluation of the Potential Risk to Human Health from Fish Caught and Consumed from San Diego Bay. Prepared for Port of San Diego, San Diego (Ca): San Diego County Department of Health Services, Document No. 25467.
- Schmitt, C. J., 2002. Organochlorine chemical residues in fish from the Mississippi River Basin, 1995. Archives of Environmental Contamination and Toxicology 43: 81–97.
- Schmitt, C. J. & W. G. Brumbaugh, 1990. National Contaminant Biomonitoring Program: Arsenic, cadmium, copper, lead, mercury, selenium, and zinc in U.S. freshwater fish, 1976–1984. Archives of Environmental Contamination and Toxicology 19: 731–747.
- Schmitt, C. J., J. L. Zajicek & M. A. Ribick, 1985. National Pesticide Monitoring Program: Residues of organochlorine chemicals in Freshwater fish, 1980–81. Archives of Environmental Contamination and Toxicology 14: 225–260.
- Schmitt, C. J., J. L. Zajicek & P. H. Peterman, 1990. National Contaminant Biomonitoring Program: Residues of organochlorine chemicals in U. S. freshwater

- fish, 1976–84. Archives of Environmental Contamination and Toxicology 19: 748–781.
- Schoof, R. A., J. Eickhoff, L. J. Yost, E. A. Crecelius, D. W. Cragin, D. M. Meacher, D. B. Menzel, 1999a. Dietary exposure to inorganic arsenic. In Chappell, W.R., C.O. Abernathy & R.L. Calderon (eds), Proceedings of the Third International Conference on Arsenic Exposure and Health Effects. Elsevier, Amsterdam: 81–88.
- Schoof, R. A., L. J. Yost, J. Eickhoff, E. A. Crecelius, D. W. Cragin, D. M. Meacher & D. B. Menzel, 1999b. A market basket survey of inorganic arsenic in food. Food and Chemical Toxicology 37: 839–846.
- Schroeder, R. A., M. Rivera, B. J. Redfield, J. N. Densmore, R. L. Michel, D. R. Norton, D. J. Audet, J. G. Setmire & S. L. Goodbred, 1993. Physical, chemical, and biological data for detailed study of irrigation drainage in the Salton Sea area, California, 1988–90. U.S. Geological Survey. Open-File Report 93-83.
- Setmire, J. G., J. C. Wolfe & R. K. Stroud, 1990. Reconnaissance investigation of water quality, bottom sediment, and biota associated with irrigation drainage in the Salton Sea area, California, 1986–87. U.S. Geological Survey. Water-Resources Investigations Report 89-4102.
- Setmire, J. G., R. A. Schroeder & J. N. Densmore, 1993. Detailed study of water quality, bottom sediment and biota associated with irrigation drainage in the Salton Sea area, California, 1988–1990. U.S. Geological Survey. Water-Resources Investigations Report 93-4014.
- Skorupa, J. P., 1998. Selenium poisoning of fish and wildlife in nature: Lessons from twelve real-world examples. In Frankenberger W. T. & R. A. Engberg (eds), Environmental Chemistry of Selenium. Marcel Dekker, New York: 315–354.
- Skorupa, J. P. & H. M. Ohlendorf, 1991. Contaminants in drainage water and avian risk threshold. In Dinar A., & D. Zilberman (eds), The Economics and Management of Water and Drainage in Agriculture. Kluwer Academic Publishers, Norwell (Massachusetts): 345–368.
- Smith, G. J. & V. P. Anders, 1989. Toxic effects of boron on mallard reproduction. Environmental Toxicology and Chemistry 8: 943–950.
- Sorensen, E. M. B. 1988. Selenium accumulation, reproductive status, and histopathological changes in environmentally exposed redear sunfish. Archives of Toxicology 61: 324–329.
- Sorensen, E. M. B., R. E. Henry & R. Ramirez-Mitchell, 1979. Arsenic accumulation, tissue distribution and cytotoxicity in teleosts following indirect aqueous exposures. Bulletin of Environmental Contamination and Toxicology 21: 162–169.
- Sorensen, E. M. B., J. S. Bell & C. W. Harlan. 1983. Histopathological changes in selenium exposed fish. American Journal of Forensic Medicine and Pathology 4: 111–123.
- Sorensen, E. M. B., P. M. Cumbie; T. L. Bauer, J. S. Bell & C. W. Harlan, 1984. Histopathological, hematological, condition-factor, and organ weight changes associated with selenium accumulation in fish from Belews Lake, North Carolina. Archives of Environmental Contamination and Toxicology 13: 153–162.
- Stemberger, R. A. & C. Y. Chen, 1998. Fish tissue metals and zooplankton assemblages of northeastern U.S. lakes. Canadian Journal of Fisheries and Aquatic Sciences 55: 339–352.
- Storelli, M. M. & G. O. Marcotrigiano, 2000. Organic and inorganic arsenic and lead in fish from the South Adriatic Sea, Italy. Food Additives and Contaminants: Analysis, Surveillance, Evaluation, Control 17: 763–768.
- Suhendrayatna, A. O. & S. Maeda, 2001. Biotransformation of arsenite in freshwater food-chain models. Applied Organometallic Chemistry 15: 277–284.
- Suhendrayatna, A. O., T. Nakajima & S. Maeda, 2001. Metabolism and organ distribution of arsenic in the freshwater fish *Tilapia mossambica*. Applied Organometallic Chemistry 15: 566–571.
- Suhendrayatna, A. O., T. Nakajima & S. Maeda, 2002. Studies on the accumulation of arsenic in freshwater organisms II. Accumulation and transformation of arsenic compounds by *Tilapia mossambica*. Chemosphere 46: 325–331.
- Surico-Bennett, J., 1999. A risk analysis of selenium in the Salton Sea, Imperial Valley, California. M.S. Thesis, San Diego State University, San Diego (California): 96 pp.
- Swanson, S., 1986. Salton Sea fish advisory issued by authorities. Indio Daily News, Indio, California, May 8, 1986.
- Tiffany, M. A., B. K. Swan, J. M. Watts & S. H. Hurlbert, 2001. Metazooplankton dynamics in the Salton Sea, California, 1997–1999. Hydrobiologia 161: 103–120.
- Tseng, W., H. Chu, S. How, J. Fong, C. Lin & S. Yen, 1968. Prevalence of skin cancer in an endemic area of chronic arsenicosis in Taiwan. Journal of the National Cancer Institute 107: 727–729.
- U.S. CSWG, 2003. United States Cancer Statistics: 2000 Incidence. U.S. Cancer Statistics Working Group, Department of Health and Human Services, Centers for Disease Control and Prevention and National Cancer Institute, Atlanta (Georgia).
- U.S. EPA, 1980. Ambient Water Quality Criteria for DDT. U.S. Environmental Protection Agency. Criteria and Standards Division, Springfield (Virginia): EPA 440/5-80-038.
- U.S.EPA, 1985a. The carcinogenic assessment groups calculation of the carcinogenicity of dicofol (Kelthane), DDT, DDE and DDD (TDE). U.S. Environmental Protection Agency, Office of Health and Environmental Assessment Washington (D.C.) .
- U.S. EPA, 1985b. Health effects assessment for selenium (and compounds). U.S. Environmental Protection Agency, Office of Health and Environmental Assessment Washington (D.C.): NTIS PB-86-134699/AS. .
- U.S.EPA, 1987. Ambient Water Quality Criteria for Selenium. U.S. Environmental Protection Agency. Office of Water Regulations and Standards Washington (D.C.): EPA 440/5-87/006.

- U.S.EPA, 1989a. Risk Assessment Guidance for Superfund: Volume 1 – Human Health Evaluation Manual (Part A). U.S. Environmental Protection Agency, Office of Emergency Response and Remedial Response Washington (D.C.): EPA/540/1-89/002.
- U.S.EPA, 1989b. Exposure factors handbook. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment Washington (D.C.): EPA/600/08-89/043.
- U.S.EPA, 1997. Exposure Factors Handbook – Volume I. U.S. Environmental Protection Agency, Office of Research and Development Washington (D.C.): EPA 600/P-95/002Fa.
- U.S.EPA, 1998. Report on the Peer Consultation Workshop on Selenium Aquatic Toxicity and Bioaccumulation. U.S. Environmental Protection Agency, Office of Science and Technology, Office of Water, Washington (D.C.): EPA 822-R-98-007.
- U.S.EPA, 2000a. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisory. Volume I: Fish Sampling and Analysis, 3rd edition. U.S. Environmental Protection Agency, Office of Science and Technology, Office of Water, Washington (D.C.): EPA 823-B-00-007.
- U.S.EPA, 2000b. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisory. Volume II: Risk Assessment and Fish Consumption Limits, 3rd edition. U.S. Environmental Protection Agency, Office of Science and Technology, Office of Water, Washington (D.C.): EPA 823-B-00-008.
- U.S.EPA, 2002. Integrated Risk Information System database. U.S. Environmental Protection Agency Available: <http://www.epa.gov/IRIS/subst/html> (visited 2004, February 22).
- U.S. Fish and Wildlife Service, 1964. Pesticides-wildlife studies, 1963: a review of Fish and Wildlife Service investigations during the calendar year. FWS Circular 199.
- U.S. FDA, 1970. Methods of Analysis. AOAC-Eleventh edition PAM. Vol.1, Section 212. U.S. Food and Drug Administration, Washington (D. C.).
- U.S. FDA, 1994. Pesticide Analytical Manual, Vol. 1, 3rd edition, Chapter 3, Multi-residue Methods, Section 303-C1. U.S. Food and Drug Administration, Washington (D. C.).
- U.S. FDA, 1998. Action levels for poisonous or deleterious substances in human food and animal feed. U. S. Food and Drug Administration – Available: <http://vm.cfsan.fda.gov/~lrd/fdaact.html> (visited 2000, August 18).
- Vicario-Fisher, M., 1999. Total arsenic concentration in muscle tissue of *Tilapia mossambica* and *Bairdiella icistia* : A human health risk assessment for fish consumed from the Salton Sea. M.S. Thesis, San Diego State University, San Diego (California): 74 pp.
- Wainwright, S. E., M. A. Mora, J. L. Sericano & P. Thomas, 2001. Chlorinated hydrocarbons and biomarkers of exposure in wading birds and fish of the Lower Rio Grande Valley, Texas. Archives of Environmental Contamination and Toxicology 40: 101–111.
- Wang, C. & R. T. Lovell, 1997. Organic selenium sources, selenomethionine and selenoyeast, have higher bioavailability than an inorganic selenium source, sodium selenite, in diets for channel catfish (*Ictalurus punctatus*). Aquaculture 152: 223–234.
- Wiemeyer, S. N. & D. J. Hoffman, 1996. Reproduction in eastern screech owls fed selenium. Journal of Wildlife Management 60: 332–341.
- WPCL, 1999. Water Pollution Control Laboratory – Analysis of extractable synthetic organic compounds in tissue. WPCL SOP# SO-TISS Rev. 5. .
- Watts, J., B. K. Swan, M. A. Tiffany & S. H. Hurlbert, 2001. Thermal, mixing, and oxygen regimes of the Salton Sea, California, 1997–1999. Hydrobiologia 162: 159–176.
- Wilber, C. G., 1980. Toxicology of selenium: A review. Clinical Toxicology 17: 171–230.
- Winger, P. V., P. J. Lasier, D. H. White & J. T. Seginak, 2000. Effects of contaminants in dredge material from the Lower Savannah River. Archives of Environmental Contamination and Toxicology 38: 128–136.
- Winton, J. 2003. Summary of the 1996–1997 fish pathology findings. U. S. Geological Survey, Biological Resources Division, Northwest Biological Science Center. <http://pacific.fws.gov/salton/saltsum.htm> (visited 2004, January 12).