Minamata Disease and the Mercury Pollution of the Globe

Masazumi Harada, M.D., Ph.D.

Minamata Disease was discovered for the first time in the world at Minamata City, Kumamoto Prefecture, Japan, in 1956 (Minamata Disease Research Group; 1968, Harada M; 1995), and for the next time at Niigata City, Niigata Prefecture, Japan, in 1965 (Tsubaki T & Irukayama K; 1977). The both cases were attributed to the methyl mercury that was generated in the process for producing acetoaldehyde using mercury as catalyst. Methyl mercury had accumulated in fishes and shellfishes and those who ate them had been poisoned with it. These cases of the poisoning with organic mercury poisoning were the first to take place in the world through the food chain transfer of its environmental pollution. The cases of organic mercury poisoning that had been known prior to Minamata Disease occurred as the result of the direct poisoning of those who were engaged in organic-mercury handling occupations or those who took it in accidentally (Hunter D et al; 1940, Lundgren KD et al; 1949).

The Shiranui Sea that caused the first Minamata Disease i.e. that in Minamata has an area of 1,200 square kilometers and once enjoyed abundance of fishes, shellfishes and so forth and then a population of approximately 200,000 were engaged in fishery and other relative jobs. The residents living there were accustomed to eat fishes and shellfishes as main dishes throughout years. Some of them ate 500 grams of them a day. On the other hand, the residents living along the Agano River, Niigata, that caused the second Minamata disease also ate the fresh water fishes caught in great quantities there (Harada M; 1972, 1994).

Department of Social Welfare Studies
KUMAMOTO GAKUEN UNIVERSITY
Ohe2-5-1,Kumamoto 862-8680,JAPAN
Tel 096-364-5161,Fax 096-363-1289.

According to Prof. Tokuomi, sensory disturbance and constriction of the visual field were observed as an example of the typical symptoms of Minamata Diaease among 100% of its patients, coordination disturbance among 93.5%, dysarthria among 88.2%, hearing disturbance among 85.3% and tremor among 75.8%. Besides, the patients' serious symptoms were evidenced by the fact that 82.4% of them showed walking disturbance (Tokuomi H; 1960). They are typical symptoms of methyl mercury poisoning but, in fact, there are patients with various combinations of symptoms and with various degrees of symptoms that range from mild to serious cases. It can be said that the population of patients with non-typical symptoms is greater than that with typical symptoms. Particularly, glove and stocking type and perioral sensory disturbances were characteristically observed with Minamata Disease (Harada M; 1995). The Minamata Disease with such unique sensory disturbance alone has however been found out so far. The pattern of such sensory disturbance is similar to that of peripheral nervous disturbance. According to the study conducted lately, there are great possibilities that they may belong to the category of central nervous disturvance (Ninomiya T et al; 1995). Some chronic period or mild cases of coordination disturbance are difficult to identify. If this is the case, electroophthalmography (EOG) and optokinetic nystagmus pattern (OKP) can conveniently be used for obtaining referential data (Harada M; 1995). Particularly, in new-developing case of Minamata Disease, the possibilities of the occurrence of mild and non-typical symptoms are greater than those of typical serious symptoms.

Table 1, Figure 1 shows the frequency of clinical symptoms and the pattern of sensory disturbance of those suspected of suffering from chronic Minamata Disease (Harada M; 1995). The hair mercury value of the residents living around the Shiranui Sea has come down at present as shown in Figure 2 but there still remain various symptoms (Harada M; 1998).

In 1962, it was found that methyl mercury causes unborn childrens' poisoning via

the placenta. It is called congenital Minamata Disease (Minamata Disease Research Group; 1968, Harada M; 1986). The author has identified 64 cases of it so far. All its patients are such that another member or other members of their family had also suffered from Minamata Disease and their mothers ate much fishes and shellfishes during pregnancy. The site and timing (1952-1963) of congenital Minamata Disease agreed with those of acquired Minamata Disease. The handicapped accounted for 9.0% of the children newly born in the settlements with the highest density of the disease. Observed among its initial symptoms were such ones as mental retardation with 100% of its patients, primitive reflexes with 100%, coordination disturbance with 100%, dysarthria with 100%, limb deformation with 100%, growth disorder with 100%, chorea-athetose with 95%, and hypersalivation with 95%. What a mercury value they showed soon after their birth was not known because the disease was found out five through eight years later (Harada M; 1986). In Japan, it is an old custom that the umbilical cords of newly-born children are preserved by their parents in memory of their birth. Noting it, the author and others measured their methyl mercury value. As the result, the high methyl mercury values of 1.0 ppm or so were detected from them (Harada M; 1995). In addition, it became pathologically and experimentally clear that it belonged to the category of viviparous methyl mercury poisoning. The congenital Minamata Disease that occurred via the placenta was the first that had ever been found out in the world. Besides, all its patients' symptoms were serious and those with neither mild nor imperfect type symptoms were not found out among them. The school children living in polluted areas showed mild diskinesia and intellectual disturbance. The author and others considered that they had occurred under the influence of methyl mercury. Lately it has been suggested that an influence may be exerted on unborn children although the hair mercury value of already-born children is up to tentative safety criterion 50 ppm (Harada M; 1996).

The Minamata Disease patients officially recognized at Minamata or Niigata amount to a population of 2,200 or 800 respectively. In addition, those who are suspected of

suffering from Minamata Disease (with chronic and mild symptoms) amount to a population of 12,000. The production of factories' methyl mercury containing waste water was discontinued after the processes that produced methyl mercury were disused in 1968. The mercury discharged into the Minamata Bay still remained there and, for its removal, reclamation and dredging were carried out so late as 15 years after its discovery. If the discharge of methyl mercury containing waste water is discontinued earlier, it was not considered that so many patients were affected by serious Minamata Disease symptoms (Harada M; 1994).

In Canada, mercury pollution was also caused by a caustic soda factory and the methyl mercury produced through the methylation of the inorganic mercury in the nature contaminated aborigines. Cat's Minamata Disease was also found out there (Takeuchi et al; 1977). The hair mercury value of the residents living there exceeded 50 ppm and some of them showed as high hair mercury values as 100 ppm. The author and others found out mild neurological symptoms among them (Harada M et al; 1977). The Canadian Government recognized mercury pollution, but denied the occurrence of Minamata Disease insisting that no serious typical cases were not found out. On the other hand, the environmental mercury pollution by caustic soda factories brought on severe criticism not only in Canada but also in Asia and Middle and South Americas. Japan has already made switch-over to the caustic soda production processes that require no mercury to be used. In the meanwhile, some other countries have not yet accomplished such switch-over.

In Jirin City, China, methyl mercury flew out of a chemical complex into a river. This pollution was the same as that by Chisso Corporation, Minamata, in that it was caused by an acetoaldehyde factory. However, it caused the fishes living there to disappear entirely so the occurrence of Minamata Disease was avoided fortunately. On the other hand, Jirin and Heilong Provinces announced that some residents showed hair mercury values more than 50 ppm and some of those living in the downstream of the river

showed the symptoms peculiar to Minamata Disease though being mild. On the other hand, delayed Minamata Disease was observed there. Its symptoms advance even after mercury intake is discontinued (Pan Yun Zhon et al; 1988). According to Haerbin University's investigation, fishermen showed mean and maximum hair mercury values of 20 and 71.2 ppm respectively and those with the hair mercury values more than 20 ppm showed sensory disturbance with 44% and visual field constriction with 17% (Song Zeng Ren et al; 1993).

The author and others started the analysis of residents' hair mercury values along the Amazon River, Brazil in 1988, and in 1992 carried out joint research with Tokyo University, Yokohama National University, and Para State University. Its results are shown in Table 2. At Garimpo, garimpeiros were confirmed to have caused inorganic mercury poisoning. However, the fishermen living on the downstream side of Garimpo showed high hair mercury values in 1994, those of some of them being confirmed to exceed 100 ppm in 1994, and more than 90% of detected mercury was methylated (Akagi H.et al; 1995,Harada M; 1996, 1997, 1998). The maximum mercury value of 3.17 ppm was detected form the fishes caught there so hair mercury contamination was considered as attributable to fish eating (Akagi H et al; 1995). It exceeded the tentative safety criterion of mercury value so it is feared that fishermen's health may be affected by it. Nevertheless, Minamata Disease was not found out in the author and others' clinical examinaiton carried out until 1994. The occurrence of Minamata Disease take five steps until becoming apparent (Table 3). It can be said that the Amazon River has already come to the fourth step and it is feared that unborn children may be affected because they are more sensitive than already-born children and adults. The clinical epidemiological investigation carried out in Canada, Iraq, New Zealand and Faeroes Islands revealed that even mothers' hair mercury values less than 50 ppm may affect their unborn chilren (WHO; 1990, Grandjean P et al; 1997). In the author and others' examinaiton of the conserved umbilical cords of children, the mothers of congenital Minamata Disease

patients showed the minimum hair mercury value of 21.5 ppm (Harada M; 1997).

Mercury is used for gold minig in East Africa and the Philippines also (Ikingura JR et al; 1996, Harada M et al; 1998). In Tanzania and Kenya, there was a great concern that the Victoria Lake may be contaminated with it. Nevertheless, garimpeiros and fishermen showed low hair mercury values (Table 4) because mercury is used at a lower rate there than in the Amazon River. The fishes caught in the lake also showed the low mercury values of 0.0089 through 0.063 ppm. Nevertheless, some women showed extraordinarily high mercury values up to 470 ppm. These cases of hair mercury did't considered as attributable to their contamination through the food chain transfer of mercury because it was confirmed to be almost inorganic mercury. Investigation also revealed that women were using the cosmetic soaps with a mercury content of 2% to whiten their skin and that their health was affected by them. The Government had already prohibited their use but there is a possibility that they may become a new source of mercury contamination.

Minamata Disease was discovered with many cases of typical symptoms coming up in Minamata. At present, the mercury-polluted areas of the world are in need of the data of its minimum requirements and its influence on unborn children.

Reference

1. Akagi H., Kinjo Y, Branches F, et al: Methylmercury pollution in Tapajos river basin, Amazon. *Environ. Sci.* **3**:25-32 (1995)
2. Grandjean P, Weihe P, White RF, et al: Congenitive deficit in 7 year-old children with prenatal exposure to methylmercury. *Neurotoxicol. & Teratol.* **19**:417-428 (1997)
3. Harada M: Minamata Byo, Iwanamishoten, Tokyo (1972) (in Jap.)
4. Harada M., Fujino T, Akagi T et al: Mercury contamination in human hair at Indian reserves in Canada. *Kumamoto Med.J.* **30**:57-64 (1977)

5. Harada M: Congenital Minamata disease, Intrauterine methylmercury poisoning. In John L. Sever: Teratogen Update, Environmental birth defects risks, New York, Alan R. Liss, pp123-126 (1986)

6. Harada M: Environmental contamination and human rights, Case of Minamata disease. *Ind. Environ. Crisis Q*, **8**:141-154 (1994)

7. Harada M: Minamata disease: Methylmercury poisoning in Japan caused by environmental pollution. *Crit. Rev. Toxicol.* **25**:1-24 (1995)

8. Harada M: Neurotoxicity of methylmercury. Minamata and Amazon. In Yasui, M. et al (ed.): Mineral and Metal Neurotoxicology, pp177-188, CRC Press, Tokyo (1996)

9. Harada M: Characteristics of industrial poisoning and environmental contamination in developing countries, *Environ. Sci.* **4** Suppl., 157-169 (1996)

10. Harada M, Nakachi S, Nakanishi J et al: Gold mining and mercury polution in environment, Monitoring of Amazon basin (Brazil) and Victoria Lake (Tanzania). *Res. Environ. Disrupt.* **27(3)**:9-15 (1998) (in Jap.)

11. Harada M, Nakanishi J, Konuma S et al: The present mercury contents of scalp hair and clinical symptoms inhabitants of the Minamata area. Environ. Res., 77:160-164 (1998)

12. Hunter D, Bomford RR & Russell DS: Poisoning by methylmercury compound. *Q. Med. J.* **9**:193-213 (1940)

13. Ikingura JR & Akagi H: Monitoring of fish and human exposure to mercury due to gold mining in the Lake Victoria goldfield, Tanzania. *Sci. Total Environ.* **191**:59-68 (1996)

14. Lundgren KD & Swensson A: Occupational poisoning by alkyl mercury compounds. *J. Ind. Hyg. Toxicol.* **31**:190-200 (1949)

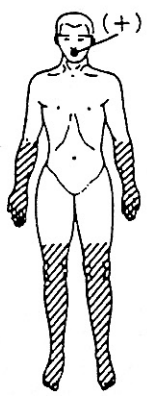
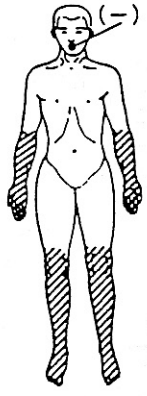
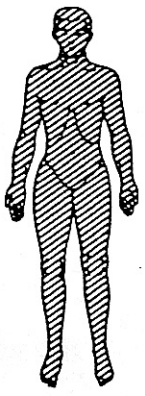
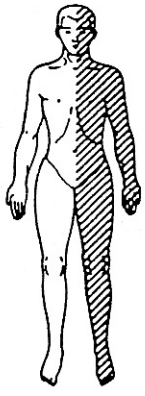
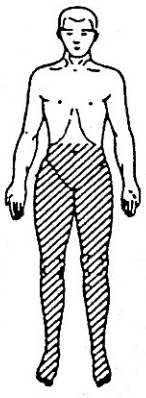
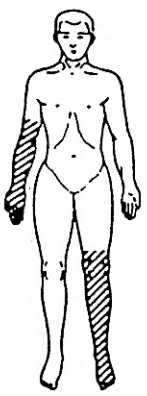
15. Minamata Disease Research Group: Minamata Disease. Medical School of Kumamoto University, Kumamoto, Japan (1968)

16. Ninomiya T, Ohmori Y, Hashimoto K et al: Expansion of methylmercury poisoning

outside of Minamata, An epidemiological study on chronic methylmercury poisoning outside of Minamata. *Environ. Res*, **70**:47-50 (1995)

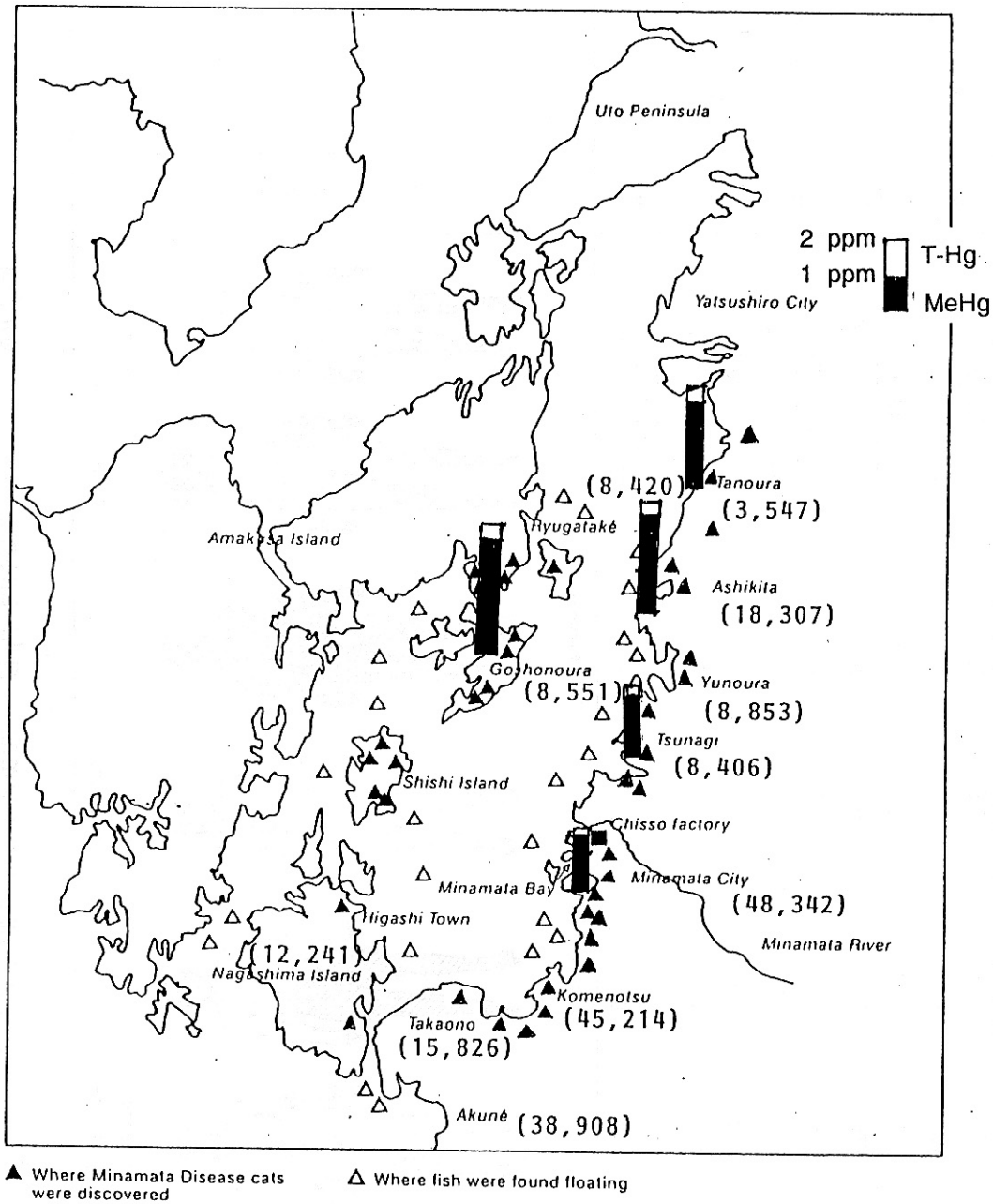
17. Pan Yun Zhon, Jiang Xi Luo, Wang Shu Hai et al: The problem of methylmercury poisoning along Songhua River bank in China, International Conference of Minamata Disease (Kumamoto) (1988)
18. Song Zeng Ren: Epidemiological research on the health effect of residents along the Sonhua River polluted by methylmerucry. "Assessment of Environmental Pollution and Health Effects from Methylmercury", WHO, Kumamoto (1993)
19. Takeuchi T et al: Outbreak of Minamata disease in cats on north-western Ontario's reseraves. *Environ. Res.* **13**:215-225 (1977)
20. Tokuomi H: Clinical observation and pathologenesis of Minamata disease. *Psychiat. Neurol. Jpn.* **62**:1816-1850 (1960) (in Jap.)
21. Tsubaki T & Irukayama K: Minamata disease, Methylmercury poisoning in Minamata and Niigata, Japan. Amsterdam, Elsevier (1977)
22. WHO: IPCS. Methylmercury, Environmental Health Criteria 101, Geneva, WHO (1990)

Figure 1. Types of Sensory Disturbance in Minamata Area

Objects \ Types	Numbers of case	Glove & stocking +Perioral		Generalized	Hemiplegic	Vertebral	Irregular & uncertain	Reference
								
Mothers of Cong. M. d.	28	6 — 22	28 (100)	0	1 (3.5)	0	1 (3.5)	Harada. M. (1974)
Family members of acute M. d.	145	44 — 65	109 (75.1)	0	11 (7.5)	4 (2.7)	4 (2.7)	Harada. M. (1972)
Residents of Minamata areas	928	70 — 145	215 (23.1)	14 (1.5)	51 (5.4)	21 (2.2)	16 (1.7)	Tatetsu. S. (1973)
Harada's material	2383	147 — 1393	1540 (64.6)	184 (7.7)	199 (8.3)	84 (3.5)	196 (8.2)	Harada. M. (1992)
Laborers of Chisso	296	9 — 71	80 (27.0)	2 (0.6)	7 (2.3)	8 (2.7)	31 (10.4)	Harada. M. (1990)
Canada Indian	89	2 — 13	15 (16.8)	4 (4.4)	3 (3.3)	0	16 (17.9)	Harada. M. (1977)

※Some cases overlapped each other types.
( ) are %.

Figure 2. Map of the Shiranui Sea and Hg Value of Hair in the Residents Living Around the Shiranui Sea Area



* Figures in ( ) are population of 1960.

Table 1. Frequency of Main Neurological Symptoms in Minamata Area (Harada)

Symptoms	N	%
Sensory disturbance		
Glove and stocking type	1540	64.2
		72.3
Generalized type	184	7.7
Auditory disturbance	1173	49.2
Incoordination	803	33.6
Dysarthria	527	22.1
Constriction of the visual field	398	16.7
Weakness	842	35.3
Tremor	662	27.7
Total	2383	100.0

Table 2. Total Hg and Methyl Hg Concentration in Hair From Inhabitants Living Along Amazon River Basin

Place	Origin	Sex			Age		T-Hg (ppm)		Sampling date	Excepted case (ppm)
		N	m	f	Mean±SD	min-max	Mean±SD	min-max		
Imwelu	Goldmine	18	17	1	36± 7.9	25 - 52	3.35± 3.5	0.38 - 9.7	1996. 8	(953.0) (623.0) (84.7)
Chipaka		17	17	0	32± 8.6	22 - 56	1.31± 1.5	0.33 - 7.0	1996. 8	
Mutakuja		7	6	1	40±13.0	20 - 55	1.01± 0.5	0.29 - 1.8	1996. 8	
Katoma		21	21	0	33± 7.3	22 - 56	3.29± 3.6	0.61 - 14.3	1996. 8	(51.0)
Buckleef		14	14	0	34± 4.3	27 - 42	3.62± 5.5	0.83 - 22.0	1996. 8	
Ikungu		15	7	8	38±14.2	18 - 70	2.00± 2.1	0.73 - 9.5	1997. 1	
Simbasirori		17	14	3	31±12.0	7 - 47	4.03± 4.1	0.57 - 13.5	1997. 7	(94.3)
Dodoma (1)		18	18	0	31± 6.8	22 - 48	1.02± 1.1	0.31 - 5.1	1996. 8	
Dodoma (2)		17	17	0	36± 8.3	27 - 57	1.61± 2.0	0.28 - 8.6	1997. 7	(189.7)
Seweya (1)	Fisherman's village	14	14	0	31±10.0	16 - 55	3.79±14.9	0.40 - 37.9	1996. 8	(70.4)
Seweya (2)		17	12	5	28±14.8	6 - 58	1.75± 1.1	0.54 - 3.9	1997. 7	(415.6) (84.9) (48.2)
Burgorola (Ukerewe)		22	19	3	39±15.7	24 - 70	2.08± 3.6	0.42 - 17.4	1997. 7	
Hurseni (Ukerewe)		26	21	5	39±12.7	18 - 59	1.00± 0.6	0.32 - 2.7	1997. 7	
Hombolo		20	20	0	23± 9.4	12 - 49	1.00± 1.0	0.29 - 5.1	1997. 7	
Mwanza	City	15	10	5	29±15.1	6 - 46	3.44± 6.9	0.48 - 27.7	1997. 1	(473.8) (231.6) (286.3) (80.2)

Table 3. The Processs of Outbreak of the Minamata Disease

First stage :	Garimpeiros suffer from inorganic-Hg poisoning (Direct exposure)
2nd stage :	Contamination of the air, soil and water. Changes to organic Hg from inorganic-Hg.
3rd stage :	Accumulation of organic Hg in fishes
4th stage :	Hg accumulation in humans. Hg level in the hair, blood and urine rise.
5th stage :	Minamata disease

Table 4. Total Hg Concentration in Hair (Tanzania)

Place	Origin	Sex			Age		T-Hg (ppm)		Sampling date	Excepted case (ppm)
		N	m	f	Mean±SD	min-max	Mean±SD	min-max		
Imwelu	Goldmine	18	17	1	36± 7.9	25 - 52	3.35± 3.5	0.38 - 9.7	1996. 8	(953.0) (623.0) (84.7)
Chipaka		17	17	0	32± 8.6	22 - 56	1.31± 1.5	0.33 - 7.0	1996. 8	
Mutakuja		7	6	1	40±13.0	20 - 55	1.01± 0.5	0.29 - 1.8	1996. 8	
Katoma		21	21	0	33± 7.3	22 - 56	3.29± 3.6	0.61 - 14.3	1996. 8	(51.0)
Buckleef		14	14	0	34± 4.3	27 - 42	3.62± 5.5	0.83 - 22.0	1996. 8	
Ikungu		15	7	8	38±14.2	18 - 70	2.00± 2.1	0.73 - 9.5	1997. 1	
Simbasirori		17	14	3	31±12.0	7 - 47	4.03± 4.1	0.57 - 13.5	1997. 7	(94.3)
Dodoma (1)		18	18	0	31± 6.8	22 - 48	1.02± 1.1	0.31 - 5.1	1996. 8	
Dodoma (2)		17	17	0	36± 8.3	27 - 57	1.61± 2.0	0.28 - 8.6	1997. 7	(189.7)
Seweya (1)	Fisherman's village	14	14	0	31±10.0	16 - 55	3.79±14.9	0.40 - 37.9	1996. 8	(70.4)
Seweya (2)		17	12	5	28±14.8	6 - 58	1.75± 1.1	0.54 - 3.9	1997. 7	(415.6) (84.9) (48.2)
Burgorola (Ukerewe)		22	19	3	39±15.7	24 - 70	2.08± 3.6	0.42 - 17.4	1997. 7	
Hurseni (Ukerewe)		26	21	5	39±12.7	18 - 59	1.00± 0.6	0.32 - 2.7	1997. 7	
Hombolo		20	20	0	23± 9.4	12 - 49	1.00± 1.0	0.29 - 5.1	1997. 7	
Mwanza	City	15	10	5	29±15.1	6 - 46	3.44± 6.9	0.48 - 27.7	1997. 1	(473.8) (231.6) (286.3) (80.2)