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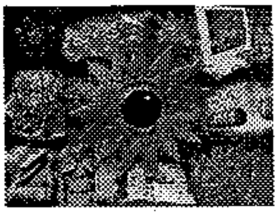

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CONTENTS

Consciousness in Philosophy and Science

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At the outset, the most perplexing fact about consciousness is that it is too familiar and intimate to common people. Our knowledge of consciousness is more direct than any other thing of the world. Hence, it appears that we all have acquaintance with consciousness and it is an easily understandable phenomena. Common people are not puzzled by the expressions "loss of consciousness" or "return of consciousness". But many who hope to express the meaning of consciousness in the form of a definition is not a simple matter. It is too complex and elusive to understand. Its nature can not be ascertained easily. Although it is embedded in all our experiences including experiences which are specifically cognitive. Today physicists, cognitive scientists, neuro- physiologists and also information technologists are interested to explore the nature and the role of consciousness in human life. It seems that studies related to consciousness in the contemporary intellectual world have gone beyond philosophy, psychology and neuro-physiology. From contemporary perspective, therefore, perplexity about the nature of consciousness is rooted in the multiplicity and diversity of the connotation of the concept of consciousness. In recent times physicists, cognitive scientists information technologists and new age people have discussed the nature of consciousness from their own perspective. Yet they could not reach in to a shared meaning. All of them try to explain consciousness, but they can not give a definition of the term 'consciousness'. They can not explicate what exactly it is? Why does it exist? How could it arise? What is the function of consciousness? All these questions are addressed to the ontology of consciousness. But it is very difficult to answer these questions. These questions are in fact 'what is' questions. 'What is' questions are answered in the form of a definition. The term consciousness as definidum itself being a primitive notion. It is difficult to define in terms of notions which are more primitive than the term consciousness. In this regard, John Dewey proclaimed that 'consciousness can neither be defined nor described'.[1] Julian Jaynes in 'The Origin of Consciousness in the break down of the Bicameral mind' asked : "This consciousness that is myself of selves, that is everything and yet nothing at all—what is it? [2] Even the International Dictionary of Psychology can not give a satisfactory definition about consciousness. International Dictionary of Psychology defines "Consciousness: The having of perceptions, thoughts, and feelings; awareness. The term is impossible to define except in terms that are unintelligible without a grasp of what consciousness means. Many fall into the trap of confusing consciousness with self-consciousness—to be conscious it is only necessary to be aware of the external world. Consciousness is a fascinating but elusive phenomenon; it is impossible to specify what it is, what it does, or why it evolved. Nothing worth reading has been written about it". [3]

In English language there are a large number of synonyms of the term consciousness. "Sensation", "perception", "apperception" "understanding", "intelligence"—all are its synonyms. Almost all the synonyms of consciousness have reference to awareness. But each of these terms expresses the nature of consciousness from a specific ground and presents only its partial meaning. It is well understandable that consciousness is an internal state of mind. It is associated with different mental states but it is not exhausted by different mental states. Hence, it is more than mental states. It is over and above mental states. The most interesting sense of consciousness is "experience". According to Thomas Nagel, a mental state is conscious if there is something it is like to be in that mental state. In other words, conscious mental state is associated with experience. A mental state is conscious if it has a qualitative feel. Qualitative feel is associated with quality of experience. Quality of experiences are known as phenomenal qualities or 'qualia'. 'Qualia' can be properties of 'internal' mental states. 'Qualia' is associated with mental states but it is not mental states. "To be conscious" means "to have qualia". "To have qualia" means "to have subjective experience".

From the survey of the works of philosophy of mind in the west since the era of positivism it appears that large number of philosophers develop materialist interpretation of consciousness. This means they deny the existence of mind including consciousness. Whatever is real is responsible to objective behaviour. Behaviourists assert that minds are not entities and mental states are not accompanied with the substantive and non-physical entities called minds. The positive aspect of behaviourism is mental states are behavioural states i.e. observable bodily behaviour. As linguistic philosopher, Gilbert Ryle considered that mind or mental world is not treated as a separate entity. We have no need to assume the existence of mind in order to account for the significance of sentences containing mentalistic expressions such as thinking, remembering, feeling, hoping etc. Such sentences are all disposition sentences. Mental words are described in terms of one's behaviour or disposition of behaviour. Dispositional word like 'believe', 'know', 'aspire', 'clever' signifies abilities or tendencies to do. To say that a person has a certain disposition is to say that habitually he does act in a certain way in a certain situation. Ryle has shown that the word, suppose, belief consists in having a tendency to speak and behave in a certain way. Our propensity to act and speak signifies what the belief actually is. Like belief, another mentalistic word 'intelligent' is a dispositional word. Usually 'intelligency' means the person's mind has the ability to solve certain problem speedily and accurately. Ryle utterly refutes that view. He invokes any performance which we say intelligent have no clues to the working of mind. One supposed to be intelligent if he works skillfully. As for example, a car driver who drives a car skillfully on a road may be called intelligent. His performance is said to be intelligent which reveals as his behaviour. Hence there is no mind behind behaviour and disposition. Ryle concludes that consciousness as the essence of mind which is claimed by Descartes does not exist.

In 1960s J.J.C Smart asserts Psycho-neural identity theory which shows the identity of the mental states with physical states. He wanted to deny the existence of any mental states in addition to behaviour. Mental states are identical with brain processes as well as neural events in the brain. However, this identification does not refer to they are synonymous. Such as 'pain' (mental states) and neural expression that is 'c-fiber activation' do not have the same meaning. It refers to the same phenomena which is just one thing, not two. Smart suggested that 'pain-states' which are over and above 'c-fiber activation' does not exist. So, mind is the same as brain. In other words, mind is identical with brain. Consciousness as an irreducible phenomenological property does not exist. [4]

In support of psychoneural identification David Armstrong considered a useful analogy in biological science. According to biological research gene is identical with DNA molecule. From Mendel's work it is also known that gene produces certain characteristic or hereditary characteristic of a person or an animal. Here a question arises, what the gene is. We know the expression of gene or what the gene refer to. But we do not know what the gene actually is. Through biological research DNA molecule found in centre of cells, produces hereditary characteristics. This means DNA (Deoxyribonucleic acid) molecule is the cause of producing hereditary character. Hence it may conclude the gene is identical with DNA molecule. Similarly Armstrong tried to put causal analysis of mental concepts. Mental states are necessarily involve in the cause-effect relation. Pain is caused by tissue damage and in turn it causes behaviours such as winces and groans. Now-a-days researcher in neurophysiology tries to establish that c-fiber activation in the brain is caused by tissue damage. Like pain, c-fiber activation is internal state in brain. It is also in turn causes winces and groans. So, pain is the c-fiber activation in brain .Thus Armstrong shows pain-states or mental states are identical with neural processes in brain .Ultimately, Armstrong argues radically consciousness as intrinsic property of the so called mental phenomena does not exist at all.[5]

There are philosophers, Searle and Popper among others who depart from the materialistic interpretation about consciousness. Searle says subjectivity is the special feature of consciousness which is not found in any physical state.[6] Behaviourism and identity theory are failure to explain consciousness in terms of the notion of subjectivity. But in the context of understanding of the nature of consciousness subjectivity is the primary concept. Conscious mental states are always states of a person and it can be studied within the first person parameter. According to Searle brain and consciousness are two separate domains of existence. Brain has objective existence and consciousness has subjective existence. Brain and consciousness are both real. In-eliminable subjectivity is the special character of consciousness. Searle suggests that consciousness has place in nature and explain its reality by particular cause-effect relation between brain states (physical states) and conscious states(mental states). Ultimately Searle tries to explain the interaction between physical states and consciousness without disturbing the autonomy of consciousness.

Consciousness has been talked about within science from the past. The 'existence of mind' denied by most of the physical scientists and it is almost universally accepted among scientists that there is nothing besides matter. Newtonian physicists thought everything in the world could be explained in terms of laws governing basic motions. Dualism (mind & body are separated by existence) is inconsistent with the mechanistic world view of Newton.

When the region of physical theory was extended to close in microscopic phenomena, through the creation of quantum mechanics, the concept of consciousness comes again. The laws of quantum mechanics does not formulate without the reference to the consciousness. The success of mechanistic and macroscopic physics and of chemistry overshadowed the obvious fact that thoughts, desires and emotions are not made of matter. Cartesian-Newtonian Science failed in the world of subatomic stratum of reality which is celebrated by uncertainty principle of Heisenberg. Scientific materialism also breaks down by Schrodinger's wave equation. In this regard the physicists Josephson suggests that subatomic reality is found to be to subtle and elusive. It can not be recorded by experimental observation.

These subtle, elusive entities of nature may recorded by direct experience. So, the first person perspective of observation is added to the third person perspective in material science.[7] Now, it is suggested in quantum mechanics, involvement of the observer in any observational lebel is an important feature. In other words, conscious mind of an observer plays an distinct role in observation. The discoverer or the subject as scientist must be conscious about his own identity at different steps or different moment of observation. It seems that modern physics is influenced by philosophical framework of understanding reality. In recent years, quantum theory is closely related with the Indian philosophical thinking i.e absolute consciousness is an act of direct mystical experience.[8]

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Acharya Jagadis Chandra Bose – Homage on his 150th Birth Anniversary

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Jagadis Chandra Bose was born on 30th Nov, 1958 at Mymansingh town of undivided India. His ancestral house was Rarikhal village near Dhaka. His father Bhagaban Chandra Bose was a Deputy Magistrate, a courageous man with deep swadeshi spirit. Contrary to his status and current custom, he sent Jagadis to a local Bengali school, where on his right side, sat son of his father's Mohamedan Chaprasi and on his left, sat fisherman's son so that Jagadis could know his country and neighbours.

Jagdis learnt the Ramayana and the Mahbharata from his grandmother and village jatras for which he had great attraction all throughout his life. He told to Patrick Geddis, his approved biographer that "it is through them (Jatra players and the reciters of the epics) that the highest national culture has been kept alive among the people. They are fast disappearing and we must either revive the institution or have its modern equivalent....... It should be! It must be! Then and then only shall we realize the true India" He was greatly inspired by these epics. Karna was his favorite hero. So favorite that afterwards he requested his friend, Rabindra Nath Tagore to write about Karna which resulted 'Karna Kunti Sambad', a classic Bengali literary work.

But when his father was transferred from Faridpur to Burdwan, he was sent to Calcutta, first at Hare School and then at St. Xaviers' School. After passing Entrance with scholarship, he got admitted at St. Xaviers' College wherefrom he passed F.A. in 1877 and B.A. in 1879. Here he was attracted to Physics due to Father Lafont,s demonstrative teaching.

Then it was a craze to become ICS or Barrister from London. But his father directed him to take science and to serve country. He left for England in1880. First he took medical course. But due to attack of Indian black fever being aggravated at dissection room, he had to give up the course after first year. Then he took science with Physics, Chemistry and Biology at Christ's College, Camridge and received Natural Science Tripos and B.S. from University of London in 1884. Lord Raleigh and Dr. Vines were among his teachers.

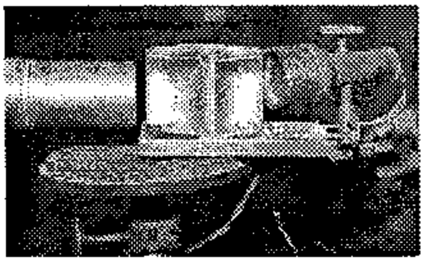
He had to carve his way through hard rocks of colonial British racist antagonism of the rulers and burocrates. After graduating from London, he was appointed in 1885 as professor of Physics in Presidency college in Calcutta by the intervention of the Viceroy of India, Lord Ripon. His appointment was strongly opposed by Sir Alfred Croft, then Director of Public Instruction of Bengal and Mr. Charles R. Tawney, Principal of the Presidency College. But he was granted one-third pay compared to his European counterpart, and thus Indians were being discriminated for their mystic mind. Britishers were of the opinion that due to oriental mysticism, Indians were not fit for science teaching and learning. In protest against racial discrimination he did not take salary for three years in spite of his tight financial condition. That time his father was suffering from various illness and was sunk in heavy debts due to his several swadeshi enterprises like opening bank, tea garden, technical school etc, all of which failed in absence of strong commercial motive. However, ultimately his talent was recognized and was granted full scale with retrospective effect .

He had to manage his research work in a 24 square feet small enclosure in Presidency college with his hard earned money with great conscientiousness. He had to spend and he spent a good time for teaching. He was not allowed any concession to teaching hours or class adjustments. S. N. Bose, famous for Bose-Einstein statistics, was one of his students.

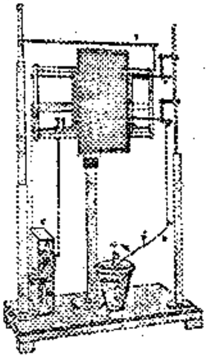
With the help of an untrained tinsmith he devised and constructed new apparatus for his research toward epoch making discoveries. Jagadis had great talent to make great use of simple things, In Royal Society he used Hairs,Bradshaw Time Table as polarizer. In 1888 Heinrich Rudolf Hertz (1857-94) produced and detected electromagnetic waves in the 60 cm wavelength range and in doing so he verified James Clerk Maxwell's (1831-79) electromagnetic theory. However, Bose was the first to produce millimeter-length radio wave. Such waves are now called microwaves, and are used in mobiles, radars, ground telecommunication, satellite communication, remote sensing and microwave method of transmission and of reception of electromagnetic waves. In 1894 he generated Electric waves in the room of Acharya Prafulla Chandra Roy that penetrated through intervening room where father Lafont was present and triggered a pistol at Prof. Pedler's room. In 1895 in presence of Governor General of Bengal, Lord Mackenzie in Town Hall, Calcutta, Bose transmitted electric

waves through three intervening walls to a room 75 feet away where that tripped a relay which fired off a pistol, threw a heavy ball and exploded a small mine. In 1896 the Daily Chronicle of England reported the incident. Popov in Russia was doing similar experiments, but had written in December 1895 that he was still entertaining the hope of remote signalling with radio wave. Hertz had used a wavelength of 66 cm; other post-Hertzian pre-1900 experimenters used wavelengths well into the short cm-wave region, with Bose in Calcutta and Lebedew in Moscow independently performing experiments at wavelengths as short as 5 and 6 mm. The first successful wireless signalling experiment by Marconi on Salisbury Plain in England was not until May 1897. The 1895 public demonstration by Bose in Calcutta predates all these experiments. But Bose refrained from patenting his dicovery . That resulted the official credit going the other way.

Invited by Lord Rayleigh, in 1897 Bose reported on his microwave (millimeter-wave) experiments to the Royal Institution and other societies in England.) Bose was the first Indian to be admitted in person to the sanctum sanctorum of English, thus western science. Lord Kelvin congraturalted Bose by stating that he was "literally filled with wonder and admiration...for his success in the difficult and novel experimental problem".



The 'Electric Engineer' expressed "surprise that no secret was at any time made as to its construction, so that it has been open to all the world to adopt it for practical and possibly moneymaking purposes". But he did not take patent for it. He firmly upheld his ideal of not making money with scientific discovery. *Science is for the mankind only* **That resulted the official credit going the other way**



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The Institute of Electrical and Electronics Engineers, IEEE (USA) has recognised that Bose's work actually predated that of Marconi. He was first to use a semiconductor junction to detect radio waves. His Galena detector was the first semiconductor device and the earliest photovoltaic cell. Neville Francis Mott, Nobel Laureate in 1977, remarked that "J C Bose was at least 60 years ahead of his time" and " in fact, he had anticipated the existence of P-type and N-type semiconductors." He dealt with equal aptness in both physics and bioscience.

In course of his research, he found that his apparatus(Coherer) did not work well for continuous hours. But after allowing rest, it regained its working strength. Inanimate matters also have fatigue. Response graphs of matters like a piece of tin against poisoning or anidotes were recorded in his Electric apparatus. Later on Bose invented Crescograph which can record plant's growth as small as 1/100,000 inch per second under which a crawling insect can be seen running at a speed of train. He showed that plants can "feel pain, understand affection etc." His finding of consciousness not only in plants but also in inanimate matters has great implication in Bharatiya Vedanta philosophy having far reaching consequences which are yet to be worked out. Thus Bose's work gave a fresh scientific impetus to the age-old Bharatiya wisdom of *oneness*. He declared – " It was then I understood for the first time a little of that message proclaimed by my ancestors on the bank of Ganges thirty ceturies ago. They who see but one, in all the changing manifoldness of the universe, unto them belong the eternal truth, unto none else, unto none else."

He showed that responses of plants and matters to the stimulant like heat, cold, poisoning etc are similar and concluded that there is no boundary between living and nonliving. Leading biologists like Sir J. W. Sanderson and Dr. Waller opposed him. They opined that oriental mysticism had misled him. Royal society of London postponed the publication of his paper. He became ill and operated surgically at Nivedita's house. He needed time for research to prove his theory beyond doubt. But those biologists conspired against him and persuaded Secretary of India not to extend his leave. That was really hard time for him. He was hurt at mention of oriental mysticism. It made him more adamant. Then Nivedita appeared at Basu Battle. It was decided to publish his work in form of book. Nivedita helped him a lot in drafting, editing and writing review of the book. At this time by the efforts of Nivedita, Mrs.Sara Champman Bull, disciple of Swami Vivekananda, came to his help. They had relation of mother and son. She persuaded J C Bose to take patent of his Coherer. Mrs. Bull financed the publication of his book. She also kept a good amount of money in her will for Bose Institute.

Provocation of oriental mysticism led him think that his failure in anyway would deprive his country from science cultivation. "Not only we have perfect imagination, but also we have exact analysis" Ultimately he overcame all the hurdles through hard work. The Fortnightly Review wrote defending oriental mysticism –"In Sir Jagadis the culture of thirty centuries has blossomed into a scientific brain of an order which we cannot duplicate in west." The Times, The Spectator wrote also similar praises. His findings subsequently influenced subjects like physiology, chronobiology, cybernetics, medicine and agriculture

He differed from Charles Darwin regarding sruggle for existence that "It is misreading of the law of Nature to regard conflict as the only factor in evolution;far more potent than competition is mutual aid and co-operation in the scheme of life."

Acharya along with family went to pilgrimage several times with Nivedita to Buddha Gaya, Kedar Badri, Kashmir, Mayavati etc. He rediscovered India. He wrote to Rabindra Nath –"Now I am able to understand our national greatness. Seeing through eye of foreigners makes one blind. If I have to take birth again, then I wish to be born hundred times in Hindusthan"

Rabindra Nath Tagore was his intimate friend. His wife, Mrs. Abala Bose, a fourth year medical student at time of marriage, provided strong support and family security. Sister Nivedita had great cotribution behind J C Bose's success. Tagore wrote " Nivedita was great inspiration for his works. From then Bose's work spread in world platform" By her persuasion, his relation grew with Swami Vivekananda. J C Bose went to Belur Math and Swamiji also came to J C Bose's house. Swami Vivekananda had all out praise for his presentation in Paris in 1900.

Nivedita played the great roll for the patriot, nationalist scientist savant Acharya J C Bose. He also did not forget her. He founded, Basu Bijnan Mandir(Bose Institute)- 'not merely laboratory, but temple of science", first scientific research institute in India in 1917 ***on the altar of holy ashses of Sister Nivedita*** and dedicated it to the nation. The "lady with the lamp" is just to remember Nivedita. J C Bose wrote to her sister Wilson – "The institute is the embodiment of her prayer." Nivedita discovered 'Bajra' at Buddhagaya, a symbol of selflessness, symbol of dedication. She planned this to be adopted in our national flag. Acharya decided this to be the symbol of his institute.

Bose was conferred with rare honors: ● Companion of the Order of the Indian Empire (CIE) (1903) ● Companion of the Order of the Star of India (CSI) (1912) ● Knighthood, 1917 ● Fellow of the Royal Society (1920) ● Member of the Vienna Academy of Science, 1928 ● Member of the League of Nations' Committee for Intellectual Cooperation. He was highly appreciated by Lord Kelvin, George Barnard Shaw, Lord Huxely, Romand Rolland and others. Barnard Shaw presented his books to J C Bose with writings"from the least biologist to the greatest biologist" and Roman Rolland sent his gift with caption"To the revealer of new world". He is considered the father of millimetre radio wave and is also considered the father of Bengali science fiction.

Britain's Cambridge University put up a special memorial plaque in honour of its almuni, Indian scientist Jagdish Chandra Bose, to mark his 150th birth anniversary alongwith eminent personalities like biologist Charles Darwin and poet John Milton, whose plaques were put up several years back.

Sir J C bose's constant struggle to uphold the image of India through science will remain great inspiration for the coming generations.

Ionic liquids – the 'green' solvents

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Abstract

Ionic liquids, consisting of ions and ion pairs, have negligible vapour pressure and have been found to be extremely useful for conducting a wide variety of organic reactions in them with the simultaneous scope of solvent recovery which characterizes them as 'green solvents'.

Key words: Ionic liquid, designer solvent, green solvent

Introduction

It is known that chemists require solvents as the medium to prepare a variety of products – both inorganic and organic in nature. Many of these reactions are carried out in aqueous medium and that is also beneficial for the society because water causes no environmental pollution. But it is not possible to carry out each and every reaction in aqueous medium due to various reasons. Use of organic solvents for undertaking chemical reactions is therefore very common. These solvents are usually volatile and required in large quantities particularly in industrial processes and therefore have a damaging effect on environment. Chemists around the globe are in search of solvents having no hazardous effect on environment, i.e. green solvents. Accordingly solvents which are liquid at and below room temperature and are non volatile at the same time, possessing fascinating properties have enough reasons to be accepted as such solvents. Ionic liquids are such 'green' solvents.

Another most interesting feature of ionic liquids is that the two components viz. the cation and anion, with which ionic liquids are made of, can be varied to get a set of desired property in the solvent. Thus the solvent can be 'designed' according to the requirement of a reaction in mind and hence these solvents are sometimes called 'designer solvents'[1]. The melting point, viscosity, density and hydrophobicity can be changed by simple variation in the structure of the constituent ions.

The main characteristics of ionic liquids are:-

1. They are liquid salts consisting of ions and ion pairs.
2. The cation and or anion components are usually large.
3. They are thermally stable over a long liquid range (often > 200---°C).
4. They are mainly composed of poorly coordinating ions.
5. Unlike organic solvents, they are non volatile (have negligible vapour pressure). Hence they may be used in high-vacuum systems and eliminate many containment problems.
6. They have generally moderate specific and molar conductivities (molar conductivity usually < 10 Scm^2 mol^{-1})
7. They are moderately polar good solvents for a wide range of both organic and inorganic reactions.
8. Unusual combinations of reagents can be brought into the same phase in it.

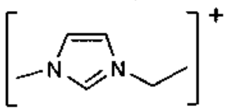
Types of ionic liquid

There are two types of ionic liquid – i) Simple salts, made of a single cation and anion. ii) Binary ionic liquid, where melting point and other properties depend on the relative amounts (mole fraction) of the components. For example, the composition of tetrachloroaluminate(III) ionic liquid can be 'basic', 'acidic' and 'neutral' depending on the apparent mole fraction of $AlCl_3$ {$X(AlCl_3)$} present. Ionic liquids with $X(AlCl_3) < 0.5$ contain an excess of Cl^- ions over $[Al_2Cl_7]^-$ ions, and are called 'basic'; those with $X(AlCl_3) > 0.5$ contain an excess of $[Al_2Cl_7]^-$ ions over Cl^-, are called 'acidic'; when $X(AlCl_3) = 0.5$, they are called 'neutral'.

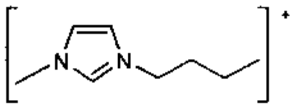
Examples of simple room temperature ionic liquid cations:

$[NR\text{-}_{X}H_{4-X}]^+$ $[PR\text{-}_{X}H_{4-X}]^+$

Alkylammonium alkylphosphonium



1-ethyl-3-methylimidazolium
[emin]



1-*n*-butyl-3-methylimidazolium
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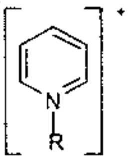


N-alkylpyridinium

The anion counterparts are usually Cl^-, $[ClO_4]^-$, $[NO_3]^-$, $[PF_6]^-$, $[BF_4]^-$, $[CF_3SO_3]^-$, $[AlCl_4]^-$ etc

The ionic liquids which have received significant attention in recent times are:

$[EtNH_3][NO_3]$, $[emin][NO_3]$, $[emin][ClO_4]$, $[emin][PF_6]$

It has been found that the melting points and also the miscibility of these ionic liquids with water depend on the length of the 1-alkyl group e.g. 1-alkyl-3-methylimidazolium tetrafluoroborates are miscible with water where alkyl chains are below 6, and at or above 6 they become immiscible with water. This property is often exploited for the separation of products by solvent extraction. It has already been stated that the properties of these ionic liquids can be adjusted to suit a particular reaction type and this has made them as designer solvents[2]. Reactions in ionic liquids require no special apparatus or methodologies and often quicker than that of conventional organic solvents[3].

Preparation

Halogenoaluminate(III) ionic liquid: In general an imidazolium or pyridinium halide is mixed directly with the appropriate Al(III) halide in the desired ratio. An exothermic reaction starts on mixing and the two solids melt into a liquid. It is better to add the ingredients into a small amount of the ionic liquid obtained from the previous treatment which allows more effective stirring and greater dissipation of heat generated during the reaction.

$$[\text{N-butylpyridinium chloride}] + AlCl_3 \quad [X(AlCl_3)] = 0.43 - 0.66] \rightarrow [Nbpy][X(AlCl_3)]$$

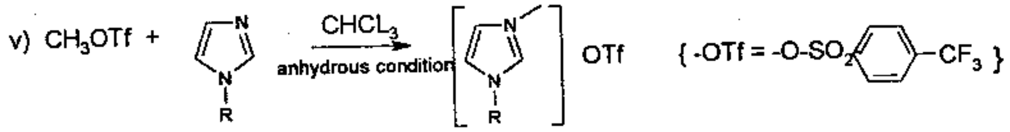
Preparations of some other ionic liquids are outlined below:

i) $[emin]I + Ag[BF_4] \xrightarrow{CH_3OH} [emin][BF_4]$

ii) $[emin]I + [NH_4][BF_4] \xrightarrow{acetone} [emin][BF_4]$

iii) $[emin]Cl + HPF_6 \longrightarrow [emin][PF_6]$

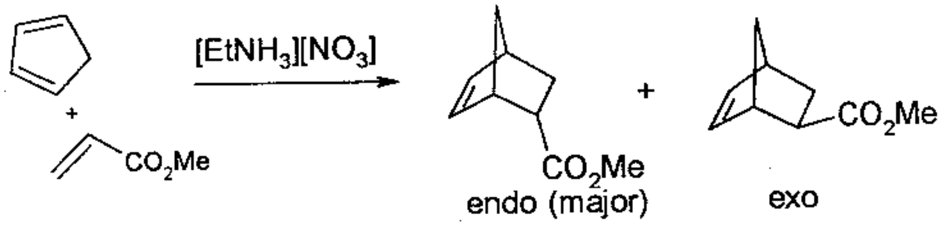
iv) $[R_4N]Br + Li[BF_4] \longrightarrow [R_4N][BF_4]$



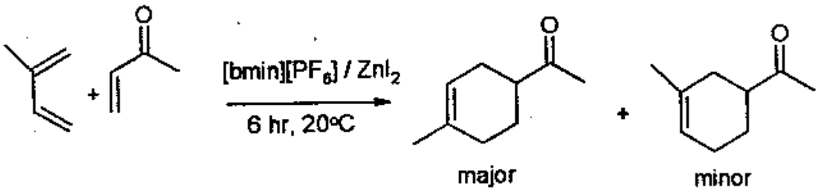
Reactions

A wide variety of reactions have been carried out in ionic liquid solvents and the findings are quite interesting. In many cases significant rate accelerations as well as enhance selectivities have been observed. In the following section some of the more common and interesting reactions are cited to illustrate the importance of these solvents.

Diels-Alder Reaction: Though water is the common solvent in this reaction, some neutral ionic liquid solvents have been tried in this reaction and a greater selectivity has been observed. This solvent system offers an advantage for using moisture sensitive reagents in it. e.g.

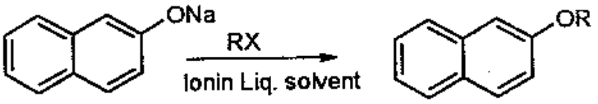


This reaction has also been investigated in some other ionic solvents like [emin][PF$_6$], [emin][BF$_4$], [emin][ClO$_4$], [emin][CF$_3$SO$_3$] and [emin][NO$_3$]. The same general trend is observed in all the cases[4].
Another example:

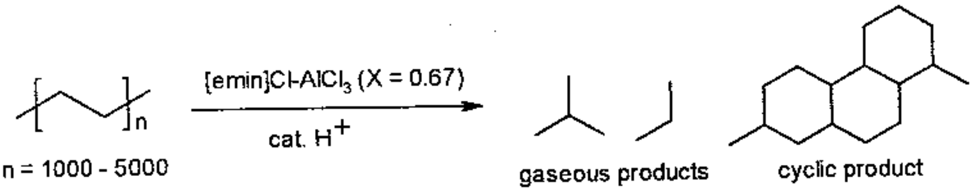


The addition of mild Lewis acid ZnI$_2$ improved the selectivity of the '*para*' like isomer from 4:1 to 20:1 in ionic liquid solvents[5].

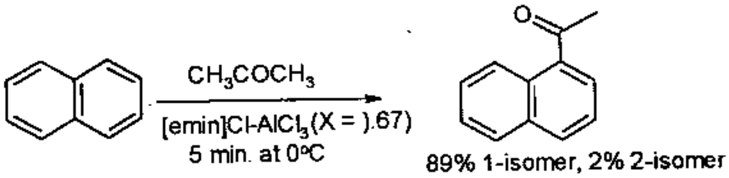
O-Alkylation: Like polar aprotic solvents, the regioselectivity of O-alkylation can be increased to more than 90% in ionic liquid solvents like n-Bu$_4$PBr, n-Bu$_4$NBr, [emin]Br and n-Bu$_4$PCl [6].



Isomerization and Cracking of alkanes can be undertaken at a much lower temperature (90°C) as compared to conventional reaction temperature (300 - 1000°C):



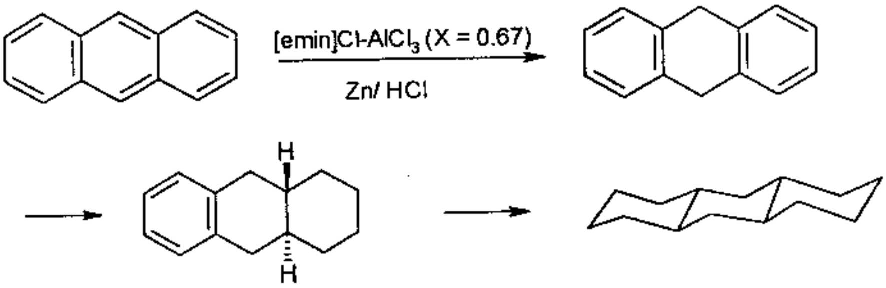
Acylation of naphthalene at 1- position: Acylation of naphthalene under normal Friedel-Craft conditions gives both 1- and 2-naphthyl methyl ketone and the product ratio depends on the nature of the solvent; e.g. with acetyl chloride in CSO_2 as solvent, 1- and 2- products are formed in a ratio of 3:1, in nitrobenzene, 1:9; [emin]Cl-$AlCl_3$ (X = 0.67) gives the highest known selectivity for 1-product[7].



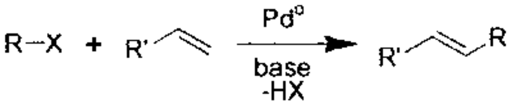
Friedel-Craft reactions have been fond to occur best in [bmin][PF_6] at 150°C, without any catalyst and the amount of waste formed is reduced considerably. Moreover, the reactions in ionic liquid can also be undertaken without strictly anhydrous conditions and inert atmosphere.

Catalytic Hydrogenation:

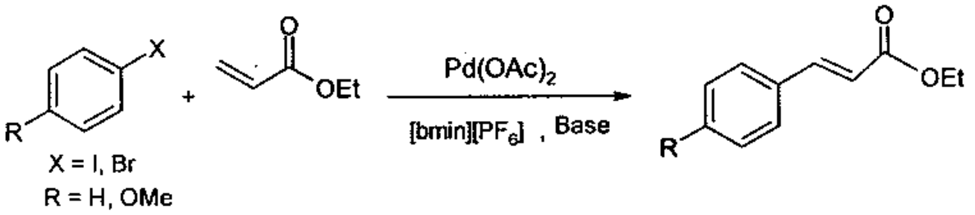
Homogeneous transition metal catalysts can be used for undertaking hydrogenation reactions in neutral ionic liquids. The process offers easy separation of the products from the ionic liquid and catalyst. Cyclohexene, benzene, anthracene etc have been reduced completely under this condition. Use of Wilkinson's catalyst [$RhCl(PPh_3)$] and [$Rh(cod)_2$][BF_4] (where cod = cyclooctadiene) is very common.

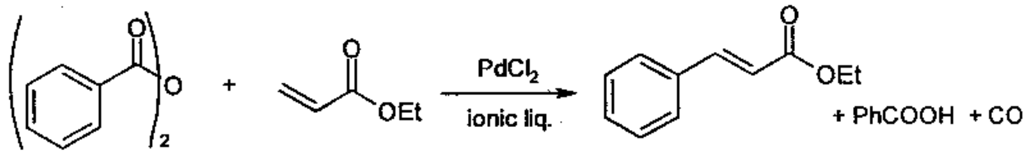


Heck Reaction (also called the **Mizoroki-Heck reaction**):
This reaction involves C-C coupling of aryl halides or triflate with alkenes in the presence of Pd catalysts to form a substituted alkene. The halide or triflate is an aryl, benzyl, or vinyl compound and the alkene contains at least one proton and is often electron-deficient such as acrylate ester or an acrylonitrile. The catalyst can be tetrakis (triphenylphosphine) palladium(0) or Pd(OAc)$_2$. The ligand is PPh$_3$. The base is triethylamine, potassium carbonate or sodium acetate.



This coupling reaction is stereoselective with a propensity for *trans* coupling. In the presence of an ionic liquid a Heck reaction proceeds in absence of a phosphorus ligand. The Heck reaction was found to proceed markedly more efficiently in the ionic liquid 1-butyl-3-methylimidazolium bromide ([bmim][Br]) than in the analogous tetrafluoroborate salt [bmin][BFO$_3$].[8,9,10]





Nucleophilic Substitution

It is known that nucleophilic substitution reactions are undertaken in polar aprotic solvents like DMF, DMSO etc. but they often create problem during their separation from the product. These reactions, however, can be better carried out in ionic liquid solvents because the products of the reaction as well as the inorganic salts can be separated very easily by solvent extraction with organic solvents and water respectively and the ionic liquid can be recovered for next use.

Conclusion

It is evident from above that room temperature ionic liquids provide the chemists with a great scope for undertaking a wide variety of organic reactions and synthesis under ordinary conditions. In the preceding paragraphs only a few examples have been cited to illustrate the objectives and there are many such examples in literature. The reactions generally require no special conditions, apparatus, and methodologies and generally very easy to perform and often the reactions in ionic liquid are quicker also. As the solvents can be recovered, the cost of experiments gets reduced and consequently the threat of environmental pollution becomes insignificant. Obviously, there is ample scope of further development in this system. It should be mentioned here that the reasons behind these startling differences from the molecular solvents, rather 'ionic liquid effect', are not totally clear.

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Rethinking Environmental Movements in India: A Theoretical Journey from Critical Perspective

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Introduction :

Environmental movements or social movements related to environment are not easy straight-forward subjects. There are intricacies and nuances woven into it. One needs fine mind, empathy and sensitivity to appreciate their origin and nature. Today when consumerist culture is set to become all-pervading, it is all the more difficult to get such a mental mould. Again it is a subject that needs re-reading since one can hardly get nearer to the core of any of such social enterprise at the first attempt. So, at a glance what one sees might appear somewhat different in subsequent review. Also it is quite uphill on the part of any serious observer of societal dynamics to provide with a systematic form because never ever such movements follow any definite pattern.

Part-I: Identifying the problem & locating the contemporary situation

The author of the present discourse has the feeling of having got entangled in an irony. It is primarily because the time chosen for writing on environmental movements in India coincides with the signals of their decline. This puzzling uneasiness gets confirmed in exactly what Prof Satyabrata Chakraborty says about environmental movements in India that, of late, they seem to have lost much of their earlier fervour. In a recent interesting article entitled 'Environmental Movements' in India: Revolution Eclipsed? he talks about the good days and the bad turn. One seriously thinking on the issue cannot but concur when he says that for some years now, we have neither any significant report of environmental movements in general or any intensification of any of such movements. The Chipko has lost its steam. The Tehri movement could never crystallize. The Narmoda Banchao Andolan seems to have been stuck in an impasse. A general mood appears to have set in suggesting that it is neither feasible nor desirable to halt the process of globalization or to stay away from it and the concomitant spurt in economic growth. The fear of environmental degradation has proved a feeble and manipulable deterrent, if at all, in this blind pursuit of growth.

It is also important to raise a critical point at the outset about the vigour of such movements since talk of decline presupposes an earlier stage of vibrancy. So, obviously the question that follows is—have there really been many environmental movements in India in the past so that we can talk about decline now? According to Prof. Chakraborty the answer depends on how we define **environmentalism**. If **environmentalism** presumes the 'right' of nature not to be disturbed, there has actually been none. Deep ecologists like Vandana Shiva have proved more successful in stirring academia rather than common man on the street or ordinary villagers whose primary concerns have always been to eke out livelihood and not environment per se. It does not, however, mean that local people are not concerned with such movements, but their approach to environment is mediated mainly by issues pertaining to their daily life-struggle. Taken in this sense, thought of preserving ecological balance in the long run interest of humankind is conspicuous by its absence in the general pattern of environmental movements in India, except the Silent Valley campaign in Kerala. **In most cases, therefore, what we get to hear are movements for environmental protection—the movements that sometimes emerge from local peoples life and subsistence questions and then shift to their right to local natural resources** like land as their home and forest as their source of subsistence and concomitantly protection of nature from commercial exploitation which the state in connivance with commercial organizations is alleged to have been doing since the colonial times. **In all these cases obviously the line of demarcation between environmental movements and social movements for local peoples' right to livelihood or more generally speaking human rights movements is very thin and narrow.** So, this is one major problem that tends to confuse us while we categorise certain movements as environmental because they are neither exclusively so, nor do they prioritise the agenda of environment in a truly crusading spirit. **Often the problem is that there exists a gap between what the movement is and how it is reported in some media by the reporters and intellectuals.** The role of media and also some intellectuals in this connection is that of a double-standard. Sometimes, they support such movements, sometimes not. What lies hidden beneath their stand to support or to oppose is that they represent corporate interest rather than any genuine concern for environment. Hardly media plays a balancing role in reporting news inputs candidly.

It has become fashionable to classify certain movements as environmental or to ascribe environmental orientation to some social movements linked with issues like water pollution or conservation of water bodies and marshy lands etc. But, these are all, by and large, devices more from the point of view of drawing popular attention in the name of environment than thinking environment in total sense. As such, there is no dearth of such initiatives in coming to terms with pollution, but no sooner these initiatives take off the ground than they start getting stumbled or stuck at a point in the absence of long-term vision and tenacity to carry forward to produce long-term effects. In India, like most developing countries what matters often is popular hype created around the issue rather than cool and serious thinking on the issue. Examples abound when it has been found again and again that the idea behind showcasing environmental issues is just to get funds from some agencies even outside one, for doing patch-works here and there and meeting the cost of such superficial engagements rather than hotly pursuing the issue in terms of developing best possible alternative, issues involved in the implementation of such plans, so on and so forth.

Part-II: why so-called environmental movements are getting currency?

Here a question arises as to why such so-called environmental movements are getting currency. In our rejoinder we think it is primarily because now a day lot of money is coming into such movements and not due to gravity of issues. Now public and private agencies, even the MNCs at global level are competing and contending for establishing their environment-friendly pretensions. Eco-friendliness has become the buzz word of development in the post-globalization period and from World Bank to any business organization nobody is opposed to it at least in vocal propagation. As such, much of recent spurt in awareness and concern for environment protection at the policy level is a donor-induced one and at the grassroots level it can largely be attributed to local activists and NGOs (Reddy, 1997). So far as NGOs are concerned, they are found to work on the basis of two-pronged strategies. First, they receive money for environmental protection and secondly, often they also work for rehabilitation of project-affected people, imparting training to them for making alternative arrangements for their livelihood etc. Here again the question is how far they are truly playing this role, i.e. awakening and educating the local people and generating veritable alternative source of income for the affected masses. If so, the pertinent question is why not local masses themselves take the lead, because they are the first victims of degradation of nature. In recent time one may have noticed also that the villagers living next to one thermal power station in West Bengal forced the authority to close down some its units for not taking appropriate steps in disposing fly ash other than depositing it in a pond nearby. They wanted clean pollution free environment, not power—the point which may be construed in different ways. However, what is beyond doubt is that these local people are supposed to have intimate knowledge of their natural surrounding which they are to take care of. But the question is why such social initiatives are grossly missing at the level of local community? It is true that local communities' understanding of nature are far deeper than any outside agency which often has to bank on local people's knowledge to work in interior locations. But also what is natural in the grassroots conditions of poverty and lack of awareness among local people is the marginalization of environmental aspects to the contemporary social movements in India. **Therefore, it would be simply a theoretical fancy to conceive of social movements in purely environmental terms at the expense of local people's conditions of existence, if not contradiction in terms. It is because alienation of locals would then divest these movements of their social character.** The problem is—even if one talks of a social movement having strong environmental connection, any single issue based movement is unlikely to enthuse people in most cases unless that spills over into other areas related to their daily lives and broader social existence and thus linger.

Part-III:The problem of defining environmental concerns from rural & urban situations

Again we do confront another problem that is how to define environmental concerns from our different social situations— rural and urban. In rural India, for example, nature continues to be the source of life, it provides subsistence and meaning, and it contributes to self-definition of village-folk who they are. It is also unpredictable and often causes starvation during natural calamity, drought, pestilence etc. For others who predominantly live in urban areas, priorities encompass pollution of air, water and land. In this connection it may be noted that PILs have been lodged against holding book fair in Maidan—the green heart of the city of Kolkata. This might well be seen as publicity stunt and critics finger at lawyer cum environment-activist Subhas Dutta that he is out to create gimmick. There is a point in it in so far as it seems that out of so many issues connected with environment his focal concern is the city. It is not quite true however because Mr Dutta was also found fighting industrial pollution in Howrah. But note these are by and large initiatives on the part of a section of advanced middle class city-dwellers, not the city's common rank and file who struggle twenty four hours for hands-to-mouth existence.

For them there is hardly any time to ponder over city's environmental health. Of late, another issue hitting newspaper headlines is the pollution controversy of two-stroke autos which are sought to be replaced by four-stroke LPG engines for being more-eco-friendly vehicles. Similar other issues concerning urbanites are those of expanding slums, loss of green due to illegal construction and hoarding, choking of drainage by plastic bags, destruction of the ozone layer, climate change, global warming etc. So the issues are clearly divided and they indicate the existence of complex and vastly differing relationships with nature in these two domains—rural and urban. All these make it problematic to generalize environmental stakes in city and village at a single stroke. Of course, some might point to this rural –urban divide to be a flimsy and naïve one today since modern development is increasingly ironing out the distinction between the two by making tremendous inroads into the rural backwater in recent times(e-facilities in remote villages for example). The point is tenable no doubt. But it is also to be taken into account that issues like tree-felling for hoarding or choking of drainage by plastic bags, however important, are still not burning concerns in rural areas. It does not, however, mean that poor villages are not used to plastic bags or that their society is not as consumerist as to have less hoarding. Still what is being stressed here is that development, free market etc. have to fit with diverse nature of different localities. That is why when global warming is a burning global concern today and for that matter global legislations are necessary for combating them everywhere, going about to address these issues at grass roots level, the approach, in practice, has to fit locally. So, one cannot simply wish away the distinction between city and village society. It is not being said at all that city is having more environmental hazards than the countryside but the nature of their problems clearly vary from one another. That is why it may be problematic if we equate city-based environmental middle class movements with ones having rural origin. Also a point to be noted in this regard is that in city environmental movement is likely to be organized more easily not only because the middle-class intellectuals are active here or the chance of media attention is likely to be more but also because there are other options of livelihood for ordinary folk in city. So, one can afford to switch over to other options. In rural areas, on the other hand, environmental movements in the extreme extent can go against the livelihood of ruralites.

Part-IV: Are environmental movements & movements against environmental pollution synonymous?

Now let us tread into another fuzzy area by asking whether environmental movements and movements against environmental pollution are synonymous or not. The answer is although used interchangeably both cannot be clubbed together for obvious reasons.

First, when we talk of movements against environmental pollution we actually talk in terms of understanding different sources behind any such pollution and also envisage the need of creating public awareness of it. Here our main concern is to check the cause, formulate alternative workable solution and mode of public action like awareness campaign at community level. All these are undertaken to see that questions of pollution cannot stall the process of development; rather these can be taken care of so that development and concomitant changes acquire greater legitimacy and smoothness to carry on. So, here the approach is limited to the purpose of devising protective measures either to restrict environmental pollution or to provide with safeguards in minimising the ill-effects of pollution. Still such protective measures cannot be discounted because springing from protection things can lead towards preservative approach to environment.

On the other hand, the term environmental movement is quite comprehensive in which priority is attached to the agenda of environment. Development and related matters are considered appendage to it. Thus **environmental movement is a social movement tuned to the promotion, cultivation and nurturing of nature and its biotic and a biotic surrounding. Here nature assumes an autonomous value, not an instrumental value** or one which can be manipulated, but essentially a value in itself. For example, it would be mistaken to equate the experience of the Green Revolution with environmental movement of any sort. Thus what happened in the aftermath of the Green Revolution was that people had started becoming aware of the ill-effects of the use of chemical manure, pesticides along with the crop variety which was highly water dependent. So, later there was a shift in the strategy to manipulate nature from hitherto high yielding variety to a different type of farming. It is okay. But this attitude did not reflect natural bond that human beings have with nature. If nature is considered our mother at whose lap we are born and brought up only then it is possible to understand the deeper value of nature that cannot be neglected, far less exploitable. This point can be further extended to include the right of all biotic species, i.e. bio-centrism in stead of anthropocentrism alone. This position might seem a bit romantic to someone since in bourgeois society it is common tendency to think from selfish, possessive individualistic and anthropocentric point of view. This bare selfish individualism is clearly visible in liberal accounts of Locke and many other contemporary liberal thinkers.

Part-V: Locke's theory of property and nature's right to free self-subsistence

Locke's theory of property, for example, is based on the argument that all the resources on this earth are meant for the use of men for their advantage and convenience. It further tells that whenever man mixes his labour with natural resources to make them usable he can claim them as his own as a result of his own labour. Its contradiction lies hidden in the fact that servants won't be entitled to similar right to property when they mix their labour with anything in serving his master's. However, such a take on nature seems natural from two angles.

First, it depends on how one sees nature. So, one is free to view nature as sort of commodity, and

Second, the condition of scarcity of this commodity introduces the mad race for occupying it in which man divests nature of its own right of survival from human intervention and domination.

So, the basic contention of the author is that individual right to property directly conflicts with nature's right to free self-subsistence. Theoretically it is like not only defending nature's right vis-à-vis human society, but also arguing a case where both would enjoy equal rights without disturbing the other. But this is not the case in liberal political theories, even so in neo-liberal entitlement theory of Robert Nozick. In this framework Nozick would not object if an owner of a plot (since he is a just owner due to just transfer or just acquisition at the initial stage) decides to cut some of his trees and sell them off to a saw-mill owner. This is as anti-nature as unjust to say that since industries have government permission, they can pollute nature also. Forget not that government cannot formally permit industries without assessing their impact on environment. But there are lobbies to soften the attitude of government and extract permission without much delay. Similarly lacunae in the laws are also exploited and taken advantage of to run industries like those of sponge iron.

Often the plausible argument against closing down such industries is to keep in view the questions of livelihood of those working in such small industries. So, it is like an unresolved dilemma as to whether one should think in terms of long-term potential benefit of mankind and keep environment clean by taking harsh decisions to close down such units or should one think in terms of short-term real losses of some sections of society. Still the point that can be said here is that in the third world states given the nature of democratic politics and compulsion of votes, it is very difficult to shut one's eye to people's economic conditions. Therefore, in case of conflict between long-term environmental benefits with short-term real losses of some sections of society, the former is less likely to figure in the agendas of vote-seeking parties. It is precisely because they won't like to test their electoral fortunes by riskily lending support to environmental concerns without keeping in view real losses of some sections of society. Political parties are not all too liberal, altruistic organizations. At times they may show off to be different but after all that is also a tactic to get some mileage. As Prof. Chakraborty has aptly pointed out that behind the success of the Silent Valley Movement in Kerala one important factor at that time was believed to have been the then Prime-Minister Indira Gandhi's wish to avail of the opportunity to establish her eco-sensitive credential at the international forums without much risk of internal political trouble.

Part-VI: Nature & environment bore the brunt of State-led development in the name of welfare

True nature-friendly attitude is rarity indeed. Not only liberal and neo-liberal theorization but also acts of welfare states can be interpreted as appropriative of nature. What happened in the erstwhile colonies in the heydays of post-colonial reconstruction in the 1950s and 60s were nothing short of trampling nature under the feet of development by stalling rivers with big dams, setting up heavy industries and constructing infrastructural facilities. In this mega show of 'state-led development' nature was not only over-exploited but development at the cost of nature was glorified so much that the entire process, though lop-sided from the very inception, appeared rational and justifiable. The whole bogey of misconception that seemed to have guided national policy-makers and planners through the 1960s and 70s was that if there are omissions, okay, there is science to take care of the problem. This attitude continued till the illusion was over and it was discovered that already irreparable damage was done to nature in the pursuit of west-centric development. So, it was time to sort of turn around to question the path of our development and seriously review the strategies.

In short, the drive towards development and concern for environment are tied together. It is because whatever line of development may be adopted, it would have some repercussion on environment. So, how to deal with that? It also opens up Pandora's Box of questions. For example is it desirable to sacrifice development in the name of environment and if so, how far practicable that would be? For instance, now in Kolkata tracks of electric driven tram hailed for being an eco-friendly public transport system, are being laid in concrete. But it would not only obstruct rain water to easily trickle down into the earth underneath and refill water therein, but might also aggravate the existing problem of water-logging during heavy downpour in the city. So, how far this sort of development project is sustainable for environment?

A no less important question that comes to mind is whether there is a kind of hidden politics behind such idea to dichotomise development and environment so much so that we can't have two at the same time. Is there any possibility also that the line of development would not hamper environment? For this some are talking in terms of environmentally concerned famous economist E.F. Schumacher's idea: 'small is beautiful'. No doubt it has profound implication as a guiding mantra for development. For instance small cottage industries are less polluting compared to giant mills. Similarly using solar cells prove useful and environment-friendly for electrical illumination at small pockets. But when it comes to the issue of supplying electrical power to the dozens of villages one quickly understands the necessity of installing a big power plants. Therefore the application Schumacherean idea in the context of developing countries like India is not justifiable because it amounts to opposition to all big programme of national development. In spite of this today some activism in India and other developing countries in the South that included environmental aspects in the later stages like Narmoda Banchao Andolan have grown along Schumacherean lines. The NBA is the best-known case which shows that its activists are not only against the Narmada dam project but is typically against all mega dams causing mass displacement. Although it has assumed an environmental orientation in so far as it speaks of keeping the natural flow of river system and river valley with human settlement, it remains incomplete as it fails to generate and extend the movement to similar concerns of displacement of people.

Now sustainable development is the slogan of the day. But in fact it is an attempt to modify the current capitalist development slightly. As such, it has nothing to offer any alternative thought on development. Real alternative thought, on the contrary, is one with genuine concern for nature and environment, which not only thinks in terms of wellbeing of human beings but also rooted in bio-centrism. In the system of environment all biotic and a-biotic components play vital complementary role. Similarly Environmentally concerned social movements are of various types. Sometimes it comes out as local initiatives for saving even a small pond from illegal filling; sometimes people take to judicial activism with the resultant outcomes of unmixed blessing and still in some other cases we find dedicated activists fighting for the cause of human rights as well as environment. Even contemporary anti-globalisation movements are fast acquiring environmental dimensions. Thus multiplicity, plurality of approach, inherent tension, dialogue with the proponents of development, shared Gandhian value of non-violence, civil-society initiatives, judicial activism, and NGOization are markers of environmentally concerned social movements in India.

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Photophysical studies of some dyes in aqueous solution of triton X-100

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Abstract

The spectral (both absorption and fluorescence) and photoelectrochemical studies of a few selective dyes, namely, anionic erythrosin B, neutral riboflavin and cationic safranin O have been carried out in aqueous solution of triton X-100, a neutral surfactant. The results show that the ionic dyes, erythrosin B and safranin O form 1:1 electron donor – acceptor (EDA) or charge – transfer (CT) complexes with triton X-100 both in the ground and excited states, whereas neutral dye riboflavin in its excited state forms 1:1 complex with triton X-100.In these complexes, the dyes act as electron acceptors whereas triton X-100 acts as an electron donor. The fluorescence spectra of erythrosin B and safranin O in presence of triton X-100 show enhancement of fluorescence intensity with red and blue shifts respectively while riboflavin shows normal quenching of fluorescence. A good correlation has been found among photovoltage generation of the systems consisting of these dyes and triton X-100, spectral shift due to complex formation and thermodynamic properties of these complexes.

1. Introduction

Investigation of photoinduced electron transfer or redox reactions in surfactant solution are not only inherently interesting and relevant to the understanding of photobiology, but they are also important for efficient photochemical conversion and storage of solar energy. Surfactants or surface active agents, on the other hand, help to achieve the separation of photoproducts by means of hydrophilic-hydrophobic interaction between the products and interfaces .From the spectral and photoelectrochemical studies of phenazine, thiazine and xanthene dyes in aqueous solution of surfactant, it has been concluded that all the dyes, both cationic and anionic, act as electron acceptors and form 1:1 charge-transfer(CT) or electron-donor acceptor (EDA) complexes with neutral surfactants which act as electron donors. There is no interaction of these dyes with the surfactant of same charge whereas the interaction of these dyes with oppositely charged surfactants is coulombic in nature. For a better understanding of the nature of dye-surfactant interaction, the studies of spectral, photophysical and thermodynamic properties of dyes of diverse nature in aqueous solution of surfactant are essential. In recent years, Safranin O, a cationic dye, has received considerable attention because of its photoredox properties and for its application in some areas of research including solar energy conversion and initiation of photopolymerisation . The anionic xanthene dyes, namely Erythrosin B, are very efficient laser dyes and fluorescent probes . On the other hand, Riboflavin is a yellow coloured neutral isoalloxazine dye and is biologically important as vitamin B_2. Therefore, the photophysical studies of these dyes in micellar medium of surfactant would be of considerable importance. For this purpose, the spectra (both absorbance and fluorescence) of these three dyes, each representing its class, have been studied in micellar solution of triton X-100, a neutral surfactant, and the spectral studies have been complimented with the photoelectrochemical studies and the results are reported here.

2. Experimental

All the dyes, namely Erythrosin B, an anionic dye, Riboflavin, a neutral dye and Safranin O, a cationic dye, were supplied by Sigma Chemicals. These were recrystallized twice from ethanol-water and the purity of these dyes were checked by absorption and fluorescence spectra. The surfactant, triton X-100 (TX-100) supplied by Sigma Chemicals was used without further purification. Aqueous solutions were prepared in doubly distilled water.

The absorption and fluorescence spectra of the solutions were recorded on a Shimadzu 160A UV-VIS spectrophotometer and Shimadzu RF-540 spectrofluorophotometer, Japan, respectively, using silica cells of 1 cm optical pathlength placed in a thermostated cell holder. The photogalvanic effect of these dyes in TX-100 was studied in a photoelectrochemical (PEC) cell, which consisted of an H-shaped cell with a 300 W tungsten lamp of 30mW cm^{-2} intensity. All measurements were done with freshly prepared solutions.

3. Results and discussion

The visible absorption spectra of mixed solutions with a fixed concentration of Safranin O and varying concentrations of TX-100 above the **cmc**, 2.5×10^{-4} mol dm^{-3} in aqueous media at 298 K are shown in Fig.1. The cationic dye Safranin O exhibits a maximum absorption at 520nm with the molar extinction coefficient of $2.5x10^3 m^2 mol^{-1}$ in water at 298K.The absorption band is shifted to 537 nm in presence of TX-100 with a sharp isobestic point at 502nm which indicates 1:1 molecular complex formation between Safranin O and TX-100. The lack of change in absorbance of Safranin O at the concentration below the cmc of TX-100 indicates absence of interaction between the dye and surfactant. The absorption spectra of

Erythrosin B, an anionic dye, is also perturbed in presence of TX-100 with concentration above the cmc of TX-100. The visible absorption band of Erythrosin B in water appears at 527 nm at 298 K and is shifted to 541 nm in presence of TX-100 with an isobestic point at 533 nm.The absorption spectra of Riboflavin, a neutral dye (absorption maximum at 448nm in water), is not perturbed in presence of TX-100. This indicates that there is no molecular interaction between Riboflavin and TX-100 in the ground state. The spectral data with a fixed concentration of Safranin O and Erythrosin B and varying concentrations of TX-100 have been utilized to calculate the equilibrium constant (K_c) and molar extinction coefficient (ε_c) of the dye complexes with TX-100 using Scott equation (30) in the following modified form:

$$\frac{[S]\,[D]l}{d-d_0} = \frac{[S]}{\varepsilon_c-\varepsilon_0} + \frac{1}{K_c(\varepsilon_c-\varepsilon_0)} \qquad (1)$$

Where [D] and [S] are the initial molar concentrations of the dye (Safranin O and Erythrosin B) and surfactant (TX-100) respectively, l is the optical pathlength of the solution which is 1 cm here, d and d_0 are the absorbances of the dye at the absorption maximum of the complex with and without TX-100 respectively, and ε_c and ε_0 are the respective molar extinction coefficients of the complex and the dye at the absorption maximum of the complex. However, it is a pre-requisite for the equation in the present form that the condition [S]>>[D] should hold and the complex absorbs at a wavelength where TX-100 is completely transparent. The values of K_c and ε_c were calculated from eqn.(1) by plotting $[S][D]/(d-d_0)$ against [S]. The plots shown in Fig.2a were found to be linear in all cases confirming 1:1 molecular complex formation. From the slope and intercept K_c and ε_c of the dye-surfactant interaction in aqueous media were calculated. The thermodynamic quantities of these complexes were obtained from the equilibrium constants at three temperatures (288, 298, 310 K) by the usual method and the values are summarized in Table 1.

The fluorescence spectra of Safranin O and Erythrosin B in presence of TX-100 above the cmc show the enhancement of fluorescence intensity with blue and red shift respectively, while the fluorescence spectra of riboflavin in presence of TX-100 show normal quenching of fluorescence. The fluorescence spectra of mixed solutions with a fixed concentration of Safranin O in aqueous solution of TX-100 of varying concentrations at 298 K are shown in Fig.3a. The fluorescence spectra of Erythrosin B in aqueous solution of TX-100 above cmc behave in a similar manner except that they exhibit a red shift. The fluorescence spectra of mixed solutions with a fixed concentration of riboflavin and varying concentrations of TX-100 above the cmc in aqueous media at 298K are shown in Fig 3b. In many cases, exciplex formation takes place by enhancement of fluorescence, and in some cases, a spectral shift along with enhancement has been observed in addition to the normal fluorescence quenching. In the cases of Safranin O and Erythrosin B in presence of TX-100, it should be the excited state charge transfer (CT) interaction since the dyes interact with TX-100 in the ground state. But in the case of Riboflavin in presence of TX-100 it might be the exciplex formation between Riboflavin and TX-100. A possible mechanism of fluorescence enhancement of Safranin O and Erythrosin B as well as the quenching of the Riboflavin dye by TX-100 in micellar solution may be represented as follows:

$D + h\nu \rightarrow {}^1D$ (2)
${}^1D \rightarrow D + h\nu_f$ (3)
${}^1D \rightarrow D + \text{heat}$ (4)
${}^1D + S \rightarrow ({}^1D \ldots S \leftrightarrow D^{''}\cdot \ldots S + \cdot) \rightarrow D + S$ (5)

Here, D represents the dye (Safranin O, Erythrosin B, Riboflavin) and S represents TX-100. The equation (5) shows the excited state CT interaction, $({}^1D \ldots S \leftrightarrow D^{''}\cdot \ldots S + \cdot)$ where TX-100 (S) acts as an electron donor and the dye(D) in its singlet excited state acts as an electron acceptor and this interaction enhances the fluorescence intensity of safranin O and erythrosin B and quenches the fluorescence intensity of riboflavin in micellar medium with increasing concentration of TX-100. This step $({}^1D \ldots S \leftrightarrow D^{''}\cdot _ S + \cdot)$can be written separately for three separate dyes as $({}^1\text{Safranin O}+ \ldots S \leftrightarrow \text{Safranin O}\cdot \_ S + \cdot)$ for cationic Safranin O, $({}^1\text{erythrosin B}^{''} \ldots S \leftrightarrow \text{erythrosin B}^{-2\cdot} - S + \cdot)$ for anionic erythrosin B and $({}^1\text{riboflavin} \ldots S \leftrightarrow \text{riboflavin}^{''}\cdot \_ S + \cdot)$ for neutral riboflavin.

Assuming that the relative increase of fluorescence intensity of these dyes in presence of TX-100 is due to the excited state complex formation, the stability constant(K_c^*) of the excited state interaction for safranin O and erythrosin B can be calculated from the following empirical relation ,

$[D][S]\, F_0 / (F - F_0) = [S]/\beta\varepsilon_F + 1/\beta K_c^* \varepsilon_F$ (6)

Where F_0 and F are the fluorescence intensities of the of the dye (safranin O and erythrosin B)in absence and presence of TX-100; [D] and [S] are the initial concentration of the dye and TX-100 respectively and ε_F and β are proportional constants; the details of the equation (6) are mentioned earlier . The plots of $[D][S]\, F_0/(F-F_0)$ against [S], the initial concentration of TX-100, should yield a straight line for excited state complex formation between singlet excited dye and TX-100. From the slope and intercept of this plot, K_c^*, the stability constant of the excited state interaction between TX-100 and excited dye i.e., safranin O and erythrosin B can be estimated.The plots are shown in Fig.2b at 298 K and the results are presented in Table 2 along with the fluorescence characteristics of safranin O and erythrosin B dyes in water as well as in micellar solution of TX-100.The fluorescence intensity of riboflavin, a neutral dye is quenched in presence of TX-100, confirming the excited state molecular interaction. By measuring the decrease in relative fluorescence intensity of riboflavin at varying concentrations of TX-100, the Stern Volmer quenching constant K_{sv}, has been calculated using the equation

$F_0/F = 1 + K_{sv}[S]$ (7)

Where F_0 and F are the relative fluorescence intensities of riboflavin alone and in presence of TX-100 respectively, and [S] is the molar concentration of TX-100 or quencher. The plot of F_0/F against [S] is linear (Fig 2c) with unit intercept and from the slope K_{sv} value has been calculated and mentioned in Table 2.

A photovoltage generation has been observed on illumination of one compartment of the cell consisting of TX-100 (concentration: 1×10^{-2} mol dm^{-3}) and dye (safranin O/erythrosin B / riboflavin of concentration, 1×10^{-5} mol dm^{-3}) using platinum electrode in the illuminated compartment and counter saturated electrode in the dark compartment. The illuminated electrode acts as the anode. The photovoltage attains a maximum value within a few minutes.The growth and decay of photovoltage can be reproduced at least 5-6 cycles with a slight change establishing the reversibility of the photoinduced effect. The maximum photovoltage attained by the dyes safranin O, erythrosin B and riboflavin are 47, 53 and 36mV respectively.It is to be mentioned that deoxygenation is not done prior to illumination in these systems so that the singlet excited dye would be mainly responsible for the generation of photovoltage in presence of TX-100 in aqueous media. The possible mechanism of photoinduced electron transfer for TX-100 (S) to singlet excited dye (D) leading to the generation of photovoltage in a photoelectrochemical cell is as follows,

$D + h\nu_a \rightarrow {}^1D$ (8)

${}^1D + S \rightarrow ({}^1D...S \leftrightarrow D^{-}\cdot - S^{+}\cdot) \rightarrow D^{-}\cdot + S^{+}\cdot$ (9)

$D^{-}\cdot \rightarrow D + e^{-}$ (anode reaction) (10)

For these three dyes, ${}^1D$ and $D^{-}\cdot$ can be represented as ^{1}safranin O^{+} and safranin $O\cdot$, ^{1}erythrosin B^{-} and erythrosin $B^{-2}\cdot$, ^{1}riboflavin and riboflavin$^{-}\cdot$ for cationic safranin O, anionic erythrosin B and neutral riboflavin respectively.

At the cathode compartment, the reaction is

$\frac{1}{2} Hg_2Cl_2 + e^{-} \rightarrow Hg + Cl^{-}$ (11)

and at the junction, i.e., platinum foil separator between the anode and cathode compartments of the PEC cell:

$S^{+}\cdot + Cl^{-} \rightarrow S + \frac{1}{2} Cl_2$ (12)

The platinum foil separator plays an important role since it prevents the recombination of the dye radicals (neutral and anionic) by behaving as a double electrode with oxidation in the dark compartment and reduction in the illuminated compartment. The overall forward (light) and backward (dark) reaction in the cell under continuous illumination is represented by

$D + S$

$D^{-}\cdot + S^{+}\cdot$ darklight (13)

Thus, the photoelectrochemical cell containing dye and TX-100 absorbs photon energy and this energy is partly converted into electrical energy by the electrode reactions through the formation of an excited state CT complex, ($^{1}D...S \leftrightarrow D^{''}\cdot - S^{+}\cdot$) where S (TX-100) acts as an electron donor and dye as an electron acceptor. There is no photovoltage generation in the absence of TX-100.

From the thermodynamic and spectrophotometric results in the ground state, it can be concluded that in the ground state the electron accepting ability of cationic Safranin O is greater than that of anionic Erythrosin B, while neutral Riboflavin does not interact in the ground state. However, photoelectrochemical and fluorescence results in the excited state show the electron accepting abilities of the three selected dyes are in the order : Erythrosin B > Safranin O > Riboflavin.

It is interesting to mention that the fluorescence spectra of Safranin O and Erythrosin B in presence of TX-100 show enhancement with blue and red shift respetively while riboflavin shows normal quenching of fluorescence in presence of TX-100. Very recently it has been reported that the absorbance and fluorescence spectra of Safranin O are highly dependent on the polarity of the solvent. A blue shift in the ground state absorption and a red shift in the emission band is clearly apparent as the solvent polarity increases. The Stokes shift also increases with increasing solvent polarity. The results presented here in the case of Safranin O in water as well as in the micellar medium agree with this observation, i.e., the micellar medium provides less polar environment to the excited Safranin O molecules compared to the aqueous medium. In our case, on going from the polar aqueous medium to the less polar micellar medium, there is a red shift in the ground state absorption and a blue shift in the emission band with decreasing Stokes shift from 1870cm^{-1} in water to 954 cm^{-1} in TX-100 solution.This indicates a change in solvent polarity from water with E_T (30) of 63.10 to micellar medium with E_T (30) of 50 . The lower value of the excited state stability constant of safranin O – TX-100 interaction compared to the ground state interaction also confirms the blue shift of fluorescence spectra of safranin O in TX-100 solution. According to Mulliken's CT theory, the CT interaction in the excited state is prominent as compared to the CT interaction in the ground state.The stability constants, both K_c and $Kc^{*}$of erythrosin B with TX-100 support this (Tables 1 and 2).

TX-100 in water forms ordered aggregate structure (micelles) in water above a given concentration, the critical micelle concentration (cmc). The absorption and fluorescence spectra of these dyes are not perturbed in presence of TX-100 below the cmc which indicates that the surface formation of micelle in TX-100 is a necessary criterion for complex formation between dye and surfactant. The interface (micelle/ water) induces the complex formation, due to adsorption of dye from the solution. In the excited state CT complex, Safranin O forms neutral radical and it can penetrate into the hydrophobic region of the micelle, whereas in the case of Erythrosin B and Riboflavin which form anionic radicals they will remain on the surface of the micelle in the hydrophilic region. This is correlated with the Stokes shift of the dyes, both in water as well as in TX-100 (Table 2). Lastly, we can conclude that a good correlation has been found between the spectral shifts (both absorption and fluorescence) due to complex formation and the stability constants of these complexes both in the ground and the excited state. As expected, the photovoltage generation in these systems is related with the excited state stability constants (Table 2) which in turn generate electroactive species for the generation of photovoltage.

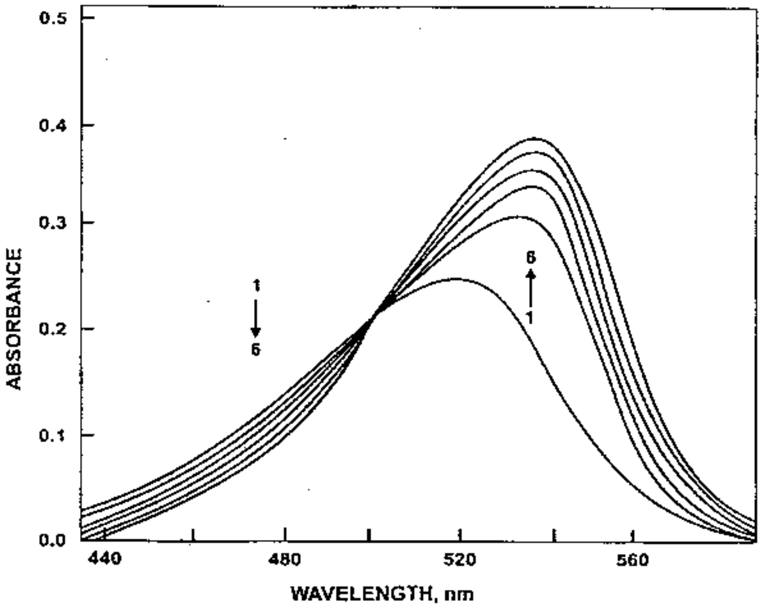

Fig.1 The visible absorption spectra of safranin O in aqueous solution TX-100 of different concentrations at 298 K . Concentration of safranin O : 1 x 10^{-5} mol dm^{-3} and concentrations of TX-100 (10^{-3} mol dm^{-3}) : (1) 0.0 (2) 2.0 (3) 4.0 (4) 6.0 (5) 8.0 (6) 10.0.

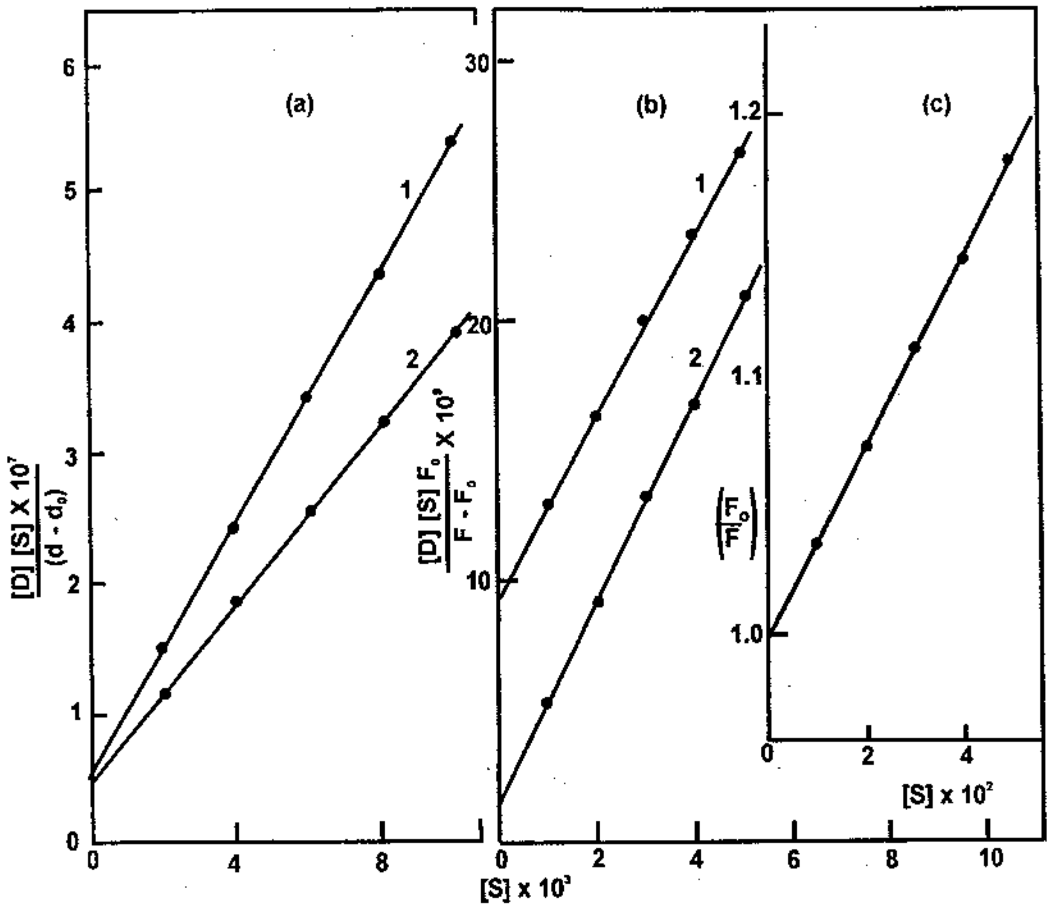

Fig. 2 (a) Plots of $[D][S]/(d-d_0)$ against the concentration of TX-100 at 298 K for determination of ground state stability constants and molar extinction coefficients of dye TX-100 complexes using Scott equation. The curves 1 and 2 refer to safranin O – TX-100 and erythrosinB-TX-100 complexes respectively.

(b) Plots of $[D][S]F_0/(F-F_0)$ against the concentration of the dye-TX-100 complexes at 298 K for determination of excited state stability constants of the dye-TX-100 complexes in the excited state. Here D_0 and S represent dye (safranin O / erythrosin B) and TX-100 respectively and the numbers 1 and 2 refer to safranin O and erythrosin B respectively. Concentration of the dye: 1×10^{-5} mol dm^{-3} in all cases and concentration of TX-100 (mol dm^{-3}) : 2×10^{-3} to 10×10^{-3} for (a) and 1×10^{-3} to 5×10^{-3} for (b).

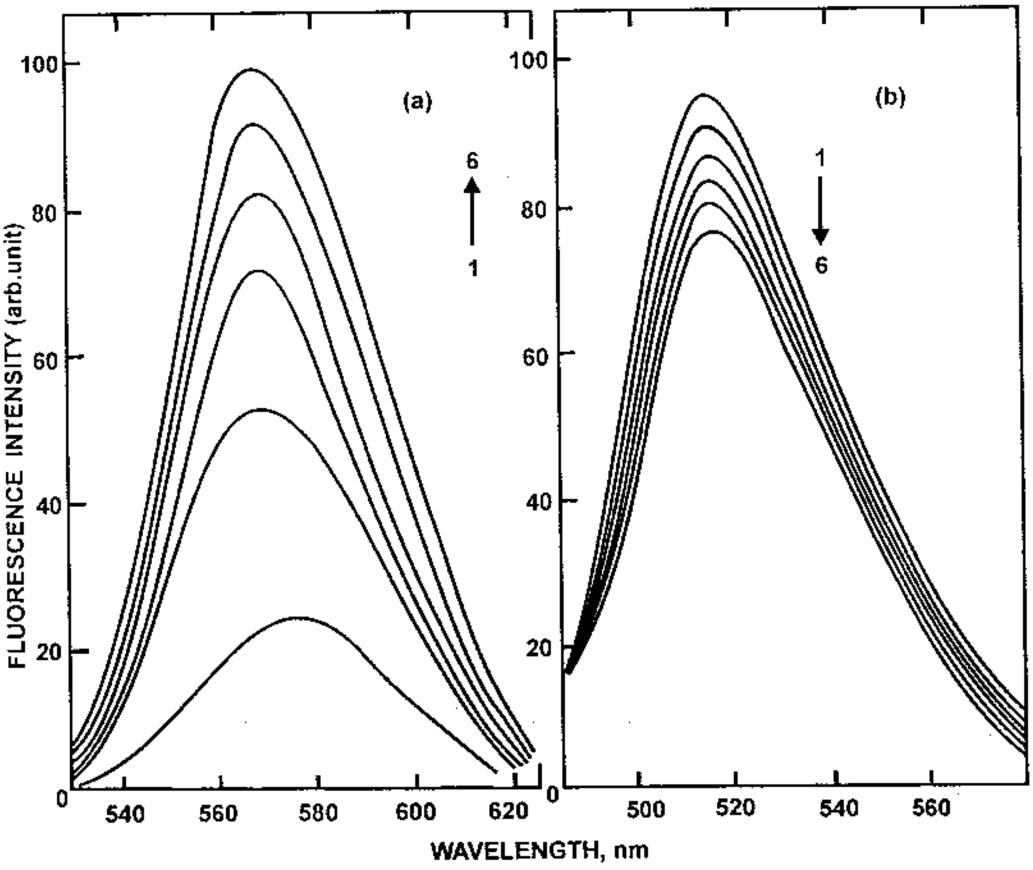

Fig. 3 (a) The fluorescence spectra of safranin O in aqueous solution of TX-100 at 298 K.

(b) The fluorescence spectra of riboflavin in aqueous solution of TX-100 at 298 K. Concentration of dye (safranin O/ riboflavin) : 1 x 10^{-5} mol dm^{-3} in both cases; the concentration of TX-100 (10^{-3} mol dm^{-3}): (1) 0.0 (2) 1.0 (3)2.0 (4)3.0 (5)4.0 (6) 5.0 in (a) and the concentration of TX-100 (10^{-2} mol dm^{-3}): (1) 0.0 (2) 1.0 (3) 2.0 (4) 3.0 (5) 4.0 and (6) 5.0 in(b).

Table 1

Thermodynamic and spectral characteristics of dye interaction with TX - 100 in aqueous media at 298 K from absorption spectra

	Thermodynamic characteristics				Spectral characteristics				
					In water		In TX-100 solution (5 X 10^{-3}mol dm^{-3})		
Dye	K_c (dm^3 mol^{-1})	$-\Delta G^0$ ($kJmol^{-1}$)	$-\Delta H^0$ ($kJmol^{-1}$)	$-\Delta S^0$ ($Jmol^{-1}$ deg^{-1})	λ_{max} (nm)	ε_{max} (m^2mol^{-1})	λ_{max} (nm)	ε_{max} (m^2 mol^{-1})	Shift $\Delta \bar{v}$ (cm^{-1})
Safranin O	9.60 X 10^2	17.12	48.60	105.64	520	2.50 X 10^3	537	3.80 X 10^3	678
Erythrosin B	7.20 X 10^2	16.29	36.03	66.24	527	2.36 X 10^3	541	2.56 X 10^3	491
Riboflavin	-	-	-	-	448	1.35 X 10^3	448	1.30 X 10^3	0

Concentration of dye used in each case is 1 X 10^{-5}mol dm^{-3} and concentrations of TX-100 used are in the range of 2 X 10^{-3} to 10 X 10^{-3} mol dm^{-3} in all cases.

Table 2

Excited state stability constant (K_c^*), fluorescence characteristics, Stokes shift and open-circuit photovoltage of the systems consisting of some dyes and TX-100 in aqueous media at 298K

Dye	Excited state stability Constant K_c^* (dm^3 mol^{-1})	Fluorescence characteristics		Shift $\Delta \bar{v}$ (cm^{-1})	Stokes shift $\Delta \bar{v}$ (cm^{-1})		Photovoltage (mV)*
		In water	In TX-100		In water	In TX-100	
Safranin O	3.62 X 10^2	576	566	307	1870	954	47
Erythrosin B	2.54 X 10^3	545	558	428	626	563	53
Riboflavin	3.64	516	516	0	2991	2991	36

Concentration of dye used in each case is 1 X 10^{-5} mol dm^{-3} and concentration of TX-100 used are in the range of 1 X 10^{-3} to 5 X 10^{-3} mol dm^{-3} in all cases except with riboflavin where concentrations of TX-100 are in the range 1 X 10^{-2} to 5 X 10^{-2} mol dm^{-3}.

* For photovoltage generation the fixed concentrations of dye and TX-100 used are 1 X 10^{-5} and 1 X 10^{-2} mol dm^{-3} respectively.

Sir J.C. Bose's experiments on millimeter waves

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In 1865 James Clerk Maxwell predicted the existence of electromagnetic radiation propagating at the speed of light. This was verified experimentally by Hertz in 1888. He demonstrated the possibility of generation of electromagnetic waves and proved that their properties were similar to those of light. In the last decade of the nineteenth century, many experimenters began working with the newly discovered "Hertzian Waves". The book "The Work of Hertz and Some of His Successors" by Oliver Lodge caught the interest of J.C. Bose and he began research particularly into short wavelength electromagnetic radiation. In the last few years of the 19th century he used, and in some cases invented, many now commonplace microwave components, including dielectric lenses and prisms, horn and lens waveguide radiators (antennas), microwave absorbers and semiconductor detectors. Some of his discoveries are surprisingly relevant today.

In 1895 J.C. Bose, pioneer in modern experimental science in India, gave his first public demonstration of electromagnetic waves, using them to ring a bell remotely and to explode some gunpowder. In 1896 the Daily Chronicle of England reported: "The inventor (J.C. Bose) has transmitted signals to a distance of nearly a mile and herein lies the first and obvious and exceedingly valuable application of this new theoretical marvel." In January 1897 Bose described to the members of the Royal Institution in London his research carried out in Calcutta at millimeter wavelengths. The wavelengths he used ranged from 2.5 cm to 5 mm. A sketch of Bose's apparatus demonstrated at the Royal Institution in London in 1897 is shown in Fig. 1. He used a circular waveguide and the "collecting funnel" (F) is a pyramidal electromagnetic horn antenna, first used by Bose. The radiation was generated by spark transmissions, using resonant structures to define the wavelength of emission; the wavelength was measured by a reflecting diffraction grating consisting of different dimensions and spacings of metal strips. Fig. 2 shows Bose's diagram of one of his radiators, used for generating radiation of wavelength 5 mm. Sparking between two hollow hemispheres and the interposed sphere produces oscillation. There is a bead of platinum on the inside surface of each hemisphere. For some experiments, a lens of glass or of sulphur was used to collimate the radiation - the first waveguide-lens antenna. The lens was designed according to the refractive index measured by Bose at the wavelength in use. Fig. 3 shows Bose's drawing of such a radiator; the sparks occur between the two outer spheres and the inner sphere which is at the focal point of the lens **L** at the right. Bose was able to measure the wavelength of the radiation with a reflecting diffraction grating made of metal strips, as mentioned earlier. He invented and used the waveguide-lens and horn antennas, examples being shown in Fig. 3 and 4. Fig. 4 is a photograph of one of his radiating antennas; part of the spark oscillations is generated inside the overmoded circular waveguide. A polarizing grid is built into the antenna, which is visible at the radiating end of the waveguide.

For detectors he used what are now recognised as semiconductor junctions - Bose later was awarded the world's first patent for a semiconductor device. He characterised the I-V properties of his semiconductor junctions, showing that they did not obey Ohm's law. Fig. 5 shows examples of point-contact junctions used inside the receiving antenna of Fig. 4. The knee in the I-V curve was found to lie at about 0.45 Volt ***(Note the similarity to modern semiconductor junctions, with a knee voltage between 0.2 and 0.7 Volt.)*** Bose had found that optimum detector sensitivity was obtained when a DC bias of this value was applied to his detectors. He also used free-space detectors (Fig. 6), which have since been described as a "space-irradiated multi-contact semiconductor (using the natural oxide of the springs)".

A complete setup of Bose's apparatus on mm wave transmission and reception is shown in Fig. 7. The transmitting antenna is at the left, the receiving antenna is at the right. There is an adjustment screw on top of the receiving antenna which is used to adjust the pressure of the point-contact detector. In the center is a rotating table (the "spectrometer circle" of Fig. 1) on which various microwave components (prisms, lenses, grids, polarisers, etc.) may be mounted for study. A dual-prism attenuator, described below, was used to measure the wavelength of the generated wave.

Two prisms (Fig. 8) were arranged to be separated by a small, variable air gap. With a large air gap, the incident radiation suffers total internal reflection, and is not transmitted into the dielectric of the second prism. With the prisms touching, the radiation sees no obstacle and passes right through the system. By adjusting the air gap, the attenuation is controlled. Fig.7 shows the prism between the transmitter antenna (left) and receiver in the typical arrangement used to measure the properties of the dual prism. The wavelength of emission from Bose's spark transmitters was defined by the resonant structure associated with his spark gaps and waveguide. He measured the wavelength associated with some of his structures using a concave reflection grating; in his 1897 paper he describes how with different structures he measured wavelengths of 1.84 and 2.36 cm. He showed that the wavelength of the emitted radiation scaled at least approximately with the dimensions associated with his spark generator. In other investigations, he assumed, reasonably, that the wavelength would scale with the physical dimensions of his emitter. Schaeffer and Gross published the theory of the dual-prism attenuator in 1910. This theory, not available to Bose during his 19th century investigations, now permits an independent estimation of the wavelengths used by Bose for his dual prism experiments. Bose measured the size of the air gap between the two prisms at which the intensities of the reflected and transmitted waves were equal. In a separate experiment, using a measurement of the critical angle, at which total internal reflection inside the prism begins, he measured the refractive index of the dielectric – to air – interface at the wavelengths in use. His value is 2.04.

Fig. 9 shows the calculated attenuation of the transmitted, and of the reflected rays, through a dual-prism system, the prisms having a refractive index of 2.04, as a function of the air gap between the prisms. The horizontal axis gives the air gap measured in wavelengths. It is seen that the reflected and transmitted powers are equal when the air gap is ~0.11 wavelengths. To calculate the attenuation, equal powers were assumed in two orthogonal linear polarisations, parallel to and perpendicular to the plane of incidence. If the emission were to be 100% linearly polarised in either plane, the corresponding air gap would change by approximately +/-20%. Effects of diffraction have been ignored. For two different radiators, Bose found that gaps of 3.7 and 4.5 mm gave equal transmitted and reflected powers; the corresponding wavelengths derived from Fig. 9 are 3.45 and 4.17 cm. For the shorter wavelength, the radiator consisted of a conducting sphere of diameter 0.97 cm, with two smaller beads placed symmetrically on either side. It is interesting to note that the fundamental mode of oscillation for an isolated conducting sphere of this size corresponds to a wavelength of 3.54 cm, an estimate perhaps fortuitously close in value.

These wavelengths are somewhat greater than would be implied by a simple scaling of dimensions from the radiators used in Bose's diffraction grating based wavelength measurements; however, the geometries used for the dual prism measurements were also somewhat different - a square waveguide, one inch on a side, was used during the diffraction grating measurements, as opposed to the somewhat larger circular waveguide during the dual prism experiment. It is also possible that in his diffraction grating experiments, a diffraction peak of emission corresponding to a higher resonance mode was detected, while the dual prism measurements responded to the total radiated energy. It would be very interesting to model the response of Bose's different transmitter configurations using modern software - or to make direct measurements of the radiated spectra from the surviving equipment. Recently, at U. S. National Radio Astronomy Observatory scientists had found the need for a quasi-optic attenuator for local oscillation injection at 230 GHz (1.3 mm) in a multi-feed system used for sensitive radio astronomical measurements at the 12 Meter Radio Telescope at Kitt Peak. Bose's prism was the ideal solution. In the published description of this NRAO system, Bose's paper published in the Proceedings of the Royal Society in London, in 1897, has been cited.

In his research into electromagnetic waves, his use and development of microwave components and the first use of semiconductors, Bose was clearly some 50 years ahead of his time.

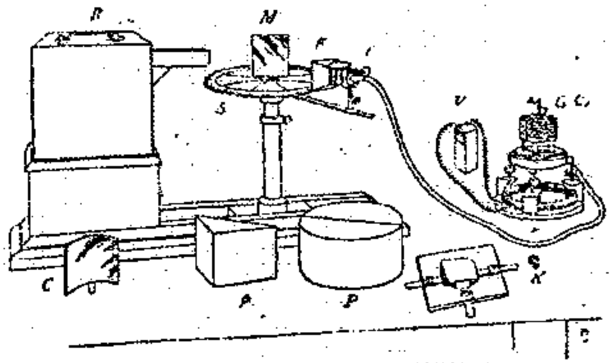

Fig. 1 : R : radiator, S : spectrometer-circle, M : plane mirror, C : Cylindrical miror, p : totaly reflecting prism, P : semi-cylinders, K : crystal-holder; F : collecting funnel attached to the spiral spring receiver, t : tangent screw, by which the receiver is rotated, V : voltaic cell, r : circular rheostant, G : galvanometer.

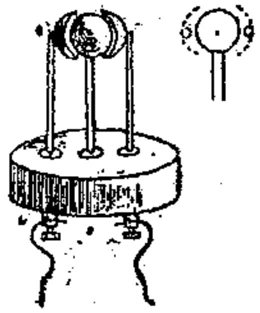

Fig. 2 : The Radiator

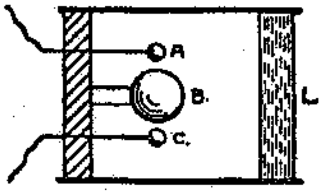

Fig. 3 : Bose's radiator with a lens(L) at the exit of the waveguide

Fig. 4. One of Bose's transmitter antennas (being held on the right of the picture). Note the polarising grid; the spark gap is just visible behind the grid. In the background behind this antenna, part of the high voltage equipment used to generate the spark can be seen. At the left of the picture is the receiving horn.

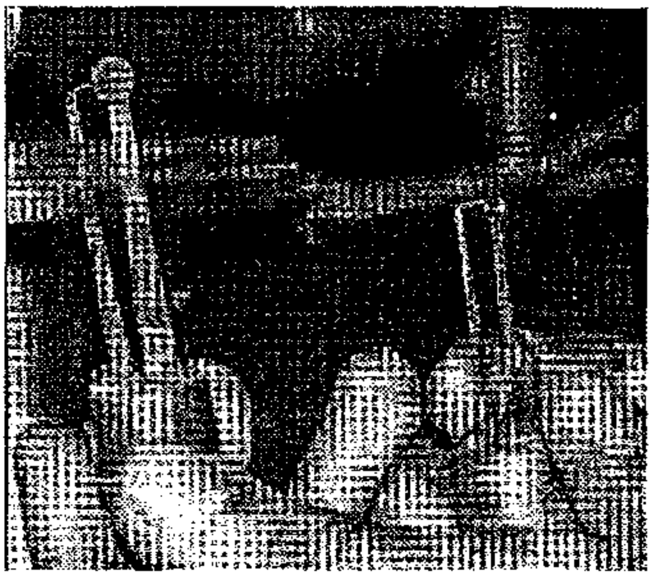

Fig. 5. Two of Bose's point contact detectors removed from the receiving antennas

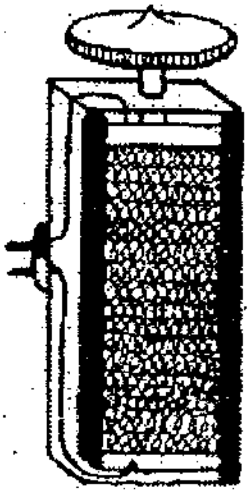

Fig. 6. Bose's diagram of his spiral-spring receiver used for 5-mm radiation.

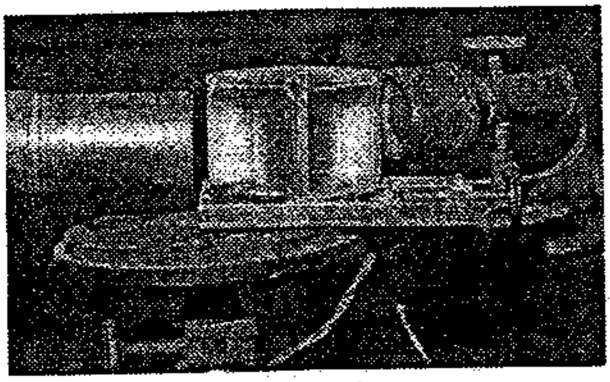

Fig. 7. A Complete setup

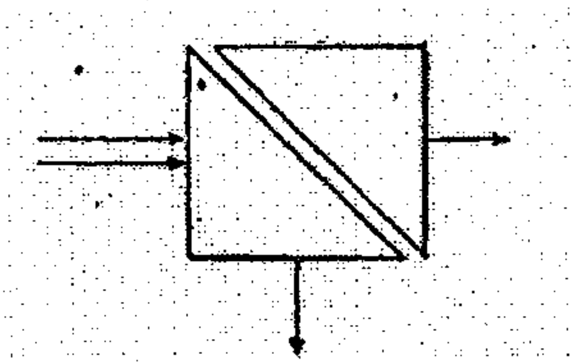

Fig. 8. Bose's 1897 diagram of the double – prism attenuator.

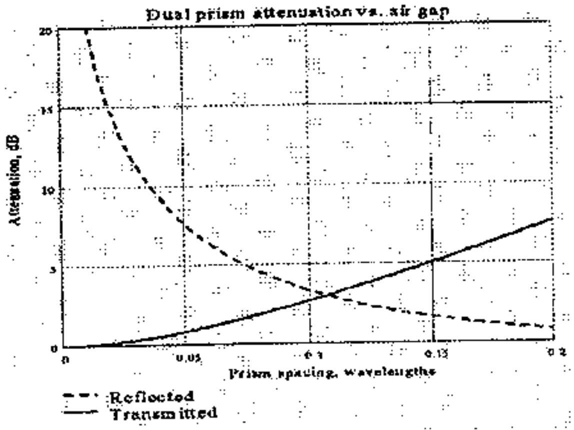

Fig.9 Attenuation of the transmitted and reflected waves vs. dual prism air gap·(in wavelengths.)

Heal Effects of out door Air Pollution
A Panoramic View

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Abstract

Interest in the health effects of outdoor air pollution is growing among policy makers, international lenders, nongovernmental organizations, industry, and others due to increasing knowledge about the health effects of air pollution and to the high levels of air pollution. Exposure to outdoor air pollution is now widely accepted as being associated with a broad range of acute and chronic health effects, ranging from minor physiologic disturbances to death from respiratory and cardiovascular disease. This acceptance is based on observational epidemiologic studies of disease occurrence in human populations and the in vitro and in vivo studies of animals and humans.

Despite this growth of knowledge, uncertainty remains about some critical questions of importance to public policy. How large is the effect of short-term exposure on daily mortality and morbidity? How does it vary across the globe? How accurately can we measure the magnitude and variability of these effects with our current statistical tools? Which diseases, social conditions, or genetic factors place people at greatest risk when exposed to air pollution? To what extent does long-term exposure affect the development of chronic disease, and what shortening of life may result? Which constituents of the pollutant mixture are most toxic and which sources contribute to it? The answers to most of these questions might well be different in Asia than in the West.

This paper attempts to describe different aspects of Health hazards caused by different pollutants. Section 1 describes the association between health and air quality. Particulate matters and their observed health effects have been described in section 2 to section 4. Section 5 deals with major air pollutants and their health effects with a detailed analysis of particulate matter. Mechanism of health effects has been described in section 6 and finally section 7 documented different empirical findings of health hazards caused by air pollutants.

In recent years, there has been an increasing concern about the effect of air pollution on health. The World Health Organization (WHO) estimates that every year 800,000 people die prematurely from lung cancer, cardiovascular and respiratory diseases caused by outdoor' air pollution [Cohen et al 2003]. Other adverse health effects include increased incidence of chronic bronchitis and acute respiratory illness, exacerbation of asthma and coronary disease, and impairment of lung function. There is strong evidence linking urban air pollution to acute and chronic illnesses and premature death and these adverse health impacts in turn carry high economic costs to society [SAUAQM 2003].

§1 HEALTH AND AIR QUALITY

Health is defined as a state of physical, mental and social well-being. Since a quantitative measurement of well-being is not possible, proxies such as the nature of sickness and intensity of sickness are used for determining the state of health. Also, the health of an individual is dependent on a number of factors such as genetic make up, nutrition, life style and personal habits, and the environmental exposure to water, air, and other sources of pollution. Industrial Toxicology Research Centre (ITRC) in their report on health impact from air pollution have stated that there are several reports of studies on air pollution and its health impact from various cities of India, sponsored by the Ministry of Environment and Forests, and CPCB etc. They have attributed various morbidities in the studied population to ambient air pollution in urban areas. It is difficult to enumerate or discuss all the substances present in the ambient air due to air pollution. Some of the important ones have been taken into account. However, it must be kept in mind that all chemicals have some ill effect or the other and it is the dose and/ or route of exposure, which determines the toxicity of any chemical. Air quality is an important determinant of health. The sources of air pollution are many as may be seen in chart 1.1.

CHART 1.1
Classification of Air Pollution Sources

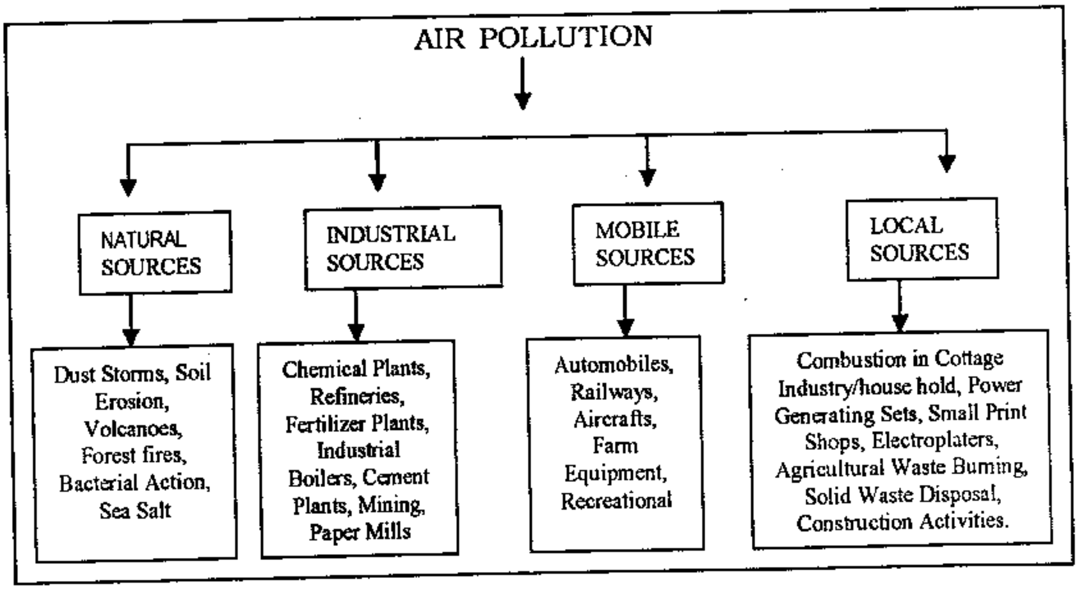


Table 1.1
Common air pollutants and their sources

Sulpher Dioxide (SO_2)	Fuel combustion in industries, power plants, chemical plants including refineries, fertilizer plants, cement plants, industrial boilers, motor vehicles and their mix, domestic combustion, power generating set, solid waste disposal, agricultural material burning, geothermal activities and bacterial action.
Nitrogen Oxides (NO_x)	Motor vehicles and their mix, fuel combustion in industries, power plants, chemical plants including refineries, fertilizer plants, cement plants, industrial boilers, railway transport, power generating set, road dust, agricultural material burning, geothermal activities and bacterial action.
Particulate Matter (PM, SPM, RSPM-PM_{10}, RSPM-$PM_{2.5}$)	Soil erosion, construction activities, traffic/ road dust, quarry/ mining activities, land tilling, Motor vehicles and their mix, fuel combustion in industries, power plants, chemical plants including refineries, fertilizer plants, cement plants, smelters, industrial boilers, railway transport, power generating set, road dust, agricultural material burning, geothermal activities and bacterial action and of biogenic origin.
Carbon Monoxide (CO)	Motor vehicles and their mix, fuel combustion in industries, power plants, chemical plants including refineries, fertilizer plants, cement plants, industrial boilers, railway transport, power generating set, solid waste disposal, waste incinerators, refuse burning, agricultural material burning, forest fires.
Ozone (O_3)	Secondary pollutant formed during photochemical reaction.
Lead (Pb)	Lead additives in petrol batteries disposals, metallurgical industry, soil originated particles, paint, plumbing.

Source: Auto Fuel Policy Report

Based on certain assumptions the quantification of pollutants that are emitted every day can be done for a city or region. While such numbers provide a quick assessment of the magnitude of the problem of pollution, it is not possible to directly use them for assessing their adverse effects on health. Along with the mass, the level of concentration (microgram of pollutant per cubic meter of air - mg/m3), as well as the duration of exposure of an individual to a pollutant needs to be known for gauging the health effect of emissions. Thus quantity, concentration and exposures are crucial components in any exercise that is designed to measure the effect of pollution. The National Ambient Air Quality Standards have taken into account these factors in setting air quality standards. Table 1.2 may be seen for the prevailing air quality standards.

Table 1.2
Air Quality Standards

Pollutants	Time weighted average	Concentration in ambient air			Method of measurement
		Industrial Area	Residential Area	Sensitive of Rural & other Areas	
Sulphur Dioxide (SO_2)	Annual Average 24 hours	80 $\mu g/m^3$	60 $\mu g/m^3$	15 $\mu g/m^3$	1. Improved West and Geake method
Oxides of Nitrogen as NO_2	Annual Average 24 hours	120 $\mu g/m^3$ 80 $\mu g/m^3$	80 $\mu g/m^3$ 60 $\mu g/m^3$	30 $\mu g/m^3$ 15 $\mu g/m^3$	1. Ultraviolet fluorescence 2. Jacob & Hochheiser modified (Na-Arsenite) Method 3. Gas Phase Chemiluminescence
Suspended Particulate Matter(SPM)	Annual Average 24 hours	120 $\mu g/m^3$ 360 $\mu g/m^3$ 500 $\mu g/m^3$	80 $\mu g/m^3$ 140 $\mu g/m^3$ 200 $\mu g/m^3$	30 $\mu g/m^3$ 70 $\mu g/m^3$ 100 $\mu g/m^3$	High Volume sampling (Average flow rate not less than 1.1 m^3/minute)
Respirable Particulate matter (size less than 10 μm)(RPM)	Annual Average 24 hours	120 $\mu g/m^3$ 150 $\mu g/m^3$	60 $\mu g/m^3$ 100 $\mu g/m^3$	50 $\mu g/m^3$ 75 $\mu g/m^3$	Respirable particulate matter sampler
Lead(Pb)	Annual Average 24 hours	1.0 $\mu g/m^3$ 1.5 $\mu g/m^3$	0.75 $\mu g/m^3$ 1.00 $\mu g/m^3$	0.50 $\mu g/m^3$ 0.75 $\mu g/m^3$	AAS Method after sampling using EPM 2000 or equivalent Filter paper
Carbon Monoxide (CO)	8 hours 1 hour	5.0 mg/m^3 10.0 mg/m^3	2.0 mg/m^3 4.0 mg/m^3	1.0 mg/m^3 2.0 mg/m^3	Non dispersive infrared spectroscopy

Source: WBPCB 2008

An important reason for controlling air pollutants such as particulate matter or Sulpher dioxide is the damaging effects they have on human health. These effects include premature death, as well as increases in the incidence of chronic heart and lung disease. Estimates of the health damages associated with air pollution are important because they can provide both an impetus for environmental controls and a means of evaluating the benefits of specific pollution control policies.

§2 OBSERVED HEALTH EFFECTS

The most significant health effects of air pollution have been associated with particulate matter (PM) and to a lesser extent, with ground-level ozone [Cohen et al 2003, Holgate et al 1999, World Bank 2002]. PM's is a mixture of many subclasses of pollutants which vary in size and chemical composition. Most studies have examined the health effects based on particle size. Much less is known about the impact on health of varying PM chemical composition. The largest health impacts have been associated with particles small enough to penetrate deep into the respiratory tract: *fine* particles $PM_{2.5}$, smaller than 2.5 microns or 2.5 x 10.6 meters in diameter) and PM_{10}, (smaller than 10 microns). Combustion, metallurgical processes, automobile exhaust, and secondary sulfate and nitrate particles formed by the atmospheric transformation of sulfur dioxide (SO_2) and oxides of nitrogen (NO_x) are the main sources of these smaller particles. Elevated levels of NO_x and SO_2 also results in higher hospital admissions and emergency room visits, but these effects are small compared to those of PM. Similarly the health effects of particles larger than 10 microns, arising primarily from resuspended dust and soil are small. The following section will discuss the science of health impacts.

§3 WHAT IS PARTICULATE MATTER?

Airborne particulate matter is not a single pollutant, but rather a mixture of many subclasses of pollutants—in solid and liquid forms, with each subclass containing many different chemical species. Particulate matter may be classified as primary or secondary. Primary particles are emitted directly by emission sources, whereas secondary particles are formed through the atmospheric reaction of gases, such as the reaction between ammonia and oxides of nitrogen or sulfur, which leads to the formation of PM **[USEPA 2002]**. PM is typically classified according to size. The particle size can vary from approximately 0.005 microns (0.005 x 10-6 meters) to 100 microns in diameter (that is, from the size of a few atoms to about the thickness of a human hair). All ambient PM irrespective of size is referred to as suspended particulate matter (SPM) or total suspended particles (TSP) when a gravimetric procedure has been used for measuring mass. Particulate matter less than 10 microns in diameter are referred to as PM_{10}, and PM less than 2.5 microns is referred to as $PM_{2.5}$. The term *fine* PM has also come to be associated with PM2.5, *ultra fine* with PM less than 0.1 microns in diameter ($PM_{0.1}$), and *coarse* with PM in the size range between 2.5 and 10 microns. Of late, the focus of PM measurement in ambient air of South Asian cities has shifted from measuring TSP to mass-based measurement of PM_{10}.

§4 ORIGIN AND TRANSPORT OF PARTICULATE MATTER

Airborne PM has numerous sources, ranging from naturally occurring process, sea salt, and pollen, to products of combustion such as forest fires, domestic cooking and heating, garbage burning, power generation, and mobile sources (such as vehicles and ships). Combustion processes normally contribute much more to the fine and ultra fine PM fraction, whereas non combustion processes contribute more to larger size PM fractions **[HEI 2002]**. For example, a study of the chemical composition of PM in Mexico City in 1997 showed that: (a) $PM_{2.5}$ particle mass consisted of about 50 percent carbonaceous aerosols likely from combustion sources, followed by approximately 30 percent secondary aerosols, and 15 percent geological matter; whereas (b) PM_{10} particle mass consisted of about 50 percent geological matter, followed by carbonaceous aerosols (about 30 percent) and secondary aerosols (less than 20 percent) [Chow et al 2002]. Unfortunately, such detailed studies are very limited in South Asia. Fine PM tends to be distributed uniformly over large areas, thereby making it difficult to trace it to individual sources. The recent finding of the Asian brown haze, attributed primarily to combustion sources in Asia, confirms the long residence time and regional distribution of fine and ultra fine particles [UNEP 2002]. Larger particles, however, normally have shorter atmospheric lifetimes (minutes to hours) and do not travel long distances (less than tens of kilometers). As a result they tend to be less evenly distributed, and are usually found closer to their sources [USEPA 2002]. Near highways, motor vehicle emissions may dominate the pollution mixture, but in other locations emissions from power plant and steel may be the main source of particulate pollutants. A significant fraction of PM, referred to as *secondary particles*, is produced by chemical reactions in the atmosphere; nitrogen oxides, sulfur dioxide, and organic compounds react with ozone and other reactive molecules (including free radicals) to form nitrates, sulfates, and other particles. People are also exposed to PM indoors, mostly from cigarette smoke, home heating sources (such as wood burning stoves), and cooking, but also from outdoor PM sources that easily penetrate the indoor environment. Indoor exposure may be substantial because this is where most people spend the majority of their time. However, when outdoors, people tend to be more active, which increases respiration. Active people may inhale a larger amount of pollutants because they inhale a larger amount of air in any given time period than people who remain indoors. Studies by HEI and others are under way to better understand the contribution of indoor and outdoor sources of PM to total exposure.

§5 WHY IS PM SO DAMAGING TO HEALTH?

Epidemiological studies from many different parts of the world, conducted by different groups of researchers using different data sets and analysis techniques, have generated results that confirm the magnitude of PM health impacts [SAUAQM 2003]. While a statistical association has been found between adverse health effects and PM_{10}, recent studies using $PM_{2.5}$ data have shown an even stronger association between health outcomes and particles in this size range. Evidence that smaller particles are more harmful is further supported by medical and toxicological research, which is increasingly focused on understanding the role of particle size (in the fine and ultra fine range) and composition in PM toxicity.

§5.1 SIZE AND NUMBER

Size determines how different particles deposit in different parts of the respiratory tract. Studies have shown that particles of different size vary in their respiratory tract deposition, movement, clearance, and consequent retention time in the human body. Ultra fine particles tend to behave more like gases and hence travel to the lower region of the lungs as compared to larger particles which tend to get deposited in the upper or middle region of the respiratory tract. Particles larger than about 10 microns in diameter are deposited almost exclusively in the nose and throat, whereas particles smaller than 1 micron are able to reach the lower regions of the lungs. The intermediate size range gets deposited in between these two extremes of the respiratory tract. Ultra fine particles are highly toxic to the lungs, even when they comprise materials that are not toxic when present in larger particles. The mechanisms for toxicity of ultra fine particles are still under investigation, but several observations may be made. For a given mass, the number of particles and the total surface area increase dramatically with decreasing particle size. If toxic components are adsorbed on the surface of ultra fine particles, the level of interaction between the lungs and the surface of ultra fine particles is likely to increase with increasing surface area. It is not immediately clear, however, why the adverse impact of non-toxic particles should also increase with decreasing particle size and increasing surface area. The above discussion suggests that the particle size and particle number may be more relevant indicators than particle mass: the smaller the particle, the greater the fraction of particles deposited in airways and lungs, and the greater the surface area available for interaction with biological systems. Most of the research, however, on the role of particle size and number has been conducted in laboratories using animals or mathematical models. In practice ambient monitoring of particle numbers is not common; rather, most of the monitoring the world over still focuses on particle mass.

§5.2 COMPOSITION

Scientists have hypothesized that some components of ambient PM are more likely to be responsible for toxic or adverse health effects than other components. Studies are being conducted to determine which components or combination of components is key in inducing adverse health effects.

The main components of urban PM are metals, organic compounds, materials of biologic origin, secondary particulate matter (sometimes as ions), and the particle core which often forms the bulk of urban PM and is frequently composed of pure or elemental carbon. Several studies, mostly on animals, have demonstrated the effect of metallic PM on lung damage. Organic compounds are known to lead to mutations and even cause cancer. Materials of biological origin such as fungal spores and pollen are known to induce a variety of allergic responses. Sulfate and nitrate ions lead to significant impairment of the respiratory tract because of their acidic potential. Carbonaceous material in the particle core can by itself lead to lung irritation and damage after chronic exposure.

In general, the composition of larger particles differs from that of smaller particles. The coarse particle fraction consists mainly of insoluble crust-derived minerals, biologic material (such as pollen and bacteria), and sea salts. By contrast, the ultra-fine and fine fractions are composed mainly of particles with a carbon core that contains a variety of metals, secondary particles, and hydrocarbons. Fine and ultra fine particles are composed mainly of particles with a carbon core that contains a variety of metals, organic compounds, and secondary particulates [HEI 2002]. The surface area of the elemental carbon core is considerably increased by its porous nature, greatly enhancing the adsorption probability of airborne substances such as organic compounds. A recent study by the U.S. Environmental Protection Agency (EPA) highlighted the likely cancer risk from diesel emissions [USEPA 2002], declaring it as a potential carcinogen. The diesel particles, many of which are smaller than 1 micron in diameter, have a carbonaceous core with a large surface area to which various organic compounds are absorbed, including carcinogenic polycyclic and nitro-polycyclic hydrocarbons. Diesel particles have also been shown to absorb allergens from grass pollen, thus potentially increasing allergen deposition in the respiratory tract [UK DEFRA 2001]. The composition of PM varies with factors such as the nature of sources and/or geographical location. For example, the particle composition in the northeastern part of the United States, which has a high concentration of coal-based thermal power plants, is dominated by secondary sulfate particles linked to sulfur dioxide emissions from the power plants. As a result, the health effects of inhaled particles may also be affected by their composition. This suggests that more epidemiological studies based on $PM_{2.5}$ and smaller particles with varying composition are needed to see whether there is a significant impact of particulate composition on health. Table 1.3 lists some of the leading candidates that have been associated with biologic responses observed in a variety of studies and experimental systems.

Table 1.3
Chemical Components of PM and their Biological Effects

Components	Major Subcomponents	Described Biological Effects
Metals	Iron, Vanadium, Nickel, Copper, Platinum and others	Can trigger inflammation, cause DNA damage, and alter cell permeability by inducing production of reactive oxygen species in tissues
Organic Components	Many are absorbed into particles; some volatile or semi volatile organic species form particle themselves.	Some may cause mutations, some may cause cancer, others can act as irritants, and can induce allergic reactions.
Biologic Origin	Viruses, bacteria, and their endotoxins, animal and plant debris, and fungal spores.	Plant pollen can trigger allergic responses in the air ways of sensitive individuals, viruses, and bacteria can provoke immune defense responses in the airways.
Ions	Sulfate (usually as ammonium sulfate), nitrate (usually ammonium or sodium nitrate), and acidity (H^+)	Sulfuric acid at relatively high concentrations can impair mucociliary clearance and increase air way resistance in people with asthma; acidity may change the solubility (and availability) of metals and other compounds absorbed onto particles
Reactive gases	Ozone, peroxides, aldehydes	Many absorb onto particles and be transported into lower air ways, causing injury.
Particle core	Carbonaceous material	Carbon induces lung irritation, epithelial cell, proliferation, and fibrosis after long term exposure

Source : HEI 2002

A number of disease conditions are attributed to air pollution. These comprise diseases related to respiratory and vascular systems. Diseases of other systems like haematopoitic, neurological, cardiovascular and immune are also linked to air pollution. Major air pollutants, their characteristics and principal health effects are given in the table 1.4.

Table 1.4.
Major Air Pollutants and health effects

Sl.No.	Air Pollutant	Main Characteristics	Principal health effects
1.	Particulate Matter	Any solid or liquid particles dispersed in atmosphere, such as dust, pollen, ash, soot, metals, and various chemicals;	Directly toxic effects or aggravation of the effects gaseous pollutants
			Aggravation of asthma or other respiratory or cardio-respiratory symptoms
		Particles often classified according to size, as settle able particles (larger than 50 micron), aerosols (smaller than 50 microns), and fine particulates (smaller than 3 microns)	Increased chest and cough discomfort
			Increased mortality
2.	Sulpher Dioxide (SO_2)	Colorless gas with pungent odor, oxidizes, to form Sulpher, trioxide (SO_3), which forms sulphuric acid with water	Classed as mild respiratory irritant
			Most SO_2 inhaled is absorbed in upper respiratory tract and never reaches lungs,penetrates when clings to particulate matter
			Aggravates respiratory diseases including asthma, chronic bronchitis
			Can result in reduced lung function
			Irritation of eyes Possible increased mortality

Sl.No.	Air Pollutant	Main Characteristics	Principal health effects
3.	Hydrocarbons (HC)	Organic compound in gaseous or particulate form. E.g. methane, ethylene, acetylene, component in formation of photo-chemical smog.	Acute exposure causes eye, nose and throat irritation chronic exposure suspected of causing cancer
			some groups of combustion hydrocarbons especially implicated in induction of cancer in laboratory animals
4.	Nitrogen Oxides (NO_x)	Brownish-red gas with pungent odor	Major role as component in creation in photo chemical smog; also has distinct effects apart from those associated with smog
			Has been shown to be toxic to experimental animals
			Increased risk of viral infections Lung irritation
			Higher respiratory illness rates
			Chest tightness and discomfort
			Eye burning, headache
5.	Carobon Monoxide (CO)	Colorless, odorless gas with strong affinity for hemoglobin in blood	Absorbed by lungs Reduced oxygen carrying capacity of blood
		Usually aware of presence of CO only after early poisoning symptoms appear	Creates reduced tolerance for exercise, impairment of mental function, effects fetal development
			Aggravates cardio vascular diseases
			Ringing in the ears, Poor reflexes, Headache, Dizziness, Nausea, Breathing difficulties, Drowsiness, reduced work capacities
			Comatose state (can lead to death)
6.	Photochemical Oxidants (smog)	Oxidizing type of pollutant found in many urban areas Results from chemical combination of reactive hydrocarbon vapors with nitrogen oxides in presence of sunlight	Aggravation of respiratory and cardio-vascular diseases, irritation to eyes, respiratory tract, impairment of cardio-pulmonary function Some concern about possible mutagenic effects of ozone
		The resulting production of photochemical oxidants consists of number of toxic compounds: ozone, peroxyacetyl nitrates (PAN), aldehydes, other chemical compounds	Possibility of developing tolerance to oxidant pollution such as has been shown for experimental animals may account for the relatively few changes associated with chronic exposure

Sl.No.	Air Pollutant	Main Characteristics	Principal health effects
7.	Sulfates	Aerosols formed by oxides, in moist environment appears as sulphuric acid (H_2SO_4) mist or rain	Aggravation of respiratory diseases, including asthma, chronic bronchitis Reduced lung function Irritation of eyes and respiratory tract Increased mortality
8.	Lead (Pb)	Heavy, soft, malleable gray metallic element; often occurs (as environmental contaminant) as lead oxide aerosol or dust	Enters primarily through respiratory tract and wall of digestive system More than 40 percent of lead inhaled is absorbed into bloodstream Accumulates in body organs Symptoms of lead poisoning not very specific Early signs are impairment of mental function, behavior problems and anemia Higher levels cause vomiting, cramps, serious impairment of kidneys, nervous system, possible brain damage Damage of reproductive system

Source: Ghosh, A.K., 1989, Environmental Conservation in 'Environmental Awareness and Wildlife Conservation". p 1-15, Zoological Survey of India, Calcutta

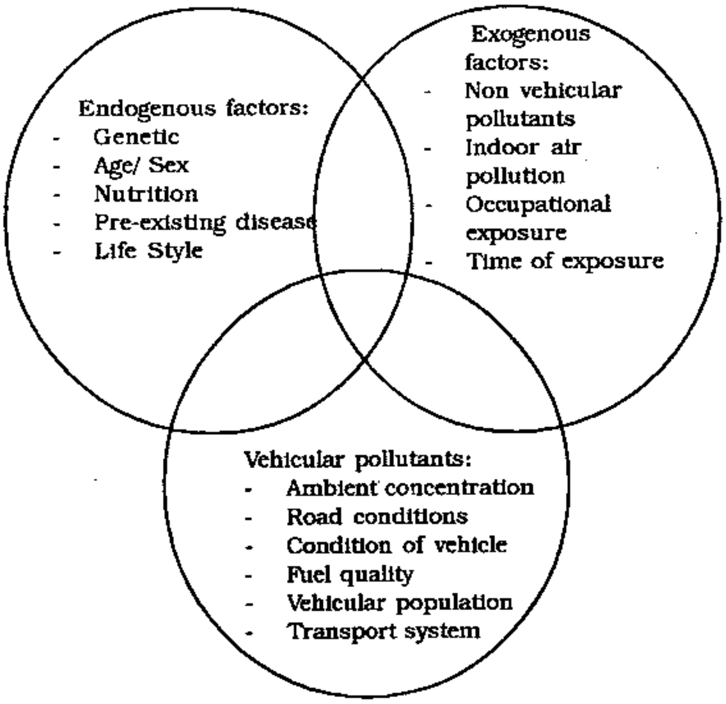
Air pollution can be caused due to both outdoor and indoor sources. The sources of outdoor pollution are industries, automobiles, mines, dusts, etc. A number of sources contribute to indoor air pollution. Some of these are burning of fuel for cooking and heating, material used in buildings construction including furniture, furnishings and their maintenance, environmental tobacco smoke, and most important the presence of biological agents (microbes, fungi, moulds, insects and their remnants). As per CPCB, health problems arising out of indoor air pollution are wide spread because of the following reasons:

1. An indoor pollutant is one thousand times more likely to reach the lungs as compared to outdoor pollutant
2. More and more people in urban areas are spending major part of their time indoors.
3. Women, children and the aged are more vulnerable to indoor pollutants.
4. Indoor air pollution has been linked with respiratory diseases (Acute infections, chronic obstructive lung disease, asthma, lung cancer).

In addition to outdoor and indoor pollution, a number of other factors play a part in the contraction of diseases. The interplay of these factors is conceptually depicted in chart 1.2 which is a diagrammatic representation of air pollution related diseases and their causes in a population.

Chart 1.2

Interplay of different factors responsible for contraction of disease



§6 MECHANISM OF HEALTH EFFECTS

§6.1 DOSE

While ambient concentrations are normally used as a proxy for linking ambient air pollution to health effects, it is the dose that actually matters. Dose is defined as the quantity of material that reaches a target. Knowledge of the dose of PM delivered to a target site or sites in the respiratory tract is important for understanding possible health effects associated with human exposure to ambient PM. The effect, however, of varying dose and potential duration of the effect are functions of the retention and clearance of particles from the respiratory tract, which in turn are affected by the characteristics of the inhaled particles (size, number, and composition) and the physical and physiological characteristics of the exposed population.

§6.2 LUNG DEFENSES

The adult human lung, with a surface area of 40-120 square meters, comes in contact with between 10,000 and 20,000 liters of ambient air daily. The lung has evolved a multilayered defense mechanism to counter inhaled particles, and it responds to particulate pollution with the same defense mechanism as it does to pollens and spores **[UK DEFRA 2001]**. The first layer of defense is a barrier of cells and fluids that the foreign matter must penetrate before it enters the tissues of the body. Fluid secretion, such as mucus lining the airways, is an important part of the first layer that essentially traps and removes the larger particles. Coughing, activated by the presence of particles, also helps in the removal process. If the particles infiltrate the first layer of defense, "scavenger" cells come into play. These cells ingest the particles and attempt to destroy them. If the burden o foreign matter overwhelms this line of defense, as can occur in response to inhaled particles, the lung defenses may be weaker from subsequent attacks

§6.3 SPECIFIC HEALTH EFFECTS

The PM damage to lung defenses manifests itself in the form o health effects such as acute respiratory infection (both upper and lowe respiratory tract infections), chronic obstructive lung disease (especiall bronchitis), asthma attacks, cardiovascular disease, and lung cancer. Further recent research has increasingly shown that particles can also affect othe parts of the body, including the nervous system, by physically moving out o the airways and into the blood stream [4]. Thus particle deposition i airways can set off a chain of events, potentially affecting parts of the bod other than just the respiratory tract. As can be expected, the changes in th body are likely to be more severe in cases where the body's defenses ar already weak or previously damaged. Hence, certain population subgroups such as the elderly, children, and individuals with existing respiratory o cardiovascular diseases, are at increased risk from exposure to PM.

§7 EMPIRICAL FINDINGS

The World Health Organization (WHO) estimates that each yea approximately 800,000 deaths and 4.6 million lost life-years worldwid are attributable to urban air pollution **(WHO 2002)**. This burden is n equally distributed, however: approximately two thirds of the deaths an lost life-years occur in the developing countries of Asia. Owing to limitation of the available epidemiologic studies in those countries, WHO's estimate are based largely on the results of research conducted in Europe and North America that have been extrapolated to developing countries. Asia differs from Europe and North America in the nature of its air pollution, the conditions and magnitude of exposures to that pollution, and the health status of its populations. These differences create large uncertainties in estimating the burden of air pollution and any other effort to estimate health impact of air pollution in Asia **(Cohen et al 2004)**. The past 10 to 15 years have seen a remarkable increase in research on the health effects of air pollution. It is now widely accepted that exposure to outdoor air pollution is associated with a broad range of acute and chronic health effects, ranging from minor physiologic disturbances to death from respiratory and cardiovascular disease **(Bascom et al 1996a, b; American Thoracic Society 2000)**. This knowledge is based on observational epidemiologic studies of disease occurrence in human populations and in-vitro and in-vivo experimental studies of animals and humans. Epidemiologic research on the health effects of air pollution provides estimates of the health effects of both short- and long-term exposure to a variety of air pollutants in human populations in many parts of the world. Because these estimates apply to humans living in real-world conditions, they have been the scientific basis for increasingly stringent air quality regulations for some pollutants. A limited but rapidly growing body of toxicologic evidence from in-vitro animal and human research has suggested mechanisms by which exposure to air pollution might produce the respiratory and cardiovascular outcomes observed in epidemiologic studies **(Bascom et al 1996a,b; Kodavanti and Costa 1999; Godleski 2000; Health Effects Institute 2002)**. The health effects of air pollution are associated with a number of the pollutants that make up the air pollution mixture in cities around the world, the ambient concentrations of which are monitored periodically. Disentangling the effects of one pollutant from the effects of others that follow similar spatial and atmospheric patterns is often difficult **(Health Effects Institute 2001)**. At the same time, not all members of a population are equally sensitive to such effects. Some subgroups (eg. the elderly, asthmatics, children, and people with heart disease) may be more at risk from exposure to air pollution **(Health Effects Institute 2002)**.

In **China** Chang **(Chang et al 2003a, 2003b)** found that at Beijing air borne levels of CO, SO_2, NO_x and PM_{10} each correlated significantly with mortality, especially from respiratory disease, cardio vascular disease (CVD), COPD etc. TSP levels were associated with respiratory disease. They also found that when CO, SO_2, NO_x or PM_{10} increased by 100 $\mu g/m^3$, visits for cold, bronchitis and pneumonia also increased by 1%-8%. Chen **(Chen et**

al 1995) found strong adverse pregnancy outcomes among women living near a petrochemical plant. Conditional logistic regression **(Kan and Chen 2003)** identified in relative risk of death from COPD and CVD for each 10 $\mu g/m^3$ increase in NO_2, SO_2 and PM_{10} (in decreasing intensity of impact). In a study on lung cancer mortality **(Li et al 1994)**, lung cancer was found higher in 1985-1989 compared to 1970-1974 and correlation analyses attributed rate of lung cancer to air pollution. In another study **(Qian et al 2000)** TSP levels were found significantly associated with the adjusted odds ratios for cough, phlegm, hospitalization and pneumonia. Wang, in his study **(Wang 1992)**, found the effects of air pollution on human health in cities were more severe than in the suburban and rural areas and respiratory complaints correlated with the level of pollution. Xu **(Xu et al 1995 a)** found a significant dose-dependent association between preterm delivery and levels of SO_2 and TSP. Results **(Xu et al 1995 b)** suggested an exposure–response relation between TSP and SO_2 and hospital outpatient visits both at high air pollution levels and at levels well below WHO air quality standards. Standardized questionnaires **(Zhang et al 1999)** revealed increased rates of cough, phlegm, persistent cough and phlegm, and wheeze associated with increasing TSP levels. Positive associations were found **(Zhang et al 2002)** between respiratory morbidity and all outdoor PM levels, especially $PM_{10-2.5}$. A weaker but still positive association was found with NOx and SO_2. Daily admissions for asthma increased significantly in Hong Kong **(Wong et al 2001)** with increases in ambient NO_2, SO_2, and inhalable particles.

In **India**, medical studies that bring out such cause-effect relationships between the patients suffering from diseases attributed to air pollution and their exposure histories are lacking. There is a need to take up such studies. As disease is the end point of interaction between different endogenous and exogenous factors in the human body, it is difficult to attribute a specific disease to a particular etiological agent. This is specially true in the case of multiple environmental exposures. The multi-factorial theory of disease causation is well established and universally accepted. Properly designed epidemiological studies are so designed as to calculate the attributable risks for different exposure agents, which indicate the proportion of disease in a population that could be attributed to a single exposure factor or a group of factors being examined.

Epidemiology has often associated particulate matter pollution with asthma exacerbation and other cardio respiratory illnesses, even in cities with relatively mild air pollution. A study conducted by the All India Institute of Medical Sciences (AIIMS) in Delhi showed that exposure to PM has an impact on respiratory health and contributes to respiratory morbidity. It indicated that most common symptoms related to air pollution were irritation of eyes, cough, pharyngitis, dysponea and nausea6. Despite very large uncertainties in all valuations of impacts of air quality, the choice of PM_{10} as a measure of particulate air pollution to be controlled is based on a biological plausible argument, given the aerodynamic characteristics of the human respiratory tract. Some of the smallest particles, called respirable particulates may tend to be deposited in the alveoli (tiny air sacs in the lungs and are of health concern because they can reach the deepest recesses of the lungs). Scientific studies have linked fine particles (alone or in combination with other air pollutants), with some health problems.

A few studies have been done in India which are area/region specific. For example, higher prevalence of diseases correlated with the higher environmental pollution in Eloor- Kalamassery area of Cochin is reported **[Medical College, Thiruvananthapuram, 1992-94]**. In another study, the average concentration of lead in atmospheric air particulates in different suburbs of Mumbai for almost a decade along with their spatial and temporal profiles in relation to emission sources. According to the study, the concentration of lead in the entire residential suburban atmosphere is well below the CPCB prescribed limit of 1.5 mg/m3, barring a few exceptions for some residential and industrial sites, such as those of Thane and Kurla scrap yards. The correlation between blood lead and air lead reveals that the blood lead level in children could increase by 3.6 mg/dl for an incremental rise of 1.0 mg/m3 of air. The temporal profile of air lead values indicates a decreasing trend in residential suburbs **[Tripathi et al 2001]**. As per CPCB **[CPCB 2001]**, lungs are the only significant routes for CO uptake from the environment. It diffuses rapidly across alveolar, capillary and placental membranes. Inhaled CO has no direct toxic affects on lungs but rather appears to exert its effects by interfering with oxygen transport through the formation of carboxy-haemoglobin (COHb). In a study conducted, healthy subjects exposed to CO, sufficient to result in 5 per cent COHb levels exhibited reduced duration of exercise performance and consumption of oxygen. Studies involving subjects with deficient blood supply to the heart (Ischemic Heart Disease) who were engaged in exercise during exposures have shown that COHb levels as low as 2.2 per cent can lead to earlier onset of electrocardiograph change indicative of increased deficiency of oxygen supply to the heart, earlier onset of chest pain, increase in the duration of chest pain and decrease in oxygen consumption. In another comprehensive review **[Hext et al 1999]** information on $PM_{2.5}$ particles in the context of the potential to induce adverse health impacts had concluded that the result of over 50 studies reviewed by them show that the overall pattern that emerges is that $PM_{2.5}$, at normal ambient levels or those seen during episodic pollutant increases, poses limited risk, if any, to normal healthy subjects. Individuals suffering already from cardio respiratory disease or predisposed to other respiratory diseases such as asthma may be at risk of developing adverse responses to exposure to increased ambient levels of $PM_{2.5}$.

Bladen found **(Bladen 1983)** in Bombay that air pollution from SO_2, PM, CO, and HC were markedly associated with acute respiratory illness. This finding was especially strong in November–February when pollution concentrations were higher due to thermal inversions. In some study by Kamat **(Kamat and Doshi 1987, Kamat et al 1980, Kamat et al 1992)** Air pollution was found related to several respiratory symptoms as well as an increase in mortality due to cardiac, respiratory, and malignant diseases. In Mumbai, the 3 urban areas of low, medium, and high air pollution levels, low pollution was associated with higher lung function at all ages. In the rural area, however, lung function was significantly lower despite lower pollution. Other factors possibly contributed to this difference. Respiratory symptoms and cardiac diseases appeared to occur more commonly in more polluted areas. In a study by Kumar **(Kumar et al 2000)**, a respiratory questionnaire and lung function spirometry tests revealed a higher prevalence of respiratory problems associated with higher levels of SO_2 and NOx.

In **Indonesia**, Aditama found **(Aditama 2000)** from data collected from pulmonologists, health offices and hospitals indicated a "significant" increase in respiratory conditions that the authors attributed to air pollution haze. Tri-Tugaswati and Yasuo **(Tri-Tugaswati and Yasuo 1996)**, in a self-administered questionnaire, found the prevalence of persistent cough was 7.3–10.8% and of persistent phlegm was 4.5–5.0%. A significant relation was found between NO_2 exposure and the prevalence of cough, phlegm, and wheeze.

In **Japan** regional differences in age-adjusted lung cancer death rates were explained by NO_2 and temperature. Temperature increased the effect of NO_2 on lung cancer deaths compared with NO_2 alone in one region. Comparing questionnaire data and meteorological data Setiani identified **(Setiani 1996)** a significant association between SO_2 and lacrimacy, runny nose, and cough, and between O_x and phlegm. NO_2 levels were negatively associated with phlegm. Shima and Adachi found **(Shima and Adachi 2000)** a significant association between wheeze and asthma and outdoor NO_2 levels but no such association with indoor NO_2 concentration. The data did suggest that girls may be more susceptible to indoor NO_2 than boys are. Some studies **(Shimizu et al 2001, Tango 1994, Shima et al 2003)** found positive correlation between the number of asthma attack visits and increasing level of pollutants. They also found positive association between NO_2 and rate of increase in lung cancer mortality. Ye found **(Ye et al 2001)** concentrations of NO_2 or PM_{10} were associated with daily hospital emergency transports for angina, cardiac insufficiency, asthma, acute and chronic bronchitis, and pneumonia among men and women.

In **South Korea** study **(Hong et al 2002a)** revealed estimated increase in stroke mortality was 1.5% for each interquartile increase in PM_{10} and ozone in the same day. Stroke mortality increased 3.1% for NO_2, 2.9% for SO_2, and 4.1% for CO in a 2-day lag for each interquartile increase in single pollutant models. The elderly and women were more susceptible to particulate pollutants. Hong **(Hong et al 2002b)** also found TSP, SO_2, NO_2, CO, and O_3 levels were significantly associated with ischemic, but not hemorrhagic, stroke mortality. An increase in PM_{10} was found **(Kwon et al 2001)** associated with an increase in mortality from congestive heart failure. CO, NO_2, SO_2, and O_3 were also associated with mortality from congestive heart failure. In a study **(Lee et al 2002)** estimated relative risk of asthma hospitalization was 1.07 for PM_{10}, 1.11–1.16 for SO_2, NO_2, O_3, and CO. In the multipollutant models, control for other pollutants did not change the estimated effect for O_3 or NO_2. In **Thailand** a significant increase in school absence and medical expenses was found **(Pothikamjorn et al 2002)** associated with high PM_{10} exposure.

Both the international and national literature indicates evidence of health damage as a result of air pollution. But robust evidence is required to substantiate this. We have learned much about the adverse health effects of outdoor air pollution in the past 20 years, with epidemiologic research playing a critical role in the growth of this knowledge. Nonetheless, important gaps remain, especially the following questions that are critical to public policy. How large is the effect of short-term exposure on daily morbidity and mortality, and how accurately we can measure the magnitude and variability of these effects with our current statistical tools? Which constituents of the pollutant mix, and which sources that contributes to it, are the most toxic? Which diseases, social conditions, or genetic factors place people at greatest risk when exposed to air pollution? To what extent does long-term exposure affect the development of chronic, life-threatening disease? The answers to these questions might well be different in Asia than in the developed West, where to date the vast majority of studies have been conducted. A consensus on its risk in an epidemiological perspective is, however, not available. Studies in different locations and geographical situations are required for policy decisions and devising preventive strategies.

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Understanding the Dynamics of Epidemiological Systems with reference to a Host-Pathogen Model

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Abstract

Applications of mathematical concepts to understand the general epidemiological system, in the form of a host-pathogen model, have been discussed. A mathematical model, representing a basic host pathogen system of biological origin, is considered. Generalization of the model to include recovery of infected host to the healthy class as well as a time delay in the lysis death of the infected host population, have been considered. It is argued that the process of cell lysis requires a finite time for completion and hence the delay in the lysis death. It is observed that inclusion of delay does not introduce any instability in the system. However, the delay in the cell-lysis is observed to compete with the recovery of the infected population. Reminiscence of the stability characteristics of the non-delayed model are observed to exist for the delayed model. But significant quantitative shifts are observed in some of the calculated thresholds of the model.

Introduction

Biological systems of various types and forms are essential constituents of nature and their well-being is of paramount importance for the upkeep of its balance and for its very existence. It is thus imperative to gather sound knowledge and understanding of these biological systems. The interdisciplinary fields of research, where application of Mathematical and Physical concepts are gaining momentum, have already signified to be of great potentials and the relevant field is termed to be theoretical biology.

Applications of mathematical concepts to gather understanding about various natural systems including those of biological origin have been a century old practice. Mathematical equations have been in use to track the behaviour of epidemiological systems. Daniel Bernoulli, in a pioneering effort, used mathematical time differential equations to understand dynamics of small-pox epidemics [1], specifically to know about the spread of the disease. W. A. Hamer formulated and analyzed a discrete time model to gather knowledge about the recurrence of measles epidemics in order to contain the spread of the disease [2]. The fact that Sir Ronald Ross used the mathematical tool of differential equations to gain understanding about the epidemiological outbreak of the disease Malaria before discovering the malarial vaccine, has been recorded in the literature [3].

Mathematical models have been developed to describe the behavior of various biological systems to predict dynamical changes over a period of time [4-8]. Host-Pathogen models are mathematical models pertaining to epidemiology depicting the interplay of pathogens and host densities with time and are needed to be studied with renewed interest in view of the emergence and re-emergence of epidemiological diseases in the contemporary global society. In this communication we consider such a Host-Pathogen model including recovery [9] of the infected host to that of susceptible and with a time delay in the cell-lyses. Analysis of this model would give us insights regarding the procedures of containment of various epidemiological diseases. Specifically we will consider here to build up the basic host pathogen model by use of mathematical concepts and tools and then discuss about the inclusion of recovery as well as time-delay in the cell-lyses term. Further we will consider numerical analysis of the mathematical prototype of the host-pathogen system, discuss our results and make probable predictions regarding eradication of the disease.

One needs to define here a host-pathogen model. A host-pathogen model is the representative of epidemiological systems pertaining to biology that depicts invasion of some host species' populations by pathogens. Such system may often find logical description in mathematical terms. Such descriptions by a set of equations are termed as the host-pathogen model. Let us elaborate the definition.

A general host-pathogen model consists of a set of time differential equations signifying the interactions as well as interplay of pathogen and host densities with time. These mathematical models are of paramount importance to understand the emergence and re-emergence of epidemiological diseases within various host species. In this type of model the host population is divided into two classes, namely susceptible which are healthy organisms and infected individuals. The mathematical model actually narrates the change in densities of susceptible host (S), infected individuals (I) and pathogens (V) including various realistic parameters arising out of influence from environment, immunization, inter-class contact etc. These classes of models are important in their own rights, but, in addition, they find relevance for predator-prey and host-parasite models. In case of the interplay between predator and prey in ecological systems, the effect of diseases on the population dynamics of model constituent, is an important area from mathematical as well as ecological point of view.

Towards the Basic Host-Pathogen Model

Evolving a model to represent a biological system requires a suitable translation of the biological system into a mathematical prototype with the help of a set of hypotheses or assumptions. These assumptions are sort of building blocks in the process of transcribing a biological system into its mathematical prototype. Assumptions are unverified proposition that tentatively explain certain facts. Set of assumptions for a specific case are, however, justified and substantiated by a string of logical, relevant and realistic arguments. Mathematical prototype of a general epidemiological host-pathogenic system is well represented by a set of time differential equations.

Let us now consider the formulation of a model to represent a basic host-pathogen system. Let, N = Number of host cells/designated area, V=Number of pathogens /designated area. Pathogenic Infection divides Hosts into two disjoint classes Succeptible (S) and Infected (I). Assumptions leading to Basic host-pathogen Model are:

A1. In the ideal case of no pathogen the growth of susceptible host population follows the logistic law [10] implying that this growth is entirely controlled by an intrinsic birth rate constant $r(\in R_+)$ with a carrying capacity $K(\in R_+)$. Logistic growth is mathematically represented as $\frac{dN}{dt} = rN(1-\frac{N}{K})$

A2. Introduction of pathogens divides the host population into two disjoint classes, Susceptible *(S)* and Infected *(I)* with $N(t) = S(t) + I(t)$ at any instant of time *t*

A3. Infected hosts *I* do not take part in reproduction and hence does not contribution to the growth of N, the total host population but contribute to the carrying capacity of S. Infected host offers for pathogen replication within them and suffers death at the completion of replication known as cell-lysis. *I* competes with the S for resources to enable Pathogen Replication within them

A4. Spread of the disease in the model is assumed to be among the host species only and infected prey is more vulnerable to predation by pathogen than the susceptible prey as evidenced in the natural systems.

A5. At any instant of time *t*, the whole of susceptible host *(S)* are equally susceptible and all infected hosts *(I)* are equally infectious. Two ways for the spread of disease is assumed: (i) by contact of S with I and (ii) by contact of S with V, following the Law of Mass Action. Note that some researchers argue for proportional mixing between S and I rather than simple mass action law based on the results of Greenwood experiment [11]. However it is observed that in both the cases the quantitative results remain unchanged.

Following the above assumptions, the mathematical prototype of a biological host-pathogen System can be written as

$$\begin{aligned} \frac{dS}{dt} &= rS(1-\tfrac{S+I}{K}) - \lambda SI - \gamma SV \\ \frac{dI}{dt} &= \lambda SI + \gamma SV - dI \\ \frac{dV}{dt} &= -\gamma SV + \eta dI - \mu V \end{aligned} \qquad \text{.............}(1)$$

Where λ = Rate of infection by Infected host (I), γ = Rate of infection caused by V, d = Lysis Death rate constant of I, η = Rate of cell lysis (Pathogen Replication Factor), μ = Natural death rate of Pathogens.

Inclusions of Recovery and Delay in Cell-Lysis

In the basic model of a biological host-pathogen System, it is observed that there is no scope of healing of the infected host from the disease. However in reality, recovery from the disease does exist. Such recovery may be owing to various clinical and/or other human activities or implementations. Inclusions or recovery from the disease within the model means the transition of a fraction of infected host (I) to the susceptible host (S). For the purpose of including a term representing recovery, we need to make an assumption as follows:

Assumption : Infected hosts do not grow or reproduce but, can recover from pathogenic infection and add to the susceptible hosts. Such recovery would stem out from immunization, vaccination or other clinical processes or could be achieved through adaptation. Consider a parameter δ = rate of recovery [with $\delta(\in R_+)$].

Further, note that in the basic model an instantaneous lysis-death of an infected host has been considered which means zero latency for pathogens involved in lysis. But again, in reality lysis growth (replication) of pathogen within the infected host requires a finite definite time. In other words, there must be a time lag between pathogenic infection caused to the host and its lysis-death. This requires us to include a delay in the death of I and Lysis Growth of V. Again we make the relevant assumption as:

Assumption : A positive discrete time lag between the infection of host cells and the death of them. This necessitates inclusion of delay in the death of infected hosts I and a positive discrete latency period (delay) of the intruding pathogens inside the infected hosts where they get replicated.

Delay at an instant of time (*t*) can be included through a delay kernel or memory function *F(u)* as *I(u)F(t-u)*. For the average effect of the delay at a time *t* one needs to integrate *I(u)F(t-u)* over the range ($-\infty$ to *t*). Under suitable conditions the memory function becomes a delta function giving $F(u) = \delta(u-\tau)$, with $\tau \geq 0$ and the delay in infected host population appears as $I(t-\tau)$

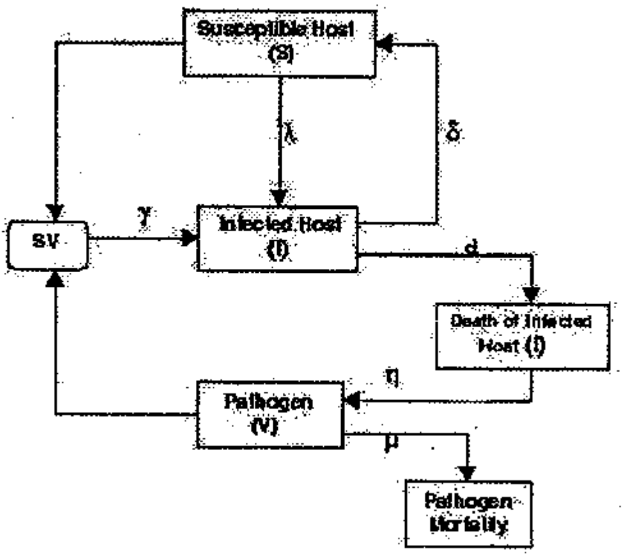
The model equations including the recovery term and the delay factor τ in the lysis-death of infected host population becomes

$$\frac{dS}{dt} = rS\,(1 - \tfrac{S+I}{K}) - \lambda SI - \gamma SV + \delta I$$

$$\frac{dI}{dt} = \lambda SI + \gamma SV - dI\,(t-\tau) - \delta I \qquad \text{..................} \; (2)$$

$$\frac{dV}{dt} = -\gamma SV + \eta dI\,(t-\tau) - \mu V$$

The above equations represent a generalized mathematical prototype of realistic host-pathogen system from biology including finite latency in the lysis of pathogens. A schematic Diagram of the model with the recovery term can be represented as follows



Numerical Solutions of the Model Equations

To understand the dynamics of the model system evolved in the earlier section, one needs to solve these equations with well defined values of the model parameters. Basic outcomes are time-series solutions of the model variables (S, I and V here) of the system. Detailed analysis of these solutions would generate an understanding of the dynamical behaviors of the associated model system and by virtue of this one can gather knowledge of the analogous epidemiological system in general.

Below we list the numerical values of model parameters used to solve the model equations.

Table.1 : Values of Model Parameters used for Numerical Solutions

Parameter	Definition	Reported Range	Default Values
r	Maximal Growth rate of Susceptible Host	$10 - 15$ Day $^{-1}$	11 Day $^{-1}$
K	Carrying Capacity	$30 - 40$ mm^{3}	35 mm^{3}
λ	Force of Infection through contact with Infected Host	$0.15 - 0.25$ mm^{3} Day $^{-1}$	0.2 mm^{3} Day $^{-1}$
γ	Force of Infection through contact with Pathogens	$0.02 - 0.04$ mm^{3} Day $^{-1}$	0.03 mm^{3} Day $^{-1}$
d	Rate of Lysis-death of Infected Host	$2 - 4$ Day $^{-1}$	2.5 Day $^{-1}$
η	Pathogen Replication Factor	$85 - 150$	115
μ	Natural Death Rate of Pathogens	$1.5 - 3.0$ Day $^{-1}$	2.2 Day $^{-1}$
δ	Rate of Recovery of Infected Host	$5 - 110$ Day $^{-1}$	Varied in a Range
τ	Delay Factor	In units of Day	Varied in a suitable Range

For numerical calculations we always comply with the positive octant restriction on the variables S, I, V at time $t = 0$ (i.e., $S>0$, $I>0$, $V>0$) and thus choose $S(0)=200$, $I(0)=10$, $V(0)=2$. We consider variation of recovery rate δ and a variation in time delay factor τ to understand the bearing of these parameters on the model dynamics. Our predominant focus is to carry out a comparative study of the dynamics for the non-delayed and delayed models. Note that the parameter δ is taken in units of day^{-1} and is varied in a wide range from $\delta = 0$ to about $\delta = 110$. The delay factor τ is also varied in a suitable region.

Let us now elaborate on various aspects of the solutions of model equations. In figure.1 we plot time series solutions of the hosts and pathogen populations for a recovery rate $\delta = 0.4$ and with $\tau = 0$, that is for the non-delayed model system. We have found solutions for a time well beyond that needed to stabilize the system. Figure.1 shows that all three populations assume oscillatory solutions and they are bounded by upper and lower limits yielding stable limit cycle solutions. As we increase the value of δ for the same non-delayed system, upper and lower limits of them gradually come closer and asymptotically the solutions are single valued and stable [see figure.2(a)]. Keeping $\delta = 0.8$ as in figure.3(a), we now look at the time series solutions of different population for nonzero $\tau = 0.1$, i.e., the delayed system [see figure.2(b)]. We find that inclusion of delay makes the system to move again to oscillatory limit cycle solutions.

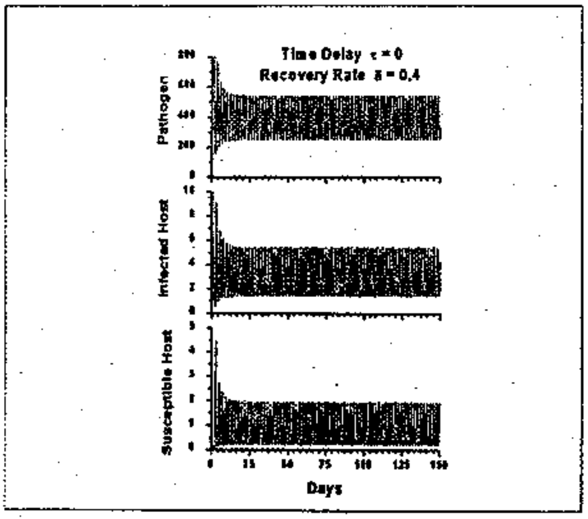


Figure.1: Population densities of S, I and V are plotted as a function of time for the Recovery rate δ = 0.4 (in units of per designated area). Values of other parameters are as given in Table.1.

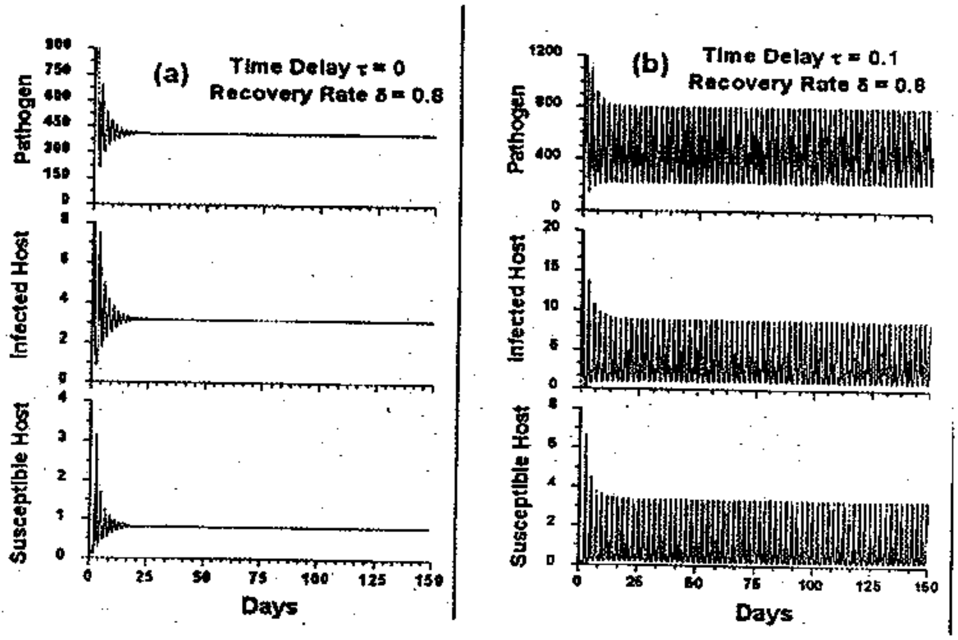


Figure.2: S, I and V densities are plotted as a function of time for recovery rate (δ = 0.8). Part **(a)** corresponds to non-delayed model (τ = 0) and **(b)** is for delayed model (τ = 0.1). Smearing out of single valued equilibrium solution is apparent for delayed model.

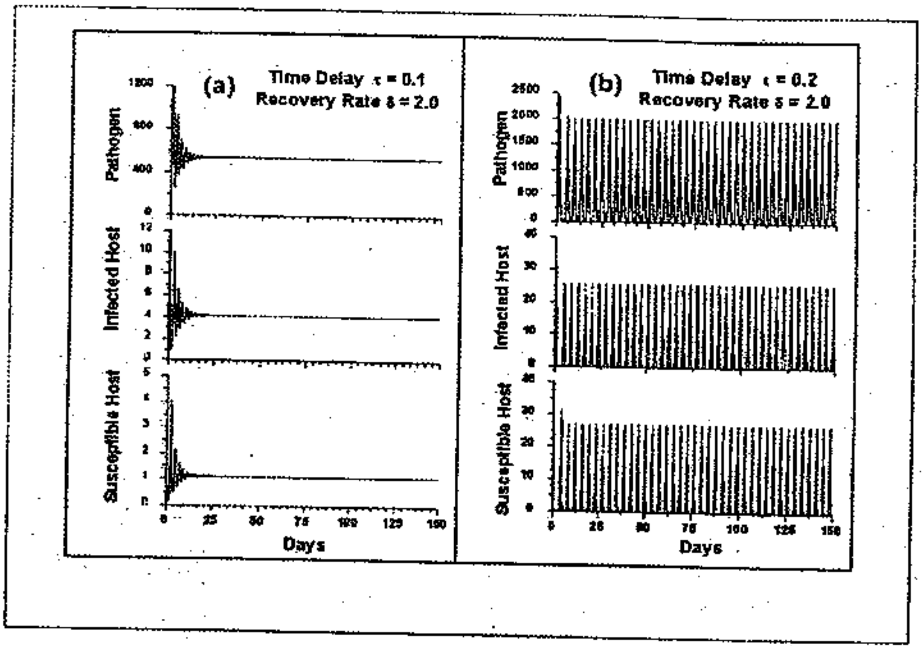


Figure.3: Time series solutions of S, I and V populations are plotted as a function of time t. Value of the delay factor τ is increased from **(a)** to **(b)** to demonstrate the competing dominance of delay over recovery rate by way of introducing local instability around the equilibrium solutions.

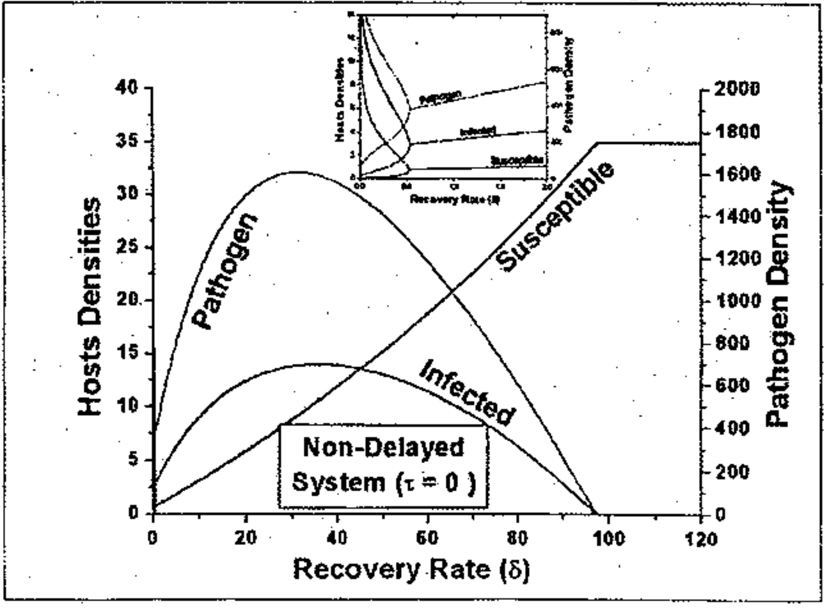


Figure.4: Equilibrium solutions of S, I and V are plotted with the recovery rate for non-delayed system. Host densities correspond to the left y-axis and Pathogen to that at right. Inset gives small-δ solutions of populations showing upper and lower limits of multi-valued oscillatory solutions.

In figure.4 we have plotted stable solutions for S, I and V in a wide range of values of δ for the non-delayed system ($\tau = 0$). Inset shows the same for small values of δ. We find that around $\delta \le 0.6$ stable solutions are oscillatory for all three populations where bifurcating lines towards lower δ denote the upper and lower boundary of such oscillatory solutions corresponding to each population. Towards larger δ we find that stable (solution) value of susceptible host increases monotonically, but the same for infected host and pathogens rise faster. Beyond certain δ, rise in I and V stable values are checked and for further increasing δ they turn downwards. Beyond a very large value of $\delta \sim 100$ extinction of infected population as well as the pathogen population take place. The stable value of I goes to zero at δ = 99 which is slightly lower than that for V stable value, facing extinction at δ =100.5. For $\delta \ge 99$, at which extinction of I takes place, stable solution S becomes completely flat and attains the numerical value of the carrying capacity K=35 (in the present case).
Figure.5 contains plots of the stable solutions of different populations for the time-delayed system ($\tau = 0.2$), where the small-δ part is expanded in the Inset. Towards small δ the solutions for all three populations remain oscillatory and bounded by upper and lower limits. This state persists till a value of δ much higher (δ ~ 4.0) than the non-delayed system. Towards higher δ, qualitative features of the stable solution curves are similar to those of non-delayed system, but with significant quantitative shifts. At $\delta \ge 100$ eradication of infected host and pathogen populations take place and susceptible host population assumes the globally stable numerical value 35 which is the carrying capacity K (in the present case).

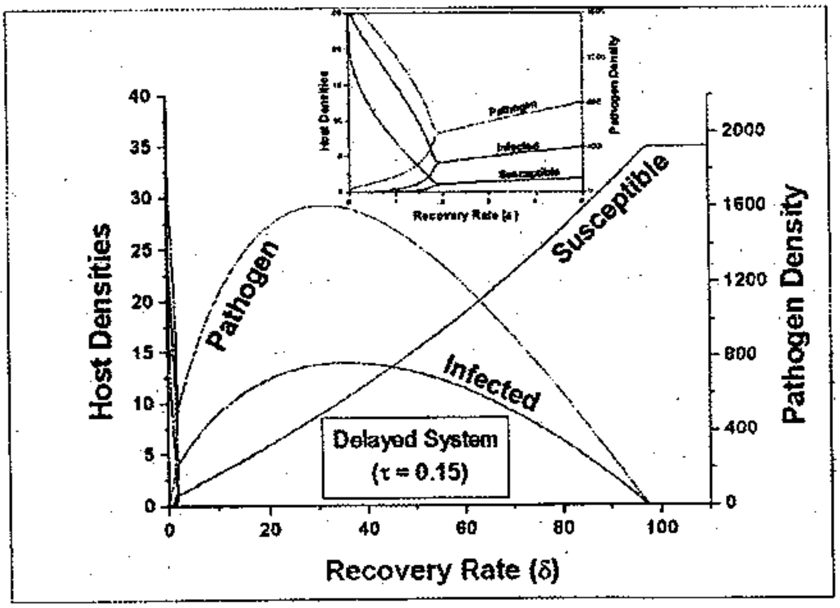


Figure.5: Equilibrium solutions of S, I and V as a function of the recovery rate for delayed system ($\tau = 0.15$). A magnified multi-valued solution regime is seen in the inset for small δ

Discussion and Conclusions

We have formulated a mathematical model representing a general host-pathogen system that has a finite recovery of the infected host to the susceptible class including a time delay in the lysis-death of infected host. The model system has practical relevance to the epidemiological systems in general. The set of differential equations constituting the parent non-delayed model are solved analytically to judge the characteristics of the model dynamics. Model equations are also solved numerically focusing primarily on the effects of delay on the model dynamics. Comparison of results of the systems with and without the delay have been made.

Complete numerical solutions of the model equations, for the mentioned choice of parameters (as in Table.1) and for varying recovery rate (δ) and (τ), are considered. These results are in line with the analytical findings. We observe that the inclusion of delay in the lysis-death of infected population inject instability in the system which is manifested as the transition of time series solutions from the stable single-valued to a regime of bounded periodic multi-valued solutions. In other words, delay inflicts a local instability around the solutions of interior equilibrium. As depicted in the figures 2 - 5, time delay factor (τ) apparently compete with the recovery rate (δ) to smear away the effect of recovery and the stability of the system as well. Delay introduce significant quantitative shift in the phase diagram of stable solutions versus recovery rate (S^*, I^*, V^* - δ) where delay actually extends the domain of multi-valued periodic solutions towards higher δ. In the non-delayed case we find that for very small to moderate values of recovery rate ($\delta \leq 0.6$), stable solutions for all the different populations are multi-valued and oscillatory, but for delayed system ($\tau = 0.2$), the domain of oscillatory solutions persists till $\delta \sim 4.0$.

Towards very large values of the rate of recovery, both delayed and non-delayed systems behave almost in similar fashion. This is probably because of the fact that corresponding delays (τ) are not comparable to very high recovery rate and the system steadily moves towards global stability with the extinction of infected host and pathogen populations. For both the delayed and non-delayed cases, when recovery rate attains a value $\delta \sim 100$ (in units of per designated area), the susceptible host population (S), with the passage of time, only survives while infected host (I) and pathogen (V) populations are pushed to zero. This implies that beyond this value of recovery, the host population automatically becomes free from any pathogenic attack which, in biological terms, means that the system enters a disease-free zone. Our numerical calculations show that removal of infected host population by way of its death and recovery to susceptible host makes it more and more unavailable for pathogenic lysis and hence the extinction of the pathogen population.

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