

CURRICULUM VITAE

Mukul Das, Ph.D

Personal data

Date & place of birth : July 9, 1956, Lucknow, India

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Office Address : Chief Scientist & Area Coordinator
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Marital status : Married

Academic Qualification

1976 : B.Sc Chemistry, Botany and
Zoology, First Division
Lucknow University, Lucknow.

1978 : M.Sc Biochemistry, First Division
Lucknow University, Lucknow.

1983 : Ph.D. Kanpur University/ITRC, India
Thesis: Chemical studies on brain xenobiotic
metabolism

Advance training/Deputation abroad:

1. Visited on invitation to Kuopio University, Finland, to attend 4th International Conference on Cytochrome P-450 from June 1-3, 1982.
2. Awarded research associate fellowship from Department of Chemistry, Wayne State University, Detroit, USA from Sept.1982 to July 1983
3. Awarded research associate fellowship from Department of Dermatology, Case Western Reserve University, Cleveland, USA from July 1983 - Oct. 1986.
4. Visited on invitation to Cologne and Gottengen, West Germany to attend the 4th Annual Meeting of Skin Pharmacology Society and Workshop on Photodermatology from May 31 - June 21, 1987.
5. Visited on invitation to Prague, Czechoslovakia to attend XIV International Congress of Biochemistry from July 5-15, 1988.
6. Visited on invitation to WHO, Geneva, Switzerland to attend 63rd Meeting of Joint Expert Committee on Food Additives from June 7-18, 2004.

7. Visited on invitation to Dubrovnik, Croatia to attend EUROTOX 2006 and 6th CTDC Conference from September 20-26, 2006.
8. Visited on invitation to Brussels, Belgium as an expert to review Indo-EU projects on Functional Foods, March 15-20, 2009.

Positions held:

Feb 1980 - Jan 1982	Junior Research Fellow, Industrial Toxicology Research Centre, Lucknow, India.
Feb 1982 - Aug 1982	Senior Research Fellow, Industrial Toxicology Research Centre, Lucknow India.
Sept 1982 - July 1983	Research Associate, Department of Chemistry, Wayne State University, Detroit, Michigan, USA.
July 1983 - June 1985	Research Associate, Department of Dermatology, Case Western Reserve University and Veterans Administration. Medical Centre, Cleveland, Ohio, USA.
July 1985 - Oct 1986	Senior Research Associate, Department of Dermatology, Case Western Reserve University and Veterans Administration Medical Centre, Cleveland, Ohio, USA.
Nov. 1986 - May 1987	Adhoc Sc C, Industrial Toxicology Research Centre, Lucknow, India.
May 1987 - May 1991	Scientist C, Industrial Toxicology Research Centre, Lucknow, India
May 1991 - May 1996	Asst. Director (Sc E1), Industrial Toxicology Research Centre, Lucknow, India.
May 1996 - May 2001	Sr. Asst. Director (Sc E11), Industrial Toxicology Research Centre, Lucknow, India.
May 2001 – May 2007	Deputy Director (Sc F), Industrial Toxicology Research Centre, Lucknow, India
May 2007- till date	Chief Scientist, Indian Institute of Toxicology Research, Lucknow, India

Honors/Awards:

1980 - 82	Council of Scientific & Industrial Research (India) merit fellowship award.
1985	Recipient of Burroughs Wellcome Fund Fellowship Award in honor of Dr Marion Sulzberger, USA.
1985	Plenary Presentation at joint meeting of Society of Investigative Dermatology, USA and Japanese Society for Investigative Dermatology "Chronic oral feeding of ellagic acid inhibits 3-methylcholanthrene induced skin tumorigenesis".
1986	Recipient of Schering-Plough Foundation Research Fellowship Award from Dermatology Foundation, USA.
1986	Plenary Presentation at annual meeting of Society of Investigative Dermatology, USA "Comparative tumorigenicity of crude coal tar with other polycyclic hydrocarbons in skin of SENCAR mice".
1988	Recipient of the International Union of Biochemistry Fellowship Award to attend XIV International Congress of Biochemistry, Prague, Czechoslovakia, July 10-15, 1988.

- 1990 Recipient of ITRC Young Scientist Award on Silver Jubilee Foundation Day.
- 1991 Recipient of ITRC Technology Award
- 2002 Awarded Fellow of Society of Toxicology, India at the XXI Annual conference of Society of Toxicology, at Kolkata December 3-5, 2002.
- 2004 WHO Temporary Adviser for 63rd Meeting of Joint FAO/WHO Expert Committee on Food Additives at Geneva, Switzerland, June 7-18, 2004
- 2007 Awarded Fellow of National Academy of Agricultural Sciences (FNAASc), New Delhi
- 2007-till date Chairman, Food colours and Flavours Sub-committee of Bureau of Indian Standards. FADC (8:1).
- 2007-2014 President, Indian Nanoscience Society (INS)
- 2008 Paper cited on Cover Page of **Current Science** 94 (2008) 570-572
- 2009 European Union Panel of Experts for review of projects on Functional Foods, Brussels, Belgium
- 2009 Paper cited on Cover Page of **Toxicol Appl Pharm** 234 (2009) 192-201.
- 2009 Co-editor of a Special Issue of an international journal "Nanotoxicology" Vol 3, No 1, 2009
- 2010 Awarded Fellow of National Academy of Sciences (FNASc), India
- 2011 Paper cited on Cover Page of **Clinical & Exp Allergy** 41 (2011) 1157-1168.
- 2011 Paper cited on Cover Page of **GM Crops** 2 (2011) 92-98.
- 2013 13th Alumni Oration of Alumni Association of Department of Biochemistry (AADB), University of Lucknow, Lucknow.

Research experience:

Biochemistry and biochemical toxicology, Chemical carcinogenesis and DNA adduct formation, Chemoprevention of carcinogenesis, Xenobiotic biotransformation with reference to toxication and detoxification mechanisms, Neurotoxicity and neurochemistry, Dermatotoxicology and dermatopharmacology, Prostaglandin synthetase dependent xenobiotic metabolism, Phototoxicology, Toxicity of food adulterants and contaminants, Nanotoxicology.

Supervision as Guide:

Supervised 24 research students for PhD dissertation from Lucknow University; Awadh University; Benaras Hindu University; Gorakhpur University and Jamia Hamdard University

Supervised 12 students for MSc dissertation from various universities

Teaching experience:

Biochemistry courses to undergraduates, Wayne State University, Detroit, September 27, 1982- July 15, 1983.

Food Toxicology; Cancer Biology; and Nanotoxicology courses to AcSIR PhD fellows, 2011- till date.

Editorial:

Editorial Board Member:

Journal of Liver and Pancreatic Diseases (2015- till date)
Austin Journal of Environmental Toxicology (2015- till date)
The Open Biochemistry Journal (2014 - till date)
World Journal of Methodology (2013 - till date)
Prudence J Biochem Biotech (2013 - till date)
International J Clinical Pharmacol Toxicol (2012 - till date)
Journal of Toxicology (2012 - till date)
Biotechnology & Mol Biol Reviews (2012 - till date)
Journal of Environment Protection (2010 - till date)
Journal of Biophysical Chemistry (2010 - till date)
The Open Enzyme Inhibition Journal (2009 - 2014)
Toxicology Mechanisms and Methods (2008 - till date)
Journal of Medicinal Plant Research (2007 - 2008)
Toxicology International (2006 - 2010)
International Journal of Toxicology, Occupational and Environmental Health (1989 - 1995)

Member Refree's Pannel:

J. Investigative Dermatology (USA); Environmental Health Prespective (USA); International J. Radiation Research (USA); Biochim Biophy Acta (USA); Toxicology & Applied Pharmacology (USA); Toxicology Letters (UK); Food Chem Toxicol (UK); Arch fur Toxicol (Germany); Env Sci Tech (USA); BMC Cancer (UK); Food Res Intl (UK); J Agri Fd Chem (UK); J Chromat A (Germany); J Hazardous Mat (UK); Toxicology Mechanisms & Methods (USA); J. Applied Toxicology (USA); Toxicology Chemistry & Ecotoxicology (Germany); Inflammation (USA); J Hazardous Mat (USA); J. Food Biochemistry (USA); Intl J Environ Res & Public Health (USA); J AOAC Intl (USA); J Food Quality (USA); J. Food Quality; J. Food Sciences; Cell Biochem Function; International Dairy Journal (Netherland); African J. Food Sciences (South Africa); African J Env Sci & Technicol (South Africa); J. Medicinal Plant Research (USA); Indian J. Biochemistry Biophysics; Indian J. Experimental Biology; Indian J. Medical Research; The National Medical Journal of India; Toxicology International; Current Sciences;

Examiner:

MSc (Biochemsirty): Lucknow University, Lucknow
Ch.Charan Singh University, Meerut
M. Phil (Biochem): APS University, Rewa
PhD Thesis: Jamia Hamdard University, New Delhi
Osmania University, Hyderabad
Guahati University, Assam

Member of Societies:

Americian Association for Cancer Research,
Society for Investigative Dermatology, USA,
International Society for the Study of Xenobiotics,
Society of Biological Chemists, India (Life Member),
Society of Toxicology, India (Life Member),
Indian Pharmacological Society,
Indian Association for Cancer Research (Life Member),
Indian Photobiology Society,
National Academy of Sciences (Life Member)
Indian Academy of Neurosciences (Life Member).
Association of Food Scientists and Technologists, India (Life Member)
Environmental Mutagen Society of India (Life Member, **Executive Member** 2004-08)
U.P. Association for the Science & Technology Advancement (Life Member)
Alumni Association of Department of Biochemistry, Lucknow University
(Life Member, **Treasurer** 2001- till date)
President, Indian Nanoscience Society (2007- 2014)

Member of Regulatory Bodies :

1. Preservatives, Antioxidants, Emulsifying and Stabilizing Agents, Sub committee of Bureau of Indian Standards. FADC (8:2), 1992- till date.
2. Food colours and Flavours, Sub-committee of Bureau of Indian Standards. FADC (8:1), 1992 - till date.
3. Food and Agriculture Division Council (FADC), Committee of Bureau of Indian Standards, 1993- till date.
4. Central Committee for Food Standards (CCFS) on GM Foods, 2001- till date.
5. Working Group to work with European Committee for quantification of GM foods under ISO, Bureau of Indian Standards, 2000- till date.
6. Food Additives Sub-Committee of CCFS, DGHS, New Delhi, 2001- till date.
7. Food Laws and Legal Advisory Sub-Committee of CCFS, DGHS, New Delhi, 2001- till date.
8. Safety Issues & Testing of GM foods Sub-Committee, DGHS, New Delhi, 2001- till date.
9. Shadow Committee on Methods of Food Analysis & Sampling, DGHS, New Delhi, 2006 - till date.
10. Working group on Food Safety, Bureau of Indian Standards, New Delhi, 2006 - till date.
11. Technical committee of ECOMARK, Central Pollution Control Board, New Delhi, 2006- till date.

Nominated representative of Government Organizations :

1. DBT representative on Institutional Biosafety Committee (IBSC) for Indian Institute of Sugarcane Research, Lucknow, 2002-2005.
2. Member, DBT Task Force committee on "Biotechnological approaches for food and nutritional security" July, 2002-June 2004 and April 2006-March 2009

Patents:

- 1 A process for the preparation of a colour detection test strip useful for the detection of Butter yellow, a toxic adulterant in edible oils.
S.K. Khanna, Mukul Das and P.K. Ray
Complete application No: 309/DEL/92 dated 08.04.1992.
Indian Granting Date: January 04, 2002
Indian Patent No: 185972
- 2 A process for extraction of argemone alkaloids useful for the detection of adulteration in edible oils
Mukul Das and S.K. Khanna
Complete application No: 3509/DEL/97 dated 08.12.1997
Indian Granting Date: January 01, 2004
Indian Patent No: 189711
3. A novel device with circular electrode for electrophoresis of nucleotides (DNA & RNA) and proteins.
A. Dhawan, H.O. Misra, A.K. Pandey, M. Bajpai, D. Parmar and Mukul Das:
Patent application No 0847/DEL/2008, April 08, 2009.
4. A process for making a circular electrode and product thereof for conducting electrophoresis of nucleotides (DNA, RNA) and proteins.
A.Dhawan, H. O. Misra, A. K. Pandey, M. Bajpai, D. Parmar and Mukul Das:

Singapore Granting Date: April 03, 2013.
Singapore Patent No: 165130

5. An electrophoresis device for separation of charged macromolecules using petri dish.
A.Dhawan, H. O. Misra, A. K. Pandey, M. Bajpai, D. Parmar and Mukul Das:
U.S. Granting Date: January 14, 2014
U.S. Patent No: 8628651

Technologies developed:

1. CD Strip: To detect the presence of Butter yellow, a toxic dye in edible oils
2. FDD : Fluorescent detection device for detecting fluorescent food adulterants/contaminants
3. AO Kit : To detect argemone oil adulteration in mustard oil

Grant-in-aid projects carried out:

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| 1983-84 | Co-investigator of the American Skin Cancer Foundation project Goeckerman Therapy of Psoriasis: Mechanisms of skin cancer induction. |
| 1986-1988 | Co-investigator of the American Institute for Cancer Research grant Prevention of skin and lung cancer by ellagic acid and tannic acid: Mechanistic studies. |
| 1992-94 | Principal Investigator of Department of Science & Technology, New Delhi grant Evaluation of Carcinogenic potential of azo dyes, CI Acid yellow 36, CI Acid Orange 7 and their blend. |
| 1992-94 | Co-investigator of Indian Council of Medical Research, New Delhi project "All India coordinated project on evaluation of magnitude and usage pattern of artificial synthetic colours in foodstuffs" |
| 1995-95 | Convener of Indian Council of Medical Research, New Delhi "Training programme on Food Contaminants/Adulterants: Food colours and metals" |
| 1994-96 | Co-investigator of Indian Council of Medical Research, New Delhi project" Assessment risk due to intake of artificial colours in children population". |
| 1994-97 | Co-investigator of the Department of Agriculture and Corporation, New Delhi grant A study on the possible health implications on consumption of Lathyrus sativus. |
| 1994-96 | Co-investigator of Indian Council of Medical Research, New Delhi grant "Assessment of exposure risk to polyaromatic hydrocarbons through raw, refined and repeatedly heated edible oils". |
| 1995-98 | Co-Principal Investigator of Technology Mission, New Delhi on Oil seeds & Pulses research grant "Development of quick and sensitive on the-spot test/kit to detect the presence of toxic adulterants/contaminants on edible oils". |
| 1997-98 | Co-investigator of WHO-DGHS, New Delhi project "Survey of presence of heavy metals and aflatoxin in food products: Multicentric study". |
| 1998-2001 | Co-investigator of Ministry of Food Processing Industries, New Delhi project "Multicentric study on intake of food colours". |
| 1999-2002 | Principal Investigator of Technology Mission on Oilseeds & Pulses, New Delhi research grant "Toxicity and preventive studies on Epidemic Dropsy caused by consumption of mustard oil contaminated with argemone oil". |

- 1999-2002 Principal Investigator of Technology Mission on Oil seeds & Pulses, New Delhi research grant "Studies on polycyclic aromatic hydrocarbon residues in edible oils".
- 1999-2002 Principal Investigator of Technology Mission on Oilseeds & Pulses, New Delhi research grant "Validation of Argemone Oil Detection Kit".
- 1999-2001 Co-Principal Investigator of Indira Gandhi Institute of Development Research grant "A study of environmental exposure to polycyclic aromatic hydrocarbons in economically under privileged population of urban/rural areas of Uttar Pradesh".
- 2000-2001 Principal Investigator of Tata Memorial Hospital, Mumbai project "Monitoring of edible oil samples from Gall Bladder carcinoma/stone patients".
- 2000-2001 Co-investigator of WHO-DGHS, New Delhi project "Survey of occurrence of metal contaminants, lead, cadmium, arsenic and tin in infant milk substitute".
- 2002-2004 Principal Investigator of Technology Mission on Oilseeds & Pulses, New Delhi research grant "Removal strategies for polycyclic aromatic hydrocarbons in contaminated edible oils".
- 2002-2003 Principal Investigator of WHO-DGHS, New Delhi project "Survey study for the pattern and prevalence of permitted and non-permitted colours in various food products"
- 2002-2003 Principal Investigator of WHO-DGHS, New Delhi project "Survey on pesticide residues in food products".
- 2003-2005 Project Coordinator of Department of Biotechnology, New Delhi project 'Development and validation of protocols for allergenicity evaluation of genetically modified foods'.
- 2007-2010 Project Coordinator of Department of Biotechnology, New Delhi project 'Development of molecular tools for detection of genetically modified foods'.
- 2008-2011 Principal Co-investigator of Department of Science & Technology, New Delhi project 'Fate of nanomaterials in biological systems'.
- 2007-2012 Principal Investigator of CSIR-Supra Institutional Project (CSIR-SIP 08) entitled 'Molecular mechanism of argemone oil toxicity and its mitigation by antioxidants'.
- 2007-2012 Principal Investigator of CSIR-Network Project (CSIR-NWP 17) entitled 'Detection of food contaminants and their effects on health'.
- 2012-2017 Principal Investigator of CSIR-INDEPTH Project (BSC 0111) entitled 'Elucidation of Molecular mechanism of toxicity of *Cassia occidentalis*'.
- 2012-2017 Principal Investigator of CSIR-NANOSHE Project (BSC 0112) entitled 'Safety and efficacy of nano-encapsulated bio-antioxidants'.

Industry Sponsored projects carried out:

- 1993 Co-investigator of Central Cattle Breeding Farm, Orissa project "Identification of toxic constituent(s) in two animal feed oil cake samples.
- 1993 Co-investigator of Thirumalai Chemicals, Madras project "Safety guidelines for approval of DL-Malic acid as permitted food additive under PFA Rules, India.
- 1993 Co-investigator of Sengar Poultry Farm, Etawah project "Identification of toxic constituent(s) in animal feed responsible for toxicity in the poultry birds".
- 1994 Co-Principal Investigator of Zafrani Zarda Manufacturers Association, Delhi project "Survey studies on the consumption pattern of Menthol through Pan Masala.

- 1994 Co-Principal Investigator of Colour Chem Ltd, Bombay project "Studies on acute safety evaluation of Acesulfame-K in rat.
- 1994 Co-Principal Investigator of Colour Chem Ltd, Bombay project " Studies on acute safety evaluation of Acesulfame-K in mice"
- 1995 Co-investigator of Indian Petrochemicals Corporation Ltd, Vadodara project "Evaluation of consumer safety, occupational and ecological risks on application of treated industrial effluents for irrigation of crops (A joint ITRC programme)".
- 1996 Co-Principal Investigator of Colour Chem Ltd, Bombay project "Standardization and applicability of analytical methodology on Acesulfame-K"
- 1997 Co-investigator of East India Pharmaceuticals Works, Calcutta project "Aflatoxin residues profile of herbal culture filtrates".
- 1995 Co Principal Investigator of Colour Chem Ltd, Sub of Hoechst AG, Bombay project "Safety evaluation studies on blend of two artificial sweeteners: Acesulfame-K and Aspartame".
- 1996 Co Principal Investigator of Parry's Confectionery Ltd, Chennai project "Analytical standards/contaminants in CRUMBLE sugar confectionery".
- 1996 Co Principal Investigator of Tata Tea Ltd, Calcutta project "Accelerated storage stability studies on sugar-Aspartame blend vis-a-vis evaluation of shelf-life and health safety of the project under variable conditions of temperature and humidity".
- 1998 Co-investigator of K.K. Dye Chem Pvt Ltd Budivada, A.P project "Assessment of Naphthol AS-G, an azoic coupling component as part of banned azo dyes".
- 1998 Co investigator of Cattle Feed Plant, Pradeshik Cooperative Dairy Fedn. Ltd, Merrut project "Identifation of aflatoxin and argemone alkaloids in mustard oil cake".
- 1998 Co-investigator of Cattle Feed Plant, Pradeshik Cooperative Dairy Fedn. Ltd, Merrut project "Identifation of aflatoxin and argemone alkaloids in mustard oil cake and deoiled mustard cake.
- 1999 Co Principal Investigator of Bush Boake Allen Ltd, Chennai project "Toxicological profile of Sunset yellow FCF, Tartrazine and Carmoisine used in Indian sweets".
- 1999 Co-investigator of Jute Manufactuers Development Council, Calcutta project "Safety evaluation studies on food grade Jute products".
- 2000 Co investigator of Central Salt and Marine Chemicals & Research Institute, Bhavnagar project "Evaluation of the modified Jojoba body cream".
- 2000 Co-investigator of Fragrance and Flavour Development Centre, Kannauj project "Toxicological study of Neem based cold cream".
- 2002 Principal Investigator of Indian Lac Research Institute, Ranchi project "Safety evaluation studies on lac dye for food usage".
- 2012 Principal Investigator of Indian Council of Medical Research, New Delhi, project "Analysis of oxytocin in ampules of oxytocin for animal and human use".
- 2012 Principal Investigator of Indian Institute of Vegetable Research, Varanasi project "Analysis of oxytocin in bottle guard".
- 2013 Principal Investigator of Organic Manure Mills PVT Ltd, Vellore District, Tamilnadu project "Analysis of ochratoxin in bonemeal sample".

SIGNIFICANT R&D ACHEIVEMENTS

1. Studies on Xenobiotic Metabolism

Metabolism of xenobiotics in hepatic and extrahepatic tissues is of importance in expressing their pharmacological and toxicological action. The xenobiotic metabolizing enzyme system requires NADPH and molecular oxygen for catalytic activity, and has its terminal oxidase; the microsomal membrane bound heme protein, cytochrome P-450.

His studies indicated that benzo(a)pyrene (BP), a ubiquitous polycyclic aromatic hydrocarbon (PAH) pollutant, enters brain after parenteral administration (29). Cytochrome P-450 (P-450) dependent aryl hydrocarbon hydroxylase (AHH) activity biotransform PAHs including BP to various metabolites. The properties of P-450 dependent AHH activity in brain microsomes and mitochondria were characterized (1,2,7,8). **Brain was in fact, the first tissue to show higher mitochondrial AHH activity as compared to microsomal activity** (2,8,70). It was hypothesized that P-450 in brain mitochondria may be responsible for steroidogenesis as other investigators had shown the presence of adrenodoxin like ferridoxin protein in brain mitochondria. AHH enzyme was found to be responsible to metabolize PAHs to their highly reactive products, which can then bind to cellular DNA to initiate the process of carcinogenesis (26). The metabolic products of PAHs may also undergo further biotransformation by cytosolic detoxification enzymes such as glutathione-S-transferase (GST) which has been characterized in brain (3,70). Furthermore, this enzyme system in brain was found to be regulated by sex hormones (10) and neurotoxicants including acrylamide and styrene (4,6,12,15,23). Subsequent studies have shown that brain can metabolize ethoxycoumarin and ethoxyresorufin suggesting the multiplicity of P-450 (65,83) and that the neuronal cells contained 2-3 fold higher activity of cytochrome P-450 dependent enzymes as compared to glial cells in brain (71). This enzyme system may have some physiological role in the neurotoxicity of drugs and chemicals selectively acting on the CNS.

Skin was considered to be inert for xenobiotic metabolism. Our studies suggest that skin of rodents are equipped with phase II and cytochrome P-450 monooxygenases, which metabolizes various xenobiotics including polycyclic aromatic hydrocarbons to ultimate toxic metabolites (27,37,45,53), which were later used as machineries for carcinogenic index due to their binding potential to DNA molecules.

Studies suggest that metabolism of alkoxyphenoxazone compounds including pentoxyresorufin and ethoxyresorufin by P-450 IIB1/2 and IA1/2 isozymes can be monitored simultaneously alongwith electron donor, spectrophotometrically (129). The approach was found to be useful for not only estimating the alkoxyresorufin-O-dealkylase activities, but also to study the stoichiometry of enzyme reaction in the same incubation system (124). Further, the mechanism of modulation of stoichiometry of alkoxyphenoxazone metabolism with special reference to cytosolic quinone reductase was suggested (141).

2. Studies on carcinogenesis and protection

It is now widely accepted that the initiation of carcinogenesis by xenobiotics relates to metabolic activation of the parent compound to highly reactive ultimate carcinogenic metabolites, which bind to DNA. This knowledge has led to the search for non-toxic inhibitors of these metabolic processes.

Studies of the applicant suggested that naturally occurring plant phenols, ellagic acid, tannic acid, quercetin, myricetin and anthraflavic acid have the antitumorigenic activity against 7,12-dimethylbenzanthracene, benzo(a)pyrene, 3-methylcholanthrene (3-MCA) and N-methyl-N-nitrosourea induced skin carcinogenesis (16,36,59,63). The possible mechanism of the action of these plant phenols may be due to alteration in metabolic activation and in target tissue macromolecular binding to carcinogenic species (17,18,31,34,46,49,50,51). A series of newly synthesized compounds, polycyclic aza-oxa and aza-oxa-thia heteroarenes, two compounds showed anti carcinogenic potential against liver cancer while three showed anti-carcinogenic potential in intestinal cancer (254).

Aflatoxin B1 (AFB1) has been shown to possess skin tumor initiating activity in mouse model. Moreover, only pre-neoplastic changes in hepatic tissue were observed in mice. The basic mechanism for not producing hepatic tumors in mouse was due to the fact that glutathione-S-transferase activity was relatively higher as compared to rats thereby eliminating the main carcinogenic metabolite, AFB1-8,9-epoxide. This may explain the reason

for rats to be susceptible towards the development of hepatic cancer by AFB1 (154). Futher, *Ocimum sanctum* leaf extract was found to inhibit the process of skin carcinogenesis induced by AFB1, 3MCA and benzo(a)pyrene which was due to antiproliferative activity in cells (164). Visualizing the importance of newer mycotoxins contamination in foodstuffs and the relevance of fungal growth in tropical countries due to humid conditions, work has been initiated on dermal toxicity as suggested by WHO/FAO. Studies on patulin, citrinin, ochratoxin and deoxynivalenol indicate toxicity and carcinogenicity in skin by different signaling mechanisms (178,202,208,220,227,240,259).

3. Studies on oscillation reactions

Earlier investigations had suggested that only specific chemicals/ substrates during chemical or biological reactions show oscillatory phenomenon. My recent work have indicated that not only the specific but all the chemicals and biological molecules involved or participating in chemical/biological reactions exhibited a state of oscillation (96,114,125,149). This challenging area needs to be explored for not only studying the mechanism of toxicity of xenobiotics and/or certain diseased conditions but can lead to suggest the protective response of various compounds against toxic manifestations and diseases. Also, this aspect may find application in clinical diagnosis of the patients.

4. Biochemical toxicity of Argemone oil, an adulterant in mustard oil

Consumption of argemone adulterated mustard oil is known to cause Epidemic Dropsy. The problem of adulteration of mustard oil with argemone still exists as evidenced by Dropsy at Barabanki in 1988, Delhi in 1998, Kannauj in 2002 and Lucknow in 2005. The toxicity of argemone oil has been attributed to benzophenanthridine alkaloids, sanguinarine and dihydrosanguinarine (105). Since, biochemical mechanism of toxicity of argemone oil was not understood; the systematic therapeutic approach towards the disease is not known (111).

The mechanism of argemone oil toxicity was explored and studies suggested that liver, lungs kidney and heart were the target sites for argemone oil intoxication (66). Argemone oil caused a decrease in hepatic glycogen level, which may be due to the activation of glycogenolysis leading to accumulation of pyruvate in blood (66, 105). The increase in pyruvate uncouples oxidative phosphorylation leading to breathlessness in patients (105). Sanguinarine inhibited Na⁺K⁺ATPase activity of the liver and intestine, thereby causing a decrease in active transport of glucose (89). Further, results indicate that Argemone oil and sanguinarine have genotoxic and tumor initiating potential and shall be hazardous if human babies are smeared with mustard oil contaminated with Argemone oil (140,147,151). Argemone oil induced loss of hepatic cytochrome P-450 and impairment of Phase I and Phase II enzymes have been shown to be responsible for the slow metabolism of its toxic alkaloids (73,172). The delayed appearance of metabolite (benzacridine) and the parent compound in urine and feces also confirmed the slow biometabolic elimination of the alkaloid (88). Argemone oil caused enhanced lipid peroxidation with concomitant depletion of free glutathione thereby indicating oxidative stress (60,72,73,75,148,168,169). The involvement of oxidative stress was further confirmed by overexpression of heat shock protein hsp 70 in *Drosophila melanogaster* (123). Studies suggested that singlet oxygen and hydroxyl radicals were involved in argemone oil toxicity which led to oxidative damage of proteins, lipids and DNA (75,140,148)). Subsequent studies have shown that AO causes cancer in skin following topical application and responsible for gall bladder cancer if consumed orally (151,211). Several bioantioxidants were shown to protect against argemone oil induced toxicity in experimental animals (72,75,157,163). The protective efficacy of these bioantioxidants were examined during Barabanki epidemic and showed beneficial effect on patients (87,122) and experimentally induced carcinogenesis (231). Favorable response of this mode of therapy was also noted during the 1998 Epidemic Dropsy in Delhi (111), Kannauj (148) and Lucknow (163).

5. Studies on dye intermediate, Benzanthrone / 3-Bromobenzanthrone:

Workers coming in contact with benzanthrone, a textile dye intermediate, complain of burning sensation, erythema and hyperpigmentation of the skin loss of appetite, intolerance to fatty foods fatigue weakness, weight loss, gastritis and decreased sexual potency.

Studies on benzanthrone have shown that it is metabolized by acting as a new substrate of hepatic P-450 to at least 7 metabolites (64). *In vivo* administration of benzanthrone to rats, showed 5 of these metabolites excreted in urine (64). Benzanthrone acted as a type I substrate for P-450 and inhibited P-450 and its dependent monooxygenases in a concentration dependent manner (74).

A striking observation noted was the depletion of ascorbic acid in serum and adrenals of exposed animals (90,119). Subsequent studies have shown that in the presence of extraneous ascorbic acid, the urinary and fecal clearance rate of benzanthrone/or metabolite(s) was enhanced with concomitant decrease in organ retention in different murine species (77,81,86). Oral supplementation of ascorbic acid to benzanthrone administered guinea pigs resulted in marked improvement of histopathological and biochemical changes observed in liver, testis, kidney and urinary bladder (93). Benzanthrone was shown to possess a weak tumour initiating activity, which was completely abolished by ascorbic acid treatment (236). Recently, he has shown that 3-bromobenzanthrone is more toxic than its parent compound (113,130,134). The depletion of ascorbic acid and skin irritating potential (113,130) is much more in 3-bromobenzanthrone when compared to benzanthrone and may be due to generation of Br• radical, which have far reaching consequences (134).

A regular daily intake of ascorbic acid by workers in benzanthrone/ 3-bromobenzanthrone manufacturing units can thus be prescribed as protective measure.

6. Mechanism of toxicity of polycyclic aromatic hydrocarbon mixtures derived from repeatedly fried edible oils:

Polycyclic aromatic hydrocarbons (PAHs) are ubiquitous environmental pollutants. They are formed through a number of combustion processes and are present as a complex mixture of many individual PAHs. Studies of various environmentally relevant matrices, such as coal combustion effluent, motor vehicle exhaust, used motor lubricating oils and tobacco smoke have shown that PAHs in these mixtures are mainly responsible for their carcinogenic potential. Vegetable oils are reported to be naturally free of PAHs, and contamination is due to technological processes like smoke drying of oil seeds or environmental sources such as deposition from exhaust gases of traffic. In a recently conducted study in India, 296 oil samples comprising of coconut, groundnut, hydrogenated vegetable, linseed, mustard, olive, palm, refined vegetable, rice bran, safflower, sesame, soybean, and sunflower showed that 88.5% samples were contaminated with different PAH (146). Of 262 contaminated edible oil samples, 66.4% of the samples showed PAH content of more than the 25 µg/kg recommended by the German Society for Fat Science. The total PAH content was highest in virgin olive oil (624 µg/kg) and lowest in refined vegetable oils (40.2 µg/kg). The maximum content (265 µg/kg) of heavy PAH was found in olive oil and the minimum (4.6 µg/kg) in rice bran oil. Phenanthrene was present in 58.3% of the oil samples analyzed, followed by anthracene (53%). Among the heavy PAH, benzo(e)pyrene was observed in 31.2% of the samples followed by benzo(a)pyrene (25.5%). The intake of PAH was highest through olive oil (20.8 µg/day) followed by soybean oil (5.0 µg/day) and lowest through refined vegetable oil (1.3µg/day). Based on these monitoring studies, international and national guidelines for permissible levels of PAH can be prepared so as to restrict the intake of these toxic contaminants. Recent studies have shown that repeatedly fish fried oil generates considerable amount of PAHs which are cytotoxic due to enhanced formation of a precursor (BP-7, 8-diol) of ultimate carcinogenic species, diol epoxide, thereby causing increased DNA binding, apoptosis, cell cycle arrest and induction of p⁵³ and p^{21waf1} which are responsible for initiation of carcinogenesis in experimental animals (153,155,159,160).

7. Detection of Oxytocin(OT) in milk: Toxicological implications of OT in young immature rats

Oxytocin (OT), a biological hormone, has physiological role for performing specific functions at different stages of life. Under normal physiological condition in lactating mothers, OT causes contraction of the myoepithelial cells which surrounds the milk alveoli in the mammary gland for milk let-down. In Indian subcontinent, OT ampules, commercially known as pitocin or syntocinon, are indiscriminately used for enhanced milk let-down following intramuscular injections to cattle. It has been presumed that due to small size (OT,1Kd) there

is a possibility that it crosses blood-milk barrier and reaches into the milk thereby causing toxicological consequences during non physiological exposure especially in children.

We have therefore developed a new extraction method for the determination of oxytocin in milk by enzyme immune assay or HPLC. The extraction of OT in milk involves two steps: (i) TCA precipitation of milk proteins, and (ii) Solid phase extraction cleanup process. OT was found to be stable against adverse temperature (up to 100°C), pH (2 to 10) and simulated gastric fluid digestibility assay suggesting that OT is not degraded either by boiling or by digestive enzymes present in gastrointestinal tract thereby indicating the possible risk in humans especially children (228). Further, in a total of 55 milk samples (39 milkman & 16 branded) OT contamination in milkman samples was found to be 21 pg/mL to 18.9 ng/mL with the mean value of 8.9 ng/mL. The average daily intake of OT in terms of µg/day/person was highest (2.3-2.4 µg/day/person) in 1-3 year age group, which may have deleterious effects (256).

Subsequent studies were carried out to investigate the anomalies in different organs following daily oral administration of OT (1 and 10 ng/100 µl) to young immature rats (10 day old) for 25 days. OT exposure resulted in increase in ovarian weight, γ globulin, total number of follicles, number of corpus luteum; indicating higher ovulation at early period when compared to control (untreated rat pups). The mechanism for enhanced ovulation involves over-expression of pEGFR followed by downstream pERK1/2 and subsequently increased ovarian PGE-2 along with enhanced COX-2, HAS-2 & TSG-6 (matrix deposition proteins) and GDF-9 (oocyte factor) proteins, suggesting that oral exposure to OT may affect the physiology and function of the ovary (256).

8. Association between children death and consumption of Cassia occidentalis seeds: Clinical and experimental investigations

During the past decade an illness in young children has been observed in several adjoining areas of western UP and Uttaranchal (India), with the involvement of muscle, liver and brain resulting in almost 70% mortality. The outbreak was earlier diagnosed as acute encephalitis of unknown viral etiology. Our recent studies showed that the disease, hepatomyoencephalopathy (HME), is related to the consumption of Cassia occidentalis (CO) seeds. This was proved in an investigation where CO seeds (0.5, 1 and 2% w/w) were given to wistar rats in diet. After 28 days it was observed that CO seeds caused significant increases in the serum markers along with histopathological lesions in hepatic tissue. CO consumption also showed decrease in grip strength, vacuolization and myopathy of skeletal muscles along with increases in serum creatinine and creatinine phosphokinase suggesting muscular damage in animals. Neuronal damage in CO treated animals was evident by a marked increase in glial fibrillar acidic protein and decrease in β-tubulin. The experimental findings of CO consumption showed liver, muscles and brain to be the target organs, which were similar to that of the clinical data of poisoning cases, and are one of the etiological factors in children population suffering from HME in India (248). Microarray studies indicated that exposure of CO (0.5%) seeds in diet to rats differentially regulated 60 transcripts belonging to various metabolic pathways including, oxidative stress, xenobiotic metabolism, carbohydrate metabolism, cell cycle, apoptosis etc. The expression of AKT1, CAT, SOD1, CYP1A1, CYP2B1, TGF-β, BAX, CREB1, JNK1 and IL-6 were validated by the qRT-PCR (260).

The toxic compounds in the CO seeds were characterized to be physcion, emodin and rhein and further quantified in the body fluid of HME patients and experimental animals exposed to CO seeds. GC-MS analysis of chloroform fraction of methanol extract of CO seeds revealed the presence of three anthraquinones viz. physcion, emodin and rhein having m/z 413, 471 and 485. In addition, other anthraquinones like aloe-emodin (m/z: 471) and chrysophanol (m/z: 383) were detected in the hexane and butanol fractions of methanol extract of CO seeds. Interestingly, these five anthraquinones were detected in the serum and urine samples of HME patients and experimental rats treated with CO seeds. Further, rhein was found to be the most toxic compound with an IC₅₀ of 50-103 µM in rat primary hepatocytes and HepG2 cells, followed by emodin, aloe-emodin, physcion and chrysophanol. These results suggest that anthraquinone aglycones are the etiological agents responsible for CO induced HME; and that the presence of these compounds in the urine and blood samples of patients and rats exposed to CO seeds may serve as diagnostic marker of the disease (Chem Res Toxicol, 2015, in press).

The molecular mechanism of toxicity of rhein involves the generation of reactive oxygen species (ROS) and intracellular Ca⁺⁺ as an early event while modulation of mitochondrial membrane potential and intracellular glutathione (GSH) as a late event. At molecular level rhein caused DNA damage resulting in over expression of γ -H2AX protein thereby causing enhancement of p53 and p21 leading to intrinsic pathway mediated apoptosis involving Bax, cytochrome c, caspases 3, 9 and Poly ADP ribose polymerase (PARP). Further, it was observed that rhein induced ROS generation is also involved in modulation of signaling molecules like MAPK including ERK1/2, p38 and JNK, mitochondrial energetics proteins including complex II-V, pAMPK, Sirt-1 and induction of cytochrome p450 isozymes. Among the various protective agents, cyclosporine A (CSA) at 100 nM concentrations was found to be most effective in preventing apoptosis in hepatocytes by interfering in various metabolic pathways (Chem Res Toxicol, 2015, in press).

9. Nanotoxicology:

A new science of nanotechnology has emerged, the products of which is being used in all the spheres of life. Due to the small size of nanoparticles the reactivities are entirely different as compared to their bulk material. We have initiated work related to nano material toxicity and have shown that zinc oxide in nano form used in cosmetics is toxic to skin cells by producing reactive oxygen species (182). Subsequent studies on zinc oxide nanoparticles showed immunotoxic responses (241,244,253,255). Work on nanomaterials used in food industries and their efficacy and toxicity have been initiated (180,181) and guidelines for safe handling of nanomaterials have been prepared (196).

10. Food quality monitoring, surveillance and health risk assessment:

Regular food quality assessment surveys sponsored by ICMR, MFPI, DGHS-WHO, TMOP&M and on our own initiatives have been undertaken so as to authenticate quality standards and identify toxic adulterants in various foodstuffs as part of our social commitment towards consumer safety, awareness and educational programme. The nature and proportion of adulteration is at times high enough to cause serious health problems to the unsuspecting consumer and feedback helps regulatory agencies to formulate standards, streamline vigilance and avoid mishaps (101,120,137,150,152,161,162,165-167,176,186,188,189,199, 212). So far all the states of India excluding Jammu & Kashmir and Arunachal Pradesh have been covered for monitoring of food samples. Based on the work the following legislative decisions were undertaken: (i) ban on sale of loose food colours, (ii) compulsory ISI certification of food colours, (iii) delisting of three colours from the prescribed list, (iv) curtailment of maximum allowable limits of colours in food, (v) prescribed the limits of nickel in hydrogenated vegetable oils, (vi) prescribed the limits of menthol in pan masala and (vii) initiation of formulation of standards for heavy metals in food grade silver foil.

Further, quick detection kits have been developed for monitoring of a toxic dye, Butter Yellow and Argemone oil in edible oils and patented. These kits can be used by consumers themselves and have been utilized during 1998 Delhi Dropsy for expeditious clearance of withheld mustard oil tankers on the directives of Delhi Government. Apart from these several laboratory analytical procedures for quantitation of additives, contaminants and adulterants have been developed (104,121,124,139,177,192,206).

The occurrence of Aflatoxin M1 (AFM1) contamination in Indian infant milk products and liquid milk samples was carried out in a total of 87 samples in categories of infant milk food, infant formula, milk based cereal weaning food and liquid milk samples. The results showed that the incidence of contamination of AFM1 was of the magnitude of 87.3%. The range of contamination of AFM1 was comparatively higher in infant milk products (65-1012 ng/L) than liquid milk (28-164 ng/L). Almost 99% of the contaminated samples exceeded the European Communities/Codex Alimentarius recommended limits (50 ng/L), while 9% samples exceeded the prescribed limit of US regulations (500 ng/L). The extrapolation of AFM1 data to estimate the Aflatoxin B1 (AFB1) contamination in dairy cattle feedstuffs indicated that the contamination may range from 1.4-63.3 μ g/Kg with a mean of 18 μ g/Kg which is substantially higher than the directive of European Communities regulation (5 μ g/Kg). The results suggest a need to introduce safety limits for AFM1 levels (480 ng/Kg) in infant milk products and liquid milk under Prevention of Food Adulteration Act of India as well as to prescribe the levels of

AFB1 in dairy cattle feedstuffs so as to minimize the health hazard risk in infant population at large (137).

During the outbreak of tricresyl phosphate (TCP) poisoning (1988) in which almost 600 victims who consumed adulterated rapeseed oil in the Behala area of the south-west outskirts of Kolkata were clinically examined (68). The follow-up investigations showed that patients consuming alcohol had slower recovery. The mechanism of slower recovery was linked to the fact that alcohol caused induction of a specific P-450 isozyme IIE1 which may be responsible for the production of cyclic metabolite of TCP which is 5 times more toxic than the parent compound (82).

Based on experimental work on bio-antioxidant protection against Argemone oil induced changes, bioantioxidant therapy was first time attempted in 1988 Epidemic Dropsy patients at Barabanki (87). This mode of therapy was again used in 1998 Dropsy at Delhi (111,122) and recently in July 2002 outbreak of dropsy at Kannauj and in 2005 at Lucknow. During August 1998 loose mustard oil samples collected from outskirts of Lucknow showed almost 25% samples contaminated with argemone oil. This information led to banning of sale of mustard oil in Uttar Pradesh and subsequently several others states and averted the occurrence of Dropsy in the region.

List of Publications

Xenobiotic Metabolism (1,2,3,7,8,10,26,28,29,40,43,44,45,48,53,54,65,71,83,124,141, 153,155)	23
Neurotoxicants (4,5,6,9,12,13,14,15,22,23,38,52,68,82)	14
Carcinogenesis & Protection (16,17,18,20,21,31,34,36,37,41,46,47,49,50,51,55,58,59,62,63,159,160,198,218,254,271)	26
Photobiology (25,27,32,42)	04
Prostaglandin/Leukotrienes (19,61)	02
Argemone oil/alkaloid (60,66,72,73,75,87,88,89,111,123,140,147,148,151,156,157,163,168,169,172,182, 187,190,191,193,211,213,231,252)	29
Mycotoxins (137,154,164,176,178,202,208,212,220,225,227,240,242,259)	14
Dyes and Dyes Intermediates (64,67,74,77,79,81,86,90,92,93,94,95,97,98,103,106,107,108,109,112,113, 119,130,134,143,145, 236, 270)	28
Oscillation Reactions (96,114,125,149)	04
Oxytocin (234, 256)	02
Methods Development (104,121,128,129,139,177,192,206,221,228,268)	11
Metals (30,84,150,152)	04
Nanotoxicology (183,196,241,244,245,253,255,261,263)	09
Pesticides (110, 127)	02
Plant Toxins (Lathyrus, Cassia occidentalis & Litchi) (131,132,136,184,248,257,260,272,273)	09
Allergenicity & Immunotoxicity (171,185,194,197, 200, 205, 210,215,216,229,233,237,243,246,247,249,250,264,265,266)	20
Surveys (101,120,146,158,161,162,165,166,167,170,186,188,189, 199,217,230)	16
Chapters in Books (11,24,35,56,57,70,76,115,116,117,122,173,258)	13
Reviews (39,69,78,80,85,91,99,100,105,126,135,142,179, 180,181,204, 207,214,219,222,224,226, 232,235,238,251,262,267,269)	29
Popular science articles (33,102,118,133,138,144,174,175,195,203,209,223,239)	13
Books (201)	01

Total: 273

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Average IF of 273 papers	2.70
Average IF of 225 refreed papers	3.27
Average IF of 127 papers with IF > 3.0	4.42
Average IF of 84 papers with IF > 3.4	5.10
Average IF of 60 papers with IF > 4.0	5.61