Abstract.–Exposure of developing pollock *Theragra chalcogramma* embryos to static water-soluble fractions (WSF) of Cook Inlet crude oil in seawater slowed initial development, produced shorter larvae, and caused morphological abnormalities including membranous vesicles; body curvatures; deformations of yolk, eye, brain, jaw, intestine, and pericardial sac; absence of lower jaw; fin erosion; yolksac bloating; and light pigmentation. These abnormalities were retained after hatch, and in many cases became more pronounced as developing structures failed to form properly. The median concentration of WSF that caused abnormalities (AB_{50}) was 2.1 ± 0.1 ppm. Exposure during embryonic development reduced prehatch survival by a maximum of 26%, and caused high mortality after hatch. The median lethal concentration (1.8 ± 0.6 ppm) was not significantly different than the AB_{50}. Although exposed pollock embryos generally survived to hatching, larvae were malformed, smaller, and had poorer survival potential than controls.

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Abnormal Development and Growth Reductions of Pollock *Theragra chalcogramma* Embryos Exposed to Water-Soluble Fractions of Oil

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Early life stages of fish are usually more sensitive to environmental stress than adults (Moore and Dwyer 1974, Rice 1985, Carls 1987). Planktonic eggs and larvae of demersal fish, such as Atlantic cod *Gadus morhua* and walleye pollock *Theragra chalcogramma*, are generally sensitive to dissolved oil. For example, prefeeding Atlantic cod larvae grew significantly less than controls when exposed for 2 weeks to 0.07 ppm of water-soluble fractions (WSF) of Ekofisk crude oil (Tilseth et al. 1984). The frequency of oil spills is greatest near the shore where shipping and drilling activities are concentrated and navigation hazards are more frequent. For example, the oil tanker *Exxon Valdez* grounded within Prince William Sound on 23 March 1989 with disastrous consequences. Oil spilled by drilling and transportation generally concentrates in the surface layers and, therefore, may impact nearshore areas where the planktonic eggs of Atlantic cod and walleye pollock are spawned (Kühnhold 1970, Longwell 1977).

Fish embryos respond in similar ways to a broad range of stressors (Rosenthal and Alderdice 1976, Linden et al. 1980), including the WSF of crude oils. Most sublethal effects are probably biochemical in origin, and include morphological abnormalities and reductions in growth (Rosenthal and Alderdice 1976).

In this study we measured the effects of exposure to WSF hydrocarbons on the growth and development of pelagic marine walleye pollock eggs. Walleye pollock is an abundant species in the North Pacific Ocean and comprises 20–50% of the total fish biomass in the Bering Sea (Smith 1981). Pollock are ecologically and commercially important; they are a major component of the food web, and the total commercial catch off Alaska averaged 1,013,815 t during the years of this study (1982–83) (National Marine Fisheries Service 1982, 1983).

Methods

Adult walleye pollock were collected by trawling in March 1982 and May 1983 near Juneau, Alaska. Ripe adults were transported live to the Auke Bay Laboratory. Each year, eggs and milt were stripped from a single pair and mixed in a beaker without water. Eggs were then transferred at random into 15 cm diameter × 20 cm tall cylindrical glass jars wrapped with black plastic tape and each filled with 2.5 L of either hydrocarbon free seawater (28.5 ppt) or seawater contaminated with the WSF of Cook Inlet crude oil (Table 1). After a 2-hour

Table 1
Initial concentrations of water-soluble fractions (ppm) of Cook Inlet crude oil for each treatment group of walleye pollock eggs.

	Treatment group					
	0–21 days		1–21 days		7–21 days	
Concentration	Mean	SE	Mean	SE	Mean	SE
Control	0.0		0.0		0.0	
Low	0.2	0.00	0.4	0.01	0.3	0.02
·	0.6		0.8	0.02	0.7	0.10
·	1.6	0.08	1.5	0.01	1.8	0.20
·	2.3		2.2	0.12	2.8	0.04
Maximum	3.6	0.09	2.8	0.10	3.1	0.13

Table 2
Comparison of water-soluble-fraction (WSF) preparations of Cook Inlet crude oil in treatment groups of walleye pollock eggs from years 1982 (0–21 and 7–21 day treatments) and 1983 (1–21 day treatment). Concentrations of compounds in the WSF were measured by gas chromatography and converted to percentages. $n = 3$ for each compound; asterisks denote significant differences between 1982 and 1983 percentages.

	1982		1983	
Hydrocarbon	%	SE	%	SE
benzene	50.5	0.72	51.0	0.66
toluene	34.6	1.62	30.3	0.89
ethylbenzene*	2.3	0.17	0.2	0.22
m- and p-xylene	6.2	0.45	4.5	0.27
o-xylene	3.5	0.28	2.8	0.18
mesitylene*	0.6	0.15	0.1	0.03
naphthalene*	1.2	0.15	10.5	1.87
2-methylnapthalene*	0.6	0.06	0.0	0.00
1-methylnapthalene	0.5	0.02	0.3	0.12
2,6-dimethylnapthalene	0.0	0.00	0.0	0.00
1,4-dimethylnapthalene*	0.0	0.00	0.1	0.04
1,2-dimethylnapthalene*	0.0	0.00	0.2	0.04
Total mononuclear aromatics*	97.7		88.9	
Total dinuclear aromatics*	2.3		11.1	

water-hardening period, all eggs were transferred to fresh seawater of the same treatments.

Fertilized eggs were exposed to five WSF concentrations, ranging from controls (0 ppm) through 3.6 ppm, beginning at day 0 (fertilization), day 1 (morula stage), or day 7 (11–14 somite stage) (Table 1). Exposures were static, and after the transfer at 2 hours, eggs remained in the same solution until hatch (approximately 21, 20, and 14 days, respectively). Larvae that hatched in the 1–21 day treatment were transferred to clean seawater immediately after hatch and observed until yolks were resorbed (~21 days). There were three replicates per concentration, with 49–101 eggs per replicate. Maximum biomass did not exceed 0.1 g/L in any replicate. Temperature was constant (3.9±0.1°C). Test jars were not aerated, but oxygen was not limiting (97±3% saturation at end of experiment).

Microbial growth did not cause problems. Control eggs remained healthy throughout the experiment. Any growth observed in the jars during the experiment was removed by pipette, as were dead eggs and detritus. Bacterial plates inoculated with water collected after hatch showed no significant differences between WSF and controls.

Test conditions were static, simulating a single-event oil spill, but the WSF of Cook Inlet crude oil was produced by dripping seawater through a continuously replenished 40-cm emulsified layer of oil; the resulting WSF was collected below the layer after dispersed oil floated out (Moles et al. 1985). Seawater was filtered through 5-μ glass fiber filters or approximately 10-μ sand filters before contamination with WSF. Concentrations were routinely monitored with fluorescence spectroscopy and glass-capillary-column gas chromatography as described in Carls and Rice (1988). Because polynuclear aromatic compounds were not detectable in the WSF, concentrations are reported as total mono- plus diaromatic hydrocarbons in ppm. The composition of the WSF was similar in 1982 and 1983; the principal difference was a higher percentage of naphthalene in 1983 (10.5%) than in 1982 (1.2%) (Table 2).

Developing eggs were examined daily; mortality and number hatched were recorded. A few eggs (2–10/concentration · treatment) were subsampled daily for observation and measurement. Development was staged according to Yusa (1954). Blastopore diameters were measured with an occular micrometer near the time of closure in the 1–21 day treatment. The presence or absence of morphological abnormalities was recorded. If present, the size of vesicles was coded as very small, small, medium, or large. Dead eggs were also examined for abnormalities and development stage.

Immediately after hatch, larvae were examined for abnormalities with a microscope; total body length and yolk lengths were measured with an occular micrometer, except measurements were not made in the 1–21 day treatment. Yolk lengths were measured along the major body axis, and were considered proportional to yolk volume. Mortality and the number abnormal were counted for each replicate.

Table 3

Distribution of individual aromatic hydrocarbons in water-soluble fractions of Cook Inlet crude oil during an 18-day static exposure (1–21 day treatment) of walleye pollock eggs. Concentrations are means of three replicates measured by gas chromatography.

	0 days		4 days		18 days	
Hydrocarbon	ppm	%	ppm	%	ppm	%
benzene	1.424	51.0	0.808	50.6	0.057	26.4
toluene	0.845	30.3	0.432	27.0	0.000	0.0
ethylbenzene	0.007	0.2	0.000	0.0	0.000	0.0
m- and p-xylene	0.125	4.5	0.060	3.7	0.000	0.0
o-xylene	0.077	2.8	0.041	2.6	0.000	0.0
mesitylene	0.004	0.1	0.000	0.0	0.000	0.0
naphthalene	0.292	10.5	0.243	15.2	0.153	70.8
2-methylnapthalene	0.000	0.0	0.000	0.0	0.000	0.0
1-methylnapthalene	0.008	0.3	0.007	0.4	0.000	0.0
2, 6-dimethylnapthalene	0.000	0.0	0.000	0.0	0.000	0.0
1, 4-dimethylnapthalene	0.004	0.1	0.003	0.2	0.002	0.9
1, 2-dimethylnapthalene	0.006	0.2	0.004	0.3	0.004	1.9
Total mononuclear aromatics	2.482	88.9	1.341	83.9	0.057	26.4
Total dinuclear aromatics	0.310	11.1	0.257	16.1	0.159	73.6
Total mono- and dinuclear aromatics	2.792	100.0	1.598	100.0	0.216	100.0
Percent total hydrocarbons remaining		100.0		57.2		7.7

Median lethal concentrations (LC_{50}) and median concentrations causing abnormalities (AB_{50}) were calculated with logit analysis (Berkson 1957) or Spearman-Karber analysis (Hamilton et al. 1977), corrected by control response (Abbott 1925).

Results

Test conditions

Monoaromatic hydrocarbons were initially predominant in the static WSF tests (89%), but they declined significantly more rapidly than diaromatic hydrocarbons ($P \ll 0.001$) (Table 3). After 18 days monoaromatics comprised only 26% of the remaining hydrocarbons (Table 3). The rate of total aromatic hydrocarbon loss from solution was not linear; rates were rapid initially, but slowed over time. Approximately one-half of the hydrocarbons were lost in the first 10 days. Concentrations reported in this paper are based on initial values.

Lethal effects

Egg survival and hatching success were slightly reduced (up to 17%) by exposure to WSF (Table 4). Reduction in survival and hatching success was significant in the 1–21 day treatment, but not in the 0–21 day and 7–21 day treatments. Mortality after hatch was strongly dependent on concentration (Fig. 1) and began to differ significantly from controls 29 days after fertilization, or about 10 days after hatch. The LC_{50} was 2.2 ± 0.8 ppm 10 days after hatch and stabilized at 1.8 ± 0.6 ppm 16 days after hatch.

Growth

Exposure to WSF before blastopore closure slowed early embryonic development. Blastopores in control embryos closed earlier than in embryos exposed to WSF (≥ 1.65 ppm) in the 0–21 day treatment (Table 5). Embryos in the 1-21 day treatment tended to have larger diameters at high WSF concentrations, but diameters were highly variable (Table 6). Pore diameters at 2.7 ppm were significantly greater than control diameters, but were significantly smaller at the lowest concentration (0.4 ppm). After blastopore closure, embryonic development in the upper concentrations could not be distinguished from control development, and the time of hatch was not influenced by exposure to WSF (Fig. 2).

Yolk and body lengths of larvae that hatched from eggs in the 0–21 day and 7–21 day treatments were significantly reduced by exposure to WSF ($P \leq 0.03$ [untransformed ANOVA]). (Lengths were not measured in the 1–21 day treatment.) Yolk lengths were reduced a maximum of 9%, total body lengths were reduced a maximum of 23% (Fig. 3), and the yolk-to-length ratio increased a maximum of 20%. Mean yolk lengths at concentrations ≥ 2.8 ppm were significantly ($P < 0.05$) smaller than controls. Reductions in larval body length in both treatments overlapped closely

Table 4

Walleye pollock egg survival and hatching success for treatment groups exposed to water-soluble fractions of Cook Inlet crude oil. ANOVA indicates significance of survival and hatching by analysis of variance; arc sine transformations were used with proportional data. NS $P>005$, $^{**}P \leqslant 0.01$, $^{***}P \leqslant 0.005$, † significant differences from control (Dunnette test, 95% confidence), and ±95% confidence interval.

0–21 day treatment				1–21 day treatment				7–21 day treatment			
ppm	n	% survival	% hatch	ppm	n	% survival	% hatch	ppm	n	% survival	% hatch
0.00	3	95.3 ± 5.0	88.0 ± 5.3	0.00	3	98.1 ± 5.2	97.1 ± 6.9	0.00	3	100.0 ± 0.0	100.0 ± 0.0
0.19	3	93.9 ± 8.6	86.9 ± 15.8	0.41	3	97.9 ± 6.4	96.8 ± 11.1	0.26	3	99.2 ± 3.5	99.2 ± 3.5
0.57	3	95.3 ± 8.3	87.4 ± 2.3	0.82	3	95.5 ± 6.9	95.5 ± 6.9	0.68	3	98.6 ± 3.7	98.6 ± 3.7
1.65	3	95.5 ± 5.0	84.9 ± 7.8	1.53	3	92.1 ± 2.5	89.7 ± 2.8	1.83	3	98.7 ± 3.0	98.7 ± 3.0
2.30	3	95.0 ± 8.3	87.6 ± 11.7	2.17	3	85.7 ± 11.3†	85.7 ± 11.2†	2.80	3	95.3 ± 1.3	95.3 ± 1.3
3.59	3	91.0 ± 4.1	84.3 ± 11.4	2.75	3	81.9 ± 17.0†	80.4 ± 18.4†	3.14	3	95.2 ± 16.1	95.2 ± 16.1
ANOVA		NS	NS			***	**			NS	NS

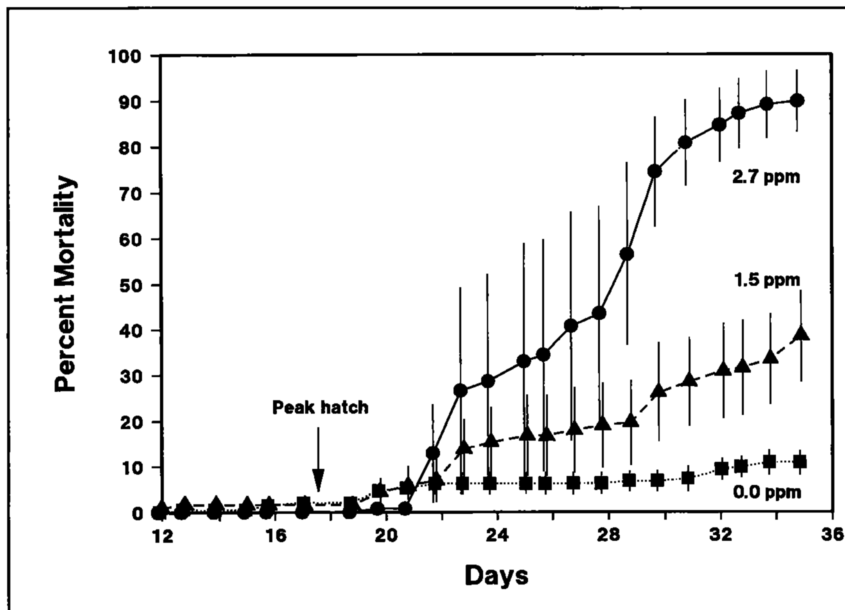

Figure 1

Percent mortality of walleye pollock larvae after hatch when embryos were exposed to water-soluble fractions of Cook Inlet crude oil (1–21 day treatment). Vertical bars are standard error.

Table 5

Blastopore closure in walleye pollock embryos 6 days after fertilization in the 0–21 day exposure to water-soluble fractions of Cook Inlet crude oil.

ppm	n	n closed	% closed
0.00	6	6	100.0
0.19	8	8	100.0
0.57	8	8	100.0
1.65	8	7	87.5
2.30	8	0	0.0
3.59	8	0	0.0

Table 6

Blastopore diameters of walleye pollock embryos 5 days after fertilization in the 1–21 day exposure to water-soluble fractions of Cook Inlet crude oil. ANOVA indicated significance of blastopore diameters by analysis of variance; $^{***}P \leqslant 0.005$, † significant differences from control (Dunnette test, 95% confidence), and ±95% confidence interval.

ppm	n	Diameter (mm)
0.00	30	0.13 ± 0.03
0.41	30	0.09 ± 0.02†
0.82	30	0.15 ± 0.02
1.53	30	0.15 ± 0.03
2.17	30	0.16 ± 0.02
2.75	30	0.19 ± 0.02†
ANOVA		***

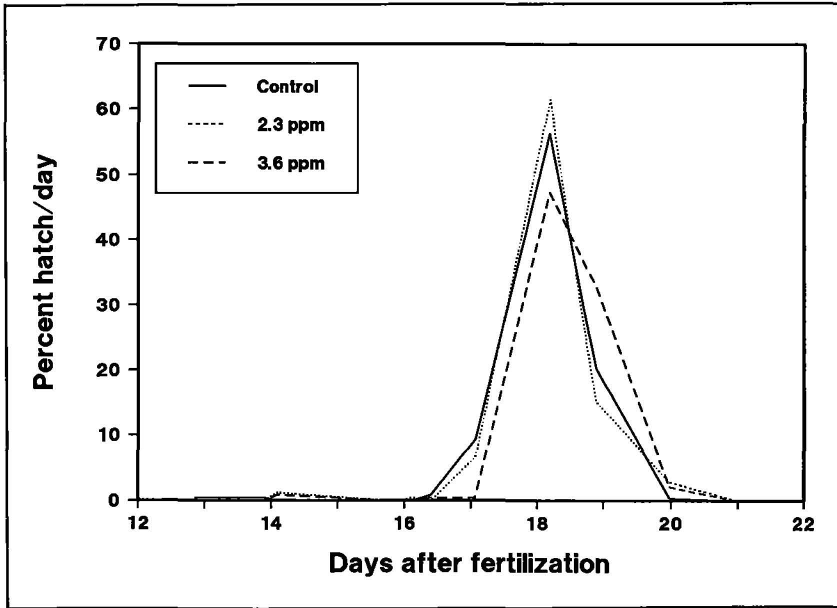

Figure 2
Timing of hatch (% per day) of walleye pollock when eggs were exposed to water-soluble fractions of Cook Inlet crude oil in the 0–21 day treatment.

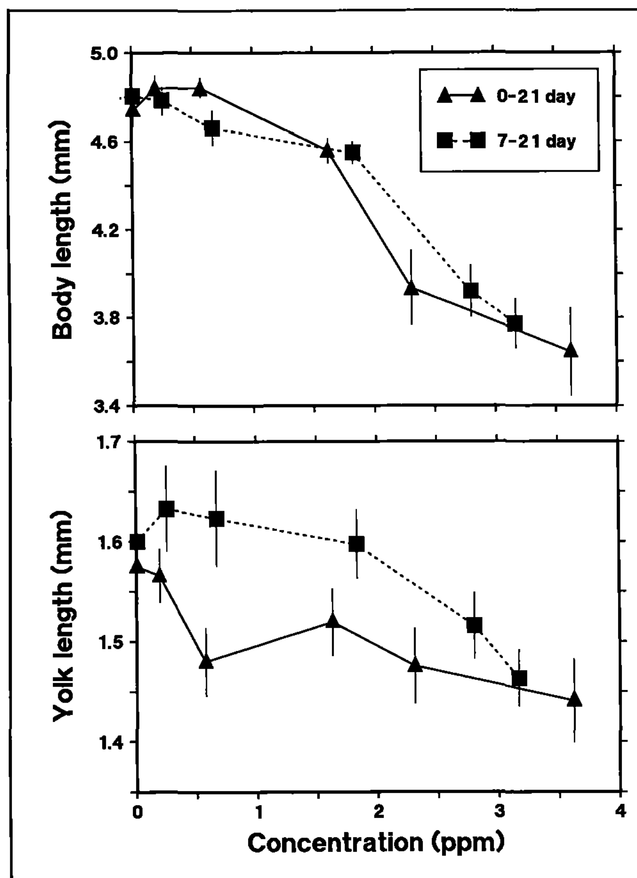

Figure 3
Length of yolks (measured along the major body axis) and total larval body lengths of walleye pollock plotted against concentrations of water-soluble fraction of Cook Inlet oil to which eggs were exposed. Vertical bars indicate 95% confidence interval.

(Fig. 3). Mean body lengths at concentrations ≥2.3 ppm were significantly ($P<0.05$) smaller than controls.

Abnormalities

Embryos exposed to WSF developed abnormalities. The earliest detected abnormality was the formation of membranous vesicles about the time of blastopore closure (days 6–9). In the 0–21 day treatment, embryos developed 1–5 vesicles along the ventral surface, usually posterior near the blastopore (Fig. 4). These vesicles were roughly spherical, apparently formed by a single membrane. Interior fluid was indistinguishable from surrounding fluid by observation of living specimens with a microscope. Frequency of occurrence and quantity of vesicles were correlated with concentration ($r=0.82$ and $r=0.67$, respectively), and vesicle size tended to increase with concentration (Table 7). The median concentration causing vesicle abnormalities was 2.4±0.4 ppm. Vesicle formation was not observed in the 1–21 day or 7–21 day treatments; embryos in the 7–21 day treatment were beyond blastopore closure when exposure began.

Yolksac abnormalities were observed in exposed eggs shortly before hatch and other abnormalities occurred at and after hatch (Table 8). These abnormalities included body curvatures; deformations of yolk, eye, brain, jaw, intestine, and pericardial sac; absence of lower jaw; fin erosion; yolksac bloating; and light pigmentation. Yolksac bloating caused inverted floating. Correlation of abnormalities with concentration was strong ($r=0.9$ [logit transformation]). Correlation between abnormalities at hatch and vesicle

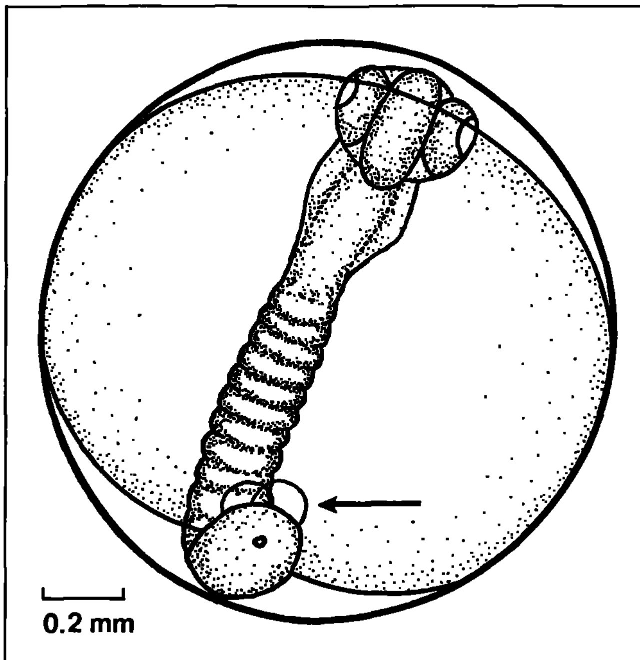

Figure 4
Walleye pollock embryos exposed to water-soluble fraction (3.6 ppm in the embryo shown) of Cook Inlet crude oil developed abnormal vesicles (arrow) along the ventral surface, usually posterior near the blastopore.

abnormalities was also high ($r=0.99$). Median concentrations causing abnormalities at hatch ranged from 1.6 ± 0.2 to 2.1 ± 0.6 ppm (Table 8).

Deformed larvae did not recover from their abnormalities in clean water. In many cases the abnormalities became more pronounced as developing structures failed to form properly: 21 days after hatch a few abnormal larvae were still alive, but most (88%) had died (control mortality was 10%).

Discussion

Early embryonic development, as indicated by differences in blastopore diameters, was delayed by exposure to WSF, but the timing of hatch was not affected. Developmental stages (Yusa 1954) of controls and treated embryos were similar at all times except during blastopore closure. In other studies involving WSF tests of crude oils (Venezuelan, Iranian, Libyan), development of cod *Gadus morhua* embryos slowed, depending on concentration (Kühnhold 1974).

Genetic differences or differences in WSF preparations may account for the greater sensitivity observed in 1983 than in 1982. The WSF contained a higher proportion of diaromatic compounds in 1983 than in 1982, and diaromatics are generally more toxic than monoaromatics (Rice et al. 1977). However, these sensitivity differences were only in magnitude of response; basic response patterns were the same.

Increased metabolic demand may explain the observed reductions in yolk sizes of newly hatched larvae exposed to WSF during embryonic development. Yolk size was negatively correlated with concentration in both treatments, and yolk sizes tended to be smaller in embryos exposed for the longest period of time (0–21 day treatment), suggesting increased consumption of yolk and elevated metabolism. Linden (1978) also concluded that shorter lengths of larval Baltic herring *Clupea harengus membras* at hatching probably resulted from increased energy demands during exposure to the WSF of several oils. Other researchers have measured increases in metabolic rate directly; for

Table 7
Occurrence of vesicle abnormalities in developing walleye pollock embryos near time of blastopore closure in the 0–21 day exposure to water-soluble fraction of Cook Inlet crude oil. ANOVA indicates significance of abnormalities by analysis of variance; arc sine transformations were used with proportional data (% occurrence) or a square root transformation to control heterogeneity in the no./embryo test; *$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.005$, † significant differences from control (Dunnette test, 95% confidence). Size codes: vs = very small, s = small, m = medium, and l = large. Large vesicles were 8.2% of the egg diameter (~0.11 mm).

	% occurrence			*n*/embryo			
ppm	*n*	$\bar{X}$	SE	Range	$\bar{X}$	SE	Relative size
0.00	14	0	(0)	0–0	0.0	(0.0)	–
0.19	17	0	(0)	0–0	0.0	(0.0)	–
0.57	16	0	(0)	0–0	0.0	(0.0)	–
1.65	16	18	(6)	0–1	0.2	(0.1)	vs–m
2.30	16	75	(25)†	0–2	1.1	(0.2)†	s–l
3.59	14	78	(11)†	0–5	1.7	(0.4)†	m–l
ANOVA		**			***		
Regression	$r=0.82$ *			$r=0.67$ **			

Table 8

Percentage of walleye pollock larvae abnormal at the time of hatch following treatment in water-soluble fractions of Cook Inlet crude oil. ANOVA indicates significance of abnormalities by analysis of variance; arc sine transformations were used with proportional data. ***$P \leq 0.005$, † significant differences from control (Dunnette test, 95% confidence), and ±95% confidence.

0–21 day treatment			1–21 day treatment			7–21 day treatment		
ppm	*n*	% abnormal	ppm	*n*	% abnormal	ppm	*n*	% abnormal
0.00	3	2.4 ± 3.7	0.00	3	1.0 ± 4.2	0.00	3	6.4 ± 5.1
0.19	3	5.3 ± 9.5	0.41	3	1.0 ± 2.3	0.26	3	8.6 ± 13.4
0.57	3	5.4 ± 6.0	0.82	3	2.3 ± 7.0	0.68	3	6.6 ± 23.2
1.65	3	10.6 ± 13.6	1.53	3	51.2 ± 2.7†	1.83	3	16.4 ± 10.7
2.30	3	86.4 ± 5.5†	2.16	3	73.6 ± 15.4†	2.80	3	91.8 ± 12.4†
3.59	3	99.5 ± 2.2†	2.75	3	90.3 ± 11.5†	3.14	3	90.9 ± 9.2†
ANOVA		***			***			***

example, metabolic rates were elevated when larval Pacific herring *Clupea harengus pallasi* and northern anchovy *Engraulis mordax* were exposed to benzene (Struhsaker et al. 1974).

Decreases in larval size and yolk reserves caused by sublethal exposure of developing fish eggs to WSF potentially reduces survival potential. Predator avoidance capability tends to increase exponentially with larval length; however, decreases in avoidance behavior may be important only when the difference in size between predator and prey is small (Hunter 1981). Larvae with smaller yolk reserves generally have less time to begin feeding before onset of irreversible starvation (Blaxter and Hempel 1963).

The abnormalities observed in this study are the same, or similar, to those observed in other studies with a variety of species (Rosenthal and Alderdice 1976; Linden 1976; Kühnhold 1974, 1977) and support the observation by Rosenthal and Alderdice (1976) that fish embryos tend to respond developmentally in similar ways to external stress. Some of the developmental abnormalities (vesicle formation, body curvatures, and bloated yolks) caused by exposure of pollock eggs to WSF in this experiment were also caused by elevated temperatures in a pollock egg study by K. Krieger and L. Sonenberg (Auke Bay Lab., Alaska Fish. Sci. Cent., Natl. Mar. Fish. Serv., NOAA, P.O. Box 210155, Auke Bay, AK 99821, pers. commun. May 1988). Cold temperatures may cause similar problems in herring larvae: abnormal optic vesicles, enlarged pericardial areas, and jaw abnormalities (Ojaveer 1981). Lighter pigmentation in exposed cod *Gadus morhua* embryos has been observed in studies involving hydroxylated aromatic hydrocarbons (Falk-Petersen et al. 1985).

Ion imbalance due to changes in membrane osmoregulation (Ernst and Neff 1977, Linden 1978) provides a good explanation for yolksac and pericardial deformities, because yolksac volumes are related to water content (Rosenthal and Alderdice 1976). Aromatic hydrocarbons alter the surface properties of cell membranes, possibly modifying their permeability (Roubal and Collier 1975, Aronovich et al. 1975). Structural changes in cell and mitochondrial membranes of larval mummichogs *Fundulus heteroclitus* exposed to the WSF of Prudhoe Bay crude oil occurred even in larvae which did not show visible abnormalities (Cameron and Smith 1980).

Previous cell damage, rather than the continued presence of sequestered hydrocarbons, is most likely responsible for the development of abnormalities after hatch, because newly hatched larvae were transferred to clean water and most of the hydrocarbon depurated rapidly (59–83% in the first 8 hours; Carls and Rice 1988). In another study, cod larvae also depurated naphthalene quickly, but heavier organic compounds (phenanthrene, benzo(a)pyrene, and 2,4,5,2′,4′,5′-hexachlorobiphenyl) were depurated progressively more slowly as molecular weights increased (Solbakken et al. 1984). In our study, however, compounds with molecular weights greater than the methylnaphthalenes were present only at extremely low concentrations, if at all. An indication that the damage happened early in development, but was expressed later as malformations, was the occurrence of abnormal membranous vesicles 6–9 days after fertilization. At hatch, other malformations were observed which could have been the result of the same or similar mechanisms responsible for vesicle formation, and correlation between early and later abnormalities was very high ($r = 0.99$).

The significant mortality of larvae exposed to WSF during egg development was probably caused by biochemical changes, structural malformations, or disruption of tissue and organ development rather than a continued presence of hydrocarbons or insufficient yolk reserves. Larval mortality did not exceed 2%

until 3 days after the majority (89%) of the hatch was complete, ample time for most sequestered hydrocarbons to be depurated (Carls and Rice 1988). Some abnormal larvae (12% at 2.7 ppm) survived on their yolksac energy reserves to the end of the experiment (21 days after hatch), suggesting that depletion of endogenous energy was not responsible for mortalities occurring soon after hatch.

Oil accidently spilled in the marine environment rarely reaches concentrations (0.4–2.3 ppm) necessary to cause the effects observed in this study. However, the probability of oil spills is greater in nearshore waters, and this oil tends to concentrate in surface layers where walleye pollock eggs occur. For example, in the Bering Sea pollock eggs are spawned at depth and rise to the pelagic zone (Incze et al. 1984). Pollock eggs were most abundant in the upper 5–10 m, but abundance of older eggs (stage IV–VI) peaked about 20 m (Nishiyama et al. 1986, Serobaba 1974).

Large, single-event, nearshore oil spills have released sufficient quantities of hydrocarbons to affect planktonic fish eggs. For example, after the *Amoco Cadiz* spill, water entering the Aber Wrac'h estuary contained more than 1 ppm hydrocarbons and 0.5 ppm throughout the estuary (Calder and Boehm 1981). After the *Argo Merchant* grounded on Nantucket Shoals, about one-half the chorions of cod *Gadus morhua* and pollock *Pollachius virens* eggs were contaminated with oil droplets or tar, and approximately 20–46% of the eggs were dead or moribund, compared with a 4% control mortality (laboratory-spawned cod) (Longwell 1977).

In conclusion, exposure of pelagic marine walleye pollock eggs to WSF during development can cause embryos to develop abnormalities, and reduces size. Although they generally survive and hatch, embryos exposed to WSF produce abnormal larvae that have poor survival potential.

Acknowledgments

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