Risk appraisal study: Sponge iron plants, Raigarh District

Sponge iron plants in Raigarh District

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Leaf burn noticed just outside a sponge iron plant

Cerana Foundation, Hyderabad
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1. Introduction

Raigarh district in Chattisgarh has several sponge iron plants, located around Raigarh town (details in Annex 1). The largest of these plants is the Jindal Steel and Power Ltd (JSPL) located in Patrapali village, about 8 km North-west of Raigarh town.

The residents of the areas around the sponge iron plants have been complaining of the air pollution caused by these plants. A local people's association, Jan Chetana, has taken up the issue of air pollution related impacts due to the sponge iron plants. As this problem is not unique to Raigarh, but exists in several other parts of India, Jan Chetana and other NGOs have formed a network of organizations to tackle this problem. National Centre for Advocacy Studies (NCAS), a Pune-based NGO, provided support to act as an information-clearing house for the network. NCAS and Jan Chetana requested Cerana Foundation, Hyderabad, to conduct a study of the impact of sponge iron plants in Raigarh District.

Cerana Foundation representative, Sagar Dhara, visited Raigarh District on 16-17 December 2005. Field visits to the areas where the sponge iron plants were conducted by Cerana Foundation and Jan Chetana, including the villages of Patrapali, Saraipali, Gorkha, Kalmi, Dhanagar, Gajamuda, Kosamnara, Kirodimal Nagar, Kansichua, Dogitaral, Banhar, Deon, Muraipali, Parsada, Kerajhar, Jampali, Kusumura, Bansiya, Khairpur and Raigarh. Discussions were held with the villagers around the plant sites. The Deputy General Manager (Environment), JSPL was contacted for holding discussions. He was not in town, but promised to cooperate with this study in the telephonic conversation had with him. Jan Chetana provided Cerana Foundation the documents that this study has relied on.

This is a preliminary appraisal to check whether there is a prima facie toxic risk, particularly cancer risk, to those living around the sponge iron plants due their air emissions.

2. Field observations

Area

The area around Raigarh town appeared to be recently cleared forest to make way for industry. The habitats were small and very basic. The population density in the area seemed to be lower than other parts of India. There was one river on the outskirts to the north of Raigarh town that had a reasonable quantity water flow at the time of the field visit.

Freshly cut forests near sponge iron plants

Habitats—small and basic

Pollution observed in the area

Solid wastes were visible along the sides of the roads leading to the sponge iron plants and the entire area was very dusty. Plants and leaves in the area were coated with an unusual amount of dust. Leaf burn is visible close to the plant sites.

Solid wastes along the roadsides on roads leading to the sponge iron plants

Leaves in the area are dust covered on the upper side and relatively clean on the underside

Dust covered leaves

Leaf burn

Iron ore handling at Raigarh station and its storage in open yards close to schools in Raigarh town was also causing air pollution. Ash, apparently from the JSPL ash pond, was being dumped in an open area as fill up an area that is later to become a bus stand.

Besides the air pollutants contributed by the sponge iron plants, the above activities also entrained significant quantities of suspended particulate matter into the air. Iron ore, coal and ash are known to contain heavy metals and are released to air.

Handling of iron ore at Raigarh station

Iron and coal stacking yards next to schools in Raigarh

Ash dumping in Raigarh

Pollution from sponge iron plants

Two types of pollutants were visible from sponge iron plants. Significant air pollution from stacks and as fugitive emissions from area sources (solid waste dumps) was very visible from most of the sponge iron plants seen in the area. There were also huge solid waste dumps visible inside the plant compounds. Some of the dumps were quite big.

Visible air pollution from point and area sources from sponge iron plants around Raigarh

Solid waste dumps at sponge iron plants

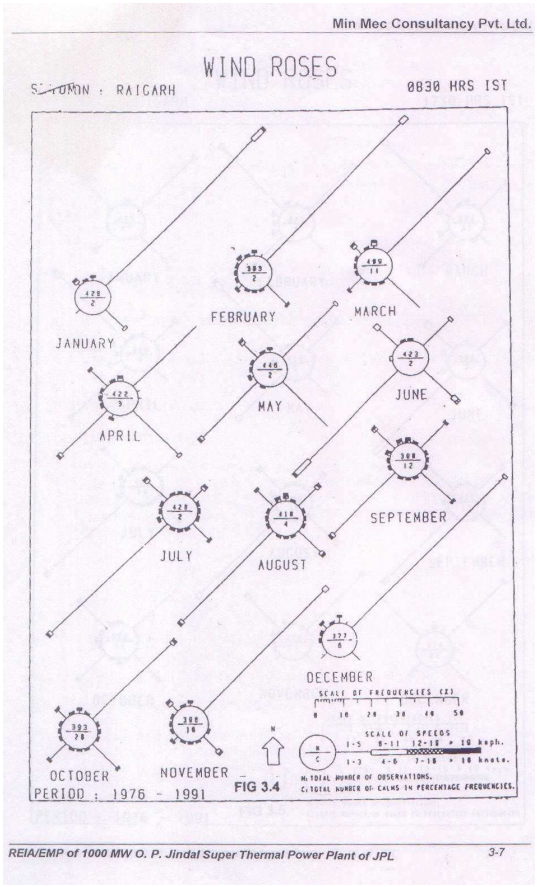
3. Documented data

Meteorology

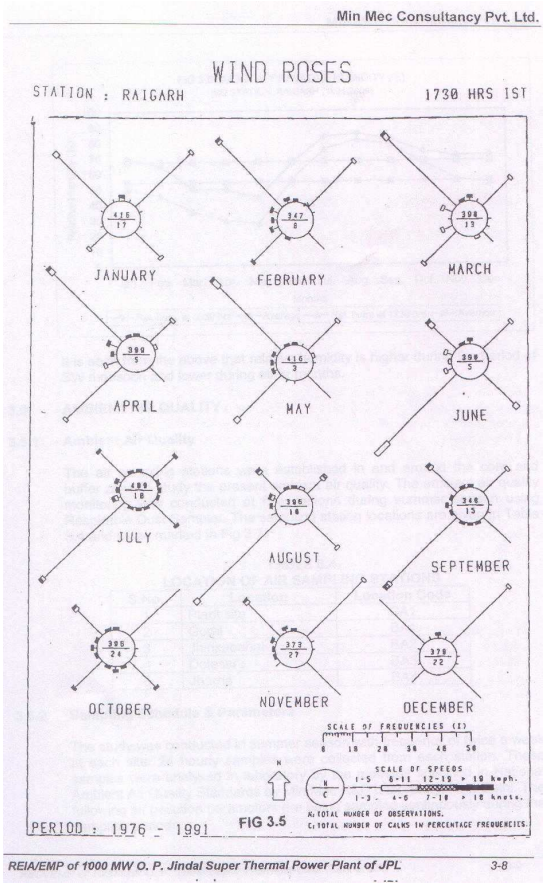
The meteorology of the area is summarized below. The area has hot summers and cool winters. Annual precipitation is about 1,500 mm, most of which occurring during the monsoon months—June-September. Average cloud cover is also 80-90% during the monsoon months and 20-30% during other months. Relative humidity is 65-90% during the monsoon and 20-60% during other months. Wind direction is predominantly from the Northeast during the non-monsoon months and from the Southwest during the monsoon. Wind speeds are higher during summer and monsoon months and lower during other months.

Meteorological data for Raigarh (***Source:*** *IMD, Raigarh, 30 year averages)*

Month	**Daily temperature** (°C)		**Relative humidity** (%)		**Rainfall** (mm)	**Wind speed** (km/hr)	**Wind direction** (from)	**Cloud cover** (oktas)
	Max	Min	Max	Min				
January	28.3	13.2	61	40	11.2	3.5	NE	1.8
February	31.6	16.0	53	30	15.7	4.1	NE	1.6
March	36.0	20.4	41	23	22.4	4.7	NE	2.0
April	40.3	25.1	38	20	13.8	5.1	NE	2.9
May	42.6	28.0	40	21	17.5	5.9	NE	3.4
June	38.0	27.1	63	50	199.0	6.7	SW	6.2
July	31.6	24.7	85	76	453.8	6.3	SW	7.3
August	31.1	24.7	86	78	494.5	5.9	SW	7.3
September	32.2	24.5	81	73	287.2	4.7	SW	6.3
October	32.4	22.0	71	59	49.1	3.9	NE	3.3
November	30.3	17.1	61	47	3.7	3.4	NE	2.1
December	28.2	13.3	62	44	4.1	2.9	NE	1.8



Raigarh windroses at 0830 hrs



Raigarh windroses at 1730 hrs

Sponge iron plant air emissions

Air emissions from sponge iron plants (see Annex 2) include highly toxic heavy metals, which may be released in three ways.

- Heavy metals are released to air in the handling of iron ore.

- Heavy metals, eg, cadmium, lead, zinc, mercury, manganese, nickel and chromium, may also be released as particulate matter from the stacks of a steel making plant. The problem is compounded if the rotary kiln does not have adequate air pollution control equipment.

- Heavy metals may also be released because of any scrap iron that may be handled by the plant.

Ambient air quality

As a part of their Rapid EIA Study, August 2004, for the expansion of the steel plant, EMTRC Consultants Pvt Ltd, Delhi, measured the ambient air quality around the JSPL plant. The data quoted in the EIA study is replicated below.

Ambient air quality around the JSPL plant

Location	Dist & dirn from JSPL (as per Rapid EIA)	SPM ($\mu g/m^3$)		RSPM ($\mu g/m^3$)		SO_2 ($\mu g/m^3$)		NO_2 ($\mu g/m^3$)	
		Summer 02	Summer 04	Summer 02	Summer 04	Summer 02	Summer 04	Summer 02	Summer 04
Gejamunda	2.5 km SW, Rural	240	178	88	74	9.8	10.2	20.5	20.8
Kasichua	3 km W, Rural	212	171	76	70	10.3	11.5	24.7	23.7
Chiraipani	2 km NW, Rural	190	168	78	72	13.3	12.0	37.8	17.6
Kosamnara	3.5 km S, Rural		143		42		8.8		10.6
Parsada	6 km NNW, Rural		152		45		9.5		11.0
Bhagwanpur	3.5 km E, Rural		153		50		10.5		16.2
Nansian	8 km SSW, Rural		133		42		7.5		12.0
Jindalgarh	1.5 km NNE, Residential	169	170	70	68	11.2	13.4	33.7	30.2
Raigarh	8 km SE, Urban	249	254	75	76	10.1	12.2	29.2	18.3
Urdhana	3.5 km NE, Forest	159	162	35	36	6.2	6.5	12.3	12.6

Source: *Rapid EIA: Expansion of steel plant—Jindal Steel & Power Ltd, Raigarh, EMTRC Consultants Pvt Ltd, Delhi, August 2004*

Metallic content of SPM in ambient air

Location	Iron (mg/g)		Nickel (mg/g)		Zinc (mg/g)		Manganese (mg/g)		Copper (mg/g)		Chromium (mg/g)		Lead (mg/g)	Cadmium (mg/g)	Cobalt (mg/g)	Benz(a) pyrene (ng/m^3)
	Max	Min	Max	Min	Max	Min	Max	Min	Max	Min	Max	Min				
Gejamunda	21.0	14.0	0.2	0.15	152	42	3.2	1.2	5.1	2.6	0.11	0.03	NT	NT	NT	3.1
Kasichua	3.0	22.5	3.0	0.1	670.0	300.0	2.3	1.2	4.1	1.8	0.03	0.01	NT	NT	NT	1.347
Chiraipani	33.0	21,5	0.01	0.01	590	384	5.0	1.0	7.4	6.6	0.21	0.11	NT	NT	NT	0.716
Kosamnara	21.5	19.5	0.01	0.01	499	185	2.7	2.6	4.9	3.8	0.37	0.01	NT	NT	NT	1.21
Parsada	11.5	10.5	0.03	0.01	171	169	1.8	1.0	1.8	1.6	0.04	0.01	NT	NT	NT	0.5
Bhagwanpur	15.5	13.5	4.5	0.1	292	209	3.7	0.1	2.6	2.2	0.03	0.01	292	3.7	0.1	1.112
Nansian	14.5	12.5	0.01	0.01	142	64	1.7	1.6	3.6	2.4	0.07	0.01	142	1.7	1.6	0.42
Jindalgarh	21.5	16.5	0.03	0.01	163	88	1.0	0.6	7.2	3.4	0.14	0.05	NT	NT	NT	1.21
Raigarh	25.0	12.5	0.1	0.1	303	250	5.1	2.4	2.1	1.8	0.12	0.1	NT	NT	NT	0.919
Urdhana	16.5	10.5	0.01	0.01	244	225	3.6	3.2	2.0	2.1	0.2	0.01	NT	NT	NT	0.157

Source: *Rapid EIA: Expansion of steel plant—Jindal Steel & Power Ltd, Raigarh, EMTRC Consultants Pvt Ltd, Delhi, August 2004*

4. Human health effects of sponge iron plant pollutants

Heavy metals released to air from the sponge iron plants are highly toxic. Some of them, eg, chromium (as Cr^{+6}), cadmium, nickel, are human carcinogens (see Annex 3). Iron acts along with other carcinogenic heavy metals to increase cancer risk. The toxic effects of heavy metals are varied and may often manifest after a prolonged period, sometimes several years, as in the case of cancer.

Sponge iron plants also emit oxides of sulphur and nitrogen and hydrocarbons. These air pollutants are likely to increase the incidence of respiratory tract ailments, eg, cough, phlegm, chronic bronchitis and also exacerbate asthmatic conditions.

5. Risk analysis

Using the ground level concentrations of toxic pollutants from the Rapid EIA Study for JSPL quoted above, exposure and risk assessments were done for air as the pathway (see Annex 4 for method). Jan Chetana wrote to JSPL requesting for clarification on the form of chromium in their emissions. As JSPL did not reply, it was assumed that the chromium in air was in its carcinogenic hexavalent form.

Uncertainty analysis was done to provide the band within which risk may lie. Though risk is generally treated as being chemical and exposure route-specific, in some situations such as the current problem, eg, when many chemicals produce the same toxic effects regardless of route,

combined risks may be computed. The table below summarizes the lifetime risk due to exposure to chromium, cadmium and manganese concentrations in the ambient atmosphere around JSPL. Since chromium and cadmium act in the same direction, their combined risk was computed to provide an idea of the risk magnitude, without necessarily considering the synergistic nature of the risks. Risk results for chemicals that were not significant are not shown.

Lifetime risk due to exposure to highly toxic pollutants around the JSPL plant

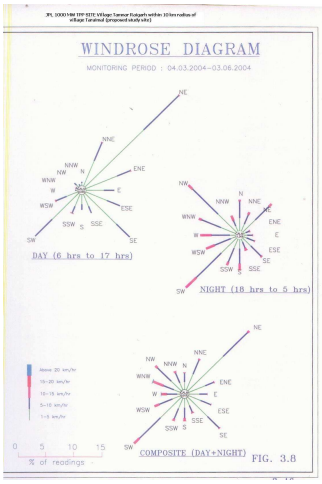
Location (distance and direction from JSPL as per Rapid EIA)	**Chemical(s)**	**Carcinogenic risk range** (lifetime probability in a million population)	**Non-carcinogenic risk range** (Hazard quotient—HQ)
Gejamunda, 2.5 km SW	Chromium	$100•10^{-6}$ – $30•10^{-6}$	
	Manganese		2.0 – 1.0
Kasichua, 3 km W	Chromium	$30•10^{-6}$ – $10•10^{-6}$	
	Manganese		2.0 – 1.0
Chiraipani, 2 km NW	Chromium	$200•10^{-6}$ – $100•10^{-6}$	
	Manganese		4.0 – 0.8
Kosamnara, 3.5 km S	Chromium	$400•10^{-6}$ – $10•10^{-6}$	
	Manganese		2.0 – 1.8
Parsada, 6 km NNW	Chromium	$40•10^{-6}$ – $10•10^{-6}$	
	Manganese		1.4 – 0.8
Bhagwanpur, 3.5 km E	Chromium	$30•10^{-6}$ – $10•10^{-6}$	
	Cadmium	$600•10^{-6}$ – $70•10^{-6}$	
	Chromium + Cadmium	$630•10^{-6}$ – $80•10^{-6}$	
	Manganese		2.0 – 0.1
Nansian, 8 km SSW	Chromium	$60•10^{-6}$ – $10•10^{-6}$	
	Cadmium	$200•10^{-6}$ – $30•10^{-6}$	
	Chromium + Cadmium	$260•10^{-6}$ – $40•10^{-6}$	
	Manganese		1.2 – 1.0
Jindalgarh, 1.5 km NNE	Chromium	$200•10^{-6}$ – $60•10^{-6}$	
	Manganese		0.8 – 0.6
Raigarh, 8 km SE	Chromium	$200•10^{-6}$ – $200•10^{-6}$	
	Manganese		6.0 – 4.0
Urdhana, 3.5 km NE	Chromium	$200•10^{-6}$ – $10•10^{-6}$	
	Manganese		2.0 – 2.0

***Note*: Carcinogenic risk is considered to be low if there is less than one-in-a-million ($<1•10^{-6}$) additional lifetime chance of cancer. Non-carcinogenic risk is considered to be low if the Hazard Quotient is less than 1 (<1.0).**

The results indicate the population within a 10 km radius around the JSPL, Patrapali plant was at significant cancer risk, besides being at an elevated non-cancer risk. Elevated cancer and non-cancer risks may extend to double this distance, or more, particularly if the emissions from other sponge iron plants are also likely to contain toxic and carcinogenic heavy metals. Exposures to air pollutants will also cause chronic respiratory ailments, particularly amongst young and old populations.

Cancer risk

The upper lifetime cancer risk at most habitats was significant, ranging from $100•10^{-6}$ to $600•10^{-6}$. The two substances that contributed to the cancer risk were hexavalent chromium and cadmium. Going by risk norms used in certain north nations, the ***cancer risk computed in this risk analysis is significant and at unacceptable levels in eight out of the 10 locations for which heavy metal concentration data was available.*** At the remaining two locations—Kasichua and Parsada, cancer risk was elevated. These two villages were in offwind directions (W and NNW from JSPL). The windrose below for Summer 2005 illustrates this point. Ambient air quality data used for this risk analysis was collected in 2002 and 2004 summers.



Summer 2005 windrose for the area

The relatively high cancer and non-cancer toxic risk obtained at Raigarh, despite it being 8 km from the source, indicates that ***local emissions due to unloading of iron ore and coal at the railway station and dumping of ash inside the town may also be contributing to the significant cancer risk due to the presence of heavy metals in Raigarh's air.***

Non-cancer risk

Non-cancer toxic risk due to exposure to Manganese concentrations in air at eight out of the ten locations was elevated, and at one location was significant (Raigarh). Again the significant risk at Raigarh suggests that local emissions due to handling of iron ore and coal and ash dumping may also be contributing to risk.

Vulnerable area

A population of 1.9 lakhs in 75 villages in a 10 km radius around the JSPL plant is at significant cancer risk, and elevated non-cancer toxic risk. A population of 1.8 lakhs in 231 villages in the area between 11-25 km radii around the JSPL plant may be at an elevated cancer risk. The total population that may be at cancer and non-cancer toxic risk in a 25 km radius around the JSPL plant is about 3.75 lakhs.

6. Impact on crops

The greatest impact of air pollution on plants occurs close to emission sources. Sulphur dioxide (SO_2), nitrogen dioxide (NO_2) and particulate matter are primary pollutants, whereas ozone is a secondary pollutant formed by photochemical reactions involving nitrogen oxides (NOx) and hydrocarbons.

Plants are known to sustain injury at relatively low SO_2 concentrations, particularly when other pollutants are also present. The importance of NOx as a pollutant is primarily because of its participation in photochemical reactions. Coal dust is quite harmful to vegetation, particularly to mango and lemon plants. Fly ash in moderate to large doses is harmful to plants. Due to the synergistic effect of SO_2, NOx and fly ash, paddy, mango, chickoo and cashew may be affected

the most in the impact zone. These plants may experience decreased yields, canopy and biomass, leaf size reduction, greater leaf fall and chlorophyll loss.

A recent study around a power plant indicates that yield reduction in sensitive plant species are greater—upto 50% than those of hardy species.

Yield reductions in different crop plants growing in the Obra, Dala and Renukoot (Agrawal, 2003)

Crop	Range of reduction		Sensitivity range
	Minimum (%)	Maximum (%)	
Triticum aestivum (Wheat)	30	50	S
Hordeum vulgare (Barley)	25	40	S
Cicer arietinum (Gram)	10	15	R
Brassica campestris (Mustard)	10	15	R
Pisum sativum (Pea)	40	50	S
Zea mays (Maize)	20	30	I
Phaseolus mungo (Urd)	30	50	S
Oryza sativa (Rice)	30	40	S
Cajanus cajan (Arhar)	20	30	I
Sorghum vulgare (jowar)	15	20	R
Sesamum indicum (Til)	10	15	R

S: Sensitive (> 30); I: Intermediate (15-30); R: Resistant « 15)

Results of field studies have been used for risk assessment to estimate crop loss due to air pollution close to various power plants. Individual farmers may have suffered losses in the range of 13-59%, which for a small or marginal farmer is unbearable.

Summary of estimated yield losses due to Sulphur dioxide within 10 km of thermal power plants in India (Ashmore and Marshall, 1997)

Thermal power plant	District	Capacity (MW)	Total district crop loss (kilo tons)	Estimated mean wheat yield reduction in 10 km range (%)
Singrauli	Sonbadhra (UP)	2050	52.5	59
Koradi	Nagpur (Mah)	1080	57.7	36
Dadri	Ghaziabad (UP)	630	334.8	19
Bhusawal	Jalgaon (Mah)	420	66.0	16
Sikka	Jamnagar (Guj)	440	46.0	13

Note: This analysis drew upon district level agricultural data, experimental field studies for dose-response relationships, and data on the location and installed capacity of thermal power plants to estimate sulphur dioxide concentrations

Results of a preliminary risk assessment of crop loss due to ozone within an 80 km zone around four major cities indicate potential yield losses in the range 12-40%.

Examples of studies showing effects of ozone on tropical crops ([a]rural; Bambawale, 1986, [b] rural; Laguette-Rey, 1986, [c] rural; Hassan *et al.*1995, [d] peri-urban; Wahid *et al*, 1995a and 1995b, [e] rural; Wahid *et al*, 2000*)*

Location	Crop	Effect
Indian Punjab[a]	Potato	Visible leaf injury
Valley of Mexico[b]	Bean	40% yield loss
		in sensitive cultivar
Nile Delta[c]	Radish	30% yield loss
	Turnip	17% yield loss
Pakistan Punjab[d]	Wheat	40% yield loss
	Rice	40% yield loss
Pakistan Punjab[e]	Soybean	57% yield loss

In Bromorposhi, a village that lies close to the Shiv Shakti Sponge Iron, it was estimated that the annual revenue loss to the village because of lowered paddy and fruit yields was Rs 18 lakhs per annum in 2001-02 (Dhara, 2002). It is probable that this has been caused by the sponge iron plant's air emissions. It is probable that the sponge iron plants in Raigarh District may cause a similar order of revenue loss due to decreased crop yields.

7. Conclusions

This risk appraisal indicates that there is a significant cancer risk and an elevated non-cancer toxic risk to a population of 1.9 lakhs in a 10 km radius around the JSPL plant. There may be an elevated cancer risk to a population of 1.8 lakhs persons living in the area between the radii 11-25 km around the JSPL plant. Similar cancer and non-cancer risk contours would exist for other sponge iron plants, which would be in direct correlation to their production and use of air pollution control devices. There would also be crop yield loss around the sponge iron plants.

8. Recommendations

1. Since an earlier study of a sponge iron plant in Orissa also indicated the existence of similar risk levels, JSPL and other sponge iron plants should be closed forthwith by revocation of their Consent for Operations and other environmental consents. These plants may be opened only when it can be demonstrated that they can be operated without risk to human health, crop yield loss and other environmental injury.

2. The environmental standards and code of practice for pollution prevention for sponge iron plants proposed by the Central Pollution Control Board does not adequately address the issue of heavy metal emissions from sponge iron plants or crop loss around such plants. The proposed standards may be suitably modified to do address these issues.

3. A risk standard for exposure to toxic substances should be developed for India.

9. References

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10. Acknowledgements

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Sagar Dhara

Annex 1: Sponge iron plants in Raigarh District

No.	Name of Industry	Location	Tehsil	Dist from Raigarh (km)	Capacity (TPA)
1	Jindal Steel & Power Ltd.	Patrapali	Raigarh	7-W	13,20,000
2	Singhal Enterprises pvt Ltd.	Taraimal	Gharghoda	15- N	1,56,000
3	Nalwa Sponge Iron Ltd.	Taraimal	Gharghoda	15-N	1,98,000
4	Shree Shyam Ispat (I) Pvt. Ltd.	Taraimal	Gharghoda	15-N	30,000
5	B.S.Sponge Pvt. Ltd	Taraimal	Gharghoda	15-N	30,000
6	Seleno Steels Ltd.	Taraimal	Gharghoda	15-N	30,000
7	Shri Ambika Ispat (I) Pvt. Ltd.	Taraimal	Gharghoda	15-N	30,000
8	Anjani Steels Pvt. Ltd.	Ujalpur-Taraimal	Gharghoda	15-N	30,000
9	Maa Kali Alloys Udyog Pvt. Ltd.	Pali-Gerwani	Gharghoda	12-N	30,000
10	Nav Durga Fuel Pvt. Ltd.	Saraipali	Gharghoda	17-N	60,000
11	Raigarh Ispat & Power Pvt. Ltd.	Saraipali	Gharghoda	17-N	30,000
12	Maa Shakambari Steel Pvt. Ltd.	Banjari Road	Gharghoda	17-N	32,500
13	Raigarh Iron Industries Ltd.	Punjipathara	Gharghoda	18-N	12,000
14	Sidhi Vinayak Sponge Iron Pvt Ltd.	Punjipathara	Gharghoda	18-N	66,000
15	Rameshwaram Steel & Power P Ltd.	Badegumuda	Gharghoda	42-N	30,000
16	Maa Mangla Ispat Pvt Ltd.	Natwarpur	Raigarh	18-E	30,000
17	Raigarh Electrodes Ltd. UNIT 2	Khairpur	Raigarh	7-N	15,000
18	Shiv Shakti Steel Pvt Ltd.	Chunchuna	Raigarh	15-E	30,000
19	Ind Agro Synery Ltd	Mahapalli-Kotmar	Raigarh	10-E	1,00,000
20	MSP Steel & Power Ltd.	Jamgoan	Raigarh	24-E	1,00,000
21	Maa Mangla Ispat Pvt Ltd.	Natwarpur	Raigarh	18-E	30,000
	Total				**23,89,500**

Annex 2: Air Emissions from Steel Making

(Extract from the Environmental and Public Health Issues subsection in the Iron and Steel section of the Encyclopaedia of Occupational Health and Safety, Ed 4, International Labour Office)

Air Pollutants

Air pollutants in the iron and steel-making operations have historically been of environmental concern. These pollutants include gaseous substances such as oxides of sulphur, nitrogen dioxide and carbon monoxide. In addition, particulates such as soot and dust, which may contain iron oxides, have been the focus of controls. Total pollution control costs, over half of which relate to air emissions, have been estimated to range from 1-3% of the total production costs; air pollution control installations have represented approximately 10 to 20% of the total plant investment.

Sulphur dioxide

The amount of sulphur dioxide, formed largely in the combustion processes, depends primarily on the sulphur content of the fossil fuel employed. The health effects attributed to sulphur oxides are not only due to the sulphur dioxide but also to its tendency to form such respirable aerosols. In addition, sulphur dioxide may be adsorbed onto particulates, many of which are in the respirable range.

Nitrogen oxides

Like the sulphur oxides, oxides of nitrogen, primarily nitrogen oxide and nitrogen dioxide, are formed in fuel combustion processes. They react with oxygen and volatile organic compounds (VOCs) in the presence of ultraviolet (UV) radiation to form ozone. They also combine with water to form nitric acid, which, in turn, combines with ammonia to form ammonium nitrate. These may also form respirable aerosols, which can be removed from the atmosphere through wet or dry deposition.

Particulate matter

Particulate matter, the most visible form of pollution, is a varying, complex mixture of organic and inorganic materials. Dust may be blown from stockpiles of iron ore, coal, coke and limestone or it may enter the air during their loading and transport. Coarse materials generate dust when they are rubbed together or crushed under vehicles. Fine particles are generated in sintering, smelting and melting processes, particularly when molten iron comes in contact with air to form iron oxide. Potential health effects depend on the number of particles in the respirable range, the chemical composition of the dust and the duration and concentration of exposure.

Metals such as cadmium, lead, zinc, mercury, manganese, nickel and chromium can be emitted from a furnace as a dust, fume, vapour or may be adsorbed by particles. Health effects depend on the level and duration of exposure.

Annex 3: Hazardous Properties of Chemicals and Health Effects of Air Emissions from Steel Making

(Extract from the Health and Safety Problems and Patterns subsection in the Iron and Steel section and the Chemicals section of the Encyclopaedia of Occupational Health and Safety, Ed 4, International Labour Office)

Airborne Pollutants

Steel workers may be exposed to a wide range of pollutants depending on the particular process, the materials involved and the effectiveness of monitoring and control measures. Adverse effects are determined by the physical state and the propensities of the pollutant involved, the intensity and duration of the exposure, the extent of accumulation in the body and the sensitivity of the individual to its effects. Some effects are immediate while others may take years and even decades to develop.
Pollutants and their derivatives can cause adverse effects by interacting with and impairing molecules crucial to the biochemical or physiological processes of the human body. Three factors influence the risk of toxic injury related to these substances: their chemical and physical properties, the dose of the material that reaches the critical tissue sites and the responsiveness of these sites to the substance. The adverse health effects of air pollutants may also vary across population groups; in particular, the young and the elderly may be especially susceptible to deleterious effects. Persons with asthma or other pre-existing respiratory or cardiac diseases may experience aggravated symptoms upon exposure (WHO 1987).

Sulphur Dioxide and Particulate Matter

During the first half of the twentieth century, episodes of marked air stagnation resulted in excess mortality in areas where fossil-fuel combustion produced very high levels of SO_2 and SMP. Studies of long-term health effects have also related the annual mean concentrations of SO_2 and SMP to mortality and morbidity. Recent epidemiological studies have suggested an adverse effect of inhalable particulate levels (PM_{10}) at relatively low concentrations (not exceeding the standard guidelines) and have shown a dose-response relationship between exposure to PM10 and respiratory mortality and morbidity (Dockery and Pope 1994; Pope, Bates and Razienne 1995; Bascom et al. 1996) as shown in the table below.

Summary of Short-term Exposure-response Relationship of PM_{10} with Different Health Effects Indicators

	Changes for each 10 $\mu g/m^3$ increase in PM_{10}	
	Mean	Range
Health Effects		
Mortality		
Total	1.0	0.5-1.5
Cardiovascular	1.4	0.8-1.8
Respiratory	3.4	1.5-3.7
Morbidity		
Hospital admission for respiratory condition	1.1	0.8-3.4
Emergency visits for respiratory conditions	1.0	0.5.4.0
Symptom exacerbations among asthmatics	3.0	1.1-11.5
Changes in peak expiratory flow	0.08	0.04-0.25

Nitrogen Oxides

Some epidemiological studies have reported adverse health effects of NO_2 including increased incidence and severity of respiratory infections and increase in respiratory symptoms, especially with long-term exposure. Worsening of the clinical status of persons with asthma, chronic obstructive pulmonary disease and other chronic respiratory conditions has also been described. However, in other studies, investigators have not observed adverse effects of NO_2 on respiratory functions (WHO/ECOTOX 1992; Bascom et al. 1996).

Photochemical Oxidants and Ozone

The health effects of photochemical oxidants exposure cannot be attributed only to oxidants, because photochemical smog typically consists of O_3, NO_2, acid and sulphate and other reactive agents. These pollutants may have additive or synergistic effects on human health, but O_3 appears to be the most biologically active. Health effects of ozone

exposure include decreased pulmonary function (including increased airway resistance, reduced air flow, decreased lung volume) due to airway constriction, respiratory symptoms (cough, wheezing, shortness of breath, chest pains), eye, nose and throat irritation, and disruption of activities (such as athletic performance) due to less oxygen availability (WHO/ECOTOX 1992). Epidemiological studies have suggested a dose-response relationship between exposure to increasing ozone levels and the severity of respiratory symptoms and the decrement in respiratory functions (Bascom et al. 1996).

Carbon Monoxide

The main effect of CO is to decrease oxygen transport to the tissues through the formation of carboxyhaemoglobin (COHb). With increasing levels of COHb in blood, the following health effects can be observed: cardiovascular effects in subjects with previous angina pectoris (3 to 5%); impairment of vigilance tasks (>5%); headache and dizziness (³10%); fibrinolysis and death (WHO 1987).

Dust and fumes

Health effects are related to size of the particles (ie, the proportion that is respirable) and the metals and aerosols that may be adsorbed on their surfaces. There is evidence that exposure to irritant dust and fumes may also make steelworkers more susceptible to reversible narrowing of the airways (asthma), which, over time, may become permanent.

Heavy metals

Emissions generated in steel making may contain heavy metals (eg, lead, chromium, zinc, nickel and manganese) in the form of fumes, particulates, and adsorbates on inert dust particles. They are often present in scrap steel streams and are also introduced in the manufacture of special types of steel products. Research carried out on workers melting manganese alloys has shown impaired physical and mental performance and other symptoms of manganism at exposure levels significantly below the limits currently allowable in most countries. Short-term exposure to high levels of zinc and other vapourized metals may cause "metal fume fever", which is characterized by fever, chills, nausea, respiratory difficulty and fatigue.

Some details of toxic effects produced by carcinogenic heavy metals, which are likely to be constituents of JSPL's emissions, are provided below.

Chromium

Hazards: In the Cr^{+6} oxidation state, chromium compounds are readily absorbed after ingestion as well as during inhalation. The uptake through intact skin is less well elucidated. The irritant and corrosive effects caused by Cr^{+6} occur readily after uptake through mucous membranes, where they are readily absorbed. Exposure to Cr^{+6} compounds may induce skin and mucous membrane irritation or corrosion, allergic skin reactions or skin ulcerations. The effects frequently involve the skin or respiratory system.

Ulcerations: Such lesions used to be common after work-related exposure to Cr^{+6} compounds. The ulcers result from the corrosive action of Cr^{+6}, which penetrates the skin through cuts or abrasions. The lesion usually begins as a painless papule, commonly on the hands, forearms or feet, resulting in ulcerations. The ulcer may penetrate deeply into soft tissue and may reach underlying bone. Healing is slow unless the ulcer is treated at an early stage, and atrophic scars remain. There are no reports about skin cancer following such ulcers.

Dermatitis: The Cr^{+6} compounds may cause both primary skin irritation and sensitization, particularly to exposed parts such as the neck. Some affected subjects had only erythema or scattered papules, and in others the lesions resembled dyshidriotic pompholyx; nummular eczema may lead to misdiagnosis of genuine cases.
It has been shown that Cr^{+6} penetrates the skin through the sweat glands and is reduced to Cr^{+3} in the corium. It is shown that the Cr^{+3} then reacts with protein to form the antigen-antibody complex. This explains the localization of lesions around sweat glands and why very small amounts of dichromate can cause sensitization. The chronic character of the dermatitis may be due to the fact that the antigen-antibody complex is removed more slowly than would be the case if the reaction occurred in the epidermis.

Acute respiratory effects: Inhalation of dust or mist containing Cr^{+6} is irritating to mucous membranes. At high concentrations of such dust, sneezing, rhinorrhoea, lesions of the nasal septum and redness of the throat are documented effects. Sensitization has also been reported, resulting in typical asthmatic attacks, which may recur on

subsequent exposure. At exposure for several days to chromic acid mist at concentrations of about 20 to 30 mg/m3, cough, headache, dyspnoea and substernal pain have also been reported after exposure. The occurrence of bronchospasm in a person working with chromates should suggest chemical irritation of the lungs. Treatment is only symptomatic.

Ulcerations of the nasal septum: In previous years, when the exposure levels to Cr^{+6} compounds could be high, ulcerations of the nasal septum were frequently seen among exposed workers. This untoward effect results from deposition of Cr^{+6}-containing particulates or mist droplets on the nasal septum, resulting in ulceration of the cartilaginous portion followed, in many cases, by perforation at the site of ulceration. Frequent nose picking may enhance the formation of perforation. The mucosa covering the lower anterior part of the septum, known as the Kiesselbach's and Little's area, is relatively avascular and closely adherent to the underlying cartilage. Crusts containing necrotic debris from the cartilage of the septum continue to form, and within a week or two the septum becomes perforated. The periphery of the ulceration remains active for up to several months, during which time the perforation may increase in size. It heals by the formation of vascular scar tissue. Sense of smell is almost never impaired. During the active phase, rhinorrhoea and nose bleeding may be troublesome symptoms. When soundly healed, symptoms are rare and many persons are unaware that the septum is perforated.

Effects in other organs: Necrosis of the kidneys has been reported, starting with tubular necrosis, leaving the glomeruli undamaged. Diffuse necrosis of the liver and subsequent loss of architecture has also been reported. Soon after the turn of the century there were a number of reports on human ingestion of Cr^{+6} compounds resulting in major gastro-intestinal bleeding from ulcerations of the intestinal mucosa. Sometimes such bleedings resulted in cardiovascular shock as a possible complication. If the patient survived, tubular necrosis of the kidneys or liver necrosis could occur.

Carcinogenic effects: Increased incidence of lung cancer among workers in manufacture and use of Cr^{+6} compounds has been reported in a great number of studies from France, Germany, Italy, Japan, Norway, the United States and the United Kingdom. Chromates of zinc and calcium appear to be among the most potent carcinogenic chromates, as well as among the most potent human carcinogens. Elevated incidence of lung cancer has also been reported among subjects exposed to lead chromates, and to fumes of chromium trioxides. Heavy exposures to Cr^{+6} compounds have resulted in very high incidence of lung cancer in exposed workers 15 or more years after first exposure, as reported in both cohort studies and case reports.
There are no "safe" levels of exposure to Cr^{+6}.

Cadmium

Chronic toxicity: Chronic cadmium poisoning has been reported after prolonged occupational exposure to cadmium oxide fumes, cadmium oxide dust and cadmium stearates. Changes associated with chronic cadmium poisoning may be local, in which case they involve the respiratory tract, or they may be systemic, resulting from absorption of cadmium. Systemic changes include kidney damage with proteinuria and anaemia. Lung disease in the form of emphysema is the main symptom at heavy exposure to cadmium in air, whereas kidney dysfunction and damage are the most prominent findings after long-term exposure to lower levels of cadmium in workroom air or via cadmium-contaminated food. Mild hypochromic anaemia is frequently found among workers exposed to high levels of cadmium. This may be due to both increased destruction of red blood cells and to iron deficiency. Yellow discolouration of the necks of teeth and loss of sense of smell (anosmia) may also be seen in cases of exposure to very high cadmium concentrations.

Cancer: There is strong evidence of dose-response relationships and an increased mortality from lung cancer in several epidemiological studies on cadmium-exposed workers. The interpretation is complicated by concurrent exposures to other metals, which are known or suspected carcinogens. Continuing observations of cadmium-exposed workers have, however, failed to yield evidence of increased mortality from prostatic cancer, as initially suspected. The IARC in 1993 assessed the risk of cancer from exposure to cadmium and concluded that it should be regarded as a human carcinogen. Since then additional epidemiological evidence has come forth with somewhat contradictory results, and the possible carcinogenicity of cadmium thus remains unclear. It is nevertheless clear that cadmium possesses strong carcinogenic properties in animal experiments.

Nickel

Allergy: Nickel and nickel compounds are among the most common causes of allergic contact dermatitis. This problem is not limited to persons with occupational exposure to nickel compounds; dermal sensitization occurs in the general population from exposures to nickel-containing coins, jewellery, watchcases and clothing fasteners. In

nickel-exposed persons, nickel dermatitis usually begins as a papular erythema of the hands. The skin gradually becomes eczematous, and, in the chronic stage, lichenification frequently develops.

Nickel sensitization sometimes causes conjunctivitis, eosinophilic pneumonitis, and local or systemic reactions to nickel-containing implants (e.g., intraosseous pins, dental inlays, cardiac valve prostheses and pacemaker wires). Ingestion of nickel-contaminated tap water or nickel-rich foods can exacerbate hand eczema in nickel-sensitive persons.

Rhinitis, sinusitis and respiratory diseases: Workers in nickel refineries and nickel electroplating shops, who are heavily exposed to inhalation of nickel dusts or aerosols of soluble nickel compounds, may develop chronic diseases of the upper respiratory tract, including hypertrophic rhinitis, nasal sinusitis, anosmia, nasal polyposis and perforation of the nasal septum. Chronic diseases of the lower respiratory tract (e.g., bronchitis, pulmonary fibrosis) have also been reported, but such conditions are infrequent. Rendall et al. (1994) reported the fatal acute exposure of a worker to inhalation of particulate nickel from a metal arc process; the authors stressed the importance of wearing protective equipment while using metal arc processes with nickel wire electrodes.

Cancer: Epidemiological studies of nickel-refinery workers in Canada, Wales, Germany, Norway and Russia have documented increased mortality rates from cancers of the lung and nasal cavities. Certain groups of nickel-refinery workers have also been reported to have increased incidences of other malignant tumours, including carcinomas of the larynx, kidney, prostate or stomach, and sarcomas of soft tissues, but the statistical significance of these observations is questionable. The increased risks of cancers of the lungs and nasal cavities have occurred primarily among workers in refinery operations that entail high nickel exposures, including roasting, smelting and electrolysis. Although these cancer risks have generally been associated with exposures to insoluble nickel compounds, such as nickel subsulphide and nickel oxide, exposures to soluble nickel compounds have been implicated in electrolysis workers.

Renal effects. Workers with high exposures to soluble nickel compounds may develop renal tubular dysfunction, evidenced by increased renal excretion of b2-microglobulin (b2M) and N-acetyl-glucosaminidase (NAG).

Iron

Hazards: Industrial dangers are present during the mining, transportation and preparation of the ores, during the production and use of the metal and alloys in iron and steel works and in foundries, and during the manufacture and use of certain compounds. Inhalation of iron dust or fumes occurs in iron-ore mining; arc welding; metal grinding, polishing and working; and in boiler scaling. If inhaled, iron is a local irritant to the lung and gastrointestinal tract. Reports indicate that long-term exposure to a mixture of iron and other metallic dusts may impair pulmonary function.

Cancer: Inhaling dust containing silica or iron oxide can lead to pneumoconiosis, but there are no definite conclusions as to the role of iron oxide particles in the development of lung cancer in humans. Based on animal experiments, it is suspected that iron oxide dust may serve as a "co-carcinogenic" substance, thus enhancing the development of cancer when combined simultaneously with exposure to carcinogenic substances.

Mortality studies of haematite miners have shown an increased risk of lung cancer, generally among smokers, in several mining areas such as Cumberland, Lorraine, Kiruna and Krivoi Rog. Epidemiological studies of iron and steel foundry workers have typically noted risks of lung cancer elevated by 1.5- to 2.5-fold. The International Agency for Research on Cancer (IARC) classifies iron and steel founding as a carcinogenic process for humans. The specific chemical agents involved (e.g., polynuclear aromatic hydrocarbons, silica, metal fumes) have not been identified. An increased incidence of lung cancer has also been reported, but less significantly, among metal grinders. The conclusions for lung cancer among welders are controversial.

In experimental studies, ferric oxide has not been found to be carcinogenic; however, the experiments were not carried out with haematite. The presence of radon in the atmosphere of haematite mines has been suggested to be an important carcinogenic factor.

Mercury

Hazards of inorganic mercury: Mercury combines readily with sulphur and halogens at ordinary temperatures and forms amalgams with all metals except iron, nickel, cadmium, aluminum, cobalt and platinum. It reacts exothermically (generates heat) with alkaline metals, is attacked by nitric acid but not by hydrochloric acid and,

when hot, will combine with sulphuric acid. Inorganic mercury is found in nature in the form of the sulphide (HgS) as cinnabar ore, which has an average mercury content of 0.1 to 4%. It is also encountered in the earth's crust in the form of geodes of liquid mercury (in Almadén) and as impregnated schist or slate (e.g., in India and Yugoslavia).

Vapour inhalation is the main route for the entry of metallic mercury into the body. Around 80% of inhaled mercury vapour is absorbed in the lung (alveoli). Digestive absorption of metallic mercury is negligible (lower than 0.01% of the administered dose). Subcutaneous penetration of metallic mercury as the result of an accident (e.g. the breakage of a thermometer) is also possible.

The main routes of entry of inorganic mercury compounds (mercury salts) are the lungs (atomization of mercury salts) and the gastrointestinal tract. In the latter case, absorption is often the result of accidental or voluntary ingestion. It is estimated that 2 to 10% of ingested mercury salts are absorbed through the intestinal tract.

Skin absorption of metallic mercury and certain of its compounds is possible, although the rate of absorption is low. After entry into the body, metallic mercury continues to exist for a short time in metallic form, which explains its penetration of the blood-brain barrier. In blood and tissues metallic mercury is rapidly oxidized to Hg^{2+} mercury ion, which fixes to proteins. In the blood, inorganic mercury is also distributed between plasma and red blood cells.

The kidney and brain are the sites of deposition following exposure to metallic mercury vapours, and the kidney following exposure to inorganic mercury salts.

Chronic exposure: Chronic mercury poisoning usually starts insidiously, which makes the early detection of incipient poisoning difficult. The main target organ is the nervous system. Initially, suitable tests can be used to detect psychomotor and neuro-muscular changes and slight tremor. Slight renal involvement (proteinuria, albuminuria, enzymuria) may be detectable earlier than neurological involvement.

If excessive exposure is not corrected, neurological and other manifestations (e.g., tremor, sweating, dermatography) become more pronounced, associated with changes in behaviour and personality disorders and, perhaps, digestive disorders (stomatitis, diarrhoea) and a deterioration in general status (anorexia, weight loss). Once this stage has been reached, termination of exposure may not lead to total recovery.

In chronic mercury poisoning, digestive and nervous symptoms predominate and, although the former are of earlier onset, the latter are more obvious; other significant but less intense symptoms may be present. The duration of the period of mercury absorption preceding the appearance of clinical symptoms depends on the level of absorption and individual factors. The main early signs include slight digestive disorders, in particular, loss of appetite; intermittent tremor, sometimes in specific muscle groups; and neurotic disorders varying in intensity. The course of intoxication may vary considerably from case to case. If exposure is terminated immediately upon the appearance of the first symptoms, full recovery usually occurs; however, if exposure is not terminated and the intoxication becomes firmly established, no more than an alleviation of symptoms can be expected in the majority of cases.

Kidney. There have been studies over the years on the relationships between renal function and urinary mercury levels. The effects of low-level exposures are still not well documented or understood. At higher levels (above 50 mg/g (micrograms per gram) abnormal renal function (as evidenced by N-acetyl-B-D-glucosaminidase (NAG), which is a sensitive indicator of damage to the kidneys) have been observed. The NAG levels were correlated with both the urinary mercury levels and the results of neurological and behavioural testing.
Nervous system. Recent years have seen the development of more data on low levels of mercury, which are discussed in more detail in the chapter Nervous system in this Encyclopaedia.
Blood. Chronic poisoning is accompanied by mild anaemia sometimes preceded by polycythaemia resulting from bone marrow irritation. Lymphocytosis and eosinophilia have also been observed.

Lead

Lead exposure principally affects haem biosynthesis, but also may act on the nervous system and other systems such as the cardiovascular system (blood pressure). Infants and young children less than five years old are particularly sensitive to lead exposure because of its effect on neurological development at blood lead levels close to 10 mg/dl (CDC 1991).

Several epidemiological studies have investigated the effect of air pollution, especially ozone exposure, on the health of the population of Mexico City. Ecological studies have shown an increase in mortality with respect to exposure to fine particulates (Borja-Arburto et al. 1995) and an increase in emergency visits for asthma among children (Romieu

et al. 1994). Studies of the adverse effect of ozone exposure conducted among healthy children have shown an increase in school absenteeism due to respiratory illnesses (Romieu et al. 1992), and a decrease in lung function after both acute and subacute exposure (Castillejos et al. 1992, 1995). Studies conducted among asthmatic children have shown an increase in respiratory symptoms and a decrease in peak expiratory flow rate after exposure to ozone (Romieu et al. 1994) and to fine particulate levels (Romieu et al. in press). Although, it seems clear that acute exposure to ozone and particulates is associated with adverse health effects in the population of Mexico City, there is a need to evaluate the chronic effect of such exposure, in particular given the high levels of photo-oxidants observed in Mexico City and the ineffectiveness of control measures.

Manganese

Chronic manganese poisoning (manganism): Chronic manganese poisoning can take either a nervous or pulmonary form. If the nervous system is attacked, three phases can be distinguished. During the initial period, diagnosis may be difficult. Early diagnosis, however, is critical because cessation of exposure appears to be effective in arresting the course of the disease. Symptoms include indifference and apathy, sleepiness, loss of appetite, headache, dizziness and asthenia. There may be bouts of excitablity, difficulty in walking and coordination, and cramps and pains in the back. These symptoms can be present in varying degrees and appear either together or in isolation. They mark the onset of the disease.

The intermediate stage is marked by the appearance of objective symptoms. First the voice become monotonous and sinks to a whisper, and speech is slow and irregular, perhaps with a stammer. There is fixed and hilarious or dazed and vacant facies, which may be attributable to an increase in the tonus of the facial muscles. The patient may abruptly burst into laughter or (more rarely) into tears. Although the faculties are much decayed, the victim appears to be in a perpetual state of euphoria. Gestures are slow and awkward, gait is normal but there may be a waving movement of the arms. The patient is unable to run and can walk backwards only with difficulty, sometimes with retropulsion. Inability to perform rapid alternating movements (adiadochokinesia) may develop, but neurological examination displays no changes except, in certain cases, exaggeration of the patellar reflexes.

Within a few months, the patient's condition deteriorates noticeably and the various disorders, especially those affecting the gait, grow steadily more pronounced. The earliest and most obvious symptom during this phase is muscular rigidity, constant but varying in degree, which results in a very characteristic gait (slow, spasmodic and unsteady), the patient putting his or her weight on the metatarsus and producing a movement variously described as "cock-walk" or "hen's gait". The victim is totally incapable of walking backwards and, should he or she try to do so, falls; balance can hardly be preserved, even when trying to stand with both feet together. A sufferer can turn round only slowly. There may be tremor, frequently in the lower limbs, even generalized.

The tendinous reflexes, rarely normal, become exaggerated. Sometimes there are vasomotor disorders with sudden sweating, pallor or blushing; on occasion there is cyanosis of the extremities. The sensory functions remain intact. The patient's mind may work only slowly; writing becomes irregular, some words being illegible. There may be changes in the pulse rate. This is the stage at which the disease becomes progressive and irreversible.

Pulmonary form. Reports of "manganese pneumoconiosis" have been contested in view of the high silica content of the rock at the site of exposure; manganese pneumonia has also been described. There is also controversy over the correlation between pneumonia and manganese exposure unless manganese acts as an aggravating factor. In view of its epidemic character and severity, the disease may be a non-typical viral pneumopathy. These manganic pneumonias respond well to antibiotics.
Pathology. Some authors maintain that there are widespread lesions to the corpus striatum, then to the cerebral cortex, the hippocampus and corpora quadrigemina (in the posterior corpora). However, others are of the opinion that the lesions to the frontal lobes provide a better explanation for all the symptoms observed than do those observed in the basal ganglia; this would be confirmed by electroencephalography. The lesions are always bilateral and more or less symmetrical.

Course. Manganese poisoning ultimately becomes chronic. However, if the disease is diagnosed while still at the early stages and the patient is removed from exposure, the course may be reversed. Once well established, it becomes progressive and irreversible, even when exposure is terminated. The nervous disorders show no tendency to regress and may be followed by deformation of the joints. Although the severity of certain symptoms may be reduced, gait remains permanently affected. The patient's general condition remains good, and he or she may live a long time, eventually dying from an intercurrent ailment.

Copper

Chronic toxicity: Chronic toxic effects in human beings attributable to copper appears only to be found in individuals who have inherited a particular pair of abnormal autosomal recessive genes and in whom, as a consequence, hepatolenticular degeneration (Wilson's disease) develops. This is a rare occurrence. Most daily human diets contain 2 to 5 mg of copper, almost none of which is retained. The adult human body copper content is quite constant at about 100 to 150 mg. In normal individuals (without Wilson's disease), almost all of the copper is present as an integral and functional moiety of one of perhaps a dozen proteins and enzyme systems including, for example, cytochrome oxidase, dopa-oxidase and serum ceruloplasmin.

Tenfold, or more, increases in the daily intake of copper can occur in individuals who eat large quantities of oysters (and other shellfish), liver, mushrooms, nuts and chocolate—all rich in copper; or in miners who may work and eat meals, for 20 years or more, in an atmosphere laden with 1 to 2% copper ores dusts. Yet evidence of primary chronic copper toxicity (well defined from observations of patients with inherited chronic copper toxicosis—Wilson's disease—as dysfunction of and structural damage to the liver, central nervous system, kidney, bones and eyes) has never been found in any individuals except those with Wilson's disease. However, the excessive copper deposits that are found in the livers of patients with primary biliary cirrhosis, cholestasis and Indian childhood cirrhosis may be one contributing factor to the severity of the hepatic disease that is characteristic of these conditions.

Zinc

Hazards: A number of zinc salts may enter the body by inhalation, through the skin or by ingestion and produce intoxication. Zinc chloride has been found to cause skin ulcers. Metallurgic processes involving zinc can lead to arsenic, cadmium, manganese, lead and possibly chromium and silver exposures, with their associated hazards. Since arsenic is frequently present in zinc, it can be a source of exposure to highly toxic arsine gas whenever zinc is dissolved in acids or alkalis.

In zinc metallurgy and manufacturing, welding and cutting of galvanized or zinc-coated metal, or melting and casting of brass or bronze, the most frequently encountered hazard from zinc and its compounds is exposure to zinc oxide fumes, which cause metal-fume fever. Symptoms of metal-fume fever include shivering attacks, irregular fever, profuse sweating, nausea, thirst, headache, pains in the limbs and a feeling of exhaustion. Attacks are of short duration (most cases are on the way to complete recovery within 24 hours of the onset of symptoms), and tolerance seems to be acquired. A significant increase in free erythrocyte protoporphyrin has been reported in zinc oxide packing operations.

Zinc chloride fumes are irritating to the eyes and mucous membranes. In an accident involving smoke generators, 70 exposed persons experienced varying degrees of irritation of the eyes, nose, throat and lungs. Of the 10 fatalities, some died within a few hours with pulmonary oedema, and others died later of bronchopneumonia. On another occasion, two firemen were exposed to zinc chloride fumes from a smoke generator during a firefighting demonstration, one briefly, the other for several minutes. The former recovered rapidly while the latter died after 18 days, due to respiratory failure. There was a rapid rise of temperature and marked upper respiratory tract inflammation soon after exposure. Diffuse pulmonary infiltrations were seen on the chest radiograph, and autopsy revealed active fibroblastic proliferation and cor pulmonale.

Skin effects. Zinc chromate in primer paints used by car-body builders, tinsmiths and steel cupboard makers, has been reported to cause nasal ulceration and dermatitis in exposed workers. Zinc chloride has a caustic action, which may result in ulceration of the fingers, hands and forearms of those who handle timber impregnated with it or use it as a flux in soldering. It has been reported that zinc oxide dust may block the ducts of the sebaceous glands and give rise to a papular, pustular eczema in humans packaging this compound.

Annex 4: Risk Analysis method

EXPOSURE CALCULATIONS

Approach

Where available, cancer potencies, unit risks, reference concentrations, and reference doses were obtained from the Integrated Risk Information System (IRIS). For chemicals not included in IRIS, toxicity data were extracted from the Health Effects Assessment Summary Tables (HEAST), distributed by the Office of Emergency and Remedial Response. The toxic hazard data used to prepare this report were current as of the date supplied for the database. However, these values may have been modified since the update of the database. Users are urged to consult IRIS and the latest HEAST tables directly.

Data

Toxic substance concentration data used for risk computations was from the Rapid environmental impact study: Expansion of steel plant done for the Jindal Steel and Power Ltd, Raigarh.

Exposure Pathways

The exposure pathway used was air. An exposure pathway combines contamination in an environmental medium, a scenario describing how a person contacts that medium, and a route of exposure (oral, inhalation, or dermal). The following list indicates the pathways considered in this assessment:

Air: inhalation of vapors indoors (air) (Inhalation), inhalation of vapors outdoors (air) (Inhalation)

Exposure Parameters

The dose (or exposure concentration) values presented in this assessment reflect concentrations of contaminants in various environmental media and the exposure pathways selected for analysis, as also the specific parameters applied to each exposure scenario.

Exposure Estimates

When an exposure assessment is used as part of a quantitative risk assessment, a numerical estimate of exposure must be calculated. When evaluating effects from inhalation exposure, contaminant concentrations are compared to Reference Concentrations (RfCs) for continuous exposure. If exposures occur for relatively short durations (less than 8 hours), care should be taken in comparing exposure concentrations to reference concentrations. When evaluating carcinogenic risks from exposures that last less than a lifetime for inhalation exposures, Adjusted Concentration is computed as:

Adjusted Concentration = Concentration * (exposure period / lifetime)

Typically, the adjusted concentration will also incorporate other adjustments for differences between the actual exposure pattern and the assumed pattern of continuous lifetime exposure. For example, if exposure only occurred for one hour each day, the Adjusted Concentration would be only 1/24th of the concentration during that hour.

Uncertainties

To understand the meaning of the quantitative dose estimates presented in this assessment, it is necessary to consider the key assumptions used in deriving them, and the uncertainties associated with those assumptions:

A key assumption is that the concentrations specified for various environmental media represent the true concentrations to which people will be exposed during the period of exposure. Actual contaminant concentrations will likely vary across both time and space.

Actual exposures to members of any specified population will vary in accordance with the degree to which they participate in the activities described by the exposure scenarios.

The uncertainty analyses illustrate the differences between calculated doses and those calculated using standard (average or reasonable maximum exposure) numerical parameter values for each scenario selected. They can also provide information on the way in which selection of exposure scenarios influences estimates.

These uncertainty analyses do not consider uncertainty regarding chemical concentration measurements or the variability of chemical concentrations across space and time. Neither do they address uncertainty associated with models of contaminant transport or inter-media transfer of contaminants.

Toxic Hazards

The risk estimates presented in this assessment reflect not only the specific exposure pathways evaluated, but also estimates of the inherent toxic hazards posed by each chemical assessed. Carcinogenic hazards are estimated as the slope of the dose-response or concentration-response function. The steeper the slope of this function, the smaller the dose, or the lower the concentration, required to produce a particular level of risk. It is generally assumed that carcinogenic risk is zero only when exposure is zero, and that at low doses, the relationship between dose and response can be approximated by a straight line. For inhalation exposures, the slope of the concentration-response function (Unit Risk) is used.

It is generally assumed that non-cancer toxic effects have some threshold. That is, up to some finite level of exposure, physiological defense mechanisms ensure that no toxic effect will occur. Accordingly, hazard assessment for non-carcinogenic effects involve estimating an exposure that is less than this threshold level. This is done by applying "uncertainty factors" to exposures that appear to be near this threshold in laboratory toxicology studies. This yields a Reference Concentration (RfC) for inhalation exposures.

Where possible, carcinogenic Slope Factors and Unit Risks, and Reference Concentrations for non-cancer hazards, have been obtained from the Integrated Risk Information System (IRIS). For chemicals not included in IRIS, toxicity data have been extracted from the Health Effects Assessment Summary Tables (HEAST).

Slope Factors and Unit Risks are generally estimated as the 95th percentile confidence limits using the linearized multistage model. As such, they are conservative estimates of toxic hazard. Risks estimated by combining these hazard values with exposure estimates are commonly referred to as upper-bound risks, but because exposure estimates may not represent upper-bound estimates, risk estimates are not true upper-bound risks.

A similar effort is made to ensure that Reference Concentrations provide a conservative estimate of non-cancer toxic hazards. The uncertainty factors applied to toxicity data are intended to take into account differences in sensitivity to toxic effects within and between species, and differences in toxic effects between chronic and subchronic exposures.

RISK ESTIMATES

Different approaches are used in the calculation of risk for chemicals that may cause cancer (carcinogens) and for chemicals with other toxic effects. If the risk results from breathing the chemical, the calculation is based on concentration, rather than dose, as follows:

$$\text{Risk} = 1 - e^{-(\text{Unit Risk} * \text{Concentration})}$$

These estimates represent the theoretical excess cancer risk (ie, risk over background cancer incidence) of developing cancer. For example, if the calculated risk is $1x10^{-6}$, this would literally suggest that a person would have a one-in-a-million chance of getting cancer because of the specified chemical exposure, in addition to her/his chance of getting cancer from other causes. However, in view of the large uncertainties associated with such risk estimates, they should always be interpreted as general indicators, rather than precise estimates. The US EPA generally considers risks below $1x10^{-6}$ to be low (Risk = Probability of getting cancer from specified exposure).

For agents that cause non-cancer toxic effects, a Hazard Quotient (HQ) is calculated, which compares the expected exposure to the agent to an exposure that is assumed not to be associated with toxic effects. For inhalation exposures, the inhaled concentration is compared to a Reference Concentration (RfC):

HQ = Inhaled Concentration / Reference Concentration

A Hazard Index, representing the sum of the Hazard Quotients for each chemical and exposure scenario to which a given person may be exposed, is used to evaluate the likelihood of non-cancer toxicity. Hazard Indices < 1.0 are generally considered by EPA to be associated with low risks on non-cancer toxic effects.

Uncertainties

Because risk values incorporate all of the estimates, default values, and assumptions used throughout risk assessment, results must be understood in terms of key uncertainties regarding both the toxic hazard values and the exposure estimates used to derive them. For a very limited set of chemicals, toxic hazard values may be estimated from epidemiologic data collected in humans. Most slope factors and RfCs are derived from experimental studies in animals. Such extrapolations are based on the assumptions that:

1) the physiological and biochemical responses of exposed persons will be qualitatively (but not necessarily quantitatively) the same as that seen in the experimental animals,

2) effects seen at high doses in a limited number of animals over a comparatively brief period of observation are predictive of toxicity at lower doses, if a sufficiently large group is exposed for a sufficiently long period.

For some chemicals, hazard values may also have been extrapolated across differing routes of exposure. This introduces additional uncertainty to these estimates.

Annex 4: Villages and their populations in a 10 and 25 km radius around the JSPL Plant

	Villages in 10 km radius around JSPL	Block	Population
1	Patrapali	Raigarh	3352
2	Saraipali	&	619
3	Kokditarai	&	4334
4	Chiraipali	&	1162
5	Korkha	&	1063
6	Riyakhol	&	214
7	Uchchbhithi	&	1243
8	Parsada	&	732
9	Khairpur	&	1410
10	krishnapur	&	745
11	Bhagwanpur	&	1467
12	Kerajhar	&	462
13	Kodtarai	&	1255
14	Dongitarai	&	799
15	Dongadakel	&	565
16	Gejamuda	&	1351
17	Kanshichu	&	1578
18	Kalmi	&	1278
19	Bermuda	&	736
20	Chatamura	Pussore	2200
21	Sahdewpali	&	1040
22	Kushwabahri	Raigarh	515
23	Kosampali	&	2145
24	Kanhar	&	422
25	Pandripni	&	1278
26	Charbhantha	&	412
27	Raigarh urban	&	115908
28	Dumarpali	Pussore	894
29	Gardumaria	&	3274
30	Kodatari	&	3004
31	Kunjedabri	&	430
32	Dumarpali	Raigarh	764
33	Kenapali	&	280
34	Borapali	&	634
35	Sangitarai	&	1218
36	Gowardhanpur	&	501
37	Udena	&	2659
38	kosamnara	&	833
39	Rampur	&	178
40	Kusmura	&	1722
41	Dewri	&	1118
42	Kotra	&	1313
43	Paltelpali	&	1307
44	Barsiya	&	1431
45	Dhanager	&	2310
46	Tarsiya	&	922
47	Badeatermuda	&	546
48	pandripni	&	847
49	Belpali	Pussore	554
50	Jamchunwa	Tamnar	143
51	Jiwari	&	789
52	Bhueikurri	&	630
53	Bagbuda	&	205
54	Saraipali	&	1303
55	Dharakpur	&	522
56	Usrotha	Raigarh	937
57	Kusmura	&	833
58	Bendrachua	&	222
59	amapal	&	331
60	baghanpur	&	790
61	Kurmapali	&	809
62	Tarekela	&	1484
63	Chuhipali	&	619
64	Bijaypur	&	1377
65	Lakha	&	756
66	Jurda	&	1424

67	Gopalpur	&	1121
68	Darama	Tamnar	744
69	Barpali	&	328
70	Gaourmudi	&	258
71	Aorabhantha	Raigarh	995
72	Tarapur	&	1049
73	Bhikharimal	&	611
74	Lebada	&	589
75	Chitkakani	&	153
	POPULATION IN A 10 KM RADIUS AROUND JSPL		**194,046**

	Villages in 11-25 km radius around JSPL	**Block**	**Population**
76	Kachhar	Raigarh	1692
77	Raniguda	&	367
78	Sardamal	&	919
79	Bokramuda	&	506
80	Chiraipani	&	333
81	Pali	&	392
82	Bhelwapali	&	799
83	Jogitari	Pussor	699
84	Jampali	&	354
85	Tetla	&	1963
86	Tadola	&	1503
87	Jampali	&	711
88	Khokhra	&	1203
89	Bijna	&	400
90	Loharsin	&	1609
91	Jakela	&	1362
92	Surri	&	1211
93	Kharmuda	&	471
94	Karrajor	&	532
95	Sarasmal	&	291
96	Sulini	&	347
97	Lankapali	&	445
98	Chhinch	&	746
99	Tilgi	&	1048
100	Gudgahan	&	689
101	Amlidiha	&	95
102	Midmida	&	1863
103	Ournda	&	2177
104	Ghanatarai	&	261
105	Daubhatli	&	240
106	Panjhar	Raigarh	663
107	Gerwani	&	1135
108	Delari	&	700
109	Lamidarha	&	410
110	Koterlia	&	1455
111	Kotrapali	&	535
112	Rabo	Tamnar	543
113	Nandeli	Raigarh	1809
114	Ganout	&	744
115	Thakurpali	&	429
116	Gorra	Pussore	1799
117	Telipali	&	616
118	Linjir	&	1139
119	Chikhli	&	822
120	Sukulbhatli	&	486
121	Nawapali	&	476
122	Nawapali	&	286
123	Badmal	&	639
124	Amapali	&	757
125	Malda	&	1084
126	Badehaldi	&	1157
127	Kosmanda	&	399
128	Bhaghadola	&	1556
129	Chhotehaldi	&	261
130	Karichhper	&	4516
131	Raitarai	&	664

132	Bbulaki	&	400
133	Kawariha	&	761
134	Badimal	&	524
135	Torna	&	282
136	Nandeli	&	837
137	Kotsura	&	1493
138	Gotma	&	986
139	Keshla	&	666
140	Mouhapali	&	353
141	Nawapara	&	970
142	Darripali	&	620
143	Bulaki	&	882
144	Khaprapali	&	383
145	Arsipali	Raigarh	619
146	Litaipali	&	555
147	Salhepali	&	409
148	Nawrangpur	&	1096
149	Regda	&	1276
150	Tarpali	&	1676
151	Vishwanathpali	&	785
152	Loing	&	1850
153	Siyarpali	&	657
154	Mahapalli	&	1945
155	Bergaon	Tamnar	625
156	Harradih	&	432
157	Gadgaon	&	405
158	Padkipahari	&	352
159	Bhaisgardi	&	230
160	Teka	Pussor	604
161	Kouwatal	&	842
162	Basanpali	&	739
163	Dhangaon	&	463
164	Rawankhondhra	&	374
165	Ruchida	&	1527
166	Litaipali	&	643
167	Pusalda	&	1423
168	Raibar	&	813
169	Thengagudi	&	801
170	Jewridih	&	409
171	Boerdih	&	331
172	Darramuda	&	866
173	Bhathanpali	&	815
174	Jhulanpali	&	229
175	Ghutkupali	&	319
176	Dewalsurra	&	517
177	Odekeara	&	1084
178	Tarda	&	1016
179	Baradoli	&	348
180	Bayan	Raigarh	1711
181	Sambalpuri	&	808
182	Junwani	&	386
183	Barpali	&	908
184	Mouhapali	&	345
185	Baghpali	&	475
186	Kosampali	&	373
187	Salheona	&	672
188	Pacheda	Pussore	668
189	Amlidiha	&	580
190	Nawapara	&	1072
191	Putkapuri	&	1158
192	Kensara	&	1432
193	Ghughwa	&	755
194	Semibhanwer	&	313
195	Shankerpali	&	265
196	Siha	&	1124
197	Bonda	&	734
198	Chhichora	&	2181
199	Saraipali	&	675
200	Kesapali	&	366

201	Takurpali	&	316
202	Binjkot	&	374
203	Jhalmala	&	878
204	Sodekela	&	951
205	Ghuranpali	&	307
206	Riyapali	&	147
207	Aarmuda	&	471
208	Guddu	&	804
209	Taraimal	Tamnar	620
210	Jamdagri	&	777
211	Ujjalpur	&	74
212	Kalmi	Pussore	390
213	Tinmini	&	1702
214	Dhanuadera	&	892
215	Thengapali	&	376
216	Basanpali	&	739
217	Mahloie	&	627
218	Pussore	&	400
219	Padigaon	&	677
220	Patrapali	Raigarh	1599
221	Jatri	Pussore	1420
222	Semra	&	934
223	Chhotebhandar	&	502
224	Suma	&	780
225	Sutupali	&	413
226	Ektal	&	524
227	Netnark	&	1252
228	Kasaipali	&	346
229	Chapora	&	1188
230	Baispali	&	652
231	Kulba	&	963
232	Nawagaon	&	399
233	Dulopur	&	429
234	Bhatpur	&	542
235	Tilga	&	1359
236	Bhagora	&	1102
237	Kukurda	&	1247
238	Katajharia	Tamnar	176
239	Amapali	&	772
240	Gorkamuda	&	97
241	Ratrot	&	128
242	Barbahli	&	111
243	Kotmara	Pussore	1023
244	Barpali	&	693
245	Badebhandar	&	1184
246	Kotmar	Raigarh	708
247	Saraipali	&	502
248	Sarwani	&	986
249	Kantahardi	&	1255
250	Karichapar	&	350
251	Dumarpali	&	442
252	Amlipali	Pussore	132
253	Sarwani	&	419
254	Amlibhouna	&	483
255	Machida	&	456
256	Silyari	Tamnar	265
257	Chirbhouna	&	139
258	Punjipathra	&	366
259	Amaghat	&	1018
260	Samaruma	&	392
261	Nawapara	Raigarh	909
262	Dhumabahal	&	220
263	Dewbahal	&	175
264	Shivpuri	&	249
265	Banora	&	1392
266	Chuhipali	&	762
267	Nawapara	&	633
268	Bunga	Pussore	2366
269	Tribhouna	&	2792

270	Tumidih	Gharghoda	1033
271	Kasdol	Tamnar	1326
272	Kachkoba	&	1056
273	Dumigol	&	389
274	Nawapara	&	293
275	Bardaputi	Raigarh	819
276	Sarbahahal	&	144
277	Bhueyapali	&	566
278	Belaria	&	886
279	Dumarpali	&	250
280	Godhi	Tamnar	1774
281	Basantpur	Raigarh	364
282	Bangursiya	&	522
283	Nayagaon	&	362
284	Jharguda	&	306
285	Natwarpur	&	347
286	Jamgaon	&	935
287	Manuapali	&	774
288	Kahirpali	&	350
289	Dokerbuda	Gharghoda	553
290	Bodajharia	Pussore	812
291	Tamnar	Tamnar	3974
292	Turanga	Pussore	1082
293	Kondpali	&	1450
294	Kathani	&	839
295	Lohakhan	&	620
296	Kandagarh	&	830
297	Rrengalpali	&	353
298	Sahaspuri	Raigarh	1059
299	Junwani	&	678
300	Adbahal	&	405
301	Sapnai	&	455
302	Shikosimal	&	157
303	Balbhadrapur	&	285
304	Kolaibahal	&	1513
305	Behrapali	&	630
306	Chharatanger	Gharghoda	1840
	POPULATION IN A 11-25 KM RADIUS AROUND JSPL		**182,340**

POPULATION IN A 10 KM RADIUS AROUND JSPL **194,046**

POPULATION IN A 11-25 KM RADIUS AROUND JSPL **182,340**

TOTAL POPULATION IN A 25 KM RADIUS AROUND JSPL **376,386**

Annex 6: Phyto-toxic Effects of Air Pollutants

Sulphur Dioxide

Of the two major gaseous emissions—sulphur dioxide (SO_2) and nitrogen dioxide (NO_2), NO_2 is more phyto-toxic. The major effects on plants are foliar injury, changes in micro-morphology, plant growth and productivity. High level of foliar injury has been observed nearer emission sources, gradually reducing with an increase in distance from the source. The injury is particularly marked in the footprint of pollution plumes.

Fruit trees, particularly mango, are quite sensitive to SO_2. Rice and legumes too are fairly sensitive to SO_2. Trees, however, are more tolerant to SO_2 doses.

Vegetation in the vicinity of polluting plants has been recorded to sustain injury at SO_2 concentrations as low as 5-20 ug/m3, particularly when other pollutants are also present. Exposure of vegetation to SO_2 decreases chlorophyll content, leaf area, root and shoot size and lowers biomass. It is also possible that elimination of certain types of vegetation may occur in areas where SO_2 levels are high.

Nitrogen dioxide

Nitrogen dioxide is less phyto-toxic than SO_2, but more so than nitrous oxide (NO). Along with SO_2, it accentuates plant injury. By itself, NO_2 concentrations need to be high to cause plant injury. The importance of NO_2 as a pollutant is primarily because of its participation in photochemical reactions giving rise to ozone and peroxyacetyl nitrate (PAN), both being highly phytotoxic secondary pollutants.

Sensitive weeds may be injured by NO_2 at 20 ppm and pinto beans at 3 ppm. NO_2 reacts with water vapour in the atmosphere to form nitric acid, which is brought down with rain.

Particulate matter

Dust is quite harmful, particularly to mango and lemon plants. For example, in-situ coal dust covered mango and lemon leaves showed brown necrotic lesions, starting at the tip and progressing down the lamina. A significant reduction in the fruit yield of both plants was observed. Fly ash in moderate to large doses is harmful to plants, which includes changes in the cuticular pattern of leaves. Garg and Varshney have reported that fly ash from power plants produce visible damage to certain plants. Dubey et al. report severe ill effects of fly ash and SO2 on Cicer arietinum L.

Alkaline fly ash from stacks will amend the acidic soils, though has to be done with caution due to the possibility of heavy metal accumulation in soils. Animals grazing grass with fly ash deposition may be affected.

Synergistic effects

Synergistic effect of SO_2, NO_2 and dust will affect some plant pecies more than others. Economically valuable plants such as paddy, mango and chickoo will also experience decreased yields, canopy and biomass, leaf size reduction, greater leaf fall and chlorophyll loss.