LEAD POLLUTANTS IN THE ENVIRONMENT AND ITS EFFECTS ON HEALTH STATUS OF INHABITANTS OF KIRTIPUR MUNICIPALITY

(Final Report)

NEPAL HEALTH RESEARCH COUNCIL
RAMSHAH PATH
KATHMANDU
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ACKNOWLEDGEMENT

With an increase in the level of lead in the environment due to various industrial and agricultural activities, Nepal has been facing pressure in prevention and control of lead exposure to human beings. So far, this problem is not adequately addressed. With this view in mind, an effort has been made in the present study to bring situation analysis of lead poisoning among the inhabitants of Kirtipur Municipality so that it could contribute towards developing plans and programs to mitigate the problem.

I would like to express my gratitude to Professor Gopal P. Acharya, NHRC and Mr. Jan A. Speets, Advisor, Environmental Health, WHO. My sincere thanks to Mr. Chandra Shekhar Yadav, Co-investigator/Environmental Health Officer-NHRC; Dr. R. P. Uprety and Dr. Ratindra Shrestha for their valuable support; Mr. Sharad Aryal and Mrs. Manju Shrestha for preparing the report. Basically, the report is prepared for the policy makers, planners and implementing agencies engaged in environmental health sector in Nepal. But it might prove equally useful to the students, professionals, media people and all those interested in environmental health problems.

Dr. Kamal Gyawali
Principal Investigator

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1. Introduction

The study was conducted selecting ward no. 14 and 15 (Chobhar) of Kirtipur Muncipality, situated southern part of the Kathmandu Valley in Kathmandu district. The study was aimed to determine the morbidity due to the exposure of lead and its concentration in the blood of the people of the locality. About 209 households comprising 360 numbers of persons were sampled for the collection of blood samples for blood testing and the analysis was performed based on laboratory report. The persons sampled were examined clinically to collect morbidity related information to find out the relationship with the lead level in the blood.

The study was mainly focussed on the clinical and experimental investigations of the people sampled. Further, it included the social and ethnic detail of the locality and possible evidences of exposure. Since the possible medium of exposure are air, water, soil and food, an attempt has been made to present some of the data in relation to the concentration of lead in air, water and soil of the locality.

2. Background

Lead (Pb) is recognized as the major toxic hazard since long time ago. Since it is a versatile type of metal, it has become widely distributed in the environment, consequently leading to human exposure. At high level of lead exposure to human, it can cause damage to almost all organs and organ systems. Some of the major health hazards are reported to be mental retardation in children, respiratory disorder, gastrointestinal upsets, neurological disorder and damage to bone and kidney. At low levels, haeme synthesis and other biochemical processes are affected, psychological and neurobehavioural functions are impaired, and there is range of other effects.

Whatever the sources of lead released in the environment, once it reaches inside an organism, it is the bloodstream and accumulated in red blood cells, tissues and bone. However, study on health impact of lead pollution in Nepal has not carried out yet. Therefore this study is probably the first study of this kind.

Chovar site is a typical one whose air quality especially with respect to TSP, PM10 and lead is entirely governed by Himal Cement factory's emission and is the highest of all other sites monitored. Lead value in the ambient air in a Chovar site is still next to Saibhu only. Lead content in these two sites is comparatively much higher than in the rest of the sampling stations. Nepal Environment and Scientific Services (P) Ltd. (1998) has reported lead content in the air of Kirtipur ranged between 2.76 $\mu g / m^3$ to 4.25 $\mu g / m^3$ mainly due to industrial pollution. Lead content in Chovar is about 4 times higher when compared with value <0.25/ $\mu g / m^3$ as acceptable lead concentration in air.

3. Objectives of the Study

Major objective:

To study the extent of lead pollution and its impact on health status of the individuals of Kirtipur Municipality.

Specific objectives:

I. To estimate the lead content in the blood of sampled population.

II. To asses the lead content in the soil and air of the village.

III. To find morbidity related to lead toxicity in the individual.

4. Literature Review

Lead is a ubiquitous and versatile toxic metal and is detectable in practicably all phases of the inert environment and in all biological systems. Lead is absorbed through ingestion, inhalation or other exposures. Lead is toxic because it can harm virtually every human system, especially the brain, kidney and reproductive system. Lead harms so many body systems because it disrupts enzyme systems mediated by other metals important to the body such as iron, calcium and zinc. This is one of the reasons why iron and calcium deficiencies increase lead absorption. Lead affects the formation of haeme, a complex iron-containing organic compound important to organs with haeme proteins, namely liver, kidneys, blood-forming tissues and the brain.

Because it is toxic to most living things at high exposures and there is no demonstrated biological need for it, the major issue regarding lead is determining the does at which it becomes toxic. Specific concerns vary with the age and circumstances of the host, and the major risk is toxicity to the nervous system. The most susceptible populations are children, particularly toddlers, infants in the neonatal period and the fetus. At high levels of human exposure there is damage to almost all organs and organ systems, most importantly the central nervous system, kidneys and blood, culminating in death at excessive levels. At low levels, haeme synthesis and other biochemical processes are affected, psychological and neurobehavioural functions and impaired, and there is a range of other effects (WHO, 1995)

Studies have shown links between childhood lead exposure and learning disabilities, impaired growth, reduced hearing acuity, hyperactivity, aggressiveness and attention deficit. These effects persist into adolescence and adulthood. It has also been shown that adults experience adverse health effects from lead at levels previously considered safe. Recent findings indicate that lead stored in women's bones from exposures throughout their lifetime can recirculate with osteoporosis or during pregnancy, potentially exposing the foetus to lead while in utero (Goyer and Clarkson, 2001).

Exposure

The principal route of exposure for people in the general population is food, and sources that produce excess exposure and toxic effects are usually environmental and presumably controllable. These sources include lead-base indoor paint in old dwellings, lead in dust from environmental sources, lead in contaminated drinking water, lead in air from combustion of lead containing industrial emissions, lead-glazed pottery, and less commonly lead dust brought home by industrial workers on their shoes and clothes.

Atmospheric lead that is deposited in soil and dust may then be ingested by children and may substantially raise their blood lead levels. Other potential sources of exposure of lead are recreational shooting on indoor ranges, hand-loading of ammunition, soldering, jewelry making, pottery making, gunsmithing, glass polishing and painting, all of which involve some exposure to lead (Goyer and Clarkson, 2001).

The pre-industrial or natural blood lead level in humans is estimated to have been about 0.016 μg/dL, 50-200 times lower than the lowest reported levels of people today people in remote regions of the southern and northern hemispheres (0.78 μg/dL and 3.20 μg/dL respectively (Flegal and Smith, 1992). This level is about 625 times lower than the current level of concern for children (i.e. 10 μg/dL) proposed by CDC in the USA. Lead levels in human skeletal remains indicate that the body lead burden today's populations is 500-1000 times greater than that of their pre-industrial counterparts (Ericson, et. al., 1979 & Patterson, et.al., 1991).

Environmental Exposure of Lead in Developing Countries

Lead continues to be a significant public health problem in developing countries, where there are considerable variations in the sources and pathways of exposure. For example, in many Latin American countries, leaded paint is not a significant source of recurrent exposure, whereas lead-glazed ceramics are such a source. Exposure attributable to miscellaneous sources may then be even more significant than universal exposure associated with leaded petrol, especially for people living in poverty. Exposure to lead from lead mining, smelting, battery factories and cottage industries is a significant environmental hazard in developing countries (Tong, et.al., 2000).

In China, childhood lead poisoning may be widespread as a result of rapid industrialization and the use of leaded petrol. Children residing in industrial area and in areas with heavy traffic had average boold lead levels of 21.8-67.9 μg/dL. The proportion of blood lead levels >10 μg/dL ranged from 64.9% to 99.5%. Even about 50% of children living in non-industrialized areas had boood lead values >10μg/dL . The problem of lead exposure in children is particularly significant in small towns with numerous small factories (Shen X, et.al., 1996).

The situation is similar in other countries where industrialization is occurring. The mean blood lead concentration among 93 randomly selected rickwsaw pullers in India was 53 μg/dL (Viswanathan, et.al., 1991). Also in India, direct testing for blood lead was carried out randomly on 2031 children and adults in five cities with high population densities where leaded petrol had contributed to environmental lead levels. Approximately 51% had levels > 10 μg/dL and 13% had values > 20 μg/dL.

In Cape Province, South Africa, over 90% of the children in some urban and rural communities had blood lead levels >= 10 μg/dL. The mean blood lead level in inner-city, first-grade school children in the country was 18 μg/dL. Childhood lead poisoning appeared to be a widespread urban health problem throughout Africa (Von Schirnding, et.al., 1991).

Toxicokinetics

Adults absorb 5 to 15 percent of ingested lead and usually retain less than 5 percent of what is absorbed. Children are known to have a greater absorption of lead than adults; one study found an average net absorption of 41.5 percent and 31.8 percent net retention in infants on regular diets. Lead absorption in children is related to age and development of the gastroinestinal tract (Ziegler et al., 1978). Nutritional problems such as low dietary iron and calcium enhance lead absorption (Bruening et al, 1999). Lead in water and other beverages is absorbed to a greater degree than lead in food. Lead ingested between meals is absorbed more than lead with meals and increasing frequency of food intake minimizes lead absorption. The total body burden of lead may be divided into at least tow kinetic pools, which have different rates of turnover. The largest and kinetically slowest pool is the skeleton, with a half-life of more than 20 years; the soft tissue pool is much more labile. Lead in bone may contribute as much as 50% of blood lead, so that it may be a significant source of internal exposure to lead. The largesest soft tissue accumulations of lead are in liver and kidney, but lead is present in most tissues of the body. The major route of excretion of absorbed lead is the kidney (Goyer and Clarkson, 2001).

Lead and Health Effects

In children, exposure to excessive levels of lead can cause brain damage; affect a child's growth; damage kidneys; impair hearing; cause vomiting, headaches, and appetite loss; and cause learning and behavioral problems. In adults, lead can increase blood pressure and can cause digestive problems, kidney damage, nerve disorders, sleep problems, muscle and joint pain, and mood changes. In addition, there are several nutritional and dietary factors that influence lead toxicity (Mahaffey, 1985). In a study of effects of lifestyle factor on blood lead levels, alcohol consumption has been shown to account for the large proportion of variability in blood lead levels, followed by age and smoking (Weyermann and Brenner, 1997).

Most research over the last 30 years has demonstrated health effects of moderately elevated blood lead levels i.e. below 25 µg/dL. These studies have been summarised by NRC (1993) and ASTDR (1999). Most studies reported a 2- to 4- point IQ deficit for each 10 µg/dL increase in blood lead within the range of 5 to35 µg/dL. The public health significance of small deficits in IQ may be considerable. An association between hearing thresholds and blood lead levels greater than 20 µg/dL has been found in teenagers (Schwartz and Otto, 1991). The toxic effects of lead and the minimum blood lead level at which the effect is likely to be observed are shown in **Table 1.**

Table 1. Summary of Lowest Observed Effect levels for Lead-related Health Effects

	Blood Lead Levels (µg/dL)	
Effect	Adults	Children
Neurologic		
Encephalopathy (overt)	80-100	100-120
Hearing Deficits	20	-
IQ deficits	10-15	-
In utero effect	10-15	-
Nerve conduction velocity↓	40	-
Heamatologic		
Anemia	80-100	80-100
U-ALA	40	40
B-EP	15	15
ALA-D inhibition	10	10
Renal		
Nephropathy	40	40-60
Vitamin D metabolism	<30?	-
Blood pressure		30?
Reproduction		
Males		40
Females		?

Source: (Goyer and Clarkson, 2001).

Lead and Children

Elevated lead levels continue to be a particular problem among socially and economically deprived children. Poor people are more likely to live in substandard housing and be near industry and heavy traffic, to be exposed to lead dust brought about home by lead workers, and to be nutritionally deprived and therefore susceptible. Fetuses, infants, and children are more vulnerable to lead exposure than adults since lead is more easily absorbed into growing bodies. Also, the tissues of small children are more sensitive to the damaging effects of lead. The Centers for Disease Control and Prevention (CDC) has set a level of concern at 10 micrograms per deciliter. The CDC recommends testing children at their one-year checkup or at six months if the child is at risk of high-dose exposure.

Manton et al. (2000) determined that major environmental sources of lead for infants and toddlers up to 4 year of age were hand to mouth transfer from floor, the lead being derived from dust sills and exterior surfaces. Blood lead levels among people in the general population have continued to decline in the US to < 5 μg/dL (Prikle, et. al., 1998). Nevertheless, the most current national survey shoes that nearly a million US children are at risk from lead poisoning (blood levels >10 μg/dL) and that specific groups of children are at greatest risk (Fig I).

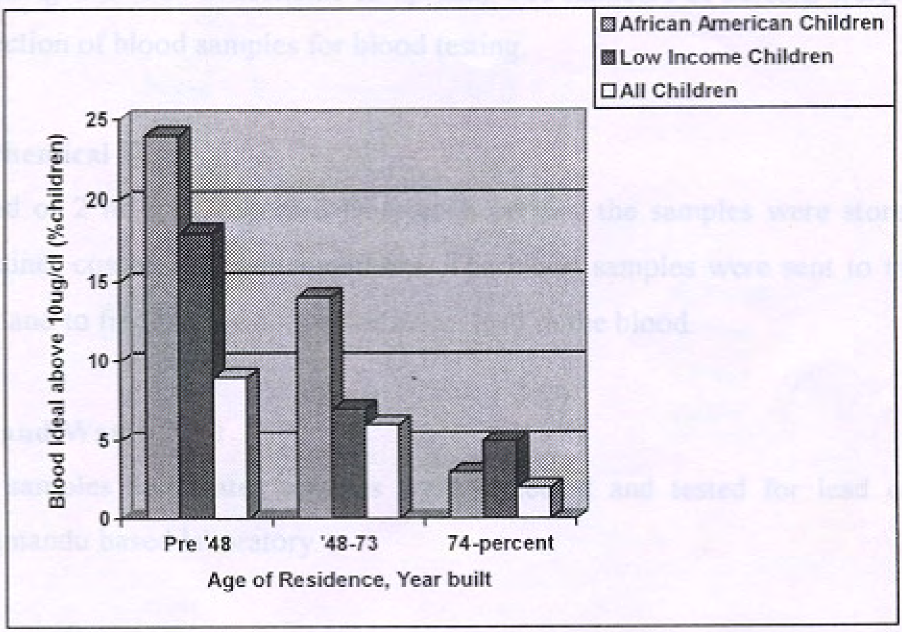


Figure I. National blood lead levels. National Health Nutrition Examination Survey, Phase 2, 1991-1994. (Source, Goyer and Clarkson, 2001)

5. Methodology

Sample Selection:

The people of different household were selected randomly for blood sampling. The sampling from 209 households comprising 360 numbers of persons were sampled for the collection of blood samples for blood testing.

Biochemical Test:

Blood of 2 ml was collected from each person, the samples were stored in the sterile container coated with anticoagulants. The blood samples were sent to the laboratory in Thailand to find out the concentration of lead in the blood.

Soil and Water Test:

Soil samples and water samples were collected and tested for lead concentration in Kathmandu based laboratory.

Socio-economic Data Analysis:

Data related with the cast and culture, educational level, occupation and symptoms of morbidity were collected and analysed to present the socio-economic conditions of the people in the study area.

Blood Report and Morbidity Analysis:

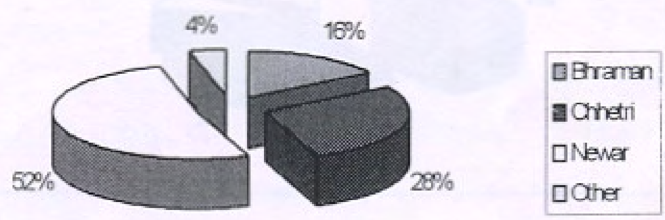
Based on other studies, the concentration of lead in the blood and the symptoms found clinically in the study area have been analysed thoroughly to find out the health effects due to lead concentration in the study area.

6. Socio-economic Conditions of the Study Area

Ethnicity:

Majority of the people of about 52% are of Newar and the other largest ethnic group were found as Chhetri of about 18%. Figure1 shows the percentage distribution of caste description of the respondents.

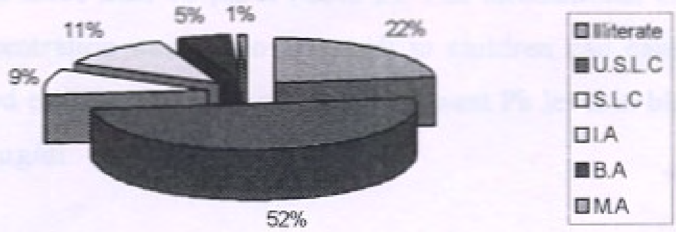
Figure 1: The percentage Distribution of Caste in the Study Area



Educational Level:

The majority of the respondent were literate and 22% were illiterate, 51 % of the people were found with an educational level less than School Leaving Certificate (SLC) and a few of them with bachelors and masters degree. It is interesting to know such an educational level of the people of study area which is very close to Tribhuvan University.

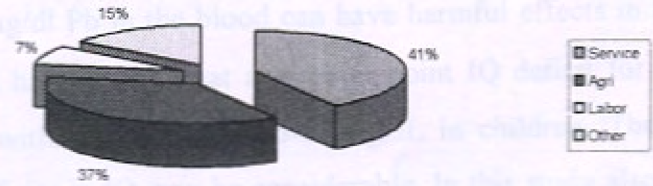
Figure 2: Educational Level of the Respondent



Occupation:

Out of the total respondent, the majority of the people of about 41% are involved in non agriculture sector and 37% in agriculture sector. The rest of the respondent of about 7% and 15% are involved carpenter and masons respectively. The figure 3 presents the percentage distribution of the respondent about their economic activities.

Figure 3: Occupation of the Respondent in the Study Area



7. Results and Discussion

Different types of symptoms were found, among which, most of the people (41.5%) were found complaining the symptoms of Arthalgia. Similarly, some of them were found suffering from joint pain (17.7%). Respondent of about 28.57% were found complaining Generalised Abdominal Disorder and 18.86% of respondents were found to be suffered from eye and skin diseases also.

Blood examination of the 360 people sampled, it revealed that 8.1% of people have the lead level in blood more than 10 µg/dl (table 2). The international experience indicates that the lead concentration more than 10 µg/dl in children can cause harmful effects. However, the blood examination showed that the lowest Pb level in blood was 1.70 µg/dl and highest 22.20 µg/dl.

Table 2: Lead concentration > 10 µg/dl in Male and Female

Lead Content	Frequency		Total	%
	Female	Male		
<10	152	179	331	91.9
>10	14	15	29	8.1
Grand Total	166	194	360	100.0

Table 2 showed that out of 360 people surveyed, 8.1 % of the population had lead content greater than 10 µg/dl. WHO and CDC (US) has set a level of concern at 10 µg/dl Pb in blood. Greater than 10 µg/dl Pb in the blood can have harmful effects in human beings. Studies in US countries have shown that a 2- to 4- point IQ deficit for each 10 µg/dl increase in blood lead within the range of 5-35 ug,dL in children. The public health significance of small deficits in IQ may be considerable. In this study also about 19% (3 out of 16 : see table 3) of the children below 10 years showed blood lead levels greater than 10 µg/dl. The possible sources of lead exposure to these people could be due to their dwellings which are situated in close proximity to industrial activity, agricultural activity as well as lead in the air due to environmental pollution (in Nepal leaded petrol is used in the motor vehicles). Results from air and soil samples from survey area also showed that a significant numbers of samples had higher levels of Pb concentration than the background concentration of Pb (See table 4 and 5) However water samples analysis did not detect any Pb in the water (table 6).

Table 3: Age wise grouping and lead Concentration in Blood

Grouped by Age	Up to 10			>10			Total
	F	M	Total	F	M	Total	
0 – 9	4	9	13	1	2	3	16
10 – 19	39	64	103	3	2	5	108
20 – 29	33	47	80	4	-	4	84
30 – 39	31	21	52	2	2	4	56
40 – 49	20	16	36	2	3	5	41
50 – 59	10	10	20	-	3	3	23
60 – 69	9	10	19	1	2	3	22
70 +	6	2	8	1	1	2	10
Total	152	179	331	14	15	29	360

Manton et al. (2000) determined that major environmental sources of lead for infants and toddlers up to 4 years of age were hand to mouth transfer from floor, the lead being derived from dust sills and exterior surfaces. It has also been shown that low income group children are at risk from lead poisoning (See fig 1). Children residing in industrial area and in areas with heavy traffic are at risk of lead poisoning.

Table 4-Soil Analysis

S.N·	Soil sample	Ward no.	Observed values of Lead ug/g
1	Inside soil	14	27.0
2	Surface soil	14	18.2
3	Inside soil	15	68.4
4	Surface soil	15	186.0

Table 5-Lead (pb) Concentration in Chovar and Saibu Areas (Based on 24 Hours Monitoring)

Place/Date	Dec. - 15	Dec. 19 – 29	Jan. 3 - 13	Jan. 17 - 27	Jan. 31 – Feb. 10	Feb. 14 - 24	April 1 - 11
Saibu	2.76	4.25	0.40	0.67	0.961	0.82	0.151
Chovar	0.135	1.8	0.34	0.153	0.392	0.24	0.271

Source : ADB TA 2847 – NEP Project, Ministry of Population and Environment, His Majesty's Government of Nepal 1999.

Table 6: Water Analysis

S. N.	Sample	Observed values of lead (mg/1)
1	Ward no.14	Not Detected(<0.01)
2	Ward no.15	Not Detected (<0.01)
3	Dudh Pokhari (ward no.15)	Not Detected (<0.01)
4	Naubishe Mul (ward no.15)Tap-2	Not Detected (<0.01)

8. Conclusion and Recommendation

This study showed that 8% of the total sampled surveyed had greater than 10 μg/dl of Pb in the blood. Since no such study were taken of this kind and only one study of this type can not generalize the situation whole population of the city. However, this certainly indicated that if studies are not carried out to pinpoint the sources of lead exposures and find suitable methods to control them, the prevailing situation can become worse, which may lead to a greater public health hazard. Exposure to environmental lead is clearly identified a major public health hazard of global dimensions. Much work needs to be done to identify and treat children with elevated levels and reduce lead exposure in the community. Public health measures should continue to be directed to reduction and prevention of exposure to lead by reducing the use of the metal and its compounds and by minimizing lead-containing emissions that result in human exposures. This can be achieved by-

- Phasing out lead additives in fuels and removing lead from petrol as soon as is practicable from the whole country.
- Identification of populations at high risk of exposure.
- Increasing emphasis on adequate nutrition, health care and attention to socioeconomic conditions that may exacerbate the effects of lead.
- Strict control of industrial emissions containing lead into the environment.
- Raise awareness among different stakeholders about understanding the harmful effects of lead poisoning.
- Improving control over lead in the workplaces

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Annex

Prof. Dr. H. Drexler
on behalf of
German Society of Occupational Medicine and Environmental Medicine e. V.
c/o Institute for Occupational, Social and Environmental Medicine
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External Quality Control acc. to the Guidelines of the German Federal Medical Council

Intercomparison programme 27 2001

Participant: Dr.Kamjad Ramakyl, Director of
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मिति 12 SEP 2001.

C E R T I F I C A T E

valid until August 31th, 2002

This is to certify you participated in the intercomparison programme 27 2001 for occupational/environmental medical - toxicological analyses. In accordance with the guidelines issued by the German Federal Medical Council (Bundesärztekammer) of January 16th and October 16th, 1987 on implementation of intercomparison programmes in the medical field you have fulfilled the requirements for the following parameters:

Occupational medical field

Lead (B)	Cadmium (B)	Hippuric acid (U)
Phenol (U)	Ethylbenzene (B)	

Noted.
file

Prof. Dr. med. H. Drexler
Ringversuchsleiter
...ftragter der DGAUM

Erlangen, July 2001

Prof. Dr. med. H. Drexler

Prof. Dr. rer. nat. J. Angerer

Laboratory Results of Lead in Blood Samples from Nepal

Division of Occupational Health, Department of Health

Ministry of Public health, Thailand

NO.	Pb in Blood.	No	Pb in Blood	No	Pb in Blood	NO	PB in Blood
	ug/dl		ug/dl		ug/dl		ug/dl
1	3.2	24	4.8	47	3.5	70	4.3
2	5.0	25	5.8	48	1.9	71	3.7
3	7.0	26	3.7	49	2.7	72	2.7
4	5.2	27	7.1	50	2.9	73	4.0
5	2.7	28	2.9	51	7.0	74	4.7
6	4.0	29	3.9	52	2.3	75	2.6
7	5.2	30	3.1	53	3.3	76	5.2
8	2.3	31	4.0	54	9.3	77	5.1
9	7.7	32	2.6	55	5.0	78	5.2
10	1.9	33	3.6	56	4.4	79	4.0
11	3.5	34	5.3	57	7.0	80	11.8
12	7.5	35	4.7	58	5.4	81	14.5
13	3.5	36	2.7	59	7.4	82	2.8
14	5.5	37	2.5	60	5.8	83	6.4
15	5.8	38	4.5	61	4.7	84	5.2
16	2.0	39	4.1	62	5.8	85	6.1
17	6.1	40	6.0	63	4.4	86	3.1
18	2.9	41	3.2	64	4.5	87	5.2
19	7.5	42	13.7	65	4.5	88	5.1
20	4.4	43	4.9	66	5.6	89	4.9
21	5.7	44	5.3	67	5.7	90	9.8
22	5.7	45	4.4	68	3.1	91	3.4
23	5.8	46	2.4	69	7.3	92	2.5

Analyst	Supervisor	Head of Laboratory Seetion
3 sep. 01		3/9/2001

NO.	Pb in Blood.	No	Pb in Blood	No	Pb in Blood	NO	PB in Blood
	ug/dl		ug/dl		ug/dl		ug/dl
93	2.5	121	4.4	149	4.4	177	7.5
94	3.5	122	5.2	150	2.4	178	3.8
95	2.3	123	5.3	151	4.0	179	7.9
96	2.3	124	4.6	152	4.9	180	8.3
97	3.8	125	2.4	153	2.9	181	4.5
98	4.4	126	5.8	154	3.3	182	5.2
99	5.5	127	4.8	155	1.9	183	11.4
100	5.7	128	5.1	156	2.5	184	11.8
101	3.8	129	7.4	157	2.0	185	4.7
102	3.3	130	5.2	158	1.7	186	7.9
103	-	131	6.0	159	2.8	187	4.6
104	4.8	132	2.9	160	4.9	188	6.3
105	-	133	4.3	161	5.0	189	10.3
106	-	134	6.0	162	5.1	190	5.1
107	5.8	135	6.5	163	3.7	191	8.4
108	3.6	136	2.8	164	14.7	192	9.4
109	3.5	137	4.8	165	1.8	193	3.7
110	3.3	138	2.6	166	3.8	194	3.9
111	2.7	139	3.9	167	3.0	195	3.3
112	-	140	5.8	168	3.5	196	7.3
113	3.5	141	2.3	169	3.0	197	6.1
114	3.5	142	3.1	170	8.4	198	13.7
115	2.3	143	7.6	171	12.6	199	6.7
116	3.4	144	8.8	172	5.7	200	6.0
117	7.4	145	2.9	173	4.9	201	7.9
118	3.4	146	2.3	174	5.3	201	17.0
119	3.9	147	3.4	175	4.8	202	8.9
120	3.2	148	3.3	176	8.0	203	6.9

Analyst — 3 Sep 01

Supervisor

Head of Laboralory Seetion — 3/9/2001

NO.	Pb in Blood.	No	Pb in Blood	No	Pb in Blood	NO	PB in Blood
	ug/dl		ug/dl		ug/dl		ug/dl
204	6.9	231	4.1	258	9.3	284	6.1
205	10.7	232	5.3	259	7.0	285	5.5
206	4.9	233	7.3	260	10.6	286	7.8
207	11.2	234	4.6	261	9.7	287	3.1
208	13.9	235	4.9	262	7.2	288	5.5
209	5.3	236	8.4	263	3.9	289	10.3
210	4.1	237	2.7	264	4.9	290	6.0
211	3.7	238	5.5	265	-	291	7.8
212	3.5	239	3.9	266	-	292	8.5
213	3.1	240	8.9	267	3.7	293	6.3
214	3.7	241	4.4	268	5.7	294	5.3
215	7.4	242	3.0	268	5.2	295	5.2
216	2.5	243	22.2	269	5.2	296	7.7
217	5.1	244	11.9	270	5.7	297	8.4
218	2.6	245	10.8	271	3.1	298	8.9
219	3.9	246	6.1	272	3.5	299	6.4
220	3.8	247	6.3	273	8.1	300	5.4
221	5.8	248	13.2	274	2.9	301	6.3
222	3.3	249	3.1	275	5.9	302	5.8
223	2.3	250	-	276	3.1	303	7.0
224	13.1	251	7.4	277	12.7	304	6.6
225	4.1	252	11.9	278	7.4	305	5.3
226	7.0	253	2.3	279	5.5	306	8.8
227	4.2	254	4.1	280	7.3	307	5.9
228	6.9	255	7.1	281	5.0	308	4.7
229	4.6	256	6.4	282	7.3	309	6.2
230	9.2	257	2.1	283	6.6	310	5.1

D. Yimrungruang
Analyst
3 Sep 01

N. Ardsawat
Supervisor

P. Rojanapanthar
Head of Laboralory Seetion
3/9/2001

NO.	Pb in Blood.	No	Pb in Blood	No	Pb in Blood	NO	PB in Blood
	ug/dl		ug/dl		ug/dl		ug/dl
311	5.0	339	5.6	367	-		
312	3.4	340	7.1	368	3.5		
313	5.3	341	6.0	369	-		
314	6.6	342	3.3	370	-		
315	12.7	343	7.4	371	5.9		
316	4.6	344	4.9	372	-		
317	8.6	345	5.2	373	2.8		
318	5.9	346	6.7	374	4.4		
319	3.9	347	8.5	375	6.9		
320	5.2	348	3.0				
321	5.8	349	3.5				
322	9.1	350	8.9				
323	7.3	351	5.7				
324	4.3	352	3.4				
325	9.7	353	13.3				
326	13.4	354	11.3				
327	8.3	355	6.2				
328	4.2	356	6.9				
329	7.4	357	5.2				
330	3.7	358	11.4				
331	5.1	359	9.0				
332	4.0	360	13.6				
333	5.9	361	4.4				
334	6.0	362					
335	9.6	363	13.5				
336	2.7	364	11.8				
337	7.4	365					
338	8.4	366					

D. Yimrungruang
Analyst
3 Sep 01

N. Ardmati
Supervisor

P. [illegible]
Head of Laboraloy Seetion
3/9/2001