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PRESIDENT WRITES

Dear Fellows and Members,

I am glad that the very relevant topic of environment and medicine is being highlighted in this issue of the JIMSA.

There has been steady progress in our understanding of disease from the days of the theory of unifactorial causation of disease to the theory of "Multifactorial causation of disease" which involves disease-causing agent factors, host factors and environmental factors.



Environmental factors are from physical, chemical and biological spheres and generally speak of the external environment. These are often overlooked in the more complex disturbance of the internal environment and homeostasis that we doctors deal with day to day.

It is heartening to see that there is growing environmental activism arising all around and may this trend grow more and more. However, both realistic activism and a vision for the greatest common good needs, are to be kept in mind while regulating human development that interferes adversely with environment.

The medical profession has a social responsibility to actively perform i.e. to help promote environmental factors that will enhance the well being of all.

Let us all encourage the nations to make Kyoto Proclamation' a reality.

Dr. K. Jagadeesan,
President, IMSA



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FROM EDITOR'S DESK

Dear Colleagues

The present special issue deals with some of the important topics related to 'Environmental Pollution and its impact on human health'. WHO drew the attention of the medical fraternity as early as 1972, when United Nations in its General Assembly passed a resolution which led to the creation of 'World Environment Day' to be celebrated the world over, on the 5th June of every year. The agenda set for the world environment day was to give a human face to the environmental issues, empower people to become active agents of sustainable development to promote an intense feeling that community can play a pivotal role to change the public attitude towards environmental issues. The problem has been multiplied by rapid industrialisation which has led to proliferation of hazardous substances; consequence of these is development of serious ill health and disease in the community, especially in the developing countries. Thus, there is an ever demanding need of creating both public awareness and political attention to deal aggressively with the environmental pollution.

This issue on 'Environment Pollution & Human Health', has been possible due to the untiring efforts of guest editors **Dr. IPS Kalra**, **Prof. S.K. Bhargava** and **Dr. Pratibha Gupta**; the topics covered are very topical and are of immense importance in day to day practice; the contributors are experts in their fields and have put in their best while analysing the data derived from their original work as well as from the current literature. I am extremely grateful to Drs. IPS Kalra, SK Bhargava and Pratibha Gupta; and also the various contributors to this special issue. I am confident that our fellows/members and several other readers/subscribers will find scientific material very useful.

I take this opportunity to thank the members of the editorial/advisory boards for their help in the compilation of this issue. I am grateful to various pharmaceutical firms for the financial assistance, provided for preparing this issue.

P.D. Gulati

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Our Guest Editors



Dr. I.P.S. Kalra

Dr. I.P.S. Kalra is presently the Secretary General of International Medical Sciences Academy (IMSA). He is a senior consultant

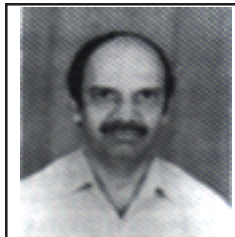
Physician-Cardiologist attached to Guru Harkrishan Hospital, Bangla Sahib and Bala Sahib (Charitable organizations) working for the last 33 years, looking after poor patients of hypertension, diabetes and cardiac ailments. He is also President of Hari-Chint Centre-NGO, involved in chairtable works.

Dr. Kalra is the Secretary General of upcoming Guru Harkrishan Institute of Medical Sciences and Research at Guru Bala Sahib Complex. He is also Chairman Guru Nanak Chairtable Poly Clinic.

He has been past President of IMA New Delhi Branch; and has been a consultant to armed forces and also Secretary, IMA Academy fo Medical Specialities National level. He was Chairman of IMA Academy of Medical Specialities, Delhi State.

As Secretary General of IMSA, since 1997, Dr. Kalra's contribution to the growth of IMSA has been commendable. The various chapters have got activated; and have already started the CME programmes. Credit for organising annual conferences **(IMSACON)**, both National & International, held regularly since he took over, largely goes to his untiring efforts. Rural CME programme has been given a boost during the last 5 years.

As one of guest editors of the present issue on 'Environmental Pollution & Human Health', he along with Prof. S.K. Bhargava & Dr. Pratibha Gupta has put in his best efforts to bring out this important publication of National interest.



Prof. Satish K. Bhargava

Prof. Satish K. Bhargava, MBBS MD (Radiodiagnosis), MD (Radiotherapy), DMRD, FUSI, FCCP, FICRI, FIAMS, FNAMS, FIMSA is currently Head, Department of Radiology & Imaging at University College of Medical Sciences (Delhi University) & GTB

Hospital, Delhi & Chairman, Board of Research Studies, University of Delhi. He is also a Consultant Radiologist at the WUS Health Centre, North Campus, University of Delhi. Prof. Bhargava was deputed to HOMS (Syria) on Govt. of India assignment and also an adviser to set up the Department of Radiology at BPKIHS, Dharan, Nepal; he continues to be visiting Professor at that Institute.

Author of many books 'Text Book of Radiology for Technicians' sponsored by WHO, 'Clinical Sonography', 'Colour Doppler Imaging', 'Diagnostic Radiology & Imaging' and many other guide books, Prof. Bhargava has been an excellent teacher; he has contributed more than 300 paper in National and International Journals; contributed many chapters in text books of Paediatrics, Orthopaedics, Obst. & Gynae. He has also been on Editorial Board of number of National and International Journals. Due to his wide academic contribution, he has many awards & orations to his credit 'Sir J.C. Bose Memorial Oration', 'Distinguished Medical Teachers Award', 'Dr. B.C. Roy Award', 'Brig. S.K. Majumdar Oration Award', 'Swami Vivekanand Memorial Oration Award', 'Prithvinath Bhargava Memorial Oration Award', 'Panna Lal Oration Award' and 'GEMS International Award'. Prof. Bhargava's unique contribution is his book in Hindi sponsored by WHO and 'Cancer Ke Tathey'; for this he has been awarded 'Maulik Hindi Pustak Puruskar' by the Ministry of Health and Family Welfare, Govt. of India. He has also been authorised by WHO to translate and edit a book of Ultrasound in Hindi 'Manual of Diagnostic Ultrasound' by M/s. Jaypee Bros.

Prof. Bhargava has widely travelled abroad on a number of assignments and fellowships like WHO fellowship to USA & Finland, Visiting Fellowship to Holland, Harvard Medical School, Boston, USA, Thomas Jafferson Medical College Philadelphia, USA, and some centres in UK. He was also invited by the french Govt. to attend a symposium on latest Imaging techniques held at Paris. Prof. Bhargava was instrumental in starting 10+2 X-ray technician Certificate course at UCMS & GTB Hospital, as part of CBSE syllabus under the Ministry of Health & Family Welfare and Ministry of HRD, Govt. of India, B.Sc. (Medical Technology) Lab. Technology (Radiography) course under University of Delhi and Diploma in Radiography through National Open School (autonomous) under Ministry of HRD, Govt. of India. Prof. Bhargava has organised many CMEs; he has served as Secretary, Indian College of Radiology and Imaging (1993-98) and as Chairman, Indian College of Radiology & Imaging (1998-2000) & 2001-2002; Prof. Bhargava is now president of Indian Radiological & Imaging Association for the year 2005.

Speical Issue : Environmental Pollution & Human Health**EDITORIAL**

While writing editorial for issue of JIMSA on environmental pollution and health, the most important aspect in positive health is pollution of mind by wrong environments. Right from childhood, if company of child is wrong, the mind is affected badly. If parents put their children and themselves in the company of right thinking people and avoid bad company then positive mental health is ensured.

Our sages in all scriptures have written about it. Guru Arjan Dev, the fifth Nanak, has at length written in Sukhmani Sahib as to what you get in Saad Sangat (company of saints, right thinking people). Just a glimpse of Sukhmani (treasure of pleasure):

“*Saadh Ke Sang Ave Bas Pancha* (one can control five evil passions in company of saints. These are *Kaam*, Meaning sex, *Krodh*, meaning anger, *Lobh*, meaning greed, *Moh*, meaning attachment, *Ahankar*, meaning ego, the most difficult passion to control.)”

Guru Nanak says, there is no difference between saint and God almighty.

Nanak Sadh Prabh Bhed Na Bhai.

If parents follow this path and teach their children these practices and values, then persons can learn to avoid other environmental pollutions like that of air, food, and water which the authors have brought up so nicely in this issue. I leave it to my co-editors to comment.

Dr. I.P.S. Kalra

Consultant Physician - Cardiologist

The 20th century has seen many revolutions in technology. Some have improved life but many more have threatened human and animal life and the environment. The industrialized nation has shown proliferation of hazardous substances which have adversely affected the developing countries, where environmental law and enforcement of safe handling practices are nonexistent, the problem has multiplied. Work place and environmental exposure to hazardous substances, alone or in combination, cause serious ill health and disease.

Environment its impact on human health has become a subject of major concern for the medical fraternity and public pollution has changed the life style of community. WHO is fighting tooth and nail to keep the environmental balance intact. According to WHO unsafe working conditions are rampant around the globe and if conditions are not improved hundreds of millions of people who work will be at risk”.

Past several years have witnessed a revolution for realizing the hazardous effects of environmental pollution all over the globe, such knowledge is greatly enhanced by the consistent efforts of activists, and by international and national summits, conferences and seminars; organised on the subject.

The International Medical Sciences Academy through its journal i.e. JIMSA under the leadership of Prof. P.D. Gulati and Dr. IPS Kalra have rightly thought to bring out a special issue on “Environmental Pollution & Human Health” so as to enate awareness which will definitely help in preventing this menace.

Prof. Satish K. Bhargava

Dr. Pratibha Gupta

Ex. Prof. UCMS, Delhi

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- Visiting Professor - BP Koirala Instt. Of Health Sciences, Nepal

- National President - Indian Radiological & Imaging Association

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Respiratory Function Tests in Rubber Factory Workers

MANISH GUPTA, PRATIBHA GUPTA, SATISH K. BHARGAVA*

Departments of Physiology & Radiology* & University College of Medical Sciences & G.T. B. Hospital, Dilshad Garden, Delhi-110095, India

Abstract: Respiratory functions of 607 workers from rubber processing unit were studied in relation to occupational exposure to various pollutants viz. suspended particulate matter, polycyclic aromatic hydrocarbon compound (PAH), sulphates and nitrates. The results showed significant decline in lung functions proportionately with the longer working duration and to the increased pollutant concentration of the various sections of the rubber factory. Workers in the compounding (mixing) unit were most affected as pollutant concentration found in this section was maximum; this was supported by the abnormal X-ray findings.

Introduction

The presence of suspended particulate matter (SPM), polycyclic aromatic hydrocarbon compounds (PAH) e.g. benzo(a)pyrene, benzo(e)pyrene, benzo(a)anthracene, sulphates and nitrates in the rubber factory has raised concerns about the respiratory health of the workers. Long term exposures of workers to these pollutants in their workplace environment not only derange lung functions, but may produce effects ranging from chronic bronchitis to cancer of lungs¹⁻³. Approximately 500 substances are used in the manufacture of rubber⁴ and most of these are capable of causing acute and chronic respiratory effects^{4,7}. Quite a few studies from abroad do reveal the presence of benzo(a)pyrene in the air as a good indicator of exposure to PAH compound⁸. Exposure to noxious agents in the rubber factory are associated with the development of acute and chronic respiratory impairment⁹. Some workers^{10,11} demonstrated significantly lower ventilator capacity tests and higher prevalence of respiratory symptoms in rubber factory workers as compared to the controls. The present study was carried out in the rubber factory workers to evaluate the effect of particulate matter and PAH various lung functions e.g. forced vital capacity (FVC: the maximum volume air expelled after a maximum voluntary inspiration), the forced expiratory volume in 1 second (FEV₁: that part of the FVC expired in first second of forced expiration), peak expiratory flow rate (PEFR), peak inspiratory flow rate (PIFR) and forced expiratory flow 25-75% (FEF_{25-75%}). Radiographic examination of their chest was also done.

Methods

Environment Sampling: Air sampling at the factory was carried out with a five-stage Kimoto cascade sampler operating at a flow rate of 1.7 l/m²/min on glass fibre filter paper (GF/A Whatman) giving fraction sizes of (in μm): >10.4, >5.2, >1.6, >0.6 and <0.5. Six samples (8 hour duration) were collected over a period of 3 months in each of the following three different stages of the rubber manufacturing process: packing and loading, vulcanization and compounding. The particle concentration (total suspended particulate matter: TSP) of each sample was estimated gravimetrically, and the benzo(a)pyrene from the five fractions of particles was determined after extraction with benzene in a Soxhlet apparatus for 8-10hr¹²⁻¹³. The extracted material was concentrated in a Buchi rotavapour and then deoxygenated by passing nitrogen through it. Finally, the sample was analysed in a Ferrands-3 scanning spectrophluorometer¹⁴. The qualitative determination was carried out using an excitation-correction module coupled with the instrument.

Lung Function Tests: Information regarding rubber factory workers employed in different sections was collected between October 1989 and March 1990. A questionnaire requesting details on age, duration of employment, smoking habits and full medical history was completed by all 667 workers examined. Their height and weight were measured, as were their FVC, FEV₁, FEF_{25-75%}, PIFR and FEF_R (with an electronic lung function machine: model Elf, P.K. Morgan, UK). A chest X-ray (posterior-anterior view) was taken of each worker studied.

Correspondence: Prof. S.K. Bhargava, E-3, GTB Hospital Campus, Dilshad Garden, Delhi-95, India

Data Analysis: To examine the effect of the duration of exposure, three subgroups of workers were formed according to the duration of employment. Statistical analysis was by one-way analysis of variance with Tukey's test technique, using an SPSS package. The relationship between the results of the pulmonary function tests and variables was assessed by multiple linear regression with an examination of residuals. Values are expressed as the mean and standard deviation.

Results

The quantity of suspended particulate matter (SPM) (mean+SD) to which each group of workers was exposed was as follows (in $\mu\text{g}/\text{m}^3$): packing and loading 77.32+21.86; vulcanization, 147.37+47.87, and compounding, 155.44+12.68 respectively. The highest SPM (66.0 $\mu\text{g}/\text{m}^3$) and benzo(a)pyrene (10.94 ng/m^3) concentrations were observed in the particle fraction size of <0.5 μm , of compounding section (Table 1). The SPM concentration of packing and loading workers were exposed was significantly less than that to which the other workers were exposed at the two smallest fraction sizes ($p < 0.01$). The concentration of benzo(a)pyrene was seen to decrease with an increase in particle size throughout (Table 1).

Table 1: Concentrations of SPM and benzo(a)pyrene, according to particulate size in rubber factory.

Particulate size (μm)	Packing+Loading unit		Vulcanization unit		Compounding unit	
	SPM($\mu\text{g}/\text{m}^3$)	Benzo(a)pyrene	SPM($\mu\text{g}/\text{m}^3$)	Benzo(a)pyrene	SPM($\mu\text{g}/\text{m}^3$)	Benzo(a)pyrene
>10.4	552+363	0.32+0.46	546+246	0.31+0.11	574+0.40	0.1+0.01
>5.2	722+465	0.72+0.30	11.02+3.83	0.82+0.18	11.77+3.45	0.71+0.12
>1.6	962+4.17	1.16+0.57	15.11+3.46	1.16+0.51	16.53+4.59	2.01+0.97
>0.6	1027+3.67	2.01+0.59	20.90+1.98	1.34+0.34	21.86+4.59	5.94+2.21
<0.5	38.49+12.79	3.65+1.70	65.09+22.67	5.85+1.20	65.99+11.12	10.94+2.10
TSPM	77.32+21.86		147.37+47.87		155.44+12.68	

Values are mean+SD. TSPM=Total SPM

Table 2: Mean Concentration of rubber factory pollutants in three sections of rubber factory.

Parameter	Section I	Section II	Section III	F value
SPM($\mu\text{g}/\text{m}^3$)	77.32+21.36	147.37+47.87	155.44+12.68	11.16
Benz(a)pyrene(ng/m^3)	0.90+0.59	0.74+0.49	9.66+1.55	202.31
Benzo(a)pyrene(ng/m^3)	46.76+14.23	128.43+94.93	283.62+104.63	13.75
Benzo(a)anthracene(ng/m^3)	1.15+0.58	1.66+1.56	6.83+3.07	17.94
Sulphates($\mu\text{g}/\text{m}^3$)	27.96+5.84	23.24+3.77	40.96+12.13	10.44
Nitrate($\mu\text{g}/\text{m}^3$)	6.41+2.87	5.23+1.65	20.70+7.50	26.46

Table 2 shows the concentration of SPM and PAH compounds, viz. benzo(a)pyrene, benzo(e)pyrene, benzo(a)anthracene, sulfates and nitrates. It is evident that the SPM and other chemicals were in highest concentrations in the compounding (mixing) unit as compared to those in other two units. From the analysis of variance of significant difference ($p < 0.001$) could be seen amongst these pollutants from one section to another. (Table 2)

Demography and Symptoms

The monthly income of the workers studied ranged from Rs. 1,469 to 1,639 per month, which reflects their average socio-economic status. Their mean age, height and weight ranges were, 23.8-27.9 years, 161, 1-161.7cm and 50.0-51.6 kg respectively, and there was homogeneity between workers in all groups.

Symptoms indicative of respiratory difficulty (Table 3) were present in factory workers involved in the three different stages of the rubber manufacturing process studied, but were both most prevalent and severest in the workers belonging to compounding section (n=148), followed by those in the vulcanization section (n=441). The subjects from the packing and loading unit (n=78) had FVC and FEV₁ values of 2.79 and 2.60 litres such that the FEV₁/FVC ratio was 93%; whereas the values of these workers from vulcanisation section were 2.51 and 2.31 litres with a ratio of 92% and in the compounding group, 2.35 and 2.12 litres and 90% respectively. (Table 3)

Table 3 : Distribution of respiratory signs and symptoms in the rubber factors workers

Section	Blood	Breathing	Chest	Chest	Throat	Cough	Sputum
Packing and loading	-	+e	+e	-	-	-	-
Vulcanization	+e	+e	+e	-	-	+e	with blood
Compounding	+e	+e	+e	+e	+e	+e	with blood, black colour

Mean values of FEF 25-75%, PEFR and PIFR of different groups of workers are represented in Table 4. As compared to other groups, a significant decrease in FEF 25-75%, PEFR and PIFR values were noted in compounding unit ($p < 0.05$). The relationship of the deterioration in lung function with the duration of exposure to pollutants is given in Table 5. As compared to different subgroups of working duration among the three groups of workers, a significant reduction in lung functions in workers having a working duration of 6 years and more was observed. In packing and loading section (group 1), no two subgroups were different from each other in their values of FEF 25-75%, PEFR and PIFR raw ingredients and other chemicals are heated and milled to obtain softened rubber. In all these locations, one would anticipate that there was exposure to particles and benzo(a)pyrene. (Table 4 & 5)

Researchers have correlated Benzo(a)pyrene with the mixture of PAH analysed, confirming the presence in air as a good indicator of exposure to PAH compounds⁸.

Table 4 : Mean measured values of lung functions of workers belonging to Section I, II and III

Group No.	Section	Parameters				
		FEF25% (L/Sec)	FEF50% (L/Sec)	FEF75% (L/Sec)	PEFR (L/Sec)	PIFR (L/Sec)
1	I	4.13+0.99	3.63+0.92	2.56+0.55	4.40+1.02	3.95+0.99
2	II	3.97+1.34	3.57+1.12	2.34+0.78	4.13+1.45	3.48+1.15
3	III	3.34+1.13	3.04+0.82	2.20+0.61	3.33+1.21	2.97+0.89
F value		4.636*, 8.045*, 4.199*, 11.00, 16.57*				

* $p < 0.05$ ANOVA

Discussion

The highest particles and benzo(a)pyrene concentrations were found in the compounding section workers. Such a particle size distribution is consistent with the results from the pulmonary function tests. This study showed that the lowest measures of lung function were found in the workers from the compounding section, which was the most polluted environment. Furthermore, the longer the workers had spent in the factory, the lower was their lung function, regardless of the stage of production process they were involved. This suggests that there may be a causal link between the exposure to particles and benzo(a)pyrene or other chemicals and the depression of lung function. That this depression has clinical implications as revealed by the finding that the severity and frequency of respiratory difficulty was greatest in workers from the compounding section, who were exposed to the highest concentrations of respiratory particles and benzo(a)pyrene. The loss of pulmonary function seen in the present study was supplemented by the X-ray findings, conforming that lung damage was greatest in the workers in the compounding section.

The inverse relationship between lung function and concentration of pollutants that has been found in the present study is consistent with other findings. Such a relationship was demonstrated by Zejde et al¹⁶ in Swine producers. In a study on rubber factory workers by Zuskin et al¹⁰ also showed significantly lower values of FVC, FEV₁, FEF 25%, FEF 50% and higher prevalence of acute and chronic respiratory symptoms. A study on Shoe and Cement factory workers has shown fall in various spirometric values (FVC, FEV₁/FVC) indirect proportion to duration of exposure¹¹. The high incidence of respiratory symptoms and X-ray findings in workers in the compounding section are similar to those reported by Shah and Co-workers¹⁷ in asbestos workers. These results are also in general agreement with those of other workers¹⁸ who have

Table 5 : Multiple regression analysis of FVC and FEV₁, FEF 25%, FEF 50%, FEF 75%, PEFR and PIFR vis a vis physical parameters and pollutants.

Effects of various parameters	FVC(L)		FEV ₁ (L)		FEF 25%(L/sec)		FEF 50%(L/sec)		FEF 75%(L/sec)		PEFR(L/sec)		PIFR(L/sec)	
	Coff	t	coff	t	coff	t	Coff	t	Coff	t	Coff	t	Coff	t
Age	-9.18	-1.63	0.011	-1.82	-0.24	-1.59	-0.021	-1.90	-6.20	-2.68*	-1.78	-.110	.024	-2.40*
Height	0.021	3089*	0.013	2.40*	-3.15	-.224	9.81	.936	0.011	1.64	5.15	.034	.021	1.86
Weight	0.029	5.05*	0.020	3.29*	.035	2.369	.026	2.28*	.014	1.98*	.017	1.04	.011	9.21
Calorie intake	1.22	0.163	7.77	0.960	-1.05	1.01	2.36	1.76	7.98	822	2.30	1.08	6.67	421
Smoking duration	-1.59	-0.25	-1.76	-0.255	-1.76	-8.95	-2.26	-1.54	-2.74	-2.88*	-3.03	-1.37	4.81	.311
Employment duration	-1.14	-1.50	-1.59	-1.98	-1.76	-8.95	-2.26	-1.54	-2.74	-2.88*	-3.03	-1.37	4.81	.311
Working hours/day	4.81	0.272	9.62	0.510	-0.024	-5.21	.023	.666	8.25	.366	.015	.298	.014	.368
SPM($\mu\text{g}/\text{m}^3$)	-3.87	-3.41*	-4.18	-3.35*	-1.58	-4.43	-3.21	-1.16	-3.68	-1.85	-4.96	-1.26	-6.95	-2.04*
Benzo(a)pyrene	-0.014	-1.45	-0.016	-1.55	-0.88	-3.66*	-0.046	-2.74*	-3.48	-3.35	-0.094	-3.73*	-0.037	-2.10*
r ² (%) for all 9 parameters	0.513(51.3%)		0.413(41.3%)		.343(34.3%)		.355(35.5%)		.398(39.8%)		.303(30.3%)		.317(31.7%)	
r ² value (%) for first 7 parameters	0.486(48.6%)		0.383(38.3%)		.222(22.2%)		0.300(30.0%)		0.380(38.0%)		0.157(15.7%)		0.273(27.3%)	
Additional contribution (%) of pollutants	0.029(2.9%)		0.054(5.4%)		12.1%		5.5%		1.8%		14.6%		4.4%	

* $p < 0.05$

Table 6 : Pulmonary lung functions of subgroups (a,b,c and d) durationwise.

Section	Parameter	Group according to work duration			
		a 6months	b 6monthsto 3years	c 3 years to 6 years	d 6 year & above
Packing & loading group (I)	FEF25%(L/sec)	4.48+1.07	4.21+0.87	3.96+1.00	3.74+0.83
	FEF50%(L/sec)	3.82+0.91	3.64+1.00	3.52+0.84	3.37+1.06
	FEF75%(L/sec)	2.70+0.53	2.68+0.52	2.49+0.58	2.14+0.35
	PEFR (L/sec)	4.68+1.04	4.44+0.83	4.20+0.91	4.21+1.51
	PIFR (L/sec)	4.33+1.17	3.82+0.65	3.81+1.10	3.63+1.03
	FVC(L)	2.96+0.41	2.90+0.37	2.68+0.36	2.63+1.08
	FEV ₁ (L)	2.81+0.21	2.68+0.44	2.48+0.38	2.41+0.51
	FEV ₁ /FVC(%)	94.9+5.7	92.4+6.2	92.5+6.3	91.5+7.5
	FEF25%(L/sec)	4.07+1.28	4.04+1.33	3.85+1.31	3.75+1.53
	FEF50%(L/sec)	3.69+1.07	3.64+1.06	3.39+1.04	3.33+1.35
Vulcanizing (group II)	FEF75%(L/sec)	2.48+0.76	2.36+0.72	2.30+0.78	2.02+0.81
	PEFR(L/sec)	4.25+1.41	4.14+1.44	4.02+1.47	3.92+1.54
	PIFR (L/sec)	3.53+1.12	3.47+1.18	3.47+1.17	3.38+1.18
	FVC(L)	2.59+0.61	2.56+0.61	2.39+0.64	2.38+0.65
	FEV ₁ (L)	2.39+0.59	2.36+0.59	2.17+0.59	2.14+0.66
	FEV ₁ /FVC(%)	92.3+9.6	92.2+11.7	90.8+10.1	89.9+12.3
	FEF25%(L/sec)	3.46+0.41	3.4+0.47	3.33+1.35	3.24+1.48
	FEF50%(L/sec)	3.22+0.45	3.16+1.02	3.03+0.93	2.83+0.86
	FEF75%(L/sec)	2.40+0.48	2.35+0.77	2.22+0.62	1.93+0.54
	Compounding (group III)	PEFR(L/sec)	4.08+1.00	3.10+1.17	3.09+1.30
PIFR (L/sec)		3.13+1.02	3.09+0.93	3.01+0.66	2.69+0.78
FVC(L)		2.40+0.46	2.36+0.51	2.34+0.37	2.30+0.41
FEV ₁ (L)		2.21+0.40	2.15+0.65	2.09+0.49	2.05+0.60
FEV ₁ /FVC(%)		92.1+8.0	91.1+13.8	89.3+19.0	89.1+17.1

reported significant reductions, in FVC values in samill and ricemill workers after 5 years of exposure and in FEV₁ values within a 1-year exposure time. In another study, rubber factory workers with an exposure of 10 years showed a significant decrease in FEV₁/FVC when compared with controls¹⁹. A time-related decline in lung function has also been reported in Indian workers who have been exposed to talc dust²⁰.

In any study such as the present one, it is possible to implicate a pollutant in medical effect erroneously because of compounding factors. In the present study, the workers in each group were examined with regard socioeconomic status and medical history. No relationship between these factors could be demonstrated, allowing the conclusion to be made that the impairment of lung function was related to the concentrations of particles and chemicals in the respirable fraction of air to which workers in a rubber factory were exposed. Of course, the possibility remains that the benzo(a)pyrene could be a surrogate for some other substance that causes the damage to the lungs. However, as benzo(a)pyrene was employed in the present study as a marker for all polycyclic aromatic hydrocarbons, it seems likely that such a chemical is responsible for the pulmonary damage in the rubber factory workers. Whatever the relationship between polycyclic aromatic hydrocarbons and pulmonary toxicity, it is clear that respirable particles are involved in the observed effects.

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Table 7 : Physical parameters of the subjects studied

Parameter	Packing and loading	Vulcanization	Compounding
Age, years	25.2+7.3	23.8+5.9	27.9+7.4
Height, cm	161.7+6.3	151.1+6.3	161.6+6.2
Weight, kg	50.4+5.63	50.0+5.92	51.6+6.00
Smoking duration, years	7.4+7.7	5.5+4.3	7.7+6.4
Employment duration, months	2.3+1.1	2.1+1.1	2.6+1.2
Beedi/day	122+11.7	8.7+5.5	7.3+6.2
Cigarettes/day	3.7+1.6	3.6+2.6	4.8+4.8
Tobacco, g/day	3.1+2.3	3.1+2.1	3.0+1.7
Work time, h/day	9.5+1.7	9.7+1.6	9.5+1.7
Caloric intake, kcal/day	1747+325	1822+406	1802+372
Major family members	3.4+1.8	4.4+3.2	4.2+2.5
Minor family members	2.6+1.4	2.9+2.0	3.1+2.4
Income, Rs/month	1469+1.21	1639+1.284	1585+1.187
Subjects	78	441	148

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Correlation of Lung Function Tests with Nutritional and Socio-economic Status in Male Children

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Abstract: This study was carried out on hundred male children (5-12 years) fifty each from lower and middle socioeconomic strata. Body mass index and total body fat (TBF) were assessed by measuring skinfold thickness to know their nutritional status. Lung function tests including forced vital capacity (FVC), forced expiratory volume in one second (FEV₁) and peak expiratory flow rate (PEFR) were measured by spirometry. Children of lower socioeconomic status (group I) showed more prevalence of malnutrition, lower value of the mean TBF and pulmonary functions tests as compared to children belonging to middle socioeconomic status. Results of our study reveal poor nutritional status and compromised lung functions in children living in vulnerable indoor environment of lower socioeconomic strata.

Keywords : *Nutritional profile, pulmonary function.*

Introduction

Multiple non-hereditary factors including radiation, malnutrition, passive smoking and hazardous outdoor and indoor environment may influence individual's health in many ways¹⁻¹⁰. Among the above devastating factors the role of poor nutritional status become quite dominating. Norms established in previous studies, carried out in well nourished Delhi Children under the age group of 10-15 years act as reference standards for various lung functions^{11,12}. Kapil et al¹³ in their study show 81.1% prevalence of protein energy malnutrition among the pre-school children of urban communities in Delhi. Nutritional differences influence qualitative aspect of lung development in childhood beyond simple isotropic lung growth¹⁴. Work on malnourished children¹⁵ have shown significant reduced lung functions (FVC, FEV₁) as compared to normal population. Pulmonary functions correlate better with physical parameters e.g. height-arm span, weight and upper segment than with age¹⁶. Measurement of pulmonary function tests are not only providing a direction regarding any deviation in health status of population studied but they also add a lot for required informations. On going through the literature, it is found that studies on Indian Children particularly belonging to low socioeconomic strata are very few¹⁵⁻¹⁹ and therefore, we have made an attempt to coordinate the effect of various vulnerable factors such as indoor pollution including passive smoking and others on their complete health profile so that causative factors could be identified.

Material and Methods

This study was performed in 100 male children, aged 5-12 year, 50 each from middle and lower socioeconomic strata of East Delhi. They were further divided into four groups: group A (age 5-8 years) and B (age 9-12 years) from group I and group C (age 5-8 years) and D (age 9-12 years) from lower socioeconomic strata (group II). Each group consisted of 25 children.

The general information regarding number of total and earning members in family, approximated monthly income, household area to live (HAL, ft²), fuel used for cooking, number of smokers in family, and child's vaccination under routine immunization,

programme were collected by introducing a standard questionnaire for each children. The subjects were also grouped in various combinations of abovementioned groups as well as in groups based upon fuel used for cooking (gas-users and non-gas-users), number of smokers in family (passive smokers and non-smokers) and child's vaccination (vaccinated and unvaccinated).

The height (ht, cm) and weight (wt, kg) of each child were measured and the body surface areas (BSA m²) was calculated using DuBois formula²⁰.

$$BSA = ht^{0.725} \times wt^{0.425} \times 0.007184$$

To assess the child's nutritional profile the body fatness was measured by using the swiss precision GPM Skinfold Caliper (Swiss make). The thickness of skinfold at each of the ten standard sites-namely, cheek, chin, chest, flank, waist, para-umbilical, triceps, subscapula, calf and knee was measured thrice and the mean value calculated. The sum, (Σ SD, mm) of the mean-skinfold-thickness for the ten above mentioned sites was calculated and used to estimate the total body fat (TBF, kg) the percentage fat PF, (%) and the fatless tissue (FT, kg) using following formula:

$$TBF = wt \times \frac{(\text{âSF } 40) \times BSA \times 0.039}{20 \times w} \quad 0.03$$

$$PF (TBF / wt) \times 100$$

$$\text{And } FT = Wt - TBF$$

The Body Mass Index (BMI)²¹ was also calculated by using wt/ht² and a value < 0.15 was considered for designating the child as malnourished.

The pulmonary functions namely, forced vital capacity (FEV, L), forced expiratory volume in 1 sec (FEV₁, L) and peak expiratory flow rate (PEFR, L/min) - were measured by using PK Morgan's Pocket Spirometer (PK Morgan Pvt. Ltd. England). The procedure was demonstrated to the satisfaction of each subject.

Nose clip was used on the subject during assessment. For each function, three measurements were made and the best result out of three was included. The Empey index (EI, ml/l/min.) was calculated using formula²².

$$EI = (FEV_1 / PEFR) \times 10000$$

All the collected data was analysed by applying the 'SPSS version

5.0' statistical package and the scatterograms were computer-drawn wherever significant correlations were observed.

Results

Table 1 shows the general information including number of total earning members in family, monthly income, HAL, gas-user families, number of smokers in family and unvaccinated children. As shown, the number of members in family was greater but the monthly income and HAL were lesser in groups II (lower socioeconomic stratum) than in group I (middle socioeconomic stratum) forming the basis for grouping them socioeconomically.

Table 1 : General profile of subjects and their families

Sr. No.	Parameter	Socioeconomic (Group I)	Stratum (Group II)	pvalue
1	Number of Subjects	50	50	
2	Mean age (Yr)	8.62	8.28	>.005
3	Total members/family	5.04 + 1.75	7.86 + 0.64	<.005
4	Earning members/family	1.46 + 0.10	1.32 + 0.11	>.005
5	Monthly income (Rs.)	7204 + 582	1527 + 139	<.005
6	Gas-user/family (%)	100	30	<.005
7	Household area to life (ft ²)	762 + 12	200 + 0	<.005
8	Smokers/family	0.32 + 0.08	0.74 + 0.10	<.005
9	*No of malnourished subjects	6	25	<.005

Values are Mean SEM statistical analysis using unpaired t-test

*Quetelet BMI < 0.15 as per ref.¹⁶

Although all families of middle socioeconomic status are found using LPG fuel for cooking, whereas only 30% of families of lower socioeconomic status used the same and rest of them used other less efficient and more smoke producing fuels such as kerosene oil, coal and wood. There were more number of smokers and unvaccinated children in group II families as compared with group I.

The ht, wt and BSA values as well as Σ SF, TBF, PF and FT values were found toward lower side in children of group II. These values further showed a declined pattern for non-gas-users, passive smokers and unvaccinated children (Table 2).

Table 2 : Nutritional profile and pulmonary functions in subjects.

Sr. No.	Parameter	TBF (kg)	FF (%)	FVC (L)	FEV ₁ (L)	PEFR (L/min)
1.	Socioeconomic status					
	Group I (n=50)	2580	8.48	1.334	1.219	1572
		+0.392	+1.14	+0.061	+0.054	+72
	Group II (n=50)	0.961	5.04	0.939	0.881	1270
		+0.142	+0.74	+0.045	+0.040	+65
	p value	<.005	<.005	<.005	<.005	<.005
2.	Fuel used for cooking					
	Gas users (n=65)	2.332	8.25	1.244	1.138	1487
		+0.313	+0.93	+0.054	+0.048	+64
	Non-gas-users (n=35)	0.726	3.98	0.937	0.885	1299
		+0.143	+0.81	+0.056	+0.049	+80
	p value	<.005	<.005	<.005	<.005	<.005
3.	Passive smoking					
	Passive smokers (n=41)	1.507	6.60	1.066	0.976	1283
		+0.257	+0.94	+0.068	+0.058	+72
	Non-smokers (n=59)	1.953	6.87	1.186	1.102	151.7
		+0.332	+0.99	+0.054	+0.048	+68
	p value	>.005	>.005	>.005	>.005	>.005
4.	Nutritional status					
	Well nourished (n=69)	2.348	8.71	1.200	1.093	1419
		+0.292	+0.87	+0.057	+0.049	+63
	Malnourished (n=31)	0.484	2.74	1.003	0.954	142.7
		+0.130	+0.78	+0.050	+0.049	+84
	p value	<.005	<.005	<.005	<.005	>.005

Values are Mean SEM Statistical analysis using unpaired t-test

Table 2 exhibits the results of pulmonary functions (FVC, FEV₁, PEFR and EI) of the children. Results of FVC, FEV₁ and PEFR showed a decrement in children belonging to group II as compared with others (group I) and the pattern of lower results continued in non-gas-users, passive smokers and unvaccinated children.

The prevalence of malnourished children in group II is more (50%) as compared with group I (12%) children (Table 2).

Further the FVC and FEV₁ values increased with increase in HAL as $r = 0.4613$ and 0.4315 , $P < 0.001$ respectively. The FVC value was observed to be more with increasing age as :

$$FVC(1) - 0.1379, \text{ age (yr)} 0.0271, r = 0.6864, P < 0.0001$$

Results show a linear relationship of FVC and FEV₁ with increasing TBF ($r=0.4618$ and 0.4119 , $p<0.0001$) respectively.

The PEFR results also indicated a similar pattern being on lower side in children of group II as well as in non-gas-users and passive smokers. There was a linear increase in PEFR with increasing age, ht, wt, and BSA as exhibited by r values ($r=0.6168$, 0.6814 , 0.6223 and 0.6696 respectively, $P<0.0001$) respectively. The PEFR results depicted a direct relationship with HAL ($r=0.3386$, $P<0.0007$) whereas an inverse pattern was observed with number of smokers in family (NOSMO) as well as with number of smokers/number of total members in family (S/FM) as exhibited in Fig. 1 and 2 ($r=0.2505$ and -0.2028 , $P<0.05$)

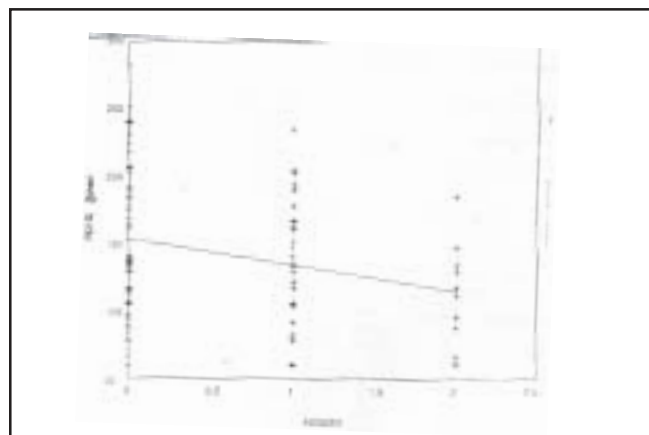


Fig. 1: Scatterogram showing PEFR (peak expiratory flow rate) in relation with NOSMO (no. of smokers in family). Regression equation : $PEFR = (-18.09 \times NOSMO) + 151.7$; $r = -0.2505$; $P < 0.05$

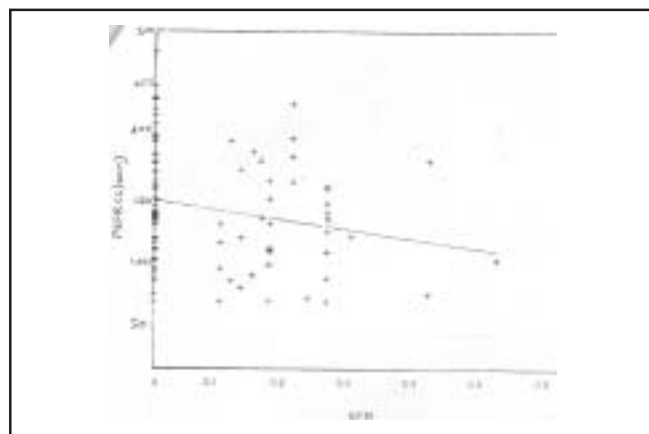


Fig. 2: Scatterogram showing PEFR (peak expiratory flow rate) in relation with S/FM (no. of smokers/no. of total members in family) Regression equation : $PEFR = (-90.42 \times S/FM) + 149.6$; $r = -0.2028$; $P < 0.05$

respectively PEFR values also indicated an improvement with increasing FVC and FEV_1 Values (Fig 3, $r=0.6482$ and Fig. 4, $r=0.7042$ retrospectively, $P<0.0001$).

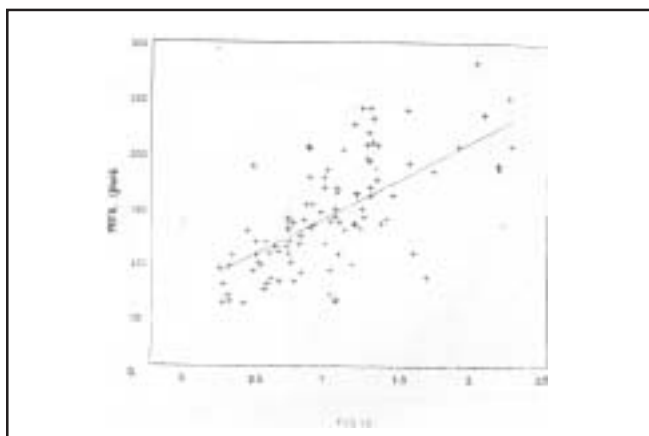


Fig. 3: Scatterogram showing PEFR (peak expiratory flow rate) in relation with FVC (forced vital capacity). Regression equation : $PEFR = (76.95 \times FVC) + 54.66$; $r=0.6482$; $P>0.0001$

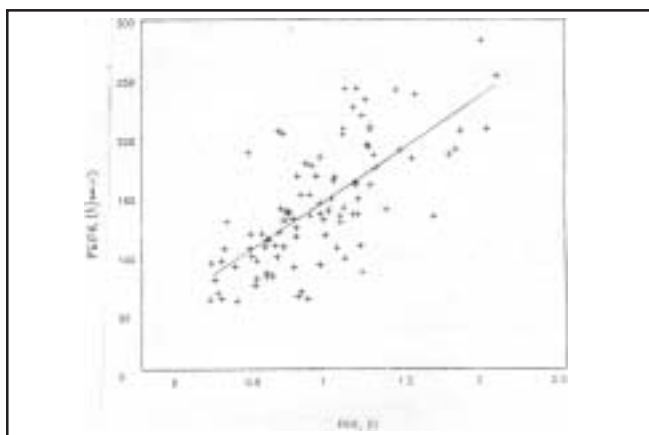


Fig. 4: Scatterogram showing PEFR (peak expiratory flow rate) in relation with FEV_1 (forced expiratory volume in 1 sec.). Regression equation : $PEFR = (95.81 \times FEV_1) + 41.54$; $r=0.7042$; $P<0.0001$.

In present study, the EI was found to be higher in children of groups B and D (age 9-12 yr) than in group A and C (age 5-8 yr) though less than 10 in all groups. The EI exhibited an increase with increasing TBF ($r=0.4216$, $P<0.0001$) and increase with increasing PEFR (Fig. 5, $r=0.3951$, $P<0.0001$). It was higher in passive smokers.

Discussion

As it has become quite clear that the children belonging to lower socioeconomic status are exposed to more hazardous indoor environment due to passive smoking and use of less efficient and more smoke-producing fuels. Along with larger fraction unvaccinated children, all these factors may be responsible for favouring the underdevelopment associated with various respiratory disorders amongst them^{1,3,15}.

The TBF has also been assessed by Burnin and Womensley²³, however, they calculated it in older age group from the body fat density presuming the latter to be uniform in the whole body. Faridi et al¹⁵ have used a method similar to our and their results were slightly lower than ours possibly because they carried out

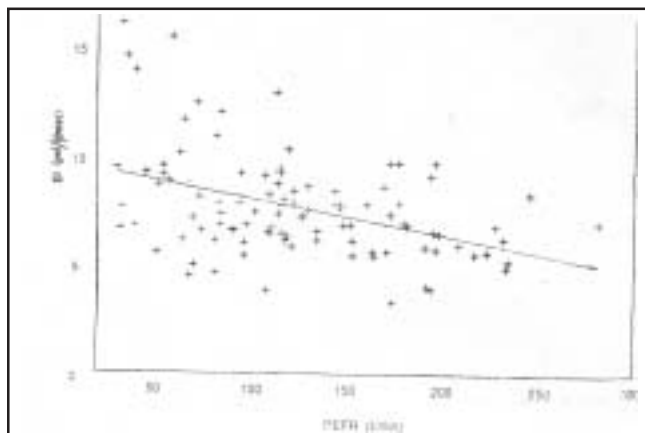


Fig. 5: Scatterogram showing EI (Empey index) in relation with PEFR (peak expiratory flow rate). Regression equation : $EI = (-0.0185 \times PEFR) + 10.35$; $r=-0.3951$; $P<0.0001$.

their study in malnourished children only. On assessing Body Mass Index (BMI) the results are further supported by showing more malnourished children in group II as compared with group I.

Ong et al¹⁴ show that lung function normalized for sitting height and stature correlated significantly with indices of nutrition in both sexes. The pulmonary function values reflect better results in children living in less polluted areas, and these are supported by many other workers as they have reported from the children belonging to health environment^{1,4,5,17,18,22}. The linear increase in PEFR values with increase in age, ht, wt and BSA has also been reported in study carried out in mildly malnourished Senegalese children²⁴. The rate of increase in PEFR also seems to be enhanced with increasing age²⁵. Smokers of the family are exposing the children to passive smoking and may cause a decrement in their pulmonary function values.

The EI is an important pulmonary function since a value of it above 10ml/L/min is suggestive of narrowing of upper airways²². Our study reveals that as the Nutritional and respiratory conditions improve, the EI value decreases depicting its significance. There are reports on similar trends by others¹⁻⁷.

Though we have tried to explain and provide suitable reasoning for our results and their correlation with other factors worked out by us, but to draw a conclusion we suggest to continue such studies involving more number of such children belong to various socioeconomic status.

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Fluoride Distribution and Fluorosis in some Rural Areas of Udaipur, Rajasthan

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Abstract: The occurrence of fluoride in drinking water, ambient air and in certain vegetable crops were studied and related with the prevalence of dental fluorosis in some rural areas of Udaipur district of Rajasthan. Although, most of the portable water samples, when analysed using fluoride electrode, showed fluoride levels below permissible limit (1.0ppm), the incidence of fluorosis was surprisingly high. The study revealed the prevalence of dental fluorosis in adults and also in school going children. Khemi was found to be the most affected village where the well water used by the villagers for drinking purposes contained highest fluoride level (2.0 ppm) observed in this study. The ambient air and the edible parts of certain crops also contained high concentrations of fluoride. This could be due to atmospheric emissions from phosphate fertilizer factories situated near these villages. The study suggests that in addition to drinking water, atmospheric deposition on crops followed by dietary intake could enhance total fluoride in body tissue and hence the cases of fluorosis.

Introduction

Recent scientific efforts, although yielded beautiful return to our basic knowledge of water quality management, have raised many questions about the safety of drinking water supply. In countries, like ours where the majority of population live in villages with bare infrastructural facilities, poor sanitation and hygiene, the concept of safe drinking water assumes great significance. This has particular concern for arid and semi-arid regions. In states such as Rajasthan, where the quantity of freshwater demand itself has become a big problem due to persistent drought from last five years, per capita availability of safe drinking water is a matter of serious concern. Rajasthan is identified as one of the highly endemic states for fluorosis¹. Fluoride, one of the abundant and widely distributed elements in nature, plays a vital role in water quality management due to its both, beneficial and adverse health effects^{1,2}. Although fluoride helps preventing dental caries, its higher concentrations lead to mottled enamel. Excess intake of fluoride beyond the permissible limit (>1ppm) causes dental and skeletal fluorosis and neurological disorders. In India, Lakdawala and Puchar³ made extensive study on the prevalence of fluorosis and total fluoride intake through drinking water and commonly consumed food. In our country, about 62 million people, including six million children, suffer from dental and skeletal fluorosis because of consuming fluoride contaminated water¹. Fluoride, being ubiquitous in nature, is always present in plants, soils and waters. In surface waters, its concentration varies widely, some time achieving values as high as 18000 $\mu\text{g L}^{-1}$ in hot springs⁴. Fluoride enters in ground water mainly through leaching from the earth crust. various rock types contain fluoride ranging from 180mg $\mu\text{g g}^{-1}$ in sandstone and greywacke to 800 $\mu\text{g g}^{-1}$ in granites⁴.

Rock phosphate, phosphatic nodules and phosphorites contain high concentrations of fluoride⁵. Industrial effluents, drugs, cosmetics and agricultural application of fertilizers coupled with pesticides also add sizeable amount of fluoride to the ground water, surface waters and to the terrestrial environment⁶. Thus, the fluoride content in different components of environment and its possible bearing on total fluoride intake in human beings have become a matter of great concern⁷. For instance, in addition to drinking water, dietary fluoride intake could significantly enhance total fluoride in our body^{3,8}. In a preliminary study conducted at

Udaipur district of Rajasthan, we observed that, in spite of low level of fluoride in drinking water, a number of persons including school going children suffer from fluorosis. Hence a systematic study was conducted to examine the cases of fluorosis in school going children and in adults in 10 villages of Udaipur district of Rajasthan. Attempts were made to relate the cases of fluorosis with fluoride in drinking water as well as in agricultural crops. Since the present study was conducted in an area receiving emissions from phosphate fertilizer factories, atmospheric depositions were likely to enhance fluoride levels in crops.

Materials and Methods

The present study was conducted in rural areas of NE Udaipur (Rajasthan). The climate of the region is tropical with three distinct seasons, a hot and dry summer (March to June), a warm and wet rainy (July to October) and a cool and dry winter (November to February), with annual average 24^o temperature, 45% relative humidity and 800mm annual precipitation. The first half of the summer season experiences strong, hot and dry winds and high temperatures, while the second half is generally hot and humid. During the study period, the day time summer temperature ranged from 34^o to 46^oC. During winter, temperature varied between 10 to 23^oC and the night temperature some time dropped below 4^oC. The annual rainfall averaged 595mm and relative humidity ranged between 12 and 95%. Wind direction shifted predominantly westerly and non-westerly in October to April and to easterly and south-westerly in the remaining months.

Ten villages were selected, and based on the population of each village, 50-218 cases of adults in the age group 21 to 65 (both men and women) were examined for dental fluorosis. Children of 10 schools (age group 5 to 13) were examined for dental caries and fluorosis. The food habits of the villagers were also enquired. Most of the villagers use well water and some use borewell water for drinking and cooking purposes. All the drinking water sources, including ground and surface sources, were tested for fluoride concentration. Drinking water samples were collected in pre-sterilized bottles from all the 10 villages and analysed for fluoride concentration using a fluoride electrode. Citrate was included in the total ionic strength adjustment buffer (TISAB) as a chelating agent in order to avoid interference by fluoride binding metal ions.

Since the area lies between two phosphate fertilizer units (Udaipur Phosphate Fertilizer Factory and Rama Phosphate Fertilizer Factory), it receives substantially high amount of fluoride through atmospheric depositions. In order to assess the possible dietary intake, samples of locally growing crops were also analyzed for fluoride concentration following bellack¹⁰. Fluoride concentration in ambient air was monitored using High Volume Samplers (Envirotech APM-415) following Narayan et al.¹¹

Results and Discussion

Fluoride concentrations in drinking water samples are presented in Table 1. For most of the water samples, fluoride concentration was well below the permissible limit. Water samples collected

Table 1 : Concentration of fluoride in drinking waer samples collected from selected villages of Udaipur.

Village	Fluoride (ppm)
Khemli	
Well water	1.80-2.15 (x=2.00+0.21)
Borewell water	1.15-1.40 (x=1.27+0.13)
Asna	
Well water	0.03-0.09 (x=0.06+0.005)
Borewell water	0.07-0.15 (x=0.10+0.02)
Shangawa	
Borewell water	0.32-0.64 (x=0.48+0.05)
Gondoli	
Borewell water	0.07-0.15 (x=0.10+0.02)
Devali	
Well water	0.05-0.1 (x=0.07+0.006)
Chandesara	
Borewell water (South)	0.05-0.1 (x=0.07+0.006)
Borewell water (Primary School)	0.16-0.26 (x=0.20+0.04)
Bhagio Ka Guda	
Well water	0.48-0.85 (x=0.59+0.07)
Gudali	
Borewell water	0.05-0.15 (x=0.09+0.006)
Well water	0.03-0.09 (x=0.06+0.006)
Junawas	
Borewell water (East)	0.03-0.09 (x=0.06+0.005)
Borewell water (West)	0.56-0.66 (x=0.61+0.10)
Odwadia	
Borewell water	0.05-0.1 (x=0.07+0.005)
Borewell water Primary school	0.03-0.1 (x=0.06+0.005)

from Khemli village contained the highest level of fluoride (2.0 ppm) observed in the present study. Interestingly, fluoride concentrations in the ambient air as well as in crops were substantially high (Fig.1 and Table 2). This could be due to atmospheric emission of fluoride from phosphate fertilizer factories situated near these villages. The cases of dental fluorosis in

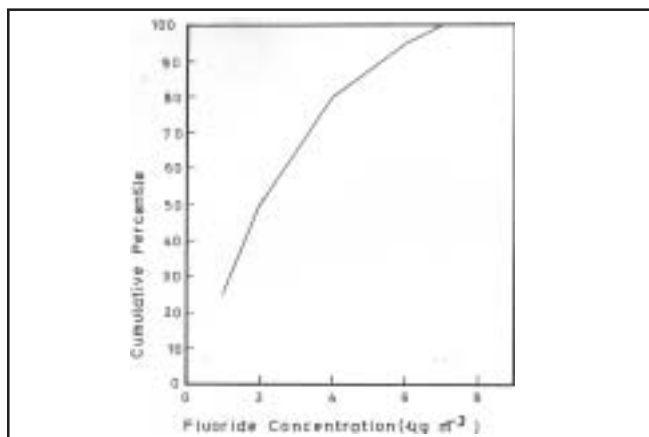


Fig. 1 : Cumulative percentile distribution of 2-h mean concentrations of fluoride in the ambient air of the study area.

Table 2 : Fluoride concentration in crops (ppm dry matter basis).

Component	Fluoride		
	Range	Mean	C.V.
Grains*	28.6 - 84.5	52.3	17.4
Vegetables**	46.1 - 136.4	86.3	16.2

C.V.: Coefficient of variation

* Include maize, wheat and pearl millet

** Include tomato, brinjal, cauliflower, lady's finger and beans

Table 3 : Cases of dental fluorosis in school going children in rural areas of Udaipur.

Name of the School	Village	No. of Students examined	Affected Boys(%)	Affected Girls(%)
Primary School	Khera	96	21 (52.5)	19 (47.5)
Primary Girls School	Kanpur	300	22 (55.0)	18 (45.0)
Primary School	Umara	115	13 (48.1)	14 (51.9)
Primary School	Deckia	74	12 (60.0)	08 (40.0)
Rajiv Gandhi Primary School	Jhapa	42	03 (42.8)	04 (57.2)
Primary School	Umara Khera	77	09 (81.9)	02 (18.2)
Primary School	Dhama Talai	120	15 (46.9)	17 (53.1)
Primary School	Dharma Talai	100	09 (37.5)	15 (62.5)
Middle School	Kanpur	100	22 (52.4)	20 (47.6)
Middle School	Khera	31	6 (42.9)	8 (57.2)

Values in parentheses indicate percent of the affected cases.

school going children of some rural areas of Udaipur district of Rajasthan are presented in Table 3. In school going children (age 5 to 13) the cases of dental fluorosis ranged between 13.3% to 45.2%. The incidence of dental fluorosis in boys and girls did not differ significantly. The data on dental fluorosis in adults (both men and women) are presented in Table 4. Khemli was found to be the most severely affected among the 10 villages studied, where 58.3% of the adult population have shown the sign of dental fluorosis. Devali was the least affected village observed in the present study. Water samples (well water) collected from Khemli village contained fluoride above the permissible limit.

Table 4 : Cases of dental fluorosis in adults in rural areas of Udaipur.

Village	No. of person examined	Affected Men	Affected women
Khemli	218	69 (54.3)	58 (45.7)
Asna	150	35 (51.5)	33 (48.5)
Shangawa	142	32 (62.7)	19 (37.3)
Gandoli	110	10 (55.6)	08 (44.5)
Devali	116	07 (53.8)	06 (46.2)
Chandesara	170	27 (50.9)	26 (49.1)
Bhagio Ka Guda	180	32 (50.8)	31 (49.2)
Gudali	160	26 (59.1)	18 (40.9)
Junawas	50	10 (71.4)	4 (28.6)
Odwadia	100	10 (66.7)	05 (33.3)

Values in parentheses indicate percent of the affected cases.

This could account for the high percentage of fluorosis in Khemli, since well is the major source of drinking water supply in this area. Borewell water of Khemli village also contained fluoride above the permissible limit.

In addition to the natural sources of fluoride in drinking water, this area also receives high amount of fluoride through atmospheric deposition from phosphate fertilizer factories. This was evidenced by high concentrations of fluoride in ambient air as well as in crops. Plants can accumulate fluoride in concentration many fold higher than that available in air environment¹². This could account for enhanced total fluoride in body tissue through dietary intake.

Data on ambient air quality indicates high concentration of fluoride in the air environment. About 5% of 2-h hourly mean concentrations exceeded $6.0 \mu\text{g m}^{-3}$ a level sufficient to cause significantly fluoride acculati in plant parts¹².

The cases of fluorosis in villagers, particularly in school going children in SE Udaipur appeared alarming. Children with fast growing tissues could get affected more severely and quickly. Children have also shown such symptoms as pain in the stomach, intermittent diarrhoea, chronic constipation and gas formation caused due to intake of fluoride¹³. It has been shown that excess intake of fluoride leads to the accumulation of dermaten sulphate, which demineralize the area around both in teeth and bones¹⁴. Such demineralised areas in teeth get perforated and chipped beside being discoloured¹⁵. In most of the water samples, fluoride level was found below the permissible limit. However, since fluoride is an accumulating pollutant, it could induce adverse effects in due course, if taken continuously even at very low concentration¹⁶. Furthermore, atmospheric emissions from phosphate fertilizer factory could make an additional source of fluoride intake through inhalation and oral uptake through food. The area studied is exposed to the emission from phosphate fertilizer factories. This together with fluoride in drinking water could exacerbate the prevalence of fluorosis in villages having fluoride content in water even below the permissible limit. A number of adults in these villages (aged 45-61) showing dental fluorosis, also complained severe pain in their joints. These symptoms may be indicative of skeletal fluorosis followed by dental fluorosis¹⁷. In most of these villages the socio-economic status is very poor. The villagers can hardly afford to take calcium and vitamin-C rich diet. Therefore, the present study invites attention, of both scientists and policy makers, to develop and implement suitable control measures so that fluoride related health problems of this area can be minimized. Alternative approaches such as sufficient availability of calcium and vitamin-C rich diet can provide some relief to this problem. However unlike most part of Rajasthan, fluorosis did not appear endemic to this area. And therefore, alternative approaches would be least productive unless the local inhabitants are getting relief from atmospheric fluoride input from phosphate fertilizer factories. This has relevance if Fluorosis Management Programme need to get success in India.

Acknowledgement

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ETHICAL GUIDELINES FOR BIOMEDICAL RESEARCH

The need for uniform ethical guidelines for research on human subjects is universally recognised. It has acquired a new sense of urgency as the critical issues in the area of biogenetic research involving human subjects have become acute. Apart from the mandatory *clinical trails* on new drugs, a number of *diagnostic procedures, therapeutic interventions and prevention measures* including the use of vaccines, are being introduced which involve human subjects. Further the advent of *new medical devices and radio-active materials* and therapeutic benefits of *recombinant DNA products* have added a new dimension to the ethical issues that need to be considered before evaluating these for their efficacy, utility and safety.

Any research using the human beings as subjects shall bear in

mind the following principles of : i) **essentiality**, (ii) **voluntariness**, **informed consent**, (iii) **non exploitation**, (iv) **privacy and confidentiality**, (v) **precaution and risk minimisation**, (vi) **professional competence**, (vii) **accountability & transparency**, (viii) **maximisation of public interest and distributive justice** (ix) **institutional arrangements** (x) **public domain** (xi) **totality of responsibility** and (xii) **compliance**.

Recent advances in the field of **Assisted Reproductive technologies, organ transplantation, Human genome analysis, and gene therapy** promise unquestionable benefits to mankind. At the same time, they raise many questions of law and ethics, stimulating public interest and concern.

(Source : ICMR Publication 2000)

NOBLE PRIZE IN MEDICINE

Dr. Barry J. Marshal and **Dr. Robin Warren** from Australia have won the 2005 **Nobel Prize in Physiology**; for discovering that bacteria (H. Pylori), not stress, was the main cause of ulcer (90% duodenal; 80% Gastric). The bacteria can be eradicated effectively, by a short course of antibiotics and acid secretion inhibitors.

(TOI News Oct.5, 2005)

Natural Protection of Human Health From Environmental Pollution

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Abstract: The environment as well as the pollution are greatly comprehensive terms. The environment includes virtually everything around us, currently in their defiled forms. This has resulted in a serious degradation of the quality of life, also manifested as human ill-health. The paper contains the environmental impacts on human life as well as the natural protection of human health from it including the concepts of conviction.

Introduction

Environment, presently the most commonly used term by one and all includes everything that surrounds us viz. the air, the water, the land, the various human activities, the economy, the culture, the politics, and what not. Likewise, pollution is another most common terminology which has been adopted for common usage even by the most illiterate gentry. The author has personally heard house maids and even riksha pullers blaming pollution for all their ills. Pollution is a term which has been understood and defined differently by the various professionals. In literature, pollutionem, the latin equivalent of pollution means defilement. the chemists perceive pollution when the various chemicals intrude the environment beyond their permissible and/or acceptable levels. Likewise, for a biologist/ecologist, pollution would mean the presence of undesirable microbes or the disturbance of the distribution of the various species of organisms in an environment or the ecosystem becoming immature or unstable. For an engineer, the polluted environment loses its usefulness and thus the term pollution has to be use-specific. For example, a water whose temperature is not low enough, is polluted for industrial cooling; a water devoid of dissolved oxygen is highly polluted for fish culture; a water of high salinity is likewise considered polluted for irrigation, the agricultural use; air of low oxygen content is considered polluted for breathing; a land littered with all and everything is aesthetically polluted for sure; non-melodious and/or loud music is nerve breaking noise pollution, the 'Aye Raam and Gaya Raam' are considered political pollutants, the black-money manifests economic pollution, the miniskirts smell of cultural pollution, and so on. The environmental engineer thus, analyses (as an example) water to determine the use(s) for which it is not polluted and is most good, even as the most polluted water will surely find some use for which it can be considered suitable and exploited usefully (Bhargava, 1983b, d,e; 1985a,b,c,d,f).

Parameters of Pollution

The impurities or the foreign material or material present beyond their permissible concentrations are termed as pollutional parameters. These parameters could be classified into various categories based on the objectives of classification. For example, into physical, chemical, and bio-logical based on the type of analytical tests for their identification or determination (the parameter or the effect of the parameter or the imparted effect such as pH, hardness, turbidity, etc.) or into settleable, colloidal, and dissolved based on the size of the impurities such that the colloidal size ranges from 10^{-7} (lesser size is the dissolved size) to 10^{-4} (beyond which, it is settleable size) cm, or into conservative and non-conservative depending on the natural degradation abilities of the material; or into toxic and non-toxic; etc. (Bhargava, 1983a, d; 1984; 1985e,f).

Concepts of Safety and Aestheticity

A water, for example, is termed palatable if it pleases our physical senses, i.e., neither has objectionable turbidity, nor colour, nor taste, nor odour. On the other hand, the term wholesome is used when the water contains neither any pathogens, nor any toxicants nor excessive amount of any organic material serving as possible substrate for undesirable biological growths. Palatability is essential for the general and universal acceptability and gives a sense of cleanliness, while wholesomeness ensures safety against any possible disorder or disease. Naturally, an environmental engineer or a medical (qualified) personnel would insist on wholesomeness while the layman public would insist on palatability (Bhargava, 1983d; 1985a,c). As a result, the layman public is often driven to a palatable water source (even if it is not wholesome) such as a well water which generally looks clean, if the public water supply is not palatable (some political leader may present such a water's sample to create noise in the Indian Parliament or local Legislature Assembly. The public water supply schemes in metropol and larger cities therefore, spent well over 80% of their budget only in making the water palatable.

Pollution occurs naturally but mainly it is man-made through various human activities such as industrial giving out emissions and effluents of varying kinds; agricultural leaving unused pesticides/insecticides and fertilizers to reach the water resources; mining; construction; domestic; etc. The effluents flow into rivers, lakes, and ponds apart from percolating into the ground rendering all water resources unacceptable and polluted for many beneficial uses.

A vast majority of diseases are water-borne and occur due to consumption of untreated or inadequately treated water. Airborne diseases and neurotic disorders (mainly caused by noise pollution) are no few in the present times. Dreaded diseases like plaque originate from land pollution or from severely unhygienic conditions. The recent surat episode and the outbreak of infectious hepatitis in the mid-fifties at the national capital city of Delhi shall ever remain unforgettable.

The Indian Scene

India and its major metropol and cities are ranked very high in the merit list of the most polluted cities of the world. Kanpur already is on top of the most polluted list (Hindustan Times, 26/01/2003) There is severe pollution of all kinds all around and Indian villages and semi-urban areas (where more than 80% of Indian population lives) in particular have all the smokes (generated mainly from the domestic activities); polluted water and insanitary conditions. Even the developed urban areas are not devoid of the noise, air pollutants, unsafe drinking water (its only by chance that one can ever smell chlorine, the only index of the safe drinking municipal water), littered solid wastes everywhere at all times, educational, cultural, political and economic pollutions. But despite all this, the Indians survive in high numbers, although the author

has witnessed many Indians using air masks; ozone purified or bottled, the so called zero-bacteria water; ear plugs; etc. Perhaps the time is not far when there would be long queues for the refills for air masks and for the real potable drinking water.

The Survival of the Indians

It is anybody's observation that Indians have proved their ability to survive despite a total pollution of their entire environment. The people all over the world remain inquisitive particularly for the survival of the millions of the Indians who have been drinking a water which was hardly whole some ever. Waterborne diseases are a major health hazard. It is a common observation that many Hindu devotees drink the Ganga water directly from the river banks at Haridwar, Allahabad, Varanasi and other religious centres on the Ganga bank. To any one's knowledge, such a water is highly polluted for direct consumption and was neither rendered pollution free by the mammoth Ganga Action Plan nor the Indian rivers can ever be rendered pollution free even after tens of such Action Plans (Bhargava, 1992; 1998a,b) mainly because even in the most developed cities, about 50% of the wastewater cannot be stopped from reaching the rivers uninterrupted as also in many heavily populated but narrow streets of say Varanasi, no sewers can be possible to lay; the faulty effluent standards evolved whimsically rather than evolving them scientifically and rationally by working back from the river/stream standards making fullest use of the river's self-purifying abilities; the right people not doing the right job; no selections/appointments through an index based evaluated merit; bureaucratic corruption (one of India's Prime Minister boldly stated that only 15% of the sanctioned money actually reaches for the execution of the projects meaning only 15% success for any one project or completion of only 15% of all projects (that is, completion of only three projects out of every 20 projects); apart from many other reasons (Bhargava, 1985d; 1992; 1998a, b).

What keeps Indians Fit Despite Consuming Polluted and Non-Potable water?

The author had chance to ponder over this problem during his studies of the Ganga and witnessed habitants living along the Ganga drinking its water directly. In a notable incident, a village belle was collecting Ganga water in a pitcher from the Ganga at Kanpur from a point near the right bank where the Ganga looked almost totally black due to a major trunk sewer outfall carrying the city's untreated/particly treated sewage. The author just asked her why she does not like to collect the water from a point a little towards the left bank of the river where the water looked much whiter as the sewage had by then not completely dispersed in the entire cross section of the river. She smilingly replied 'Babuji (Sir), how does it matter? its all Ganga any way'. Surely, she and all her family had been drinking that kind of water for years and since their births. There are many scientific arguments and observed technological data that can support the above said instance and myths related to the Ganga. Many of these would be thought provoking to seek scientific clarifications and evidences. The foremost reason responsible for preventing many waterborne diseases is of course the immunity (natural and/or acquired). The author need not say a word to elaborate this point any further in an article meant for professional research level medicos. The pathogens naturally die out in an aquatic environment. The rates varying from pathogen to pathogen depending on the various environmental factors such as the temperature, pH, composition of water, velocity of flow, turbulence generated, reaeration abilities, the self-purifying abilities of the water, etc.

The die-out rate of pathogens follows an exponential law such that their number remaining (N) after a time (t) equals $N_0 e^{-kt}$ where N_0 represents the initial number and k the proportionality constant

which is pathogen specific and depends on the various stated environmental parameters. The author expects high 'k' values because the Indian rivers (particularly the Ganga) manifested a very high (higher by an order of magnitude) coefficients of BOD (biochemical oxygen demand) assimilation and reaeration (Bhargava, 1982; 1983c; 1986a,b,c). Apart from this, the Ganga water manifested a strange disinfecting power towards the cholera vibrio (Hankin, 1896; Bhargava, 1987c) which could not survive in unboiled Ganga (Varanasi) water though they comfortably survived in the Yamuna water and the boiled Ganga water. The stated experimental study points out to the presence of some disinfecting volatile substance present in the Ganga water. The author attributes the origin of this mysterious yet magic disinfecting material to the Ganga river's bed because if the material originated from the Himalayas (Gangotri, the source of Ganga origin) its disinfecting power would have been lost after Haridwar and/or Narora (Aligarh) where all the Ganga water is diverted into the canal system and the Ganga river regenerates (after Haridwar and Narora) mostly from the groundwater infiltration and the tributaries. Bhagirath, the genius mythological God who brought down the Ganga from the Heavens (Himalayas), canalized it through such a route where the stated disinfecting material was available (Yamuna river, only about 50m away from the Ganga at some places does not manifest this property). The Ganga which is thus well known for its hostility towards the pathogens will also be equally hostile (Bhargava, 1998a) towards the anaerobes (which have similarity to pathogens in their habitat and environment needed for their survival and which cause putrefaction in the absence of oxygen). As a result, the Ganga water (Jal) stored in closed containers does not putrefy even on long storages (Bhargava, 1987c; 1998a). These arguments provide another reason why many rural people and Hindu devotees who drink Ganga water directly from the river do not fall sick as the pathogens do not survive in the Ganga river even when it is heavily polluted showing a high count of coliform (a bacteria indicative of fecal pollution and pathogens of enteric source).

In the author's opinion, one of the very significant reason why most rural people and Hindu devotees do not contract/get infected with waterborne diseases even after drinking/inhaling Ganga Jal (water) directly from the rather polluted Ganga is their strong conviction. This aspect has not as yet been proved scientifically but logically the power of conviction can to some extent be appreciated from the logic of scientific conviction. A scientific conviction is manifested when we drink water from the so called zero-bacteria/ozone water/etc. branded bottled waters although in India, plenty of duplicates of such bottled water are abundantly available and sold everywhere. The author noticed many shoe-polishing boys in Delhi's Connaught Place selling the stated duly sealed duplicate bottled water at 50% or even less of its standard printed price. A similar example of scientific conviction is seen when we drink water which is duly chlorinated (manifested by smell of some chlorine which is intentionally left as a residual to take care of any future contaminations during the transport of water through a network of pipes and valves). Likewise, a patient feels relief even when he is given a sterilized distilled water injection because the patient always has conviction of being cured through an injection administered by a qualified professional medico. Apart from this, the author while living in Haridwar heard some saintly people saying that the Ganga water has several medicinal values. The medicinal values and the matter of conviction stated as above need more scientific investigations and exploration.

Future Needs

The medicinal values of the Ganga water may be explored from a scientific comprehensive medicinal analysis of the water of the Ganges all along its route. As for the conviction aspect the

possibility of its linkage with the psychology and mental status of a person can not be ruled out. This needs investigations and analysis of data collection through oral interviews. In the author's opinion, it is possible that the conviction brings about some momentary changes in the human system which provides or enhances the resistance needed to counter the ill-effects. The big problem will be the monitoring of such changes and what change and where? It will also involve the power of psychologic and its direction towards the origin of the need. May be that some team work including the research level medicos, psychologists, analysts, etc. would come forward to find a solution/answer.

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Changing Quality of Urban Air : Future of Indian Air Quality and Its Impact on Health

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Abstract: Ambient air is highly vulnerable to environmental changes resulting in the loss of air quality for the sustenance of life. With growing uncontrolled industrialization and increasing vehicular density with little or no concern for the environment the first affected environmental factor is also air. Only when the health hazard is noticed in a large population some concern is expressed by the authorities to look in to the quality of air. Since the corrective measures are too complicated and expensive, the inexpensive preventive measure is the only solution. With a detailed study on the deterioration of the air quality in urban cities things have become easier for the developing countries to implement the policies from the lessons already learnt by developed countries. The major factors concerned are vehicular exhaust, industrial gases, cottage industries, burning of urban waste and stone crushing are considered as major air polluting activities in an urbanized society resulting in variety of health hazards. The terrain of the city also plays a major role in aggravating the ill effects of polluted air on health. Land and hill locked cities, cities with troughs and crests have added greter problem compared to coastal and well spread plain cities. The future of air pollution in Indian cities depends upon the right type of implementable policies drafted in time much before any damage is noticed. In India with good human resources and developing technologies it would be easier to maintain good air quality.

Introduction

“Unlike food, water and shelter, we have not yet reached the stage of paying for the ambient air we breathe. Perhaps the day we have to buy the air to breathe, we may develop some concern for the quality of air around us”.

Air pollution is threatening to not only human health it has a deleterious impact on the life system as a whole. The World Bank Technical Report 381 on the Urban Air Quality Management Strategy in Asia (*Urban Air Quality Management Strategy in Asia WB Technical report 381 Ed by Jitendra J Shah and Tanvi Nagapal 1997*) highlighting the Greater Mumbai report has observed that many Asian cities are on the threshold of a major environmental crisis in the form of Air pollution. However the deteriorating quality of the air we breathe is attributed to various causes such as rapid economic expansion, rise in population, increased industrial output and unpredicted growth in the number of vehicles burning fossil fuel. One of the natural biological markers of the air quality is the presence of many sensitive bird species which moved away from urbanized areas as the air quality deteriorated.

There are no scientific records or observations to support that animals and plants cause any kind of land air or water pollution in the environment they live, which eventually become hostile for its survival. On contrary man has been responsible for various types of pollution on this earth resulting in various irreversible damage to his environment and to himself. Any limited pollution caused by man is to large extent is reversible by nature around him. With uncontrolled pollution the change in nature reaches irreversible stage. Permanent damage to nature occurs resulting in hostile environment for life to sustain. Under natural hostile conditions all forms of life undergoes genetic modifications, which is termed as adaptation. On contrary unnatural hostile condition as a result of pollution becomes unpalatable and detrimental to life. Nature also has hostile conditions at several places but ideal for

some form of life to sustain. Amongst various forms of pollution air pollution seem to be most sensitive one for all forms of life. Urbanization has added to the additional problems of waste management resulting in the changing environmental conditions. Similar studies conducted in various parts of the world have concluded that the growing concentrations of air pollutants have led to increased cases of respiratory disorders such as chronic bronchitis, colds and general decline in lung function. People living in and around the polluting factories are having higher incidence of cardiac diseases, respiratory tract infections, skin allergies as a result of reduce antioxidant status in their system. In Greater Mumbai alone the estimated health damage cost is around 18 billion a year mainly due to 2800 cases of excess mortality, 60 million respiratory symptom days and 19 million restricted activity days. There are no records available from other metros in India.

Present Scenario in India : Air Quality and Vehicular Pollution

Considering the vehicular exhaust and emission as the main source of reducing the ambient air quality, abatement of air pollution with appropriate measures in Mumbai since 1990 resulted in effectiveness in terms of both emission reduction and reduced impact of the general respiratory problems. Similar studies in Delhi after the introduction of CNG and subsequent abatement of lead in gasoline throughout the country in March 2000 implemented soon after the International Conference on Lead Poisoning, Prevention and Treatment in Bangalore in 1999 as an outcome of The George Foundation studies, the air quality started improving in our country. Still with high levels of well known carcinogenic material Benzene in our gasoline the scenario is not very palatable.

Amongst many pollutants lead in Gasoline seems to have special attention. Unleaded gasoline addresses the ambient lead problem in any urban area. Lowering the maximum allowed lead in gasoline as priority many countries have successfully implemented this as a policy by replacing lead with oxygenated compounds such as MTBE (Methyl-Tetra-Butyl-Ether). With catalytic converters many toxic exhaust compounds were further oxidized.

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However the number of vehicles with catalytic converters is negligibly small in Indian cities. Improving the diesel fuel quality will result in reduced mortality, decreased respiratory problems. Strict legislation on the prevention of adulterated gasoline (such as mixing gasoline with kerosene) to be implemented to ensure the air quality. Oil industry being the main player in this area need to be monitored by the Governmental agencies. Parts of many cities have become literally gas chambers.

At present in most of our other Indian cities Ambient Air Quality Air Quality Management System requires an integrated mechanism for the continual air quality monitoring. Recommended requirement are :

- 1 An inventory of air pollution activities and emissions and documentation. *This would help in monitoring the degree of air pollution and to have a check from time to time.*
- 1 Monitoring of Air Pollution and dispersion parameters with accredited procedures. *Unfortunately the monitoring facilities are limited and do not have wide scope for example we do not measure the toxic lead levels in air in many cities. For the lack of appropriate technology*
- 1 Calculations of Air pollution using internationally accepted procedures. *In order to place our self where we stand in terms of international requirement national data base is to be generated to evaluate the effectiveness of the action taken from time to time.*
- 1 Population and infrastructural impact assessment in the area. *It is only in recent time the health impact is being evaluated to correlate with the changing air quality with International organizational support.*
- 1 Follow-up of the abatement and control measures. *This being a long term plan is very much needed to see the effective measures taken.*
- 1 Continual modifications for improvement to be ensued and achieved.

All the above require the governmental infrastructure with total freedom to implement the policies with public awareness and stress on its Public Health impact.

All over the world most of our cities in developing countries in the name of development have become garbage dumps & gas chambers. It is possible to totally prevent air pollution with timely corrective and preventive action. It is unfortunate that we have failed to restore pure and clean air what nature has gifted for the survival of plants and animals. "Urban Air" is *under pressure with developmental activities*. Efforts are made to understand the status of our urban air at huge expenditure. Large amount all over the world is spent for monitoring, testing, treating and cleaning urban air. Probably less than 1/1000th of this amount is enough to prevent air pollution. Apart from this the annual budget to rectify the health hazard to its citizens in any society caused by air pollution is enormous. Rise in respiratory diseases, cancer, cardiac problems and numerous antioxidant related diseases are all associated with pollution in air. In many parts of highly congested cities oxygen bunkers are established. Facemasks are becoming part of human body in many cities. *It appears as if man is covering his face with shame (mask) for the heinous act of pollution caused by him!* Over 80% of garbage generated in urban areas is subjected to incomplete burning which result in the production of toxic gases. There are reports on the burning of plastic resulting in the

production of dioxane which is known to bring about the sex change from male to female of a child while it is in the womb.

Major sources of air pollution in urban environment. (Few examples are given below)

Vehicular exhaust - 1st major amongst contributor of air pollution (Ref: Baker J, Santiago R., Villareal T., and Walsh M (1998) *Vehicular emission control in Metro Manila Draft final report ADB (PPTA 1723)*)

- 1 Several tons of vehicular exhaust is constantly getting in to our urban air. This comprises of suspended particulate matter, oxides of nitrogen, lead, sulfur dioxide, benzene and many other unidentified toxins.
- 1 In Bangalore alone with over 16,48,943 vehicles which is 1/3 of the total vehicles in the State of Karnataka, one can visualize the air pollution going on every day. Many other metros have higher vehicular density and more intensified air pollution.
- 1 Adulteration of different kinds of fuel, poor maintenance of the vehicular conditions and poor quality roads demanding higher fuel demand are some of the causes of increased air pollution in cities. The dust emanating from the ground rich in toxic particles from the vehicular exhaust is another major source of air pollution.
- 1 Wearing masks will not solve the problem in hot and humid conditions. The quality of mask is also poor in majority of cases. The impact of long term exposure of breathing this kind of polluted air is not clearly understood even in urban areas of developed countries.
- 1 Apart from this there are no public health impact studies undertaken in developing countries correlating the changing air quality and its health impact.
- 1 Respiratory and skin disorders are very often attributed to allergy of unknown origin and given symptomatic treatment.

Industrial gases - 2nd major cause

- 1 Most of the industries have not complied with OSHA regulations in India and as a result we have unacceptable environment for our workers apart from polluting their surroundings. Couple of years ago few workers at Najangud pharmaceutical industry died due to Hydrogen sulphide poisoning as a result of total negligence on part of management system.
- 1 At one of the cement factories in Karnataka several workers suffered due to carbon monoxide poisoning.
- 1 The unforgettable Bhopal Gas Tragedy is still fresh in our memories.
- 1 Textile industry using cotton and other synthetic fibers are also known to cause several respiratory disorders due to its impact on the quality of air.

Burning of urban waste - third major source

- 1 Every day tons of urban waste including plastics are taken and burnt in the outskirts of our cities and are burnt. Even in a small town of Udipi at Indrali area which is thickly habited by people of South Canara heaps of garbage is slowly burning for several years. The toxic fumes cause health problem. In India most of

the urban waste is burnt in the outskirts of the cities.

- 1 Residents collect the garbage in their residential sectors and lit it with fire. This is 100% avoidable.

Cottage industries - to small extent but locally causing devastating health hazard.

- 1 Large numbers of people living in the area where agarbathies are dried on pavement have been suffering from respiratory disorders. This is due to usage of synthetic aromatics such as nitro benzene etc. Cottage industries do not come under the folds of regulating authority.
- 1 Stone crushing emission in cities and the neighborhood is known to be a serious health hazard not only to the workers but to the habitants in and around the quarry industry. Suspended dust emission ranges from 0.05 in primary crushing to 2.25 fines mill resulting in varying silicosis.

Open sewage and polluted storm drains

- 1 Many toxic organic gases generated in the open drainage system is known to cause several health problems such as mild to severe headache.

Since dilution is not the solution for any form of pollution, only remedial step towards reduction of pollution is towards preventing it. All forms of air pollution are preventable.

Let us look at the results of the recent studies on the blood lead levels of in large population in our country. Following conclusions are made as result of one of the largest scientific study sponsored by Dr. Abraham George under the George Foundation. Admiral Dawson chairman of the Project lead free was great inspiration in the study. (*Ref: Proceedings of the International Conference on the Lead Poisoning prevention and treatment 1999 published by The George Foundation*)

- 1 Over 50% children below 12 years of age in seven major cities have unacceptable level of lead in their blood. This will result in the reduction of their IQ. Damage at the CNS by lead is irreversible.
- 1 Amongst these cities Bangalore children born in last 12 years are the greatest victims of lead poisoning as the vehicular density increased in geometric progression in Bangalore. However in other cities lead free petrol was introduced much earlier.
- 1 It is unfortunate to note that the lead in the blood of Indian population is not from Indian Lead mines. This is established by isotope dilution method.

As a result of the study and the white paper submitted to the Government lead free petrol is made available in most of our cities.

The above observation clearly indicates that is possible to eliminate any kind of pollution from the environment in which we continue to live.

Non-invasive simple methods to reduce air pollution in cities.

- 1 In order to reduce acidic fumes in the air alkaline fountains at several places will help. One such fountain is at present working in front of St. John's Hospital in Bangalore. Air scrubbers are other means of reducing toxic gases in air.
- 1 Coating road dividers with limewater regularly (in stead of paints which are expensive) will absorb vehicular gases to some extent.
- 1 Catalytic converters in vehicles to reduce carbon monoxide.
- 1 High quality scrubbers in industries to reduce toxic gases getting into our environment.
- 1 Strict rules to curb usage of public places for burning urban waste.
- 1 Implementing policies for sale of lead free paints.
- 1 Increasing public transport system and preventing private vehicle to congested and crowded areas identified in cities.
- 1 Displaying air quality at various places for the benefit of public.
- 1 Regularization of cottage industries and monitoring the use of quality aromatics, which does not cause any air pollution.

Internal Air Quality monitoring in residences is becoming popular in most of the developed countries. Bio-aerosol assessment control is of great health significance an aerosol in residential sector is known to cause many kinds of respiratory problems. Many cosmetic insecticide sprays are found to be toxic. Apart from this carpets and curtain mites and dust, mycotoxins, endotoxins, volatile organic solvents from freshly applied paints is known to affect the air quality. Many studies are needed in this direction. (www.aerotechlabs.com)

With increased awareness and also the availability of technology in India the future of urban air quality is likely to improve with reduced ill effect and impact on the health. This being a slow process governmental agencies will have to work in tandem with the NGO's to achieve early results. We can not afford to wait and have huge expenditure on corrective measures.

"By law of nature man has no right to disturb any part of his environment, once disturbed beyond some limits, results in irreversible damage to all forms of life. Man's presence on this earth is only towards sustenance of all forms of life in our environment. It is unfortunate that with mans efforts for easy life and comforts he can be hold totally responsible endangering not only his environment but every thing around him" - Air Pollution is 100% preventable with little concern and timely action to assure our next generation quality life.

Do You Know?

- (i) 66.06% households do not have toilets.
- (ii) 78.01 rural and 26.03% urban households have no toilet facilities and practice open defecation.
- (iii) 7.0 mill children die of diarrhoea/dehydration every year.
- (iv) Rivers get polluted mostly by the sewage from cities; open defecation adds to environmental pollution.

Asbestos : Disquieting Tale Goes On

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Abstract: Evidence on carcinogenicity of asbestos was available in early 20th century but the asbestos mining industry could successfully suppress it for 50 years. Weak politics, weak legislation, half-hearted enforcement and strong and defiant corporate may be some of the reasons behind such a situation. Today, asbestos related corporate is trying to spread a message that some forms of asbestos may be carcinogenic but the chrysotile is not. The motive behind this message is an open secret, since chrysotile occupies nearly 95% of today's asbestos market. Adverse effects of fibrous minerals on human body have always been a matter of debate, not because of scientific reasons alone but also for the interests of the marketing forces. Scientific inquiry must proceed but as long as safer substitutes exist for most asbestos uses, the proposition of releasing more asbestos in the environment, or of relaxing vigilance will be disastrous. India produces as well as imports asbestos. Imports are likely to continue under the pressure of domestic demand unless we promote and popularize safer substitutes. Continued release of several carcinogens in the environment should not be eulogized as price of development.

Introduction

The story of asbestos continues to insult us. Evidence that asbestos causes cancer was available in the thirties but the asbestos mining industry could successfully suppress it for half a century. Warning for those exposed to asbestos was deliberately delayed. As a result, millions of workers were exposed to the carcinogen and hundreds of thousand died. At the end, the industry was forced to produce confidential documents containing research data. The information became public because of legal actions and not because of the interventions by the scientific community. What is more disturbing is the clinching evidence that all this was done in collaboration of some of the leaders of occupational and environmental medicine¹. Today when the position of the asbestos mining companies and manufacturers is becoming more and more indefensible, a newer type of misinformation campaign has started. Asbestos related corporate is trying to spread a message that some forms of asbestos may be carcinogenic but the chrysotile (white asbestos) is not. The motive behind this message is an open secret. Since chrysotile occupies nearly 95% of today's asbestos market, it must be protected to save the profits.

To get to the root of this problem let us go to the basics. An important group of fibrous minerals is known by the generic term asbestos. There are four commercially important forms: chrysotile; crocidolite; amosite; and anthophyllite. Of them, the chrysotile alone accounts for 95% of global asbestos production and most of its comes from the province of Quebec, Canada². Chrysotile is a fibrous hydrated magnesium silicate mineral, which is being used in many commercial products. There are several accompanying minerals in the fibrous ores, and fibrous amphibole may be among them. In this regard, tremolite is thought to be especially important³.

Low concentrations of chrysotile are found throughout the global crust (air, water, ice caps and soil) but the human activities contributing to fiber aerosolization and distribution in the environment are chiefly occupational e.g. recovery from geological deposits, processing, manufacturing of asbestos containing products

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and their disposal. Asbestos cement industry is the largest user (85% of total use) of chrysotile fiber³.

The great debate

Partly for scientific reasons and partly for the interests of the marketing forces, the basis and the level of carcinogenicity of fibrous minerals has been a matter of controversy all through the last century^{4,5}. Today we have sufficient epidemiological and clinical evidence that besides causing a progressive fibrotic disease of lung called asbestosis, asbestos also causes: cancer of lung; malignant mesothelioma of pleura and peritoneum; cancer of larynx; and some gastrointestinal cancers. The nature and amount of evidence goes beyond any scientific controversy and in acknowledgement of this body of evidence^{6,7}, the Environmental Protection Agency (EPA) and the WHO's International Agency for Research on Cancer (IARC) have declared asbestos a proven human carcinogen^{2,8}. When doubts were raised against the approach of declaring all forms of asbestos as carcinogenic and it was suggested that some particular asbestos-types might not be causing cancer, the issue was carefully considered among the wider scientific community.

In the light of hard scientific evidence IARC-WHO acknowledged that all forms of asbestos are known carcinogens. All have been shown in epidemiological, clinical and laboratory studies to be fully capable of causing lung cancer, mesothelioma and a whole range of asbestos related diseases⁸. These developments resulted into mounting public pressure culminating in government bans in developed countries on further release of asbestos in the environment. New use of asbestos has almost completely ended in developed world. In contrast to all this, extensive and aggressive marketing by Canada and other exporting nations continues in the developing world, where sales remain strong⁹.

Some publications on exposure to chrysotile asbestos¹⁰⁻¹⁴ have asked to take a fresh look on the subject. Consequent to the newer knowledge about chrysotile form and significant decline in high-dose asbestos exposure, at least in the developed world, the focus of research and public health debate is now shifting towards supposedly low level of carcinogenicity of a particular type of asbestos and its effects on human health with non-occupational low-dose exposure. This has created a space for a renewed

debate. However, the situation may take a worrisome turn when the lure of sales may prompt corporate forces to turn this plain scientific curiosity into profitable confusion. Some of these scientific inquiries are already being used by the industry to further its agenda.

Efforts have been made by the international health community to make a considered opinion and settle the issues raised by this new debate. The collective opinion has been suitably represented by an editorial¹⁵ and a commentary¹⁶ that published in two of the leading multi-disciplinary biomedical journals. The commentary in the *Lancet*¹⁶, counters authors' suggestion that chrysotile could be commercially used with very little health risk, by saying that chrysotile asbestos found in nature is typically contaminated with amphibole and the highly carcinogenic tremolite.

As for the lung cancer, the basis of assertions about safety of chrysotile is very precarious. Though these fibers are not commonly found in large quantity at necropsy¹² for they are rapidly cleared from human lung, their carcinogenicity in animal models is well established¹⁷.

The editorial in JEJM¹⁵ quotes the landmark research conducted by Selikoff and colleagues^{6,7} to prove the point that Canadian chrysotile, like all other forms of asbestos, is a potent human carcinogen. It also highlights the fact that one of the findings published by Camus, et al¹⁴ (more than sevenfold mortality from pleural cancer in mining area), corroborates with Selikoff's conclusions. In fact, the amount of chrysotile asbestos already released in the environment creates a situation where exposure to chrysotile products remains the leading cause of mesothelioma in the world¹⁸.

Enormous and continued release of several carcinogens in the environment is being eulogized as price of development. Weak politics, weak legislation, half-hearted enforcement and strong and defiant corporate may be some of the reasons behind such a situation. But can all this happen without weak science and/or collaboration of scientific community? The case of asbestos is a specimen. To gain insight into corporate activities regarding the identification of occupational carcinogens in this century, Lilienfeld reviewed the actions of one, the asbestos industry. He studiously collected all the relevant correspondence, confidential reports, and even the exhibits used as evidence in several legal proceedings and cited them as references along with academic documents in his case study. What he found was that the industry, in concert with many of its collaborators, first generated data on carcinogenicity of asbestos and when found unfavorable, systematically suppressed it for five decades. The development of warning for those exposed to asbestos was deliberately delayed. As a result, millions of workers were exposed to the carcinogen and hundreds of thousand died. At the end, the industry was forced to produce confidential documents containing research data. The information became public because of legal actions and not because of the interventions by the scientific community. More disturbing to not ewas that the members of academic medical community, in collusion with the insurance industry, participated in this exercise of deception. Some of them were the leaders of occupational medicine. The surprised author of the above quoted study further states: "The degree to which scientific fraud permeated published reports is also of concern. However, unemployment or withdrawal of research support may be the ultimate 'reward' for those who do not participate in such activities"¹¹. The story still remains patchy and incomplete.

What should worry the wider scientific community more is the fact that such unfortunate happenings are not confined to asbestos industry alone. A similar history has been documented in aniline dye industry as well¹⁹. Despite the disclosures of suppression and fraud, no mechanisms have been developed to prevent recurrences.

Where do we go from here?

Coming back to chrysotile, the empirical evidence is sufficient itself to argue against any relaxation of public health control over any type of asbestos. Recent efforts to portray chrysotile asbestos as safe, are inaccurate. And the assertions that chrysotile asbestos can be used without risk are contrary to fact and extremely dangerous. The WHO Environmental Health Criteria - 203 concludes by stating that the exposure to chrysotile asbestos poses increased risk for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. Though the question of threshold has been raised by some researchers¹⁴, the criteria say that no threshold has been identified for carcinogenic risks²⁰. On the positive side, it is getting clearer that the direst predictions about an epidemic wave because of non-occupational exposure to chrysotile have shown little evidence of materializing. The risk is not nil, but low. However, the proposition of releasing more asbestos in the environment, or of relaxing pressure will be disastrous as long as safer substitutes exist for most asbestos products.

A word of caution for activists!

On one hand, we need to stop further release of asbestos in the environment and on the other, a studied restraint is needed while dealing with the asbestos that remains as a legacy of harmful construction practices in millions of schools, homes and commercial buildings. This is to be borne in mind that manipulation of friable asbestos products may be an important source of chrysotile emission in the environment.

Mistakes have been made in the past while handling the asbestos already used in buildings. Agitated parents in some communities have caused great harm to their children, school staff and themselves by tearing out asbestos sheets from school buildings without proper safety cover. In view of this a rational set of legally enforceable controls were evolved in USA under Asbestos Hazard Emergency Resposne Act (AHERA)²¹. Unless asbestos fibers become airborne and can be inhaled, an intact asbestos sheet in a building poses little threat to health of the inmates. However, in-place building materials containing asbestos may pose risk to those carrying out alterations, maintenance and demolition. Such materials also have the potential to deteriorate over the years and create exposures.

Do safer substitutes exist?

The process of substituting asbestos with other safer materials may encounter certain difficulties including technical performance of the substitute and operational feasibility. Cost considerations will also come into picture but they should be seen in the perspective of the total cost of asbestos related health hazards. New products may be costlier initially but the prices will rapidly come down with the increasing demand and mass production. Looking at the balance of stakes, these obstacles should not deter us from stopping further release of asbestos in our environment.

There are several established alternatives to asbestos that do not depend on fiber technology. Materials like corrugated polyvinyl chloride (PVC) and steel sheeting can replace asbestos in building material. Many non-asbestos fibers have also been developed and

they can replace asbestos in a wide range of products. Commonest of them are polyvinyl alcohol (PVA), aramid and cellulose²². They have been tested and found safer than asbestos by Committee on Carcinogenicity, United Kingdom's Dept. of Health and European Commission Scientific Committee on Toxicity, Ecotoxicity and the Environment.

Asbestos in India

Asbestos continue to be mined and manufactured in India. Because of current needs, locally mined asbestos is not enough and India imports a lot of asbestos from Canada. In fact, asbestos figures among top ten imported minerals in India²³. The most worrisome trend is about the popular demand of asbestos in building industry, though there are other areas of consumption like industries dealing with friction materials, break linings, seals and gaskets. Under the pressure of domestic demand, imports may continue. There is recoverable deposit of 2.29 million tones of asbestos in our country²³, which may further be mined and released in our environment.

Occupational health surveys have reported pulmonary function impairment and radiological abnormalities in 54.8% of asbestos milling workers and 19.5% of miners²⁴. Mesothelioma is also reported occasionally but in the absence of any systematic registry and investigation of mesothelioma cases, epidemiological and clinical evidence is sketchy. Airborne concentration of asbestos fibers in milling units of India are found much higher than prescribed standard. Of the 8 major asbestos products manufacturing units in India, examined by the Central Pollution Control Board, 6 were not complying with the emission standards. For the remaining 2, the compliance could not be ascertained²⁵.

Asbestos related policies and legislation in India remain confusing and loosely enforced. Mesothelioma is not amongst the notifiable diseases under the Factories Act. However, the Mines Act (1995 revision) includes cancers of lung, pleura and stomach in the list of notifiable diseases. Import duties for asbestos have been lowered by 68% between 1995-2000. All this is not in agreement with our New Mineral Policy (1995), that has: 'minimizing adverse effects of mineral development on forest, environment and ecology' and 'ensuring conduct of mining operations with due regard to safety and health of all concerned' as its stated objectives.

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- Statement regarding adherence to **standard ethical guidelines** prescribed by ICMR 2000. (see page 135)

Environmental Pollution and Unborn Child

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Introduction

We pay price for every good thing in life so are we paying price for all scientific breakthrough and their applications for the betterment of mankind in the form of environmental exhaustion and degradation. Pollution, the fall out of industrialization and globalization, affects all living beings. Pollutants become part of the ecological cycle and circulate in the environment from soil, water and air to vegetation, to animals, to humans and even to the fetus.

For the developing pregnancy, the environment includes anything fetus is exposed of as well as any thing that may alter conditions within the womb¹. Altered metabolism and physiological adjustments during pregnancy e.g. increased tidal volume, expanded blood volume, increased body fat, hypoalbuminemia and progesterone-induced hypomotility of the gut may facilitate the bioavailability of various pollutants in the pregnant woman which can then be passed onto the growing fetus². Functional, anatomical, physiological, metabolic and developmental immaturity renders the fetus more vulnerable to harmful effects of the pollutants than mother herself.

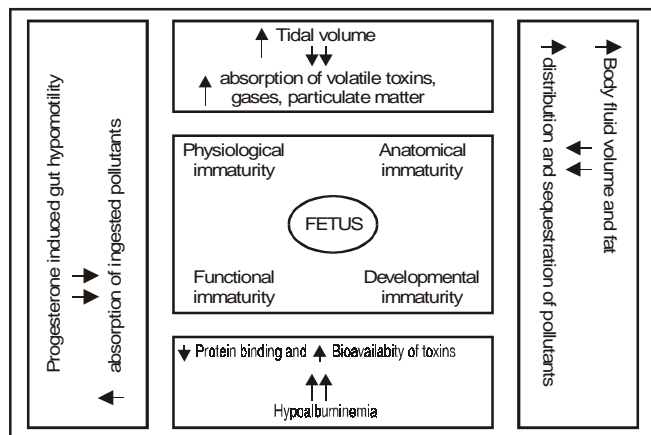


Fig. 1 : Physiological changes during pregnancy make a woman and her fetus more susceptible to pollution damage. (Adopted from Environmental pollution and human health by Prof. Satish Kumar Bhargava.

Several reports have conclusively proved deleterious effects of many industrial wastes, toxins and metals on the reproductive performance of both man and woman. From oligozoospermia to infertility in man, pollutants have been found to affect menstrual rhythm and ovulation and may cause abortion, intrauterine growth retardation, structural defects, stillbirths and neonatal deaths. "Yusho Disease" of newborn is a classical example of modern industrial disease³. There is infant growing concern of fetal well-being and environmental pollution. Unfortunately there is paucity of data on the effects of pollutants on the placental histology, fetoplacental blood flow, embryogenesis and fetal physiology, metabolism and

homeostasis. Similarly there are no specific guidelines/recommendations on safety levels of pollutants for pregnant women which are safe for growing fetus.

Mechanism of Fetal Damage⁴ :

The exposure to the pollutants may be of two types : (i) Concurrent exposure; (2) Non-concurrent exposure.

1. **Concurrent exposure** : It means that the mother is exposed to the pollutants during pregnancy, from conception till confinement, which may also affect the fetus in-utero. The exposure to the child may even continue during infancy via breastmilk. Concurrent exposure can take place either at home (indoor pollution), at workplace or the residence may itself be in the occupational premises. The another form of concurrent exposure could be paraoccupational, where other family members bring home the pollutants from their work place and the pregnant mother is exposed to these pollutants passively. The fetus can be protected if pregnant women is kept away/removed from the polluted environment.

2. **Non-concurrent exposure** : In this form of exposure either parent was exposed to some pollutants before conception but not thereafter, till the end of pregnancy. The ovum or sperm might have been damaged before conception which may cause infertility, abortion, IUGR or stillbirth. The mother might have been exposed to some pollutant during her childhood or adolescent age which was deposited in the tissues. This pollutant may be mobilized during pregnancy and may be transported to the fetus via blood. The classic example of non-concurrent exposure is lead, to which mother was exposed as a child and the latter got deposited in her bones at the time of mineralisation. During pregnancy when calcium is chelated from the maternal bones, lead also gets chelated and affects the fetus in-utero. Hence pollutants can affect fetus several years after exposure of the mother.

Pathways of Fetal Exposure to Environmental Pollution

Two possible routes are proposed for fetal exposure to environmental Pollution (2) : (i) dependent on placental circulation, (ii) independent of placental circulation :

Placental Dependent : The fetus remain confined in a cosy, sterile and well protected fluid filled sac inside the uterus. For a pollutant to reach to the fetus, it must enter the mother's blood first and then cross the placenta to enter circulation. For example carbon monoxide present in the vehicle exhaust and cigarette smoke is inhaled by the mother, enters her blood and is then transported to the fetus via placental circulation.

Placental Independent : Some pollutants need not enter the mother's blood stream and placental circulation to reach the fetus. They may exert direct effect on the fetus like ionizing radiations, hyperthermia, electromagnetic field etc.

Stages of Fetal Development and Deleterious effects of Pollutants²:

Most structural development of the fetus occurs during early pregnancy, within 3 months after conception. The embryonic stage of exposure determines the fetal structural malformation.

Pre Embryonic Phase : It extends from conception till 17 days. During this phase implantation, blastocyst formation and gastrulation take place. If an insult occurs at this time it can result in death/abortion. However few embryos can survive into normal fetuses, through multiplication of some totipotent cells, still left after the damage.

Embryonic Phase : It extends from 18-55 days after conception. This is the period during which organogenesis occurs e.g. heart forms during 3 to 9 weeks, limbs, during 3 to 6 weeks, eyes, during 4 to 7 weeks, teeth, during 6 to 9 weeks and external genitalia develops during 7 to 9 weeks⁵. The growing fetus is most sensitive to the environmental insult during this period because the rapidly dividing cell lines are more susceptible to damage and once this damage occurs it is due to are bioaccumulative substances. These toxins do not readily breakdown, they persist and build up in the tissues of animals and humans.

Mercury is an element occurring naturally in earth's crust but it does pose a health risk. It is produced by coal burning combustion of medical waste in incinerators e.g. thermometer, antiseptics and CT scan films. So far burning of fossil fuel is the largest source of mercury in the atmosphere. Mercury when reacts with water produces its most toxic form methyl mercury. In pregnant woman, both mercury and methyl mercury move freely across placenta contaminating mother and fetus together. Once mercury is in the body, it moves into all tissues, concentrating in brain, liver and kidneys due to its bioaccumulative tendencies and concentration within these organs can easily reach dangerous levels. CNS is the major target organ, though neurological sequelae may take years to manifest. In Minamata Bay, Japan, there was accidental exposure of fishes to methyl Mercury. Six percent of the children born over 10 years period in this area has cerebral palsy and mental retardation. Other features of fetal Minamata syndrome³ are CNS tumors, deafness, blindness and irritability.

PCB and dioxin together form a group known as Polychlorinated aromatic hydrocarbon (PCAH). These are formed as a by product during manufacture and incineration of most of chlorinated products like PVC and other plastic used in hospital e.g. blood bags, i.v. sets, tubes, vinyl surgical gloves, enema bags etc. These substances are also bioaccumulative substances. Environmental exposure to PCAH may interfere with sexual maturation and thus in long term may adversely affect human reproduction¹². Studies have shown that prenatal exposure to these compounds produces long lasting cognitive and behavioural damage but there is some evidence of recovery also¹³. In utero exposure to these agents caused a symptom complex called "Yusho Disease" in a Japanese community that used cooking oil contaminated with PCB. The fetal PCB syndrome consists of transient dark brown skin pigmentation, growth retardation, dental and skull deformities, exophthalmos, facial edema and delayed skeletal age. PBB at high exposure rates cause adverse pregnancy outcome including low birth weight and delayed neuropsychological development³.

Pesticide and Insecticide

It has been seen that exposure to indoor pesticides are associated

with significant reduction in fetal growth and head circumference in mothers having low paraoxonase-1 enzyme activity levels¹⁴. Another recent study has shown that chlorpyrifor, propoxur and diazinon when used during pregnancy may impair fetal growth¹⁵. DDT has also been seen to affect pregnancy outcome adversely in the form of high risk of eclampsia, preterm birth, cleft palate & lip and intrauterine deaths. However contrary to earlier belief the association of smoking with heart and limb defects is not straight forward and smoking did not seem to increase neural tube defects⁸.

Indoor Smoke Pollution :

Pregnant women are exposed to pollution not only outdoor but also indoor. Source of this indoor smoke is mainly the use of unvented gas stoves, chulhas, burning cowdung cakes, wood, kerosene, coal and cooking use of mosquito repellents, lighting agarbattis and scented sticks. In one Indian study it was found that level of COHb in the women exposed to different cooking fuel (biomas, kerosene, LPG) was statistically not different⁹. It was mainly because kitchens had poor ventilation and so even LPG use (which is supposed to be smoke free fuel) was associated with high COHb levels. Fetus has higher affinity for carbon monoxide. It has been seen in animal studies that it takes longer (twice) to normalize blood COHb levels in fetus than mother after exposure to CO. Hence poor indoor air quality can affect fetal growth and can lead to low birth weight baby.

Automobile Exhaust :

Automobile exhaust produces carbon dioxide, carbon monoxide, lead, sulphur dioxide etc. Recent studies have shown that pregnant women breathing high levels of carbon monoxide are more likely to have low birth weight babies and it also increases the risk for ventricular septal defects in dose response fashion with increasing second month carbon monoxide exposure. It is also seen that exposure of pregnant woman to ozone is associated with increased risk for aortic artery and valve defects, pulmonary artery and valve anomalies and conotruncal defects¹⁰. Lead is another pollutant, which poses a major health risk. It is released in traffic fumes and household coal combustion. Maternal lead levels may also increase if some other family member is being occupationally exposed to lead. Blood levels of lead are determined by bone resorption rather than dietary absorption. In late pregnancy cortical bone is resorbed and if mother has high levels of lead in her bones due to childhood lead exposure, it may lead to higher lead levels in the maternal blood and in the fetus¹¹ even if mother is not exposed to lead during this pregnancy. Fetal lead toxicity leads to IUGR, preterm labour, congenital malformation, mental retardation and neonatal death. Significant association has also been found between impaired semen parameter and elevated lead levels in male partners⁵.

Industrial wastes :

The main health hazards in industrial wastes include Mercury, Polychlorinated (PCB) and polybrominated biphenyls (PBB), Dioxins and DDT. These toxins pose a major health risk irreparable. Each organ and system has a particular susceptible period during its early differentiation. The precise time of maximum susceptibility varies from organ to organ.

Fetal phase : It extends from 56 days till birth. This period of fetal growth is characterized by hyperplasia and hypertrophy. During this phase the effects of various pollutants are mainly on

the growth and functional loss rather than structural abnormalities because the process of differentiation has already been completed.

Effect of Specific Pollutants

Tobacco Smoking : Tobacco smoke produces both nicotine and carbon monoxide. Nicotine readily crosses placenta with fetal concentration generally 15% higher than maternal levels. Nicotine, which is a powerful vasoconstrictor, has effect on uterine arteries and fetoplacental blood flow. Nicotine may also compromise the fetal blood flow by constricting the umbilical arteries. Reduced fetal circulation can decrease both nutrients and oxygen supply to the fetus and in the process can affect the growth of the fetus. Moreover, carbon monoxide which in an asphyxiant, forms carboxy Hb in both mother as well as in the fetus and further compromises the oxygenation of the fetal tissues.

Nicotine causes increased spontaneous abortion in first trimester, increased premature delivery rates and decreased birth weight. Researches show that not only active but passive smoking during pregnancy may adversely affect the fetal growth.

There is also association between smoking and decreased female fertility especially with a relationship to primary tubal infertility. Smoking appears to have adverse effects along a continuum of preimplantation and implantation including gamete production and function, ovulation and cyclicality, fertilization, early embryonic cleavage, embryo transport and implantation. In males, clear evidence is there that smoking results in fewer and less motile sperms as well as lower proportion of normally shaped sperms⁶. Beside the increased rate of low birth weight and small for date babies, there are two other major toxic consequences of smoking :-

- (i) Intellectual impairment and
- (ii) Increased rate of infantile cancer, primarily leukemia, lymphoma and cerebral tumors are increased in children born to women who smoke during pregnancy⁷. Recently California birth defect monitoring programme studies have shown that smoking raises the risk for oral clefts and the hazard is even more in babies who carry a cleft susceptibility gene named transforming growth factor α gene (TG F- α) A₂ form.

Men and women working in the floriculture are exposed to 127 different herbicides. A study conducted in Bogota on floriculture workers revealed serious consequences of the herbicide exposure (Table 1). This study recruited 8867 workers of which 33% were male and 67% female with mean ages of 29.2 & 27 years respectively. The mean length of time worked in the floriculture was about 3 yrs. The total number of pregnancies included in the

Table 1 : Rtes and odds ratios (OR) for various adverse pregnancy outcomes before and after work in floriculture (95% CI=95% confidence interval)

Pregnancy outcome	Female workers Prevalence rates (%)			Wives of male workers Prevalence rates (%)				
	Before	After	Or	95%CI	Before	After	Or	95%CI
Induced abortion	1.46	2.84	1.98**	1.47-2.67	0.29	1.06	3.63**	1.15-8.70
Spontaneous Abortion	3.55	7.50	2.20**	1.8202.66	1.85	3.27	1.79**	1.16-2.77
Premature baby	6.20	10.95	1.86**	1.59-2.17	2.91	7.61	2.75**	2.01-3.76
Stillbirth	1.37	1.34	0.99	0.66-1.48	1.01	0.89	0.87	0.42-1.83

*P=0.05-0.01 **P=<0.01

analysis were 13984; pregnancies of female employee and wives of male workers were 10481 and 3503 respectively².

Ionizing and Radiation :

It is a well known fact that maternal exposure to high dose ionizing radiation is teratogenic to human fetus. At 2-4 weeks of age, the embryo is sensitive to lethality of ionizing radiation. During 4-8 wks, the fetus is growth retarded and sustains mental retardation, microcephaly, cataract and microphthalmia. At 9-10 wks, it retains CNS sensitivity and is retarded. In late fetal stage, at (12-16 wks) the fetus can sustain cellular damage but is not grossly deformed¹⁶.

Electromagnetic Radiations :

These radiations are generated by sources as diverse as video display, power lines, microwave oven & cellular phones. One study showed that females with history of subfertility, having renataluse of electric blankets was associated with more than four fold increase in risk of CUTA (congenital urinary tract anomalies)¹⁷. Risk was greater if exposure occurred during first trimester. Risk also appeared to increase with increasing duration of electric blanket use. But studies also suggest that no clear-cut association is seen between using electric bed, heating devices and neural tube defects/oral clefts¹⁹.

Although neurotic disturbances, depression and anxiety disorders are seen in population living in vicinity of overhead high voltage transmission lines¹⁸ yet the recent study suggest that no clear association is seen living within 150-300 feet of high voltage power line and increased risk for birth defects¹⁹.

Regarding the use of cellular phones during pregnancy and its effect on fetus no conclusive study is available. Some studies done in nonpregnant adults show that there is no association between use of cell phone to uveal melanoma, brain tumor, skin cancer, meningioma. Till now no such studies have been carried out to look for the effect of cellphone use on fetus and thus, caution is advised for the use of mobile phones by pregnant women until some conclusive studies disprove the possibility of any harmful effect of cell phone use on fetus.

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Conference News

Medicon 2005 - 80th National Conference of IMA will be held on 27 to 29 December 2005, at *Venue* : Chennai Trade Centre, Mount Poonamallee Road, Nandambakkam, Chennai-600089. *For details* Contact Organizing Secretary **Dr. T. Sadagopan**, Usha Nursing Home, 23/2 Filter Bed Road, Vellore-632001, Tamil Nadu. Telephone : 0416-2224878; 2223222, Mobile : 98430-34878 and 94433-74878. *Scientific Committee Convenor*. **Dr. S. Chandrasekharan**, No.4, Jambulingam Street, Nungambakkam Chennai-600034, Tel : 044-28275832, 28312946 Mobile : 9840015023 E-mail : schan2004@rediffmail.com. Please offer your scientific faculty participation.

5th International Symposium on Diabetes will be held at Mumbai, India, on 21st and 22nd January 2006; *Theme* 'Emerging Treatments in Diabetes and Complications' International Faculty includes Prof. Robert Rizza, Prof. K.S. Nair Prof. William Young Jr. *For details*, please contact **Dr. Shashank R. Joshi**, Joshi Clinic, 12 Golden Palace, Turner Road, Bandra (N), Mumbai 400050, India. Email : mayoiiid_5@yahoo.com.in

7th World Congress of Nephrology will be held at Rio de Janeiro, Brazil for April 21-25, 2007. *For further information* contact website : www.wcn2007.org

Literature Review

Compiled by **Dr. P.D. Gulati**

Safety and success of kidney transplantation and concomitant immunosuppression in HIV-positive patients. *Anil Kumar MS, Sierka DR, Damask AM et al. Kidney International, 67;1622-1629, 2005.*

Human immunodeficiency virus-associated nephropathy (HIVAN) has become the third leading cause of end-stage renal disease (ESRD) in African Americans, and is expected to grow exponentially. High active antiretroviral therapy (HAART) has significantly prolonged the survival of patients with HIV infection. Despite the growing number of HIV-positive dialysis patients with prolonged life expectancy, kidney transplantation with immunosuppression has been declined because it is considered a waste of scarce donor kidney due to potential increase in morbidity and mortality. The effect of immunosuppression on HIV infection. Forty (40) HIV-positive dialysis patients received kidney transplantation between February 2001 and January 2004. Patient inclusion criteria were maintenance of HAART, plasma HIV-1 RNA of <400 copies/mL, absolute CD4 counts of 200 cells/ μ L or more. Immunosuppression was basiliximab induction and maintenance with cyclosporine, sirolimus, and steroids. HAART was continued post-transplant. Acute rejections were diagnosed by biopsy and treated with methylprednisolone. Surveillance biopsies were completed at 1,6,12 and 24 months, and evaluated for subclinical acute rejection, chronic allograft nephropathy, and HIVAN.

One and 2-year actuarial patient survival was 85% and 82%, respectively, and graft survival was 75% and 71%, respectively. Plasma HIV-1 RNA remained undetectable, and CD4 counts remained in excess of 400 cells per μ L with no evidence of AIDS for up to 2 years. Conclusion. One-and 2-year graft survival is comparable to other high-risk populations receiving kidney transplantation. One-and 2-year patient survival is higher than HIV patients maintained on dialysis. Immunosuppression does not adversely affect HIV recipients maintained on HAART in the short

term. This study shows that kidney transplantation in HIV positive patients who have plasma HIV-1 RNA of <400 copies/ml on HAART is safe and is associated with survivals better than on dialysis. However monitoring of combined immunosuppression and HAART due to major drug interactions is challenging. In those receiving protease inhibitors, very low doses of cyclosporine and sirolimus are needed.

Helicobacter pylori Stool Antigen Test. E. Mahir Gulcan, Aydin Varol, Tufan Kutlu, et al. *Ind. Jr. Paed.* 2005;72,675.

Helicobacter pylori (*H.pylori*) infection is usually acquired in early childhood. Invasive techniques used for diagnosis of *H.pylori* infection require endoscopic examination which is expensive and inconvenient and may cause complications. The aim of this study was to evaluate the performance of a new noninvasive diagnostic method, stool antigen test for *H.pylori* in untreated children with recurrent abdominal pain. Eighty children (35 female, 45 male) who have undergone upper gastrointestinal endoscopy due to recurrent abdominal pain were included in the study. The *H.pylori* stool antigen test (HpSA) is based on a sandwich enzyme immunoassay with antigen detection. HpSA sensitivity, specificity, and positive and negative predictive values were determined with reference to the results of both histology and rapid urease test as a gold standard (*H. pylori* status). While 49 of the 80 children (61%) tested were positive for *H.pylori* according to the results of both histology and rapid urease test, 28 children had negative *H.pylori* status. Among those 49 children, 48 were found to be positive by HpSA. Of 28 patients with negative *H.pylori* status, 28 were *H.pylori*-negative also in the stool test. The sensitivity, specificity, and positive and negative predictive values of HpSA were found to be 98%, 100%, 100% and 96.5%, respectively. These findings have demonstrated that HpSA as a relatively simple inexpensive and time saving noninvasive test is a reliable method for detection of *H.pylori* infections in children.

Effect of Indoor Air Pollution During Cooking in Women Belonging to Rural, Urban and Slum Areas of Delhi

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Abstract: A study was undertaken to investigate the effect of indoor air pollution caused by different cooking fuels on the lung functions in the women of East Delhi. The cross sectional study was performed in 140 females aged 12-60 years belonging to rural, urban and slum areas of East Delhi. Data was collected by questionnaire and lung functions were measured by spirometric tests. Multiple regression analysis was performed taking functional parameters FVC, FEV₁, PEFR as the dependent variable whereas BMI, per capita household living area, kitchen area, number of ventilators, total cooking exposure, cooking fuel and smoking as independent variable. Significantly lower values of FVC & FEV₁ in population from slum samples and better results of these in rural samples were observed. However in the same group females using cowdung as cooking fuel exhibited lower values of FVC.

Introduction

Several epidemiological studies have revealed inter-relationship of poor nutritional status, repeated pregnancies and workload in making women's life quite arduous¹, especially in lower and middle socio-economic class of India. Literature survey indicates that women do spend most of their time in household mainly for cooking purposes. Various fuels used for cooking have been shown to be the major cause of indoor air pollution and the exposure of women to such hazardous environment raises grave concern². These days, use of safer modern fuels are increasing, however, a large majority of rural population still depends on traditional biomass fuels for cooking². Emission of noxious gases from incomplete combustion of these fuels, more so in poorly ventilated conditions can result in accumulation of hazardous pollutants most notably being nitrogen dioxide^{3,4} and carbon monoxide⁵ (CO) to unacceptable levels. Nitrogen dioxide tends to increase respiratory illness and CO forms carboxyhaemoglobin which even in the low concentration of 0.8% is harmful specially for people having cardiorespiratory disability by accentuating exercise induced angina. Biomass fuel has deleterious effect on pulmonary function and structure leading to obstructive and restrictive pathologies (ref). Carbo et al⁶ in their study on the effect of gas cooking on lung functions concluded that cooking gas has harmful effect on the lung functions of adolescent girls with high serum level of IgE. Similar reports suggest that subjects using gas for cooking has a significantly reduced FEV₁, compared to those using electricity for cooking. Behra D et al⁷ in their study concluded that exposure to biomass fuel and LPG affect PEFR in asthmatics and both types of fuels affect airway function and causes respiratory symptoms. Reports have also indicated increasing incidence of chronic bronchitis and cor pulmonale in nonsmoking rural women engaged in cooking⁸.

Biomass fuels are used widely in developing countries mostly in rural and poor urban areas. They are composed of complete organic matter, vegetable protein and carbohydrates incorporating carbon, nitrogen, oxygen, hydrogen and certain other elements in trace amounts². Exposure to different fuels for cooking causes high incidence of cough with expectoration, dyspnoea, lung abnormalities, chronic bronchitis, cor pulmonale and decreased lung

functions⁹⁻¹⁴. Gas stove exposure is a significant risk factor for respiratory symptoms and atopic children tended to have a greater risk of respiratory symptoms compared with non-atopic children with exposure to gas stove or nitrogen dioxide. Similarly in one of the study undetected pneumoconiosis in rural women was found to be caused by a combination of dust from maize grinding and smoke from biomass fuel¹³. In Chinese women higher lung cancer rates could be attributed to the combined effects of passive smoking and domestic use of poor quality coal¹⁴. All these findings are pointing towards one common fact that lung diseases associated with indoor smoke exposure may be asymptomatic for a prolonged period masking the extent of ill health from this cause and contributing to under-reporting with particular implication for women. Viewing all above facts we planned the present study.

Material and Methods

The study was conducted on 140 females in the age range of 12-60 years old drawn from rural slums and urban areas of East Delhi. Among these 40 were from Dilshad Garden area which is inhabited by middle socio-economic group, 50 from rural area of Gazipur with lower middle socio-economic population residing in about 2000 houses and remaining 50 from slum of Kalander Colony representing lower socioeconomic group. Out of 140 study participants 55, 51, 29, 5 and 1 were using cooking fuels such as kerosene oil, cowdung cakes, firewood and heaters respectively. Detailed questionnaire was completed on each subject to document age, sex, body mass index, smoking history and other relevant informations. Pulmonary functions such as FEC, FEV₁, and PEFR were assessed using PK Morgan portable pocket spirometer. Each subject was explained about the procedure. Measurements were made with the subject comfortably seated. After application of nose clip the subject was asked to perform appropriate respiratory maneuver and maximum of 3 observations was recorded. Body Mass Index (BMI) was calculated using following formula

$$\text{BMI} = \text{Weight} / \text{Height}^2$$

Results

Regression equation of PET against Body Mass Index (BMI) in all three groups did not show any statistical significant relationship (Table 1), on comparison of PFTs in all three groups (Table 2), significantly lower values of FVC and FEV₁ in slum sample and

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Table 1 : Regression of PFTs against BMI in total sample (N= 140).

VariableName	Dependent	(P-Value)	Significance
BMI	FVC	0.7513	Not Significant
BMI	FEV ₁	0.469	Not Significant
BMI	PEFR	0.8449	Not Significant

Table 2 : FVC, FEV₁ & PEFR in rural, slum and urban samples

PFT	Rural(n=50)		SLUM(n=50)		Uran(n=40)		(P-Value)	Significance At 5:6levelTukey'sTEST
	Mean	SD	Mean	SD	Mean	SD		
FVC	2.431	0.78	1.691	0.382	2.150	0.519	<0.0001*	Group 2 is significantly different from Group 1,3
FEV ₁	1.980	0.695	1.532	0.298	1.797	0.485	0.0002*	Group 2 is significantly different from Group 1,3
PEFR	173.980	83.403	161.820	42.063	169.050	72.132	0.6673	Not significant

*Significant p value <0.05

better results for rural samples were observed. While considering BMI<25 (normal) comparison of PFTs in three groups revealed maximum values of FVC and FEV₁ in rural females. This significance was however not noticed when BMI>25 (abnormal) is taken (Table 3). Further analyzing the rural sample, for finding a relationship between PFTs and other variables such as total household area (Table 4), kitchen area (Table 5), number of ventilators (Table 6) and total cooking exposure (Table 7), no statistical significant association was obtained. However, in the same group, the females using cowdung as cooking fuel (Table 8) exhibited lower values of FVC (Table 9) indicates that there was difference in values of PFTs in smoker as well as non-smoker (Kalander Colony females found to be smoking biri and consuming alcohol) but females in the household were the total number of smokers exceeded 3, showed decrement in their values of FVC thus reflecting the effect of passive smoking (Table 10) also.

Table 3 : PFT in normal and abnormal rural, slum and urban samples.

PFT	Rural(n=50)		SLUM(n=50)		Uran(n=40)		(P-Value)	Significance At 5:6levelTukey'sTEST
	Mean	SD	Mean	SD	Mean	SD		
For BMI < 25 (Normal)								
FVC	2.443	0.804	1.711	0.382	2.218	0.573	<0.0001*	Group 2 is Significantly different from Group 1,3
FEV ₁	2.033	0.681	1.544	0.681	1.544	0.295	0.0001*	Group 2 is significantly different from Group 1
PEFR	179.630	83.746	161.467	43.370	172.889	80.233	0.456	Not significant
For BMI > 25 (Abnormal)								
FVC	2.293	0.456	1.508	0.375	2.095	0.478	0.0286	Not significant
FEV ₁	1.370	0.622	1.424	0.340	1.761	0.422	0.1303	Not significant
PEFR	109.000	47.924	165.000	31.129	165.909	66.549	0.2376	Not significant

*Significant p value <0.05

Table 4 : Per capita household living area (square feet) and PFT in rural sample (n=60).

PFT	<50sqft		<50-99sqft		100sqft		100sq.ft		(P-Value)	Significance At 5%level Tukey'sTEST
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
	(n=36)		(n=9)		(n=2)		(n=3)			
FVC	2.5564	0.7623	2.151	0.886	2.425	0.488	1.7633	0.4114	0.7297	Not significant
FEV ₁	2.081	0.681	1.704	0.829	1.935	0.134	1.62	0.4709	0.9141	Not significant
PEFR	173.980	83.403	181.167	84.157	173.000	95.089	125	25.4558	0.5271	Not significant

Table 5 : Kitchen area (sq.ft) and PFT in rural sample (n=50)

PFT	<10sq.ft		<10-19sq.ft		20-49sq.ft		50sq.ft		No Kitchen*		(P-Value)	Significance At 5% level Tukey's TEST
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
	(n=1)		(n=5)		(n=4)		(n=6)		(n=34)			
FVC	1.640	0.000	2.300	0.798	1.945	0.451	2.2067	0.73	2.49	0.80	0.1081	Not Significant
FEV ₁	1.510	0.000	2.024	0.846	1.768	0.602	1.8067	0.65	2.04	0.71	0.8011	Not Significant
PEFR	102.000	0.000	198.600	97.961	201.250	88.323	142.1667	96.64	17.4	81.42	0.5593	Not significant

*Cooking in open space

Significance p value <0.06

Table 6 : Number of ventilators in kitchen and PFT in rural sample (n=50)

PFT	No ventilator		One Ventilator		Two or more		No Kitchen*		(P-Value)	Significance At 5%level Tukey'sTEST
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
	(n=11)		(n=3)		(n=2)		(n=34)			
FVC	2.457	0.684	1.530	0.141	2.520	1.103	2.496	0.803	0.2163	Not significant
FEV ₁	1.962	0.623	1.310	0.131	2.015	1.131	2.0429	0.7144	0.6149	Not significant
PEFR	174.000	84.175	147.333	94.368	198.500	173.241	174.3824	81.4288	0.9127	Not significant

*Cooking in open space

Table 7 : Total cooking exposure (hours) and PFT in rural sample (n=50).

PFT	One (Hr)		Two (HR)		Three (HR)		Four (HR)		(P-Value)	Significance At 5%level Tukey'sTEST
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
FVC	2.784	0.814	2.466	0.798	2.647	0.826	1.9608	0.5006	0.0977	Not significant
FEV ₁	2.152	0.565	2.101	0.727	2.197	0.818	1.5308	0.4848	0.2007	Not significant
PEFR	207.400	95.931	205.167	73.097	173.111	72.757	105.6923	54.4187	0.0526	Not significant

*Significant P value <0.05

Table 8 : Main cooking fuel and PFT in rural sample (n=50)

PFT	LPG		Kerosene Oil		Cowdung/Cakes		Firewood		(P-Value)	Significance At 5%level Tukey'sTEST
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
	Group-1		Group-2		Group-3		Group-4			
FVC	2.415	0.575	2.633	0.8884	2.0636	0.719	2.483	0.416	0.0032	Group 1 is significantly different from group-3
FEV ₁	1.922	0.603	2.222	0.777	1.751	0.685	1.9533	0.5689	0.5278	Not significant
PEFR	187.429	90.427	186.389	80.387	139.143	73.686	220.6667	102.9093	0.4254	Not significant

Table 9 : Smoking and PFT in rural sample (n=50)

PFT	Smoker		Non Smoker		(P-Value)	Significance At 5%level Tukey's TEST
	Mean	SD	Mean	SD		
FVC	2.590	0.825	2.590	0.752	0.0581	Not significant
FEV ₁	2.009	0.756	1.963	0.670	0.3872	Not Significant
PEFR	153.278	76.454	185.625	86.028	0.1847	Not Significant

*Significant p value <0.05

Discussion

With increasing attention focused on women's health, the need for more of gender specific data is being felt. Our work is one such attempt to fill the existing information gap and demonstrates the

Table 10 : Number of smokers in the household and PFT in rural sample (n=50).

PFT	None		One		Two		Three or more		(P-Value)	Significance At 5% level Tukey's TEST
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
	Group1		Group2		Group3		Group4			
FVC	2560	0.840	2256	0.707	2475	0.716	398	0	0.0254	Group Disignificantly different from group abc
FEV ₁	2139	0.696	1901	0.663	1910	0.839	172	0	0.742	Not significant
PEFR	195.235	89.325	170.375	80.628	147.625	81.085	110	0	0.3246	Not significant

Significant p value <0.05

influence of contribution of indoor environment towards the health of women. As women are largely carry out most of the cooking, it is they who are maximally exposed to the hazardous indoor environment resulting mainly from the cooking fuel emission specially in under ventilated homes^{13,14}. Though LPG is commonly used fuel, but traditional biomass ones continued to be used in poor slum areas Besides this the jhuggi clusters in these slum areas contribute to poor ventilation. The reliance on biomass fuels coupled with poor ventilation is adequate to explain the low values of FVC and FEV₁ noticed in slum samples. The tobacco smoking which is quite prevalent in slum population may also contribute towards these results. The values of FVC and FEV₁ where maximum in rural women, may be attributed to cooking in open air or in well ventilated conditions. Another factor which appears to be adversely affecting the results, was passive smoking. Women in households having 3 or more smokers showed significant lower values of FVC. This reflects the combined effects of passive smoking¹⁵ and domestic cooking on reported decrement in the values of FVC. Our study strengthening the need for adequate control of environment, proper ventilation combined with health education of women as a move to remove the associated health risks.

Conclusion

The study of varied indoor environment prevailing in the rural, slum and urban areas due to use of different cooking fuels and its probable impact on lung functions was attempted by us in the present study. Pulmonary functions were measured using portable pocket spirometer and increased FEV₁ were observed in rural females as compared to urban population. Though we did not find any significant relationship between PFT and other variables such as total household area, kitchen area and smoking habits but lower values of FVC were seen amongst rural women using cowdung as cooking fuel.

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Defect in Membranous Ossification of the fossa for Lacrimal Sac and Frontonasal Ostium

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Abstract: Defect in the fossa for lacrimal sac and nasolacrimal canal was encountered on the left side of a dry female skull age about 40-50yrs. A bony crest was present at the anterior border of lacrimal bone and a thin membranous bone except for a minute canal communiating with nasolacrimal canal closed the bottom of the fossa for lacrimal sac. The left frontal air sinus had neither an ostium nor a frontonasal canal to communicate with the middle meatus of nasal cavity. As the frontal, lacrimal and maxillary bones developed by membranous ossification; these defects may be attributed to the defect in membranous ossification.

Introduction

Fossa for lacrimal sac is formed by the articulation of the frontal process of maxilla and lacrimal bone. It lies between the anterior lacrimal crest in the frontal process of maxilla and posterior lacrimal crest in the lacrimal bone. Below it continues with nasolacrimal canal, which is about 1cm in length and formed by the groove present on the nasal surface of maxilla making 2/3rd of the circumference and the remaining part by the descending part of lacrimal bone and lacrimal process of the inferior nasal concha¹.

Frontal air sinus is situated between the inner and outer tables of the frontal bone. Externally it represents a triangular area posterior to the superciliary arch. The two sinuses are rarely symmetrical and separated by a thin bony septum. Rudimentary at birth, well developed by the age of 7 or 8 yrs, it reaches its full size only after puberty. Each sinus communicates with the middle meatus of the nasal cavity of the corresponding side by frontonasal canal¹.

Case Report

A rare feature of bony stenosis at the junction of fossa for lacrimal sac and nasolacrimal canal on left side due to the developmental defect of left lacrimal and maxillary bone was observed in a skull belonging to a female age about 40-50 yrs. in the Department of Anatomy, Regional Institute of Medical Sciences, Imphal, Manipur. A small canal of about 2mm in diameter communicated the nasolacrimal canal and the fossa for lacrimal sac. The left lacrimal bone articulated in front with the posterior border of frontal process of maxilla and featured a crest from its anterior border. This crest articulated in front with a thin lamellar bone that filled the fossa for lacrimal sac and laterally with the orbital surface of maxilla. The fossa for lacrimal sac was small, but there was a larger fossa between the crest and the posterior lacrimal crest. The part of the bone behind the posterior lacrimal crest was comparatively smaller. There was no lacrimal hamulus (Fig.1).

The left maxilla was normal for all the fetures except for the thin lamellar bone, which fused with maxilla and filled the fossa for lacrimal sac. The thin lamellar bone fused laterally with the orbital surface, anteriorly and medially with the frontal process and posteriorly with the abnormal crest of lacrimal bone; and formed the floor of the fossa for lacrimal sac. The narrow canal of about 2mm in diameter presented in the lamellar bone communicated the fossa for lacrimal sac and the nasolacrimal canal. The rest of the nasolacrimal canal was normal.

Both the frontal air sinuses were within the normal limit in size, even though the left sinus was comparatively less convoluted. There was no frontonasal canal or ostium on left side. The septum between the two sinuses was incomplete and was not more than a low ridge on the floor at the junction of the two sinuses (Fig.2).

Discussion

Dacryostenosis is 4 times more common in women and it has been attributed to soft tissue infection and tissue fibrosis rather than actual bony stenosis². However; it has been reported that women have significantly smaller dimension in the lower nasolacrimal fossa and this factor may explain the increased prevalence of acquired nasolacrimal duct obstruction in females³. Coincidentally, this skull

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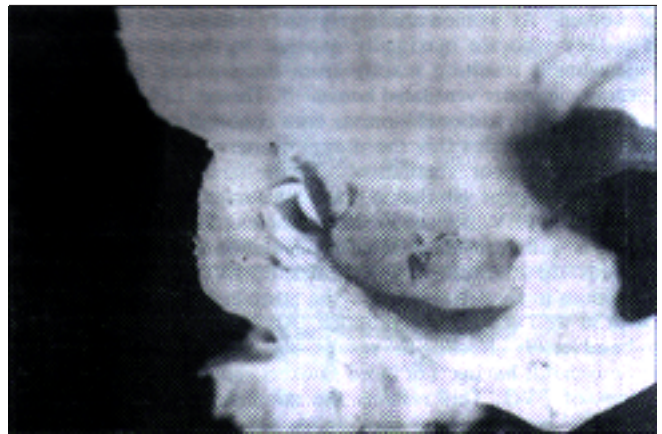


Fig.1 : Shows the medial wall and floor of the orbit. Arrow pointed to the abnormal crest of the lacrimal bone.

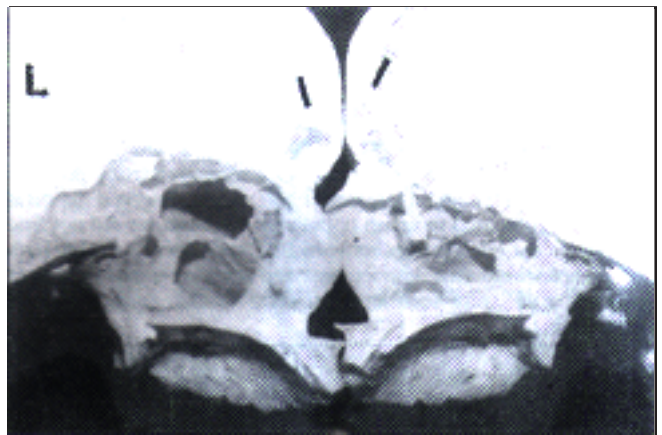


Fig.2 : Hemi-section of the skull showing the two frontal air sinuses (arrowed). The probe passes through the Frontonasal Ostium on the right side, which is absent on left side

also belongs to a female. Report is also available describing smaller nasolacrimal canal as one of the etiologic factors in primary acquired nasolacrimal duct obstruction⁴.

In the present case, the defects are present in the fossa for lacrimal sac as well as the junction of the lower part of fossa for lacrimal sac and upper part of the nasolacrimal canal. These defects are due to the developmental defects of lacrimal and maxillary bones. As both the bones are developed by membranous ossification i.e. lacrimal bone - 12th wk of intrauterine life, maxillary bone - 6 wk of intrauterine life¹, the defects may be attributed to the membranous type of ossification. Several types of developmental defects of lacrimal bone had been reported viz: this bone is frequently

perforated or occasionally represented by several ossicles or completely absent⁵; the hamulus of the lacrimal bone may be separate, double or absent⁶. But the presence of a crest at its anterior border thereby causing the defect of the fossa for lacrimal sac and nasolacrimal canal has not been reported.

Clinically, the individual to whom this skull belongs could have suffered from epiphora. Then the operative measure could have been Dacryocystorhinostomy but not Dacryocystoplasty⁷.

Frontal air sinus develops during 4th month of intrauterine life from (i) anterior ethmoidal cell within the ethmoidal infundibulum; in this situation (comprising 50%) frontal air sinus opens into the ethmoidal infundibulum, (ii) anterior ethmoidal cell - in this situation frontal air sinus opens into the middle meatus of nasal cavity by frontonasal canal (iii) most rarely, from the anterior part of frontal recess of frontal furrow - with only frontonasal ostium without a canal⁸.

In the present case, the right frontal air sinus opens by an ostium, which is absent on left side. From the developmental point of view, these frontal air sinuses are developed from the anterior part of frontal recess of frontal furrow and failed to develop an ostium on left side. As the frontal bone develops by membranous type of ossification¹, this defect also may be attributed to the defective membranous type of ossification.

Clinically, the individual could have suffered from left frontal sinusitis⁹, the treatment of choice could have been trephination or osteotomy or other surgical measures depending upon the severity.

Even though, defects in other membranous bones, foramina or canals could not be observed; all the bones involved in these defects developed by the membranous ossification and these defects may be attributed to this type of ossification defects.

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Miglitol

Miglitol is an alpha-glucosidase inhibitor. The chemical name of miglitol is 3,4,5-piperidinetriol, 1-(2-hydroxyethyl)-2-(hydroxymethyl)-, [2R-(2(alpha), 3(beta), 4(alpha), 5(beta))]. The empirical formula is C₈H₁₇NO₅. Its molecular weight is 207.2

Pharmacodynamics: Miglitol is a 1-deoxynojirimycin derivative which reversibly inhibits intestinal alpha-glucosidase enzymes responsible for the digestion of carbohydrates to absorbable monosaccharides. Its structure resembles that of glucose, and unlike acarbose (a pseudotetrasaccharide alpha-glucosidase inhibitor), it is almost completely absorbed in the upper section of the small intestine. The rank order of its inhibitory activity is sucrase > glucoamylase > isomaltase > lactase > trehalase. Although miglitol delays carbohydrate absorption in healthy volunteers, there are no significant losses of carbohydrates, protein or fat in the faeces and no significant caloric losses.

Miglitol smoothes postprandial glycaemic peaks thereby reducing postprandial peak plasma glucose levels in patients with type 2 diabetes mellitus; miglitol reduces postprandial serum insulin levels or C peptide concentration and serum T9 level after 8 weeks' treatment.

Since miglitol is almost completely absorbed, it has been suggested that it may exert extraintestinal effects on glucose homeostasis. Miglitol significantly reduces the postprandial increase in gastric inhibitory polypeptide in healthy volunteers and patients with type 2 diabetes mellitus. It also increases peptide tyrosine-tyrosine (PYY) and motilin levels. Miglitol, unlike several other compounds with cationic polarity, e.g. biguanides, has no effect on sodium-dependent small intestine transport of organic solutes, such as, hexoses.

Pharmacokinetics: Miglitol 25mg is completely absorbed (100% bioavailability); however, only 50% to 70% of a 100-mg dose is absorbed. At high doses, miglitol absorption is saturable. The mean peak plasma concentrations (C_{max}) following single oral doses of miglitol 25, 50 and 100 mg were 0.78, 1.22 and 1.86mg/L, respectively, and are attained within 2 to 4 hours (T_{max}). The protein binding of miglitol is negligible (< 4.0%). Miglitol has a volume of distribution of 0.18L/kg. Oral miglitol is excreted predominantly unchanged in urine. Thus, following a 25-mg dose, over 95% of the dose is recovered in the urine within 24 hours. At doses above 25mg, less drug is recovered in urine because of incomplete bioavailability. The plasma elimination half-life is approximately 2 hours.

Special population: Renal insufficiency: Patients with a creatinine clearance (Cr Cl) less than 25mL/min had a > 2-fold increase in miglitol plasma levels compared to patients with a CrCl greater than 60mL/min; the dosage was 25mg 3 times daily. Little information is available on the safety of miglitol in patients with creatinine clearance < 25mL/min. **Hepatic insufficiency:** Miglitol pharmacokinetics was not altered in cirrhotic patients relative to healthy control subjects. Since miglitol is not metabolized, no influence of hepatic function on the kinetics of miglitol is expected.

Indications and Usage: Miglitol as monotherapy is indicated as an adjunct to diet and exercise to improve glycaemic control in patients with Type 2 Diabetes Mellitus.

Drug Profile

Contraindications: (1) Diabetic ketoacidosis (2) Chronic intestinal diseases (3) Hypersensitivity to miglitol (4) Inflammatory bowel disease or other conditions which may deteriorate with increased gas formation in the intestine (5) Intestinal obstruction.

Precautions: Hypoglycemia: Because of its mechanism of action, miglitol when administered alone should not cause hypoglycemia in the fasted or postprandial state. Because miglitol given in combination with a sulfonylurea will cause a further lowering of blood glucose, it may increase the hypoglycemic potential of the sulfonylurea. Oral glucose (dextrose), whose absorption is not delayed by miglitol, should be used instead of sucrose (cane sugar) in the treatment of mild-to-moderate hypoglycemia.

Renal Impairment: Patients with a creatinine clearance (CrCl) less than 25 mL/min had a 2-fold increase in miglitol plasma levels compared to patients with a CrCl greater than 60mL/min; the dosage was 25mg 3 times daily.

Carcinogenesis, mutagenesis, impairment of fertility: No evidence of carcinogenesis was observed in animal studies with miglitol. No evidence of mutagenicity was observed in vitro in bacterial mutagenesis (Ames) assay and the eukaryotic forward mutation assay (CHO/HGPRT). No evidence of clastogenicity was observed in vivo in the mouse micronucleus test. In oral fertility studies of miglitol conducted in rats, there was no evidence of reproductive toxicity at doses up to 300 mg/kg/day in male or female rats (~8 times the maximum human exposure based on body surface area).

Miglitol should be used during pregnancy only if the potential benefit justifies the risk to the fetus. It is recommended that miglitol not be administered to a nursing woman. Safety and effectiveness of miglitol in pediatric patients have not been established.

Drug Interactions: Concomitant administration of miglitol with digoxin, propranolol or ranitidine reduced their absorption, and thus the dose of these agents may require adjustment.

Adverse Reactions: The most common adverse events in miglitol-treated patients involve the gastrointestinal system and include flatulence, abdominal pain and diarrhoea. Symptoms are usually mild to moderate in intensity, dose dependent, occur at the onset of treatment, decline with time and resolve promptly on discontinuation of the drug or with dosage adjustment. Compared with placebo, miglitol showed no significant effects on renal, cardiovascular, respiratory or haematological functions in long term clinical studies (6 months to 1 year).

Dosage and Administration: Adult dose: There is no fixed dosage regimen for miglitol. The maximum recommended daily dosage is 100mg 3 times daily, with treatment initiated at a dosage of 25mg 3 times daily and gradually increased. The recommended maintenance dose is 50mg 3 times daily. No dosage adjustments are required in elderly patients, in those with hepatic impairment or those with mild to moderate renal insufficiency. Miglitol is not recommended in patients with significant renal impairment (serum creatinine > 2mg/dL).

Altitude Related Disorders and Their Management

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Abstract: Altitude related medical problems cause significant avoidable morbidity and mortality. Acute mountain sickness, high altitude pulmonary and cerebral edema are potentially serious disorders. Many high altitude places are remotely located and away from medical help. It is imperative for persons travelling to such places to be able to recognize symptoms of common problems and manage them accordingly. The potentially serious consequences of these diseases and poor effectiveness of treatment modalities underscore the need for prevention. The preventive measure are screening to identify subjects at risk and providing information to mountaineers. This review aims to highlight common altitude related illnesses, their aetiologies and current management. The effect of high altitude in special high risk population is also discussed in this review.

Introduction

Mountains have fascinated and attracted mankind for millennia. Most peaks in the Alps had been climbed by the end of the 19th century. Some early climbers mentioned experiencing the symptoms now described as mountain sickness. By the beginning of the 20th century, hypoxia was known to be the main cause of these symptoms. Even today, many questions regarding the precise mechanism of altitude illness remain unanswered.

A multitude of problems is associated with ascent to altitude. Some of these are merely an annoyance while others are life threatening. Fundamentally, all are caused by a lack of oxygen. However, in most cases, considerable uncertainty exists regarding to the precise pathophysiology of these illnesses. Three major *syndromes*, acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), and high-altitude cerebral edema (HACE), are now commonly accepted. Other related problems, such as impaired sleep at high altitude, often coexist with the major syndromes and also deserve mention. Finally, the effects of ascent on certain special populations are discussed in this article.

Acute Mountain Sickness

Acute mountain sickness (AMS) is a condition affecting lowlanders 6-90hrs after rapid ascent to altitude. It is characterized by lethargy, insomnia, headache, nausea, vomiting and dyspnoea. In severe forms cyanosis, crepitation in the lungs, papilledema and other signs of cerebral edema are also features. A consensus conference was held during the 1991 Hypoxia and Mountain Medicine Symposium at Lake Louise, Canada to define the various altitude syndromes¹. This group defined AMS as follows: "In the setting of a recent gain in altitude, the presence of headache and at least one of the following symptoms: gastrointestinal (anorexia, nausea or vomiting), fatigue or weakness, dizziness or lightheadedness, difficulty in sleeping." AMS is defined by its symptoms, but the exact cause of AMS is still unknown; cerebral edema may play a role.

Many factors affect the incidence and severity of AMS, such as the rate of ascent, altitude attained (especially altitude of sleep), duration of exposure to altitude, and amount of exercise undertaken at altitude. The most important and least understood variable is the underlying physiological susceptibility of the individual. Few people experience significant symptoms below 7000-8000 ft (2130-2440m), whereas portable hyperbaric bags (eg, Gamow bag) simulate descent to a lower altitude. These bags are effective for treating AMS, although they are rarely needed unless AMS is

complicated with high-altitude cerebral or pulmonary edema.

The exact mechanism by which hypoxia causes AMS is still unknown, although hypoventilation probably is important. The role of fluid retention as a cause of AMS remains uncertain. Antidiuretic hormone and atrial natriuretic secretion factor are altered in AMS, suggesting that CNS changes that cause secretion of hormones promoting fluid retention may be important⁷. Sutton and Lassen⁸ suggested that hypoxia stimulates increased cerebral blood flow, resulting in vasogenic cerebral edema. Multiple factors lead to the development of AMS, but the exact role each of these plays in the development of this disorder is unknown.

Sleep at High Altitude : Most newcomers to altitude frequently report difficulty in sleeping at night, even in the absence of other symptoms. Sleep disruption at altitude results from a combination of many factors, including the cold windy environment and the often-crowded sleeping conditions, in addition to hypoxia periodic breathing during sleep causes further disruption of sleep continuity. At extreme altitude, loss of sleep is nearly complete, further compromising the energy of already exhausted climbers.

Frequent nighttime awakenings and arousals represent the major disruptors of high-altitude sleep. The Operation Everest II (OEII) decompression chamber study provided an opportunity to monitor changes in sleep across various altitudes up to an altitude equivalent to the South Col of Mount Everest (approximately 8040m, barometric pressure 282 mm Hg). These studies found severe sleep fragmentation and periodic breathing (with central sleep apneas) at all altitudes studied but especially at the highest altitudes. These brief 2- to 5-second arousals from sleep (not full awakenings) increased from an average of 22 + 6 times per hour at sea level to 161 + 66 times per hour at 25,000 ft (7620m, 282mm Hg)⁹.

Periodic breathing is a common breathing pattern during sleep at high altitude. Changes in sleep site, as well as conflicting effects of hypocapnia and hypoxia on the peripheral chemoreceptors, lead to a most unacclimatized person ascending to 10,000 ft (3050 m) or higher experience at least a few symptom².

Treatment and Prevention of AMS : Slow, gradual ascent with adequate time for acclimatization provides the best protection from AMS. The ideal ascent rate varies based on individual susceptibility to AMS. Once symptoms of AMS occur, additional time for acclimatization before ascending further usually is the only treatment needed for mild AMS. If symptoms worsen despite additional time for acclimatization, descent to a lower altitude (especially sleeping altitude) is needed. A descent of 1000-3000 ft (300-900m) usually is sufficient to ameliorate symptoms. Supplemental oxygen, although rarely available in sufficient quantities, also effectively relieves symptoms of AMS³.

Pharmacological Treatment of AMS : Acetazolamide (Diamox)

is effective both for the prevention and for the treatment of AMS. Forwand et al⁴ (1968) demonstrated that 250mg of acetazolamide every 8 hours dramatically reduced symptoms of AMS compared to people taking a placebo during a stay at the 12,800-ft (3900-m) summit of Mount Evans, Colorado. Others have confirmed these findings, and Sutton et al⁵ found that acetazolamide decreased hypoxemia during sleep by reducing the amount of periodic breathing. The mechanism of action of acetazolamide in AMS is unclear. The drug is a carbonic anhydrase inhibitor that causes a bicarbonate diuresis, resulting in metabolic acidosis. It also decreases production of cerebrospinal fluid. However, these actions do not adequately explain the effectiveness of acetazolamide in AMS. Current recommendations are 125-250 mg twice daily starting one day before ascent and continuing for a couple of days at altitude or even for the duration of stay at altitude. Smaller doses may be effective in some people.

Dexamethasone, 2-4 mg every 6 hours, is also effective in preventing and treating AMS. The mechanisms of action of dexamethasone in relieving AMS symptoms are unknown. Its relative effectiveness compared to acetazolamide has not been established, but it likely is equivalent to acetazolamide⁶ destabilization of the respiratory control system, which is responsible for the periodic breathing observed at high altitude¹⁰. Much of the sleep disruption at high altitude has been attributed to periodic breathing. Transient arousals from sleep commonly occur at the onset of the hyperpneic phase of periodic breathing. Nearly one half of the apneic episodes observed in the OEII study were not associated with electroencephalogram (EEG) arousals. Thus, a complex interplay exists among sleep state ventilatory responsiveness, breathing pattern, and sleep fragmenting arousals⁹.

Nighttime arterial oxygen saturation is lower than daytime (awake) values and thus represents the most profound hypoxic insult during a high-altitude sojourn. The mean arterial oxygen saturation (SaO₂) at night during the OEII studies at 25,000 ft (7620m) was only 52±2% compared with a daytime SaO₂ of 71±7%. The lower nighttime SaO₂ may, in part, result from periodic breathing, although others have suggested that periodic breathing actually improves nighttime SaO₂. Periodic breathing appears to be a risk factor for high altitude illness, and carbonic anhydrase inhibitors (eg, acetazolamide) decrease nocturnal periodic breathing, improve arterial oxygen saturation, and ameliorate daytime symptoms of AMS¹¹.

High-Altitude Pulmonary Edema (HAPE)

High altitude pulmonary edema (HAPE) is a serious and potentially life-threatening manifestation of altitude illness. The first symptoms of HAPE occur 1-3 days after arrival at altitude. In adults, these symptoms commonly occur after exercise and consist of cough, shortness of breath, chest tightness, and fatigue. In approximately half the cases, these symptoms are associated with the typical symptoms of AMS. Initially, cough is nonproductive, but thin, clear, or yellowish sputum is later produced. In some cases, the sputum is tinged with blood. Fatigue may be the first symptom, occurring even before dyspnea develops and manifesting as the inability of the affected individual to maintain the pace of the group. Physical findings in HAPE include cyanosis, temperature as high as 101°F (38.5°C, a higher fever creates suspicion of pneumonia), flat neck veins, and crackles over the mid chest. Heart and respiratory rates are increased. The incidence of HAPE is affected by factors such as rate of ascent, age, sex, physical exertion, and, most importantly, individual susceptibility¹².

A form of HAPE known as reascent HAPE or reentry HAPE occurs in acclimatized individuals who descend to lower altitude and then reascend. In these cases, individuals usually spent 3-5 days or as many as 10-14 days at low altitude before returning to higher elevations. For unknown reasons, these individuals have

an increased likelihood of developing HAPE.

Diagnosis : The diagnostic criteria for HAPE are at least 2 symptoms and 2 signs from the following list, in the setting of a recent gain in altitude :

* *Symptoms* (at least 2) - Dyspnea at rest; - Cough; -Weakness, decreased exercise performance; - Chest tightness or congestion

* *Signs* (at least 2) - Rales or wheezing in at least one lung field; - central cyanosis; - tachypnea; - tachycardia.

A chest radiograph, if facilities are available, and a measurement of arterial oxygen saturation may contribute to making the diagnosis and excluding other disorders. Marked hypoxemia is an important and common finding in HAPE.

Radiographic features : With HAPE, homogeneous or patchy opacities appear in the mid lung areas and involve one or both sides of the chest. Opacities are more likely to be present in the right lung than in the left lung. Unilateral involvement of only the left lung is rare and should raise the suspicion of a congenital absence or hypoplasia of the right pulmonary artery. The pulmonary arteries frequently are enlarged; however, the cardiac silhouette usually is normal. Kerley lines may or may not be present.

Pathophysiology : A clear understanding of the precise etiology of HAPE and the mechanism for its development is hampered by the lack of a good animal model. Any hypothesis must account for several factors, as follows : (1) elevated pulmonary artery pressures with wedge and left atrial pressures within the reference range, (2) no evidence of left ventricular failure, (3) capillary and arterial thromboses (in many fatal cases of HAPE), and (4) intense exercise (makes HAPE more likely, while bedrest is beneficial).

Hypoxic pulmonary vasoconstriction occurs, to some extent, in everyone who ascends to high altitude, however, the level of vasoconstriction is highly variable. Individuals with HAPE have more severe pulmonary hypertension than is usual at altitude, but not everyone with pulmonary hypertension of similar severity develops HAPE.

Hultgren¹² proposed the *overperfusion concept* as the mechanism for developing HAPE. This overperfusion mechanism postulates that uneven pulmonary vasoconstriction results in lung areas with decreased blood flow while other areas receive excessive flow. These overperfused lung areas are where the proposed leakage of edema fluid occurs. Bronchoalveolar lavage studies show that the edema fluid in HAPE has a high protein concentration, along with various inflammatory markers, such as complement C5a and leukotriene B₄. West et al¹³ suggested that HAPE results from a rupture of pulmonary capillaries subjected to high wall stresses from high pressure in the vessels. The nonhomogeneous vasoconstriction proposed by Hultgren would allow high pulmonary artery pressures to be transmitted to pulmonary capillaries in overperfused areas of the lung.

Treatment of HAPE : Both the overperfusion and stress failure models for HAPE imply that a reduction of the excessive hypoxic pulmonary vasoconstriction is critical for the treatment of HAPE. Oxygen and descent to low altitude both result in lowered pulmonary artery pressure. Rapid descent to lower altitude results in dramatic symptomatic improvement. Often, a descent of only 1000-3000 ft (300-900m) is necessary. Thus, descent is the most important therapeutic modality. Early descent, before HAPE becomes severe, potentially can save more lives than any other treatment.

Use of supplemental oxygen reduces pulmonary artery pressure; however, sufficient quantities of oxygen are rarely available under field conditions, precluding reliance on oxygen alone. Nifedipine and other vasodilators also are useful in treating HAPE. Patients with HAPE who were treated by Oelz et al¹⁴ with 10mg nifedipine followed by 20mg of slow-release nifedipine every 6 hours showed

improvement in oxygenation and overall condition, even without descent to lower altitude. Oter vasodilators may also decrease pulmonary artery pressure and be useful in treating HAPE. Reliance on these medications should not delay early and rapid descent.

Portable hyperbaric bags (eg, Gamow bag) are now available. These fabric hyperbaric chambers increase the pressure approximately 2 pounds per square inch (PSI), ie 103mm Hg, simulating descent, which is effective in treating HAPE¹⁵.

The best treatment is prevention of HAPE by gradual ascent and early recognition of HAPE symptoms; nifedipine is useful in preventing HAPE among susceptible individuals.

High Altitude Cerebral Edema (HACE)

High altitude cerebral edema (HACE) is an extreme form of mountain sickness. The lake louise definition¹ states that HACE "can be considered 'end stage' or severe AMS. In the setting of a recent gain in altitude, [HACE is] the presence of a change in mental status and/or ataxia in a person with AMS, or the presence of ataxia in a person without AMS." Without prompt treatment, further neurological deterioration and death are likely.

Signs and symptoms of HACE may progress rapidly (within 12h) from minimal manifestations to coma. Typically, this progression occurs slowly. Often the symptoms of HACE begin at night, occasionally resulting in a loss of consciousness during sleep. Most cases of HACE occur after individuals have been at altitude for several days.

The pathophysiology of HACE shares many similarities with the pathophysiology of AMS. Despite similarities, the reason only a few persons with AMS develop HACE is unclear. MRI in patients with HACE shows edema of the white matter, especially in the corpus callosum. Hansen et al¹⁶ suggested that cytotoxic cellular edema of the brain from hypoxia caused many of the signs and symptoms of both AMS and HACE. Lassen¹⁷ however, suggested that HACE was caused by vasogenic edema resulting from increased cerebral blood flow, causing leakage of fluid into the brain. Recently, Severinghaus¹⁸ has proposed roles for angiogenesis, osmotic swelling and ischemia in the pathogenesis of HACE.

Treatment : Mild cases of AMS do not require descent to lower altitude, whereas HACE may be lethal if not recognized and promptly treated; thus, early recognition of HACE is crucial. A change in the level of consciousness or the onset of ataxia requires immediate descent.

Supplemental oxygen, if available, should be administered along with dexamethasone at 4-8 mg initially and then 4mg every 6 hours thereafter. Diuretics, such as furosemide and mannitol, should not be administered because they may result in orthostatic hypotension from decreased intravascular volume, which makes descent difficult or impossible.

Early use of a hyperbaric bag (ie, Gamow bag) may relieve symptoms and make descent easier but should not be considered a substitute for descent, especially because recovery often requires 10 or more days, even with treatment at low altitude.

Special population at High Risk

Large numbers of individuals go to high altitudes for work and recreation, and some individuals have special medical problems. Despite similarities to altitude illness in healthy individuals, ascent to high altitude by person with underlying cardiac disease, pulmonary disease, and sickle cell anemia deserves special mention.

Coronary Artery Disease

Unacclimatized persons with coronary artery disease may develop increased anginal symptoms following ascent to altitude because of an increase in cardiac work, as well as possible vasoconstriction

of the coronary arteries. Cardiac arrhythmia, including atrial fibrillation or flutter, may worsen after rapid ascent to altitude, even without underlying coronary artery disease¹⁹. During exercise testing at 10,150ft (3100 m), cardiac patients developed angina or ST segment depression at the same double product (ie, heart rate times systolic blood pressure) as they did at 5280 ft (1600m). Thus, ascent to altitudes of 10,000 ft (approximately 3000m) has little direct effect on myocardial ischemia but may produce symptoms by increasing heart rate and blood pressure during submaximal exercise²⁰.

Despite the increase in cardiac symptoms following rapid ascent to high altitude, the increased risk for cardiac death is low. Hultgren¹⁹ reviewed the effects of altitude on patients with cardiovascular disease and suggested an approach (including when to perform a pre-ascent exercise test) for the evaluation of a patient with heart disease prior to trekking at high altitude.

Pulmonary Disease

Chronic obstructive pulmonary disease : Shortness of breath occurs in everyone, including those without heart or lung disease, after ascent to altitude. Even at sea level, patients with COPD frequently are limited by impaired lung mechanics and dyspnea.

Because of the increased ventilatory requirements of exercise at altitude, patients with COPD may experience a worsening of their symptoms during exposure to altitude. Patients with COPD without evidence of cor pulmonale were exposed to 6300-ft (1920-m) altitude by Graham and Houston²¹. These patients developed few altitude-related symptoms except fatigue (and headache in one individual), despite a decrease in resting arterial partial pressure of oxygen (PO₂) from 66 to 52mm Hg. In these patients, the authors attributed the lack of symptoms of AMS to partial acclimatization resulting from hypoxemia. They concluded that patients with mild or moderate COPD without cor pulmonale tolerate altitude exposure quite well. Patients with COPD living at altitude, as opposed to sojourners, develop cor pulmonale and have an increased mortality rate when compared to similar patients living at low altitude. Although the cause for this increased mortality rate is unknown, it probably is related to the higher pulmonary artery pressure observed in these residents²².

Pulmonary hypertension : Hypoxic pulmonary vasoconstriction raises pulmonary artery pressure in sojourners to high altitude. With primary pulmonary hypertension, ascent to altitude results in even higher pulmonary artery pressures. These patients are likely to experience additional symptoms, such as fatigue, dyspnea, or even syncope. An increase in supplemental oxygen or the use of pulmonary vasodilators may be helpful to ameliorate altitude symptoms.

Asthma : The dry, cold air often encountered at high altitude may cause bronchoconstriction; however, this climate also contains fewer allergens. As a result, many people with asthma report doign as well ro even better at high altitude than at lower elevations. The reduced barometric pressure results in decreased air density. Thus, even though the ventilatory demands of activity at high altitude are reater, the reduced air density at least partially compensates. Patients with asthma who want to travel to high altitude should be encouraged to do so, but they should bring an adequate supply of their medications and pay attention to their respiratory symptoms.

Sickle Cell Disease

Many genetic variations occur in the hemoglobin molecule. A far more common hemoglobinopathy occurs in individuals with sickle cell disease and makes ascent to high altitude inadvisable. Sickle cell disease refers to several types of abnormal hemoglobins, including hemoglobin AS and hemoglobin S. Under conditions of hypoxia, the red blood cells in these individuals become deformed and take on the shape of a sickle, causing blood viscosity to

increase, cells to clump together more readily, and microcirculation to become blocked. The concentration of hemoglobin S in the circulation is the major determination of sickling. Bone pain and splenic infarction may occur²³.

Most of these individuals have sickle cell trait and are largely asymptomatic, while a few have a far more severe condition, sickle cell anemia. Those with sickle cell anemia probably already know about their disease, but those with only sickle cell trait may be unaware of the problem and, therefore, are more likely to go to high altitude and experience problems.

Exposure to the hypoxia at high altitude may precipitate a sickle cell crisis among those patients with sickle cell anemia. These individuals should not attempt to go to high altitude. Even the modest hypoxemia associated with airline travel may precipitate symptoms in susceptible individuals.

- Consider providing supplemental oxygen to those individuals with sickle cell anemia during aircraft flights. Travel by commercial airline generally is safe for patients with sickle cell trait, however, rarely, they may experience symptoms during airplane flights. Similarly, those with sickle cell trait generally tolerate altitudes of 8000-10,000 (2440-3048m) without difficulty, although a few may become symptomatic.

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JIMSA BEST PUBLISHED ARTICLE AWARDS

The following three (3) original articles published during the years 2003 & 2004, have been awarded the 'JIMSA Best Published Article Award' as per ranking given below.

Rank I : *Effect of Electromagnetic Field on Sertoli Cell of Rat Testes : A Light and Transmission Electron Microscope study* - Amir Afshin Khaki, Rewa Choudhry, J.K. Kaul, Bagher Minaii, Ali Baybordi, Gholamreza Oskuii, Maghsoud Kafshnouchi. H. Montazam. Vol.17, page 136-140, 2004.

Rank II : *Sural Artery Flap : A Dependable Solution in Lower Leg and Foot Soft Tissue Reconstruction* - G.N. Sharma, Nepnam Sanjib Singh. A Vol.16, page 191-193, 2003.

Rank III : *Oxidative stress in Menopause with or without hormone supplementation with special reference to Bone Mineral Density* - Abha Sarkar, Purvita Dam, G.C. Sarkar. Vol. 17, page 131-133, 2004.

The award consists of a medal, citation and cash prize; will be given to the author/co-author (age below 45 years). The awardee may receive the award at the forthcoming IMSA conference at Jaipur (Contact Dr. IPS Kalra, Secretary General IMSA)

Editor

Sister Chromatid Exchange : A Useful Tool for Genetic Screening

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Introduction

Man is continuously exposed to a variety of natural and synthetic pollutants. The aim of upgradation of technology and industrialization is to provide a comfortable lifestyle and hygienic environment, though the plethora of chemicals introduced in man's environment is rising alarmingly. These industrial chemicals have cytotoxic, clastogenic, mutagenic, teratogenic and carcinogenic effects on the health of the industrial workers. These toxic agents may cause mutations of germ cells resulting in accumulation of heritable abnormal genes or may lead to mutations of somatic cells resulting in formation of malignant tumours. Chromosomal damage constitutes a set of efficient and reliable criteria to measure genetic toxicity¹.

There are two types of **cytogenetic test systems** :

1) Classic Chromosome Aberration Measurement and the more recently. 2) Sister Chromatid Exchange.

The cytological features of chromosomal aberrations are too complex to learn and need prolonged training for accurate interpretations.

Sister chromatid exchange (SCE) is preferred as a sensitive and convenient test for routine chemical mutagenicity; it gives objective results². SCE basically represents reciprocal exchanges of homologous segments between sister chromatids, first demonstrated by Taylor³ in autoradiographic studies of plant chromosomes.

Perry and Wolff⁴ developed the staining procedure named Fluorescence plus Giemsa staining which provided permanently stained heterochromosomes in which SCE's could be counted and scored.

Rationale for analysis of SCE in Humans

Being well aware that various natural and synthetic agents can modify the genetic material leading to mutational changes, determination of SCE frequency attains paramount importance in detecting these early changes. SCE is the cytological manifestation of a four strand exchange in the DNA, where one exchange is counted as two breaks. Thus formation of SCE involves breakage of genetic material and subsequent recombination of the four DNA strands. The exchanges are visible through differential staining which is brought about by incorporation of a thymidine analog-Bromodeoxyuridine.

Significance of SCE

Elevated SCE frequencies can be produced in response to environmentally induced DNA damage or spontaneously i.e. in the absence of external inducing agents. The induction of SCE is dose dependent i.e. the more the number of years of exposure to the mutagen the more will be the SCE frequency⁵.

The Peripheral Blood Lymphocyte as a Cell used to determine SCE Frequency

The following features of *lymphocytes* make them suitable for SCE analysis: 1) accessibility, 2) capable of proliferation in vitro, 3) representative of cell population.

The peripheral blood circulates to every organ carrying nutrients, metabolites and chemicals to and from the cells of the body. Thus, the peripheral blood lymphocyte serves as a useful indicator of exposure.

Factors affecting SCE analysis in Human population

- (1) Inherent factors
- (2) External Factors-Genotype, lifestyle, physiological state of the individual.

Agents known to induce SCE

- (1) UV Light, (2) alkylating agents like nitrosoureas and alkyl sulphates, (3) anti cancer drugs like mitomycin-C and cyclophosphamide, (4) smoking, (5) metals like copper, iron dust, nickel, cadmium, (6) oral contraceptives, (7) caffeine, saccharin, (8) medication, drugs

Methodology

Blood Culture is performed taking peripheral blood of the patient through a venepuncture. A suitable serum should be added in the blood culture tube along with a mitogen-phytohemagglutinin, and a thymidine analog-bromodeoxyuridine. The blood is incubated at 37°C for 72 hours and at the 69th hour a metaphase arresting agent colchicine is added. The solution is centrifuged and then the cell pellet is treated with 3:1 ratio of methanol and acetic acid.

Several slides can be prepared from each culture. The slides can be stained for analysis by fluorescence or fluorescence plus Giemsa⁴ method, using Hoechst dye 33258. Well differentiated metaphases should be accepted for scoring. The slides can be reviewed under low magnification (100-200x) and selected for scoring on the basis of good staining and chromosome number.

Conclusions

Several studies on SCE frequency as a diagnostic tool for genetic monitoring have done in the past to study the influence of chemical mutagens on the health of industrial workers^{6,7,8,9}. A latent period i.e. time period between the exposure to the agent and clinical manifestations of the disease has been described by Vogel⁹.

Early intervention in this period may prevent the industry related diseases. SCE therefore is useful in monitoring ensuing genetic damage in persons at risk. It is imperative to determine the margin of safety in person exposed to environmental or industrial mutagens and provide timely measures before it is too late. Such an assessment test will be of great help in forecasting, preventing and monitoring oncogenic hazards in persons at risk. There is a uniform consensus that Sister Chromatid Exchange is a simple, sensitive and objective test for routine mutagenicity testing. (Anwar, Yang, Kukura).

Recent Advances

Chromosomal analysis incorporating SCE is being used in a variety of clinical conditions..... e.g. leukaemias, lymphomas, choriocarcinoma, hydatiform mole, ovarian tumours.

Although newer techniques like FISH, genomic hybridizations are developing to characterize certain tumours, baseline cytogenetic bio-markers such as SCE Frequency cannot be replaced and are of utmost importance in correlation of the extent of the disease and genetic damage.

Man's most precious possession is his genetic heritage. If we carelessly squander our resources and poison our germ plasm with mutations produced as a result of environmental pollution, then our heirs will be the losers.

References

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IMSA News

IMSA Chapter Activities (July - Sept. 2005)

Tamil Naidu Chapter

- 10-7-2005 : Prof. G. Ravindran: 'Application of Robot in Medical Science.'
- 14-8-2005 : Prof. G. Sivakumar: 'Current Management of Diabetic foot Disease - Role of Preventive Strategy.'
- 11-9-2005 : Dr. M. Chandrasekaran: 'Management of Thyromegaly'.

Rural CMEs conducted by Tamil Nadu Chapter

- 21-8-2005 : Dr. M. Itambara Thi, Prof. Madras Medical College and ENT Surgeon Govt. General Hospital Chennai. Topic: 'Common ENT problems in general practice and their management.'

- 5-9-2005 : Dr. M. Rajkumar, Prof. of Vascular Surgery, Madras Medical College, Chennai. Topic: 'Deep vein thrombosis'.
- 18-9-2005 : Dr. Namitha Bhuvaneshwari Raj Kumar, Prof. Ophthalmology : Kilpauk Medical College. Topic: 'Childhood blindness'.

Rural CME - Tamil Nadu Chapter (Chennai)
CME Programme conducted by IMSA Branch Unit at Annamalai Nagar, Chidambaram.

- 16-7-2005 : Dr. N. Muragan: 'End stage liver disease - current concept and management.'
- 31-7-2005 : Dr. G. Santhanam: Type II diabetes Mellitus - Erectile Dysfunction.
- : Dr. S.V. Raghunathan: Type II Diabetes Mellitus - Problems and Management.

IMSA WORKSHOP International Medical Sciences Academy (IMSA), in collaboration with Kundan Laser Centre is organizing a workshop '6th Hands-on-Laser Workshop' Basic Orientation' Course and Workshop (Aesthetic Laser Surgery and Hair Removal) on 5th and 6th November 2005 at Kundan Laser Centre (Institute of Laser and Research Centre) 4771/23, Bharat Ram Road, Daryaganj, New Delhi-110002, India.

For registration contact : Dr. S.S. Sethi, President - IAALS, Convenor and Chairman at above address under intimation to IMSA Headquarter.

Election of Fellows

Fellows & Members elected during the quarter July-Sept. 2005

Dr. M. Bhaskar	Chennai	Dr. Patel Hemant	USA
Mr. Zinobia Mahiar Madan	Mumbai	Dr. Birinder Jeet Kaur	USA
Dr. N. Mohan	Salem	Dr. Sanjiv Kumar	Switzerland
Dr. D.P. Singh Toor	New Delhi	Dr. (Mrs.) Neeta Kumar	Switzerland
Dr. S. Anuradha	New Delhi	Dr. Nabendu Bhattacharjee	Kolkata
Dr. Ravi Ramalingam	Chennai	Dr. Deepa Sachdeva Passi	Noida
Dr. N.D. Ramanujam	Chennai	Dr. Navtej S. Butter	USA
Dr. D. Sachithanandam	Vellore	Dr. Ajay Sharma	New Delhi
Dr. R.N. Srivastava	New Delhi	Dr. Usha Gupta	New Delhi
Dr. S.V.S.R. Krishna	New Delhi	Members	
		Dr. Harish Pathak	New Delhi

HONOURS

Prof. S.C. Sahgal FIMSA, Director WHO collaborating Centre for Leptospirosis, Regional Medical Research Centre (ICMR) Port Blair, has been bestowed '**Fellowship of Royal College of Pathologists, UK**', in recognition of his contribution in medical science, for his work carried out especially in Andoman Nicobar Island.

Dr. T.D. Chugh has been awarded '**Genl. Amir Chand Oration**' by National Academy of Medical Sciences' for the year 2005' and has been appointed National Emeritus Professor by the Academy.

Dr. SNA Rizvi FIMSA has been awarded the prestigious '**Netaji Oration Award 2007**' in recognition of his significant work on Metabolic Bone disease by the association of Physicians of India.

Dr. (Mrs.) S. Sachdev, Founder Fellow of IMSA has been awarded '**Helpage India Golden Award**' for the year 2005 by Helpage India, on Oct. 1st, 2005.

IMSACON 2006

Annual Conference '**IMSACON 2006**' will be held on 3-4-5 November 2006 at Lahore (Pakistan) **Dr. Shaheena Asif**, Surgimed Hospital Lahore will be the Organising Secretary.

Theme : 'Update in Medical and Dental Sciences'

Venue : Lahore Medical & Dental College, Canal Bank North, Tulspura, Lahore-53400, Pakistan

Visa : Visa is required for Pakistan and must be obtained before travel. Please allow 3 months before conference date for application to be processed.

INTERNATIONAL ACADEMY OF MEDICAL SCIENCES

Annual Report 2005

This year we had a wonderful meeting at Mayo Clinic, Rochester, MN which was ably organized by Dr. H.S. Luthra who has now been promoted to be one of the Board of Trustees member. In Mayo Clinic we had galaxy of eminent scientists from all fields of medicine to deliver lecture on the theme of the conference 'Genomics and Proteomics: the New Revolution in Medicine'. We had planned to have IMSACON-2005 in Srinagar (J&K) but somehow that did not materialise. During my visit to Jaipur Dr. Shashi Panicker had organized a meeting of Jaipur Chapter in which eminent doctors including Mrs. Panicker, Dr. Rai and others participated. They agreed to my request to have IMSACON 2005 in this historical city

FELLOWS

1. Dr. A. Radhakrishna	Tamil Nadu
2. Dr. C.S. Balachandran	Tamil Nadu
3. Dr. V. Anandi	Tamil Nadu
4. Dr. P.T. Thamizharasu	Pondicherry
5. Dr. S. Murugan	Chidambaram
6. Dr. A.R. Annamalai	Chidambaram
7. Dr. P. Viswanathan	Tamil Nadu
8. Dr. Chitturi Surya Prakash	Tamil Nadu
9. Dr. G.N. Pitchechaya	Annamalai Nagar
10. Dr. (Col.) MAS Warrior	Chennai
11. Dr. M. Murugesan	Tamil Nadu
12. Dr. Muthukumaran	Tamil Nadu
13. Dr. Shashi Kant Singla	Delhi
14. Dr. D. Rajakumar	Tamil Nadu
15. Dr. (Mrs.) Meera (Rupta) Suhas Kulkarni	Kolhapur
16. Dr. Suhas Panditrao Kulkarni	Maharashtra
17. Dr. V.P.M. Mushafa	Saudi Arabia
18. Dr. Anmol K. Gupta	Shimla
19. Dr. N.K. Sekaran	Chidambaram
20. Dr. V. Muniappan	Annamalainagar
21. Dr. Govind Chandra Sahoo	Chidambaram
22. Dr. N. Chidambaram	Annamalainagar
23. Dr. P. Muralidhar	Annamalainagar
24. Dr. S. Viswanathan	Tamil Nadu
25. Dr. Roseline Fatima William	Annamalainagar
26. Dr. V.U. Shanmugam	Chidambaram
27. Dr. Satischandra Marutrao Gaikwad	Mumbai
28. Dr. A.K. Grover	New Delhi
29. Dr. Sethu Rajan	Chidambaram
30. Dr. Pas Rajenthiran	Cuddalore OJ

We had quite a few Editorial Board Meetings to discuss JIMSA affairs. This year we have introduced 'Best Article Award'. Our journal is really appreciable and it is all due to strenuous efforts of Dr. P.D. Gulati, Editor, JIMSA. It is on time and almost financially self sufficient. We are getting regular grant of Rs. 40,000/- from the Department of Science and Technology. Now it has been raised to Rs. 50,000/- due to efforts of Secretary General Dr. I.P.S. Kalra. The following special issues are published this year :

1. Advances in radiation Therapy Jan-March 2005 and latest one.
2. Environmental Health in July-Sept 2005 issue which is in your hand.

I once again repeat that our journal is on website 'www.jimsaonline.com'. Please read the articles and ask your friends to read it. Password is 'userjimsa'. There is no charge for this. Any one can have access to that. Account of JIMSA have been separated.

Tamil Nadu Chapter

Tamil Nadu Chapter had been very active and they are doing lot of CME programmes particularly in the rural areas. They have established a branch unit at Annamalai Nagar, Chidambaram, Tamil Nadu. It was inaugurated by Dr. MAM Ramasamy Pro-chancellor of Annamalai University under the chairmanship of Dr. K. Jagadeesan. They have had physicians exchange programmes with the universities abroad. Tamil Nadu chapter will be shortly opening second sub chapter at Atomic Energy Kilpauk. The details of activities of Tamil Nadu Chapter during the last one year which have been ably conducted by Dr. A. Govindan Dr. TMR Panicker

- Jaipur. Board of Trustees readily agreed to the request of Rajasthan Chapter. Now we are here in Jaipur for IMSACON-2005 and the theme has been aptly selected 'Emerging Health Challenges'. We have eminent scientists, teachers both from India and abroad to deliberate in this conference. Faculty from Mayo Clinic under leadership of Prof. H.S. Luthra is here I hope you will enjoy the deliberations and they will be useful to the medical fraternity.

Credentials Committee and BOT has carefully selected and cleared fellows and members and now total strength is around 1394 Fellows and 123 Members. List of Fellows and Members elected this year is given below :

31. Dr. V. Namachivayam	Tamil Nadu	62. Dr. Homi D. Doodhwala	Surat
32. Dr. G. Santhanam	Mayiladuthurai	63. Dr. Haroon Subhan Khan	Aligarh
33. Dr. P.S. Krishna Moorthy	Cuddalore OJ	64. Dr. Colonel (Dr) Chandar Mohan	
34. Dr. C. Dhana Sekaran	Chidambaram	65. Dr. Amarjit Singh Grover	Patiala
35. Dr. Waseem Qureshi	Srinagar	66. Dr. Vineet Talwar	New Delhi
36. Dr. D.K. Mehta	New Delhi	67. Dr. P. Bhattacharjee	Tamil Nadu
37. Dr. Sreeramadasu Ramaiah	Kurnool	68. Dr. Jayashree Bhattacharjee	New Delhi
38. Dr. (Mrs.) Charusheela Satischandra Gaikwad	Mumbai	69. Dr. S. Jayaram	Pondicherry
39. Dr. Deepak Singhal	Delhi	70. Dr. S. Rajagopal	Kollam
40. Dr. C.R. Sundararajan	Chennai	71. Dr. Anita Khalil	New Delhi
41. Dr. Jayaraj Govindaraj	Chennai	72. Dr. C. Ramanunni	Kerala
42. Dr. S.M. Rajendran	Chidambaram	73. Dr. V. Raveenthiran	Tamil Nadu
43. Dr. (Lt. Genl.) S.P. Kalra	New Delhi	74. Dr. Shipra Paul	New Delhi
44. Dr. P.C. Rajaram	Chennai	75. Dr. Alok Ahuja	Mohali
45. Dr. Tulsi Dass Chugh	New Delhi	76. Dr. (Mrs.) Vishva Kirti Bhasin	Delhi
46. Dr. Karri Prasada Reddy	Visakhapatnam (AP)	77. Dr. Jagbir Singh	Patiala
47. Dr. Desai Jagruti Yogesh Kumar	Gujarat	78. Dr. Ajay Kochhar	Delhi
48. Dr. Desai Yogesh Kumar Chhaganlal	Gujarat		
49. Dr. Mehta Haresh Chandran	Surat	MEMBERS	
50. Dr. Duchhwa Bharatkumar Gamanlal	Gujarat	1. Dr. Parijat Chandra	New Delhi
51. Dr. Mukino M. Karia	Surat	2. Dr. K.A. Shalini Maya	Chidambaram
52. Dr. Sadhna M. Desai	Gujarat	3. Dr. Rajul Rastogi	New Delhi
53. Dr. Anita Jayant Shah	Surat	4. Dr. Amit Bhatia	New Delhi
54. Dr. Atam Prakash Arora	New Delhi	5. Dr. Lal Bahadur	Jaunpur
55. Dr. M. Nanda	New Delhi	6. Dr. Mohd. Minhajul Haq	Surat
56. Dr. L.H. Ghotekar	New Delhi	7. Dr. Shelat Vishalkumar G	Gujarat
57. Dr. (Mrs.) Poonam Khurana	New Delhi	8. Dr. C. Paul Dilip Kumar	Chennai
58. Dr. Mohinder Singh	Patiala	9. Dr. Kunal Gupta	New Delhi
59. Dr. Surinder Singh	Pondicherry	10. Dr. Ankur Barua	Kolkata
60. Dr. Ajay Mehta	New Delhi	11. Dr. Sajeer Gupta	New Delhi
61. Dr. Nirmal G. Choraria	Gujarat	12. Dr. Sandeep Banga	New Delhi

and other members under the able guidance of our President Dr. K. Jagadeesan during the last one year are given below :

Tamil Nadu Chapter

- 10-7-2004 : Dr. Bagyam Raghavan 'Imaging in Breast Disease - A Multimodality Approach'
- 8-8-2004 : Dr. Palanisamy 'Treatment of Heart Failure'
- 12-9-2004 : Dr. Manoharan 'Surgical Aspects of Tetrolology of Fallots'
- 10-10-2004 : Prof. Krishnamoorthy, 'Pain and Perception'.
- 14-11-2004 : Dr. Srimathi, Common Cardiac Problems including Myocardial Ischemia and their Management.
- 12-12-2004 : Dr. Mayilvahanan Natarajan, 'The Custom Mega Prosthesis - The answer to the Management of Bone Tumours'.
- 14-12-2004 : Dr. Krishnamoorthy Srinivas, 'Brain, Mind and Music.
- 19-12-2004 : Rural CME Programme, Dr. R. Kndaiah, 'Common Ophthalmic Problems in General Practice and Current Concepts'
- 9-1-2005 : Prof. G. Krishnamoorthy, 'Consciousness'.
- 13-2-05 : Dr. P. Venugopal, S. Menon, 'Novel thiozole compound with antioxidant property and its role in diabetes'.
- 13-3-05 : Dr. A.R. Chandrasekaran; Emerging trends in primary health care'.
- 10-4-05 : Dr. N. Gnanasundaram, 'Mouth is the mirror of the Body'.
- 8-5-05 : Dr. P.C. Rajaram, 'Value of ultrasonography, in

- musculo skeletal and soft tissue lesions in the face'
12-6-05 : Dr. Arun Balakrishnan, 'Bio activity based screening of novel molecules towards drug development.

Delhi Chapter

Delhi Chapter has been smoothly running and have had series of scientific programmes ably organized by Dr. (Mrs.) Sachdev and Dr. (Mrs.) I.K. Sharma supported by Dr. I.P.S. Kalra Secretary General and the Board of Trustees. Detailed programme of all the activities this year is mentioned as under :

- 25-8-2004 : Lt. Col. Dr. A.K. Singhal, Dr. B.K. Dkaun "Osteo Arthritis"
16.9.2004 : Dr. S.M. Kaul, 'Malaria and Dengue'
24-9-2004 : Dr. Neeru Aggarwal, 'Recurrent Urinary Tract Infection in Women'
: Dr. Ashok Kumar 'Basti Sevika as 'Ambassador of Health'.
26-9-2004 : Dr. H.K. Chopra, Dr. I.P.S. Kalra 'Spirituality and Medicine'
10-10-2004 : Dr. Mukesh Ajmera, 'Stress Testing in IHD'.
: Dr. G. Siripathy, 'Hypothyroidism - Diagnosis & Management'.
23-10-2004 : Dr. S.J. Gupta, 'Clinical Meeting'.
16-11-2004 : Dr. A.K. Ajmani, 'Diabetes Mellitus'.
14-12-2004 : Dr. Arun Gogna, 'Echo Cardiography'.
14-12-2004 : Dr. (Lt. Col.) Ashok Rajput, 'Bronchial Asthma'.
8-1-05 : Dr. A.P. Arora, Dr. V.K. Gujral, 'Acute Coronary, Syndrome in diabetics' pitfalls and precautions in diabetes management in cardiac patient, 'Venue : Seminar Hall, National Heart Institute.
20-1-05 : Dr. G. Kapur, 'An overview of Paediatric malignancies and their chemotherapy' Dr. A. Saharia, 'Organ preservations in Paediatric Tumours; surgical aspect Dr. A.K. Anand, 'Targeted Radiotherapy - reducing late radiation morbidity in paediatric solid tumours.
12-2-05 : Dr. A.K. Jhingan, 'The Diabetes is a vascular Disease.
: Dr. Prof. S.K. Agarwal, 'Diabetic Dyslipidemia'.
22-2-05 : Dr. Lt. Col. S.K. Malau, 'Acute Myocardial Infarction'
: Dr. Col. K.K. Singh, 'Seizures'
24-2-05 : Dr. L.M. Prasher, ENT Today; What all possibilities are Dr. Kapil Kochhar, 'Imcisional Hernia - Revisited.
12-3-05 : Dr. Vinod Sharma; Percutaneous Interventions in 21st Century; Controversies, the Challenges and
12-3-06 : Follow-up guidelines Dr. O.P. Yadav 'CABG in Diabetics; Challenges and follow-up guideline'
15-3-05 : Dr. Raghugaind (UK) 'Care of Elders'
30-3-05 : Dr. Col. D.P. Vats, 'Cataract' Dr. Col. Rajat Kumar, 'Approach to care of Anaemia'
30-4-05 : Dr. Ram Raj Singh, USA 'Indian Rheumatology has its time come too late in the world'.
7-5-05 : Dr. S.J. Gupta, 'Case Presentation'
2-6-05 : Dr. Prathiba Saran, 'Recent advances in Ophthalmology'

Delhi chapter is intending to start branch chapter in Rajiv Gandhi Cancer Hospital and Guru Harkrishan Hospital Bala Sahib and Bangla Sahib.

Board of trustees meetings were held during the year on 23-11-2004, 4-2-2004 & 22-7-2005.

Rajasthan Chapter

Your are seeing not important activite the Rajasthan Chapter IMSACON 2005. They have increased their members and fellows to organised by

Chandigarh, Panchkula and Mohali Chapter

Secretary General organized meetings of the fellows at Mohali where Dr. B.N.S. Walia renowned Paediatrician and Director PGI Chandigarh (Retd.) agreed to guide the chapter as Chairman and Dr. Ashok K. Attri Surgeon a Secretary, Dr. A.K. Singhal as Treasurer they have formed a combined chapter of Chandigarh, Panchkula

and Mohali fellows in Chandigarh. They are planning to have IMSACON 2007 in Chandigarh.

Punjab Chapter

Dr. D.N. Bhardwaj retired Principal, Govt. Medical College Patiala is participating in this conference with other delegates from Punjab and Patiala and they are getting active in this organization.

US Chapter

During my recent visit to United States, I had opportunity to attend Death Anniversary Function of Dr. P. Narasimha Rao our past President at residence of Pinnamaneni Sarath son fo late Dr. P. Narasimha Rao. It was a solemn affair where we remembered oru great leader the world renowned medical scientist. I could interact with his family membrs - Dr. P. Sarath and his wife Sridevi and Dr. Prasad, son-in-Law of Dr. Narasimha Rao. They were thrilled with the idea of forming a New York chapter. and I could talk to renowned doctors in that family and urged them to join our Academy Dr. Sarath and Sridevi were keen to have hub of activities of the New York chapter in their house. Dr. Hemant Patel from New York and Dr. Brinderjit Kaur from New Jersey have been elected as fellows of our Academy. Lt. Genl. J.M. Grover introduced Dr. Sanjiv Kumar and Dr. Neeta Kumar from Geneva who have been elected as our fellows and now activities of our chapter will soon be seen in Geneva, the headquarter of UNO.

UK Chapter

Bapuji Rao from UK attended the Board of Trustees meeting held at India International Centre and he promised to boost the activities of UK Chapter.

The following are the Honorary Fellows as on date.

Honorary Fellows

1981

1. NAKAYAMA, KOMEI. Prof. MD., Nakayama Cancer Research Centre, Tokyo, Mode Centre Building, 19-7-6, Chuo-Ku Tokyo, 140 JAPAN.
2. KAY, SIR ANDREW WATT., Rozelle, 14 North Campbell Avenue, Milngavie, GLASGOW G 62 7AA, UNITED KINGDOM
3. HOUNS FIELD, SIR GODFREY, THRONE EMI Central Research Laboratories, Trevor Road, Hayed, Middle sex, UB3 IHH ENGLAND.
4. NOSSAL SIRGUSTAVE JOSEPH VICTOR, Kt. CBe MD, BS(Syd), B.Sc.(Med) Ph.D (Melb), Hon, MD(Mainz), FRCP, FRACP, FRPCA, FCMA, FTS, FAA. Director, The waliter and Eliza Hall Institute of Medical Research, PO Royal Melbourne Hospital, Victoria - 3050, AUSTRALIA.
5. GODBER, SIR GEORGE., 21, Almoners 'Avenue, CAMBRIDGE CBI 4NZ, UNITED KINGDOM.
6. AUJALEU, EUGENE, 144, Boulevard Du Montamasse, 75-PARIS, 14E, FRANCE.
7. MAHLER H., MD. Ex Director General, World Health Organizations, 1211, Avenue Apia, GENEVA-27, SWITZERLAND.
8. WHITTERIDGE DAVID, University of Oxford, Deptt. of Experimental Psychology, South Parks Road, OXFORD, OXI 3UD, UNITED KINGDOM.
9. CHIA-SSU HUANG (HUANG JIASI), President Chinese Academy of Medical Sciences, 9, Dong Dan San Tiao, Beijing China.
10. PETROVSKY, BV Member, Academy of Medical Sciences, Former Minister of Health, USSR, Rakhmanovsky Pereulok, MOSCOW, USSR.

1982

11. KOKO U., MD, Ex. Regional Director, World Health Organization, Regional Office South East Asia Region, World Health House, IP Estate, Ring Road, NEW DELHI-110002 INDIA.

1988

12. Prof. L. SURYANARAYANA, MS, Ex. Vice-Chancellor, University of Health Sciences, VIJAYAWADA-520008 (A.P. resi: "HARIVILLU", 59-A-8-5, Teacher's Colony, Patamata,

VIJAYAWADA-520008 SURGERY.

1990

13. KADEN, PROF. SC. MED. WOLFANG, Kunik Fuer Nephrologie Und Urology Des Bezirisk Rankenhauses, "Emnst Scheffler" Ave, Germany.

1991

14. KLINKMANN, Prof. DRS. HORST (DOB9-5-1935) Graduated, MD,D.Sc., President Academy of Sciences, Berlin (Germany) Home Address : Schliemann Str,7,D2500 Rostock, GERMANY
15. ANTHONIS, PR Order of the Sacred Treasure (Japan) Desmanya D.Sc.FRCS (Eng):FICS:FSCS, Consultant Surgeon, Chancellor of the university of Colombo (Sri Lanka) Res: 161, Dharmapala, Marwatha, COLOMBO (Ceylon)

1992

16. SINHA AKN - Expired 5-7-1994.

1993

17. PROF. DR. MED. DR. H.C. BORIS LUBAN - PLOZZA, Swiss Diploma in Medicine (equiv. To MBBS), Doctorate in Medicine, Surgery & Obstetrics (equiv. To MD) Vice president, International Federation of Hygiene, Social Medicine, President of Foundation for Psychosomatic & Social Medicine, ASCONA, SWITZERLAND.

1994

18. THIRUVENGADAM, Prof. KVB.Sc, MD, FRCP, FAMS, FCCP (USA), FCAI, Retired Prof. of Medicine and Head of Deptt. Madras Medical College & Physician, Government Genral Hospital, MADRAS-600003.
19. VALIATHAN, PROF. M.S. MS (Liv) FRCS (Edin) FRCS (Eng), FCRCCS (Canada) Vice-Chancellor, Manipl Academy of Higher Education, University Building, Madhav Nagar, MANIPAL-576119 (Karnataka)

1999

20. Kakarla Subbarao Prof. DOB-25 Jan 1925, MBBS, MS (Red), FA CR FRCD, FICP, FSASMA, FCCP FICR Director/CE NIGMS Panjagutta Hyd.

2001

21. Dr. S.S. Sriramacharyulu, 521, Mandakini Enclave, Kalkaji, New Delhi-19.

2002

22. JD. Williams Prof. DOB:3rd March 1931 BSc (London) 1951, MB, ChB(Liverpool) 1956, DCP 1961, MD(Liverpool) 1965, MRCPATH 1965, FRCPath 1982, MRCP 1986. JD Williams Unit 31st Olav's Court 25 Lower Road London SE16 2XB, UK
23. Dr. Ela Anand, MBBS, FRCOG., Gynaecologist, Incharge, Rural Set up of Arpana Hospital, Madhuban, Karnal (Haryana).
24. Dr. J. Heinrich Joist, MD PHD FACP, FAHA, Prof. of Pathology & Internal Medicine, Email : joistjh@slucarel.sluh.edu
25. Prof. Niali Diamid Campbell Finlayson Consultant Physician, Royal Infomary, Edinburgh, and Hony Senior Lecture in Medicine, Edinburgh University School of Medicine.
26. Dr. N.K. Ganguly, Director General, Indian Council of Medical Research, Ansari Nagar, Mahatma Gandhi Marg, New Delhi-29.

2004

27. Dr. Chella David USA
28. Dr. Richard Weinshellboum USA
29. Dr. Thomas Spelsberg USA
30. Dr. David Weatherall UK
31. Justice K. Naryana Kurup Kochi (India)

List of Chairman and Secretary of various chapter

1. AP Dr. C.M. Habilullah, principal, (Regd.) Osmania Medical College & Prof. & Head Deptt. of Gastroenterology, Hyderabad-500195.
2. Bihar Dr. B. Mukhopadhaya, Saidpur Road, Patna-800004.
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8. Maharashtra Dr. W.G. Rama Rao, 'MAYUR' 377, V.P. Road, Off. Lamington Road, Bombay-400004.
9. Nagaland Dr. L.M. Murry, Director, Christian Medical Centre, Vankhosung, Wokha-797111.
10. Punjab Dr. S.K. Khetarpal, 109, Lawrence Road, Amritsar 143001.
11. Rajasthan Dr. R.P. Sarada, Sarada Eye Clinic & Nursing Home, 39, Hospital Road, Jaipur-301001
12. TN Dr. M. Natrajan 'MAHALINGA NILAYAM', 24, Lakshmi Street, Off. New Avadi Road, Madras 600010.
13. West Bengal Prof. Bhaskar Ray Chaudhuri, Consultant Neurologist, 220, Lower Circular Road, Suit No.10, 4th Floor, Calcutta-700017.
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Dr. Pinnamaneni Narasimha Rao Internaltional Award

Last Year Dr. Pinnamaneni Narasimha Rao International Award was created and the first oration was awarded to Dr. H.S. Luthra in the IMSACON-2004 held at Mayo Clinic, Rochester USA. Sizeable amount is being collect for the Award and this will be established on the lines of Dr. B.C. Roy National Award. It is proposed to be held in President House and efforts are being made get it through from President's House.

We are trying to get financial grant from the Ministry of Health and family Welfare for CME on regular annual basis. Our President Dr. K. Jagadeesan is trying hard and we hope to achieve soon.

Efforts are being made to get a piece of land allotted from Delhi Development Authority for our organization for office building and conference Hall where seminars, workshops etc. can be conducted.

Board of Trustees meetings

Many impnortant decisions were taken at the Board of trustees meetings. On recommendations of Editorial Board meeting, Board of Trustees agreed to start 'Best Paper (Published) Award' this year; - The Awardees selected for the year 2005 are :

I have tired to do my best to work for the organization ever since my tenure of the Additional Secy. Genl. and Secy. Genl. Since 1996. The membership/fellowship has improved a lot. There are more overseas members. I have been able to get a regular grant of Rs. 40,000/- now Rs. 50,000/- annually from Deptt. of Science and Technology for our journal JIMSA. Rural conference in Arpana has been organized very efficiently, regular CME programmes of Delhi Chapter are conducted and international voerseas conferences in UK and USA had been a great success. IMSA is a great organization which has very eminent fellows headed by eminent President, Vice President and Board of Trustees. I feel if everyone of us thinks a little about this organization, to improve it, then it can be brought on a firm footing. I must have done many mistakes; for that I beg to be excused.

I am highly indebted to President, Vice-President, Board of Trustees, Additional Secretary General, Treasurer and my staff Mr. Anand Rama, Rawat, Gosain, Joshi for helping me throughout my tenure. Although I am leaving the post of Secretary General. I will be continuing my interest in this organization. God bless us all.

Dr. I.P.S. Kalra
Secretary General
International Medical Sciences Academy



IMSACON 2005

22nd to 24th October 2005

Venue : Convention Hall Hotel Rajputana Palace Sheraton, Jaipur India

CME & Scientific Programme Sponsored by (i) I.C.M.R. (ii) Medical Council of India (iii) National Academy of Medical Sciences, India

Dear Colleague,

It gives us immense pleasure to invite you to the Annual Conference of the International Medical Sciences Academy. IMSACON 2005 will be held at Jaipur, Rajasthan from 22nd to 24th October 2005. The *theme* for this year's conference is **"Emerging Health Challenges"**.

This conference promises you a fantastic opportunity to meet and interact with specialists from various branches of medical science. A galaxy of national and international speakers with vast clinical experience and outstanding research background will be participating in the scientific sessions. Along with the intellectual extravaganza, you can enjoy the delights of the Pink City. As you know, Jaipur is a great tourist destination, a heritage city famous for its rich culture, artistic excellence and legendary hospitality. We look forward to welcome you to the conference and assure you of a fruitful and memorable stay.

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International Medical Sciences Academy was established as a global organization in 1981 at New Delhi. It is a registered society with regions in America, Australia, India, UK, Africa and Europe. Dr. K. Jagadeesan, the renowned transplant surgeon and recipient of the B.C. Roy Award, is the President. IMSA is an Associate member of the Council for International Organizations of Medical Sciences (CIOMS), WHO Geneva. It has over 2000 fellows and members and has chapters in many states in India.

JIMSA, of the International Medical Sciences Academy, is published quarterly; besides its fellows, members, journal is circulated to major Medical Institutions of the country; full text of the journal is also on website www.jimsaonline.com IMSA conferences are conducted in India and abroad every year alternately. Last year's conference was organized in Mayo Clinic, Rochester, USA. Next year's conference is in Lahore, Pakistan in November 2006.

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SCIENTIFIC PROGRAMME - IMSACON 2005

Saturday, 22 October, 2005
1st Day

Time	Registration
HALL-A	
09:00 - 10:10 am	Welcome Speech : Chairman, Organising Committee, IMSACON 2005 Inauguration of CME : Dr. K. Jagadeesan, President, IMSA Challenges in Reducing Maternal Mortality : Dr. Sarla Gopalan, HOD, Ob & Gyn. & Dy. Director PGI, Chandigarh Drug Development and Research in India : Dr. Ashwini Kumar, Drug Controller - General, India Anti TNF Therapy : Dr. H.S. Luthra, Prof. & Chair, Division of Rheumatology, Mayo Clinic College of Med., USA
10:10 - 10:30	Tea / Coffee Break
10:30 - 11:30	ONCOLOGY Cancer in Elderly : Prof. Pavithran, Chief of Oncology, AIIMS, Cochin Curable Cancers today : Dr. Hemant Malhotra, Jaipur
11:30 - 12:40 pm	CURRENT CONCEPTS & MANAGEMENT Rheumatoid Arthritis : Prof. H.S. Luthra, Mayo Clinic, USA Osteoarthritis : Dr. Nisha Manek, Mayo Clinic, USA Osteoporosis : Dr. S. Amin, Mayo Clinic, USA
12:40 - 1:30	NEUROLOGY TIA & Stroke Prevention : Dr. P.N. Renjen, Apollo Neurosciences, New Delhi Movement Disorders : Col. K.K. Singh, R.R. Army Hospital, New Delhi
1:30 - 2:30	LUNCH HOTEL RAJPUTANA PALACE SHERATON
2:30 - 3:30	C.A.D. in the Asymptomatic : Dr. V. Hariharan, Apollo Hospital, Delhi Invasive Management in C.A.D. : Dr. Gurpreet Sandhu, Mayo Clinic, USA
3:30 - 4:00	Tea / Coffee Break
4:00 - 5:30	Inauguration Function
5:30 - 6:30 pm	ORATIONS Emerging Health Challenges in Developing Countries : Dr. N.K. Ganguly, Director General, ICMR, India Future Medicine : Promises & Perils : Prof. Vijaya L. Melnick, USA

HALL - B

10:40 - 11:20 am	PAEDIATRICS Monteleukast in Children : Dr. Rakesh Mudgal, UK Issues in vaccination : Dr. D. Shivpuri, Jaipur
11:20 - 12:40	SURGERY Minimal Invasive Surgery : Dr. P.K. Chowbey, Sir Ganga Ram Hosp., New Delhi Advanced Laparoscopic Surgery : Dr. Parul Shukla, Tata Memorial Hosp., Mumbai Dr. A.K. Mathur, Jaipur Laparoscopic Surgery in Gynaecology : Dr. Sorosh Roshan, New York, USA
12:40 - 1:30	BREAST Breast Nodule : Workup & Management : Dr. Nicole Sandhu, Mayo Clinic, USA Breast Reconstruction Surgery : Dr. K.S. Bhangoo, USA
1:30 - 2:30	LUNCH, Hotel Rajputana Palace Sheraton
2:30 - 3:30	PANEL DISCUSSION Challenges in Reducing Pregnancy Loss and Perinatal Mortality Moderator : Dr. Urmil Sharma, Indraprastha Apollo Hospital, New Delhi Participants : Prof. Sarla Gopalan, Chandigarh : Dr. Sorosh Roshan, USA Prof. Adarsh Bhargava, Jaipur : Dr. Sucheta Panicker, Jaipur Prof. Shaheena Asif, Pakistan : Dr. D. Shivpuri, Jaipur
3:30 - 4:00	Tea / Coffee Break
4:00 - 6:30	INAUGURATION FUNCTION & ORATIONS
6:30 - 7:00 pm	Board Meeting at Khasa Kothi (Conference Room)
7:00 pm onwards	Entertainment Programme followed by Cocktails & Dinner at Hotel Khasa Kothi.

Sunday, 23 October, 2005
2nd Day

Time	Registration
HALL - A	
09:00 - 10:45 am	Healthcare for the Masses Ethics of Research in Healthcare in Developing Countries : Prof. S.D. Gupta, Director IIHMR Challenges of Global Maternal Healthcare : Dr. Vasantha Muthuswamy, Dy. Director, I.C.M.R. Essentials of Caring : Prof. Sorosh Roshan, U.N. Delegate, USA Primary Prevention of Diabetes Mellitus : Prof. R. Gained, Guy's Hospital, London Focus on Genes or Intrauterine Environment? : Prof. A.K. Das, Director, J.I.P.M.E.R., Pondicherry
10:45 - 11:00 am	Tea / Coffee Break

11:00 am - 1:30 pm	Symposium : Early Detection & Prediction of C.A.D. Sponsored by Escorts Hospital Coordinator : Cardiology Participants :	Dr. R.R. Kasliwal , Chief of Non Interventional Dr. Sanjay Mittal, Escorts Hospital Dr. Sameer Shrivastava, Escorts Hospital
1:30 - 2:30 pm	LUNCH Hotel Rajputana Palace Sheraton	
2:30 - 3:30 pm	Metabolic Syndrome Thyroid Disease in the Young	Dr. A.K. Das, Pondicherry Dr. G.N. Saxena, Jaipur
3:00 - 4:00	HIV The Scourge of HIV in Children Skin & STD Markers of HIV Infection Vaccines against HIV	Dr. Pavitra Mohan, Unicef Dr. J.K. Maniar, Jaslok Hospital, Mumbai Dr. Meeta Singh, Jaipur

INTERNATIONAL CONFERENCE ON MEDICAL TOURISM IN RAJASTHAN

4:00 - 6:00 pm	Inaugural Function Introduction Medical Tourism in Rajasthan Raj. Advancing Heart Care in Rajasthan Public - Private Partnership in Medical Tourism Speech by Invited Dignitaries Address by Chief Guest Vote of Thanks by President, IMSA	Dr. S. Panicker, State Coordinator, I.M.A., Rajasthan Mr. Vinod Zutshi, IAS, Secretary Tourism, Govt. of Raj. Dr. Samin Sharma, USA (Eternal Heart Centre) Mr. Apurva Kumar TIE Rajasthan
6:00 - 6:30 pm	High Tea	
07:00 pm onwards	Entertainment / Cocktails / Banquet at Nahargarh Fort	

HALL - B

2:30 - 2:50	The Fight against Malaria	Dr. Kanta Patel, Baroda, WHO Consultant
2:50 - 3:20	Medical Guidelines for Nutrition in Osteoporosis Liver Disease Heart Disease	Dr. S. Amin, Mayo Clinic, USA Dr. Navtej Buttar, Mayo Clinic, USA Dr. Gurpreet Sandhu, Mayo Clinic, USA
	WORKSHOP	
3:20 - 4:00	Simulation Training in Clinical Medicine : Asthma & the Internet	Dr. Bhavesh Patel, Mayo Clinic, USA Dr. A. Patel, Mayo Clinic, USA
4:00 - 6:00	International Conference on Medical Tourism in Rajasthan	
6:00 - 6:30 pm	High Tea	
07:00 pm onwards	Entertainment / Cocktails / Banquet at Nahargarh Fort	

Monday, 24 October, 2005

3rd Day

Time	Registration
08:00 am onwards	

HALL - A

9:00 - 10:45	Robotics in Medial Science Surgical Facial Rejuvenation Chronic Fatigue Syndrome - Does It Exist? Obesity in the Young : An Emerging Health Challenge N.Delhi	Prof. G. Ravindran, Chennai Dr. K.S. Bhangoo, USA Prof. R. Gaiind, London Prof. A.C. Ammini, H.O.D., Endocrinology AIIMS,
10:45 - 11:00	Tea / Coffee Break	
11:00 - 11:40	PANEL DISCUSSION Reducing the Burden of Cardio - Vascular Disease in Developing Countries Moderator Participants : Dr. Gurpreet Sandhu, USA Dr. Ashok Jain, Jaipur	Prof. V.S. Baldwa, Jaipur Dr. R.K. Tongia, Jaipur Dr. N. Vetrivel, Chennai
11:40 -11:55	Hypertension : Appraisal of Current Guidelines	Dr. Rajeev Gupta, Jaipur
11:55 -12:20	EECP : Non Invasive Technique for CAD	Dr. S.J.S. Randhawa, Amritsar
12:20 -12:35	Inhaled steroids : Are They Safe in Children?	Dr. Rakesh Mudgal, UK
12:40 -12:55	Stem Cell Therapy	Dr. Rachna Narain, Jaipur
12:55 -1:10	Assessing Dangerousness	Dr. Susan Lesley, UK
1:15 -1:30	Challenges in Geriatric Otolaryngology	Prof. G.C. Sahoo, Chidambaram
1:30 - 2:30	Lunch, Hotel Rajputana Sheraton	

2:30 - 3:20	GASTROENTROLOGY Current Management of Hepatitis C Barrett's Metaplasia	Dr. Shiv Sarin, New Delhi Dr. Navtej S. Buttar, Mayo Clinic, USA
3:20 - 4:00	OPHTHALMOLOGY Emerging Trends in Ophthalmology Latest Generation I.O.L. for Cataract Surgery	Dr. A.K. Grover, New Delhi Dr. Ashok Puri, Jaipur
4:00 - 6:00	CONVOCATION PROGRAMME VALEDICTORY FUNCTION	

HALL - B

11:00 - 12:00	IMAGING USG in Musculoskeletal Tissue Chest Imaging for the Non - Pulmonologist	Dr. P.C. Rajaram, Chennai Dr. A. Patel, Mayo Clinic USA
12:00 - 1:30	HEALTHCARE Statistical Management for Healthcare Out Reach Programmes for Rural Surgical care E-Medicine in Rural Healthcare	Dr. Daniel Melnick, USA Dr. D.P.S. Toor, New Delhi Dr. S. Panicker, Jaipur
1:30 - 2:30	LUNCH, Hotel Rajputana Sheraton	
2:30 - 4:00	Life Threatening Infection Guillaine - Barre Syndrome Depression in the Elderly Tuberculosis in Animal Handlers Oral Microbial Flora Abruptio Placenta Adolescent Body Image	Dr. T.K. Biswas, Kolkata Prof. N.S. Neki, Amritsar Dr. Ankur Barua, Sikkim Prof. U.K. Chattopadhyay, AIHH & PH Dr. S. Jayaram, Pondicherry Dr. Yosra Jarjees, Iraq Dr. Deepa Passi, New Delhi
4:00 - 6:00	CONVOCATION PROGRAMME VALEDICTORY FUNCTION	

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22nd - 24th October, 2005

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Name Age (years) Sex
1.
2.
3.

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Request for accommodation must reach the conference secretariat on or before 30th September 2005. The period from September onwards being the peak tourist season, early hotel reservations are advisable.

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Conference Fees	Up to 30th Sept. 05	1st Oct. 05 onwards
Delegates	2800	3600
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1 One day registration fees is Rs. 1200/-

1 PG students registering for the main conference will have to register as a delegate.

1 Conference Registration fee includes fees for CME programme also.

1 All payments must be made by Demand drafts in favour of "IMSACON 2005" payable at Jaipur and sent to the Conference Secretariat.

Abstract Submission

The Scientific Committee invites Abstracts for Poster and Oral Presentations. The abstract must be in approximately 300 words in English.

The paper must clearly state the objective, methods, results and conclusions. The presentation must have a title, names of all authors and the institution where the work was done. Please mention the name of the author who will present the paper and complete address, email and phone numbers.

Last date for submission of abstract : 30th September 2005. Kindly send on diskette or CD by mail or e-mail to the conference Secretariat.

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Organising Secretary, IMSACON 2005

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July-Sept. 2005

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