Table3 Numbers, SMRs, and 95 % CIs for non-cancer disease from 1960 to 1978 in the exposed area (Tamanoura and Naru) and Shimo-Goto region excluding the exposed area

	All causes						Diabetes mellitus					
	Tamanoura		Naru		Shimo-Goto[a]		Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
1963 (−5)	62	1.1 (0.8, 1.4)	60	0.9 (0.7, 1.2)	642	1.0 (0.9, 1.1)	0	NE	2	8.4 (2.1, 33.5)	2	0.8 (0.2, 3.3)
1964 (−4)	69	1.3 (1.0, 1.6)	52	0.9 (0.7, 1.1)	567	0.9 (0.9, 1.0)	1	3.8 (0.5, 26.8)	0	NE	1	0.3 (0.0, 2.5)
1965 (−3)	60	1.1 (0.8, 1.4)	52	0.8 (0.6, 1.1)	628	1.0 (0.9, 1.1)	0	NE	0	NE	6	1.8 (0.8, 4.0)
1966 (−2)	47	0.9 (0.7, 1.2)	39	0.7 (0.5, 0.9)	597	1.0 (0.9, 1.1)	0	NE	0	NE	0	NE
1967 (−1)	43	0.8 (0.6, 1.0)	53	0.9 (0.7, 1.1)	619	1.0 (0.9, 1.1)	0	NE	0	NE	9	2.6 (1.4, 5.0)
1968 (0)	57	1.2 (0.9, 1.5)	57	1.0 (0.8, 1.3)	576	1.0 (0.9, 1.1)	NA		NA		NA	
1969 (1)	70	1.5 (1.2, 1.9)	46	0.8 (0.6, 1.1)	589	1.1 (1.0, 1.2)	3	8.8 (2.8, 27.2)	0	NE	2	0.5 (0.1, 2.1)
1970 (2)	62	1.3 (1.0, 1.6)	51	0.9 (0.7, 1.2)	585	1.0 (0.9, 1.1)	0	NE	0	NE	6	1.2 (0.5, 2.7)
1971 (3)	45	1.0 (0.8, 1.4)	39	0.8 (0.6, 1.0)	471	0.9 (0.8, 1.0)	1	2.1 (0.3, 14.6)	1	1.9 (0.3, 13.3)	12	2.2 (1.3, 3.9)
1972 (4)	67	1.5 (1.2, 1.9)	54	1.0 (0.8, 1.3)	476	0.9 (0.8, 1.0)	0	NE	0	NE	6	1.1 (0.5, 2.4)
1973 (5)	42	1.0 (0.7, 1.4)	46	0.9 (0.7, 1.3)	508	1.0 (0.9, 1.1)	0	NE	0	NE	0	NE
1974 (6)	42	1.0 (0.7, 1.3)	52	1.0 (0.8, 1.4)	501	1.0 (0.9, 1.1)	0	NE	0	NE	2	0.4 (0.1, 1.7)
1975 (7)	44	1.1 (0.8, 1.4)	40	0.8 (0.6, 1.1)	493	1.0 (0.9, 1.1)	0	NE	0	NE	6	1.1 (0.5, 2.5)
1976 (8)	42	1.0 (0.7, 1.3)	49	1.0 (0.8, 1.3)	511	1.0 (0.9, 1.1)	1	2.5 (0.3, 17.5)	0	NE	3	0.6 (0.2, 1.9)
1977 (9)	34	0.8 (0.6, 1.1)	63	1.3 (1.0, 1.7)	484	1.0 (0.9, 1.0)	3	5.9 (1.9, 18.2)	2	3.3 (0.8, 13.3)	4	0.6 (0.2, 1.7)
1978 (10)	45	1.2 (0.9, 1.6)	47	1.0 (0.8, 1.4)	499	1.1 (1.0, 1.2)	1	2.1 (0.3, 14.8)	1	1.8 (0.2, 12.6)	3	0.5 (0.2, 1.6)
	Heart diseases						Hypertensive diseases					
	Tamanoura		Naru		Shimo-Goto[a]		Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
1963 (−5)	7	1.2 (0.6, 2.6)	5	0.8 (0.3, 2.0)	61	1.0 (0.8, 1.2)	1	1.3 (0.2, 9.4)	3	3.9 (1.3, 12.1)	15	1.8 (1.1, 3.0)
1964 (−4)	10	1.8 (1.0, 3.4)	1	0.2 (0.0, 1.2)	53	0.9 (0.7, 1.2)	1	1.4 (0.2, 9.7)	0	NE	8	1.0 (0.5, 2.0)
1965 (−3)	9	1.6 (0.9, 3.2)	3	0.5 (0.2, 1.6)	83	1.4 (1.1, 1.7)	5	7.7 (3.2, 18.5)	3	4.5 (1.4, 13.9)	28	3.9 (2.7, 5.7)
1966 (−2)	3	0.5 (0.2, 1.7)	2	0.3 (0.1, 1.4)	45	0.7 (0.6, 1.0)	2	2.2 (0.6, 8.8)	0	NE	13	1.3 (0.8, 2.3)
1967 (−1)	6	1.0 (0.4, 2.2)	2	0.3 (0.1, 1.3)	58	0.9 (0.7, 1.1)	3	3.1 (1.0, 9.5)	0	NE	16	1.5 (0.9, 2.5)
1968 (0)	11	2.0 (1.1, 3.6)	6	0.9 (0.4, 2.1)	63	1.0 (0.8, 1.3)	1	0.6 (0.1, 3.9)	3	1.5 (0.5, 4.6)	24	1.2 (0.8, 1.7)
1969 (1)	12	2.0 (1.2, 3.6)	3	0.4 (0.1, 1.4)	84	1.2 (1.0, 1.5)	3	1.8 (0.6, 5.6)	2	1.1 (0.3, 4.3)	24	1.3 (0.8, 1.9)
1970 (2)	4	0.6 (0.2, 1.7)	2	0.3 (0.1, 1.1)	72	1.0 (0.8, 1.2)	4	2.0 (0.8, 5.3)	0	NE	17	0.7 (0.5, 1.2)
1971 (3)	3	0.5 (0.2, 1.7)	4	0.6 (0.2, 1.7)	55	0.8 (0.7, 1.1)	0	NE	3	1.5 (0.5, 4.5)	16	0.8 (0.5, 1.2)
1972 (4)	5	0.9 (0.4, 2.2)	3	0.5 (0.2, 1.5)	57	0.9 (0.7, 1.1)	5	2.7 (1.1, 6.4)	6	2.9 (1.3, 6.4)	19	0.9 (0.6, 1.4)
1973 (5)	4	0.7 (0.3, 1.9)	1	0.2 (0.0, 1.1)	54	0.8 (0.6, 1.1)	1	0.5 (0.1, 3.5)	1	0.5 (0.1, 3.2)	36	1.5 (1.1, 2.1)
1974 (6)	8	1.4 (0.7, 2.8)	1	0.2 (0.0, 1.1)	53	0.8 (0.6, 1.0)	2	0.9 (0.2, 3.7)	1	0.4 (0.1, 3.0)	23	0.9 (0.6, 1.3)
1975 (7)	11	1.9 (1.0, 3.4)	8	1.2 (0.6, 2.4)	78	1.1 (0.9, 1.4)	2	1.0 (0.2, 3.8)	0	NE	22	0.9 (0.6, 1.3)

Table3 continued

	Heart diseases						Hypertensive diseases					
	Tamanoura		Naru		Shimo-Goto[a]		Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
1976 (8)	7	1.1 (0.5, 2.3)	10	1.4 (0.7, 2.6)	75	1.0 (0.8, 1.2)	1	0.4 (0.1, 3.2)	0	NE	11	0.4 (0.2, 0.7)
1977 (9)	2	0.3 (0.1, 1.2)	10	1.4 (0.7, 2.5)	71	0.9 (0.7, 1.1)	2	0.9 (0.2, 3.7)	0	NE	24	0.9 (0.6, 1.4)
1978 (10)	10	1.7 (0.9, 3.2)	8	1.1 (0.6, 2.3)	80	1.1 (0.9, 1.4)	0	NE	0	NE	16	0.7 (0.4, 1.2)
	Cerebrovascular diseases						Pneumonia and bronchitis					
	Tamanoura		Naru		Shimo-Goto[a]		Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
1963 (−5)	10	0.8 (0.4, 1.5)	6	0.5 (0.2, 1.0)	108	0.8 (0.7, 0.9)	3	0.8 (0.2, 2.4)	12	2.6 (1.5, 4.5)	73	1.6 (1.3, 2.1)
1964 (−4)	14	1.0 (0.6, 1.7)	14	1.0 (0.6, 1.7)	126	0.8 (0.7, 1.0)	8	2.4 (1.2, 4.9)	7	1.8 (0.9, 3.8)	61	1.6 (1.3, 2.1)
1965 (−3)	9	0.7 (0.3, 1.3)	18	1.3 (0.8, 2.0)	114	0.8 (0.6, 0.9)	6	1.5 (0.7, 3.3)	5	1.1 (0.5, 2.7)	73	1.6 (1.3, 2.0)
1966 (−2)	14	1.0 (0.6, 1.7)	14	1.0 (0.6, 1.7)	138	0.9 (0.8, 1.1)	1	0.3 (0.0, 2.5)	4	1.2 (0.5, 3.3)	48	1.5 (1.1, 2.0)
1967 (−1)	13	0.9 (0.5, 1.6)	19	1.3 (0.8, 2.1)	134	0.9 (0.7, 1.0)	2	0.7 (0.2, 2.7)	7	2.1 (1.0, 4.4)	42	1.3 (0.9, 1.7)
1968 (0)	13	1.1 (0.6, 1.8)	14	1.0 (0.6, 1.7)	147	1.1 (0.9, 1.2)	NA		NA		NA	
1969 (1)	13	0.9 (0.5, 1.6)	9	0.6 (0.3, 1.1)	156	1.0 (0.8, 1.1)	1	0.5 (0.1, 3.2)	4	1.5 (0.6, 4.1)	28	1.1 (0.7, 1.5)
1970 (2)	14	1.1 (0.7, 1.9)	11	0.8 (0.4, 1.4)	145	1.0 (0.9, 1.2)	6	2.2 (1.0, 4.9)	2	0.6 (0.2, 2.5)	27	0.8 (0.6, 1.2)
1971 (3)	4	0.4 (0.1, 0.9)	6	0.5 (0.2, 1.1)	119	0.9 (0.8, 1.1)	1	0.5 (0.1, 3.3)	1	0.4 (0.1, 2.8)	14	0.6 (0.3, 0.9)
1972 (4)	18	1.5 (1.0, 2.4)	8	0.6 (0.3, 1.2)	130	1.0 (0.8, 1.2)	3	1.3 (0.4, 4.0)	3	1.1 (0.4, 3.4)	17	0.6 (0.4, 1.0)
1973 (5)	7	0.7 (0.3, 1.4)	15	1.2 (0.7, 2.1)	129	1.0 (0.8, 1.2)	4	1.9 (0.7, 4.9)	7	2.9 (1.4, 6.0)	30	1.1 (0.8, 1.6)
1974 (6)	9	0.9 (0.4, 1.6)	7	0.6 (0.3, 1.2)	125	1.0 (0.8, 1.2)	4	1.5 (0.6, 4.1)	7	2.4 (1.1, 5.0)	27	0.8 (0.6, 1.2)
1975 (7)	12	1.2 (0.7, 2.0)	7	0.6 (0.3, 1.2)	99	0.8 (0.7, 1.0)	2	0.8 (0.2, 3.2)	4	1.4 (0.5, 3.8)	21	0.7 (0.4, 1.0)
1976 (8)	13	1.3 (0.7, 2.2)	11	0.9 (0.5, 1.7)	123	1.0 (0.8, 1.2)	0	NE	2	0.7 (0.2, 2.7)	42	1.3 (1.0, 1.8)
1977 (9)	14	1.4 (0.8, 2.4)	5	0.5 (0.2, 1.1)	109	0.9 (0.8, 1.1)	1	0.4 (0.1, 3.0)	2	0.8 (0.2, 3.1)	19	0.7 (0.4, 1.1)
1978 (10)	6	0.7 (0.3, 1.5)	6	0.6 (0.3, 1.3)	103	1.0 (0.8, 1.2)	2	0.9 (0.2, 3.6)	11	4.2 (2.3, 7.6)	25	0.9 (0.6, 1.3)
	Liver disease						Nephritic disorders					
	Tamanoura		Naru		Shimo-Goto[a]		Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
1963 (−5)	6	5.6 (2.5, 12.5)	1	0.9 (0.1, 6.0)	10	0.9 (0.5, 1.6)	1	0.8 (0.1, 5.9)	2	1.5 (0.4, 5.9)	13	1.0 (0.6, 1.6)
1964 (−4)	3	2.9 (0.9, 8.9)	1	0.9 (0.1, 6.2)	11	1.0 (0.5, 1.7)	0	NE	1	0.9 (0.1, 6.2)	8	0.7 (0.3, 1.4)
1965 (−3)	1	1.1 (0.1, 7.5)	4	3.8 (1.4, 10.1)	12	1.2 (0.7, 2.0)	1	0.9 (0.1, 6.6)	1	0.8 (0.1, 5.8)	13	1.1 (0.6, 1.8)
1966 (−2)	1	1.0 (0.1, 6.7)	0	NE	10	0.9 (0.5, 1.6)	3	3.5 (1.1, 10.8)	1	1.0 (0.1, 7.0)	31	3.1 (2.2, 4.5)
1967 (−1)	1	0.9 (0.1, 6.4)	0	NE	16	1.3 (0.8, 2.1)	1	1.3 (0.2, 9.1)	0	NE	5	0.6 (0.2, 1.4)
1968 (0)	0	NE	0	NE	0	NE	NA		NA		NA	

Table3 continued

	Liver disease						Nephritic disorders					
	Tamanoura		Naru		Shimo-Goto[a]		Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
1969 (1)	1	0.9 (0.1, 6.7)	2	1.6 (0.4, 6.5)	9	0.7 (0.4, 1.4)	0	NE	3	3.3 (1.1, 10.3)	8	0.9 (0.5, 1.8)
1970 (2)	1	0.9 (0.1, 6.3)	1	0.8 (0.1, 5.3)	8	0.6 (0.3, 1.2)	0	NE	1	1.3 (0.2, 8.9)	11	1.4 (0.8, 2.6)
1971 (3)	1	1.0 (0.1, 7.3)	1	0.9 (0.1, 6.1)	14	1.3 (0.7, 2.1)	0	NE	0	NE	6	1.0 (0.5, 2.2)
1972 (4)	2	1.9 (0.5, 7.5)	1	0.8 (0.1, 5.6)	9	0.7 (0.4, 1.4)	0	NE	0	NE	9	1.5 (0.8, 2.9)
1973 (5)	0	NE	0	NE	9	0.8 (0.4, 1.5)	0	NE	0	NE	2	0.4 (0.1, 1.7)
1974 (6)	0	NE	1	0.8 (0.1, 5.4)	19	1.5 (0.9, 2.3)	1	2.5 (0.4, 18.1)	2	4.1 (1.0, 16.2)	3	0.6 (0.2, 1.9)
1975 (7)	0	NE	0	NE	11	1.0 (0.5, 1.7)	0	NE	0	NE	4	0.9 (0.3, 2.4)
1976 (8)	1	1.0 (0.1, 7.4)	0	NE	15	1.3 (0.8, 2.2)	0	NE	1	2.1 (0.3, 14.8)	1	0.2 (0.0, 1.5)
1977 (9)	0	NE	0	NE	8	0.7 (0.3, 1.4)	0	NE	0	NE	3	0.7 (0.2, 2.1)
1978 (10)	4	4.5 (1.7, 11.9)	0	NE	11	1.0 (0.5, 1.8)	2	4.5 (1.1, 18.0)	1	1.8 (0.3, 12.9)	8	1.4 (0.7, 2.9)

95 % CI 95 % confidence interval, *NA* not available, *NE* not evaluated, *Obs* observation number, *SMR* standard mortality ratio

[a] Shimo-Goto was excluded from the exposed area (Tamanoura and Naru)

cancer (30–34 years), and leukemia (after 10 years) were significantly elevated.

For reference, the number of cases and SMRs for cancer from 1963 to 2002 at shorter length intervals (5 years) are shown in Online Table 4. The SMRs by short interval, all-cancer mortality in Tamanoura was sporadically elevated after 15–19 years. At this same time, elevated SMRs in the comparison area of Shimo-Goto were also observed. Regarding leukemia, high SMRs were observed after 5 years in Naru.

Discussion

The present study evaluated the acute and long-term health effects of ingestion of rice oil contaminated with PCBs and PCDFs in two severely affected areas (Tamanoura and Naru). After accounting for existing trends in mortality before the incident, as well as the situation in other areas within the same archipelago, SMRs for all-cause mortality, diabetes mellitus, and heart disease were found to have increased in Tamanoura shortly after the outbreak. In addition, SMRs for several cancers were found to be higher in the long term.

Our study's novel finding was that SMRs for all cancer, uterine cancer, and leukemia were also found to have increased in the long term (Table 5). First, SMRs for all-cancer mortality were significantly elevated in both exposed areas at 30–34 years. This was consistent with a previous Yusho study. In a previous 40-year follow-up study among officially certified Yusho patients, the SMR for all cancer between 1968 and 2007 had significantly increased (SMR: 1.37, 95 % CI 1.11–1.66) (Onozuka et al. 2009). In the present study, however, SMRs in the comparison area of Shimo-Goto were also significantly elevated. Although we could not remove the effects of area-specific characteristics of the Goto archipelago from these results, further evaluation, which target residents who ingested the contaminated rice oil, should be conducted since these people might have moved to other areas away from the exposure area after such a long period.

SMRs for uterine cancer were significantly higher in Tamanoura 30–34 years after the incident, but not in the comparison area. These elevated SMRs were also observed over the whole exposed area. In a previous 40-year follow-up study among officially certified Yusho patients, the SMR for uterine cancer between 1968 and 2007 had not significantly increased (SMR: 1.14, 95 % CI 0.24–3.33) (Onozuka et al. 2009). In Seveso, Italy, where severe exposure to 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD) occurred, reported SMRs for uterine cancer had not significantly increased. For example, while two cohort studies did not observe elevated SMRs for uterine cancer

Table 4 Numbers, SMRs, and 95 % CIs for non-cancer disease for whole period before the accident (1963–1937) and after the period (1668–1978 in the exposed area (Tamanoura and Naru) and Shimo-Goto region excluding the exposed area

	Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
All causes						
Before the accident (1963–1967)	281	1.0 (0.9, 1.1)	256	0.8 (0.7, 1.0)	3,053	1.0 (1.0, 1.0)
After the accident (1968–1977)	505	1.1 (1.0, 1.2)	497	1.0 (0.9, 1.0)	5,194	1.0 (1.0, 1.0)
Diabetes mellitus						
Before the accident (1963–1967)	1	0.7 (0.1, 5.2)	2	1.3 (0.3, 5.4)	18	1.2 (0.8, 1.9)
After the accident (1968–1977)	8	2.1 (1.0, 4.1)	3	0.7 (0.2, 2.1)	41	0.9 (0.7, 1.2)
Heart diseases						
Before the accident (1963–1967)	35	1.2 (0.9, 1.7)	13	0.4 (0.3, 0.8)	300	1.0 (0.9, 1.1)
After the accident (1968–1977)	67	1.1 (0.9, 1.4)	48	0.7 (0.5, 1.0)	662	0.9 (0.9, 1.0)
Hypertensive diseases						
Before the accident (1963–1967)	12	3.0 (1.7, 5.3)	6	1.5 (0.7, 3.3)	80	1.8 (1.5, 2.3)
After the accident (1968–1977)	21	1.1 (0.7, 1.6)	16	0.7 (0.4, 1.2)	216	0.9 (0.8, 1.1)
Cerebrovascular diseases						
Before the accident (1963–1967)	60	0.9 (0.7, 1.1)	71	1.0 (0.8, 1.3)	620	0.8 (0.8, 0.9)
After the accident (1968–1977)	117	1.0 (0.9, 1.2)	93	0.7 (0.6, 0.9)	1,282	1.0 (0.9, 1.0)
Pneumonia and bronchitis						
Before the accident (1963–1967)	20	1.2 (0.8, 1.8)	35	1.8 (1.3, 2.5)	297	1.5 (1.4, 1.7)
After the accident (1968–1977)	22	1.0 (0.7, 1.5)	32	1.3 (0.9, 1.8)	225	0.9 (0.8, 1.0)
Liver disease						
Before the accident (1963–1967)	12	2.3 (1.3, 4.0)	6	1.0 (0.5, 2.3)	59	1.0 (0.8, 1.3)
After the accident (1968–1977)	6	0.6 (0.3, 1.3)	6	0.5 (0.2, 1.1)	102	0.8 (0.7, 1.0)
Nephritic disorder						
Before the accident (1963–1967)	6	1.2 (0.5, 2.7)	5	0.9 (0.4, 2.1)	70	1.2 (1.0, 1.6)
After the accident (1968–1977)	1	0.2 (0.0, 1.7)	7	1.3 (0.6, 2.8)	47	0.9 (0.7, 1.2)

95 % CI 95 % confidence interval, *Obs* observation number, *SMR* standard mortality ratio

[a] Shimo-Goto was excluded from the exposed area (Tamanoura and Naru)

(Consonni et al. 2008; Bertazzi et al. 2001), one other recent cohort study suggested a slightly elevated SMR, although not significant, for uterine cancer 20 years after the incident (SMR: 2.34, 95 % CI 0.87–6.27) (Pesatori et al. 2009). Other studies from Seveso have also suggested a potential increase in endometriosis (Eskenazi et al. 2002) and a decrease in uterine leiomyoma (Eskenazi et al. 2007). In contrast, increased risks for uterine cancer with increasing estimated cumulative PCB exposure have been reported to be significant in other PCB studies (Verkasalo et al. 2004; Ruder et al. 2014). PCBs and PCDFs are known to have estrogenic, anti-estrogenic, or anti-androgenic activity (Lauby-Secretan et al. 2013; IARC 2013; Eskenazi et al. 2007), which may explain the increased incidence of uterine disorders following exposure to PCBs and PCDFs.

While our results showed that the SMRs for leukemia were elevated 10–19 years after the incident in Tamanoura and 10–35 years after the accident in Naru, similar findings have also been reported in previous studies on PCBs and PCDF exposure. In one Taiwanese study of *yu cheng* ("oil disease"), caused by a similar exposure to contaminated rice oil, the SMR for malignant neoplasm of lymphatic and hematopoietic tissue (ICD-9: 200–208) was found to have increased among men diagnosed with Yu-Cheng disease (SMR: 3.0, 95 % CI 1.1–6.6) (Li et al. 2013). In Seveso, although a significant elevated SMR for leukemia was not observed at 2.1 (95 % CI 0.8–5.8), the SMR for myeloid leukemia (ICD-9: 205) was significantly elevated at 3.8 (95 % CI 1.1–12.5) during the 15- to 20-year follow-up period (Bertazzi et al. 2001). Consistent with our findings, the SMR for myeloid leukemia was found to be elevated 15–20 years after the incident (SMR: 3.8, 95 % CI 1.1–12.5). Although the associations between PCBs and leukemia and non-Hodgkin lymphoma were still considered as providing limited evidence (Lauby-Secretan et al. 2013; IARC 2014), there have been reports of a positive association between PCB and non-Hodgkin lymphoma (Engel

Table 5 Numbers of cases, SMRs, and 95 % CIs for cancer mortality at 10-year intervals (1963–2002) in the exposed area (Tamanoura and Naru) and Shimo-Goto region excluding the exposed area

	Tamanoura		Naru		Shimo-Goto[a]	
	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)	Obs.	SMR (95 % CI)
All cancer						
1963–1967 (−5 to −1)	33	0.9 (0.6, 1.2)	32	0.8 (0.5, 1.1)	403	1.0 (0.9, 1.1)
1968–1977 (0–9)	86	1.1 (0.9, 1.4)	101	1.1 (0.9, 1.4)	902	1.0 (0.9, 1.1)
1978–1987 (10–19)	97	1.1 (0.9, 1.4)	124	1.2 (1.0, 1.4)	1,094	1.0 (1.0, 1.1)
1988–1997 (20–29)	83	0.9 (0.8, 1.2)	128	1.1 (0.9, 1.3)	1,364	1.2 (1.1, 1.3)
1998–2002 (30–34)	63	1.4 (1.1, 1.8)	82	1.2 (1.0, 1.5)	753	1.2 (1.1, 1.3)
After whole period (0–34)	329	1.1 (1.0, 1.2)	435	1.1 (1.0, 1.2)	4,113	1.1 (1.1, 1.1)
Stomach cancer						
1963–1967 (−5 to −1)	11	0.8 (0.4, 1.4)	7	0.5 (0.2, 1.0)	167	1.1 (1.0, 1.3)
1968–1977 (0–9)	24	0.9 (0.6, 1.4)	28	0.9 (0.6, 1.3)	232	0.8 (0.7, 0.9)
1978–1987 (10–19)	20	1.0 (0.6, 1.5)	20	0.8 (0.5, 1.2)	197	0.8 (0.7, 0.9)
1988–1997 (20–29)	14	0.9 (0.6, 1.6)	11	0.5 (0.3, 1.0)	163	0.8 (0.7, 1.0)
1998–2002 (30–34)	9	1.4 (0.7, 2.7)	12	1.3 (0.7, 2.3)	78	0.9 (0.7, 1.1)
After whole period (0–34)	67	1.0 (0.8, 1.3)	71	0.8 (0.7, 1.0)	670	0.8 (0.7, 0.9)
Bronchial/lung cancer						
1963–1967 (−5 to −1)	0	NE	2	0.6 (0.2, 2.5)	27	0.8 (0.6, 1.2)
1968–1977 (0–9)	11	1.4 (0.8, 2.5)	13	1.4 (0.8, 2.4)	98	1.1 (0.9, 1.3)
1978–1987 (10–19)	12	0.9 (0.5, 1.6)	19	1.2 (0.7, 1.8)	181	1.1 (1.0, 1.3)
1988–1997 (20–29)	15	0.9 (0.5, 1.5)	23	1.0 (0.7, 1.6)	290	1.4 (1.2, 1.5)
1998–2002 (30–34)	10	1.1 (0.6, 2.0)	18	1.4 (0.9, 2.2)	164	1.3 (1.1, 1.6)
After whole period (0–34)	48	1.0 (0.8, 1.3)	73	1.2 (1.0, 1.5)	733	1.2 (1.2, 1.3)
Uteri cancer						
1963–1967 (−5 to −1)	0	NE	4	1.4 (0.5, 3.8)	28	1.0 (0.7, 1.5)
1968–1977 (0–9)	3	0.9 (0.3, 2.8)	2	0.5 (0.1, 2.0)	37	1.0 (0.7, 1.3)
1978–1987 (10–19)	4	1.6 (0.6, 4.2)	3	0.9 (0.3, 2.8)	22	0.7 (0.5, 1.1)
1988–1997 (20–29)	2	1.3 (0.3, 5.0)	1	0.4 (0.1, 3.2)	28	1.3 (0.9, 1.9)
1998–2002 (30–34)	3	4.1 (1.3, 12.6)	2	1.8 (0.5, 7.3)	14	1.3 (0.8, 2.2)
After whole period (0–34)	12	1.5 (0.8, 2.6)	8	0.8 (0.4, 1.5)	101	1.0 (0.8, 1.2)
Breast cancer						
1963–1967 (−5 to −1)	0	NE	0	NE	6	0.9 (0.4, 1.9)
1968–1977 (0–9)	0	NE	0	NE	11	0.8 (0.5, 1.5)
1978–1987 (10–19)	2	1.2 (0.3, 5.0)	1	0.4 (0.1, 3.1)	24	1.1 (0.8, 1.7)
1988–1997 (20–29)	3	1.7 (0.5, 5.2)	2	0.7 (0.2, 2.9)	26	1.0 (0.7, 1.5)
1998–2002 (30–34)	0	NE	2	1.2 (0.3, 4.8)	18	1.1 (0.7, 1.8)
After whole period (0–34)	5	0.9 (0.4, 2.2)	5	0.6 (0.3, 1.5)	79	1.0 (0.8, 1.3)
Leukemia						
1963–1967 (−5 to −1)	1	0.9 (0.1, 6.3)	1	0.7 (0.1, 4.8)	16	1.2 (0.7, 2.0)
1968–1977 (0–9)	1	0.6 (0.1, 4.3)	5	2.2 (0.9, 5.3)	17	0.8 (0.5, 1.3)
1978–1987 (10–19)	7	3.0 (1.4, 6.2)	8	2.4 (1.2, 4.8)	32	1.0 (0.7, 1.4)
1988–1997 (20–29)	6	1.8 (0.8, 3.9)	17	3.4 (2.1, 5.5)	68	1.4 (1.1, 1.8)
1998–2002 (30–34)	3	1.6 (0.5, 4.9)	12	4.2 (2.4, 7.3)	36	1.3 (1.0, 1.9)
After whole period (0–34)	17	1.8 (1.1, 2.9)	42	3.1 (2.3, 4.2)	153	1.2 (1.0, 1.4)

95 % CI 95 % confidence interval, *NE* not evaluated, *Obs* observation number, *SMR* standard mortality ratio

[a] Shimo-Goto was excluded from the exposed area (Tamanoura and Naru)

et al. 2007; Robertson and Ruder 2010; Kramer et al. 2012) and childhood leukemia (Ward et al. 2009). Although adult T cell leukemia caused by HTLV-1 was endemic in Kyushu during the study period, the SMR for leukemia was not elevated in the comparison area (after 10–19 years), thus reducing the possibility of confounding due to HTLV-1. Considering the carcinogenicity of PCBs and PCDFs, further study for leukemia is required, such as further investigation of different leukemia subtypes, or affects at different ages, in particular for those exposed earlier, such as

the 0- to 5-year-old cohort at the time of the outbreak, or regarding in utero exposure.

In short, after the event, SMRs for all cause, diabetes mellitus, and heart disease were elevated in Tamanoura, but not in Naru. As we discussed in a previous study (Kashima et al. 2011), elevated risk for diabetes mellitus (Wang et al. 2008; Consonni et al. 2008; Magliano et al. 2014; Persky et al. 2012) and cardiovascular disease (including heart and hypertensive disease) (Humblet et al. 2008; Consonni et al. 2008; Gustavsson and Hogstedt 1997) was observed in previous PCB or PCDF studies. In 2013, 14 % of the population in 1970 were certified as Yusho patients in Tamanoura and 3 % in Naru (Table 1), and more residents were considered to be exposed to the rice oil in both areas. While the different findings among the two exposed areas could be explained by the difference in the proportion of residents exposed in each area, we could not confirm this as precise data on the number of people exposed was unavailable.

The present study has several limitations. As with our previous ecological study, there may have been selection bias due to a lack of information on local migration, misclassification of exposures caused by adopting Nagasaki Prefecture as the reference population (which included residents in exposed areas), and residual confounders. A more detailed explanation of these limitations is given elsewhere (Kashima et al. 2011). Additionally, misclassification of exposure may have occurred in this setting between 1963 and 1967, which is the period before the discovery of the contaminated rice oil. Although the rice oil was officially reported to produce and distributed in February 1968 (Kuratsune et al. 1972), several patients who had similar symptoms to those of Yusho patients, including of skin diseases, were reported a few years prior to the wide-scale outbreak (Shimoda 2008; Kuratsune and Yoshimura 1996). Therefore, although the level of PCB and PCDF contamination was likely to have been substantially lower than during the outbreak, it cannot be ruled out that contaminated rice oil was not distributed before 1968. This may have resulted in an underestimation of the strength of the association between exposure to PCBs and PCDFs in the period following the incident.

Conclusions

The present study found evidence for increased all-cause mortality, in addition to deaths due to diabetes mellitus and heart disease shortly after exposure to rice oil contaminated with PCBs and PCDFs in Tamanoura. This remained the case even after accounting for the time trend in mortality before the outbreak. Additionally, mortality due to uterine cancer in Tamanoura and leukemia in the whole exposed area was elevated for many years afterward. While the potential for dilution or unadjusted confounders was considered non-negligible in this ecological study, we were able to overcome some of the limitations of previous studies. Further research is suggested, especially studies looking at PCBs and PCDFs in relation to leukemia.

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Conflict of interest No conflict of interest.

Ethical standard The manuscript does not contain clinical studies or patient data.

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P-2102-3　カネミ油喫食地域における超過死亡：短期および長期の影響評価

鹿嶋 小緒里[1)]、頼藤 貴志[2)]、津田 敏秀[2)]、烏帽子田 彰[1)]
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【目的】本研究は、カネミ油症[ポリ塩化ビフェニル（PCB）とポリ塩化ジベンゾフラン（PCDF）混合曝露]による短期の健康影響評価を行うことを目的とする。1968年、PCBとPCDFで汚染された米ぬか油（ライスオイル）によって、のちに“カネミ油症”と呼ばれる大規模な食中毒事件が西日本を中心に発生した。PCB、PCDFは、それぞれダイオキシン様PCB、ダイオキシン類に分類されるものである。我々は先行研究において、汚染油が流通した地域（町を単位）において標準化死亡比（SMR）を計算し、死亡への評価を実施した、その結果、いくつかの疾病で事故直後にSMRsの上昇が確認された。しかしながら、それら確認されたSMRsの増加が、地域特有の時間的な傾向によるものか、もしくは汚染油によるものかの評価が不十分であった。これらを考慮すべく、アクシデント発生時からさらに5年前の死因情報を取得し、汚染されたライスオイル流通地域におけるカネミ油曝露と死亡の関連に関する評価を実施した。更に、先行研究より死因を追加した。
【方法】汚染油が流通した地域である長崎県にあるT町とN町を対象地域とする。長崎県人口を参照として、1963年から2002年までの、それぞれの疾病の5歳階級別に年齢を調整したSMRとその95%信頼区間を計算した。死因別死亡数および年齢階級別死亡数、年齢別人口はそれぞれ、長崎県衛生統計年報（人口動態編）と、国勢調査を利用した。
【結果】アクシデント前の時間的な死亡傾向を考慮しても、全死因、糖尿病と心疾患による死亡が、曝露直後短期間で増加していた。加えて、子宮がんと白血病による死亡が、30-34年後と15年後でそれぞれSMRの増加があった。特に、T町において、15-19年後に白血病のSMR が4.5（95% 信頼区間: 2.0, 9.9）と長崎県と比較して高かった。
【結論】Ecologic studyなので、本研究でも喫煙等の交絡因子の影響を調整できてはいないが、カネミ油症に含まれる、PCBとPCDFの汚染された油が流通した地域において、全死因と一部の死因による死亡の増加が確認された。喫食者を対象としたさらなる健康影響評価が必要である。