PANKAJ SETH

Date of Birth March 10, 1968

Address (Office)
Molecular and Cellular Neuroscience
National Brain Research Center
N.H. - 8, Manesar, Haryana - 122 058
INDIA
Tel.: +91-124-2845212 (Direct); +91-124-2845234 (Lab)
Fax: +91-124-2338928 and +91-124-2338910
Email: pseth.nbrc@gov.in

Education

1996 **Doctorate of Philosophy**, University of Kanpur, India,
Medical Biochemistry

1991 **Masters of Science**, University of Lucknow, India
Major: Biochemistry

1989 **Bachelors of Science**, University of Lucknow, India.
Major: Chemistry, Botany & Zoology

Other Professional Training Programs/Courses

Diploma Course on **Science Policy and Management Competencies**, sponsored by Department of Science and Technology, New Delhi, on February 11-22, 2013 conducted by Management Development Institute (MDI), Gurgaon, India.

Recombinant DNA Methodology, in 1999 Foundation for the Advanced Education in the Sciences, at National Institutes of Health (NIH) Bethesda, USA.

Professional Experience

July 2017 – Till date
Scientist In-Charge (Additional Charge), Translational Research Unit of National Brain Research Centre at Gurgaon General Hospital, Gurgaon, India.

December 2018 – Till date
SCIENTIST VII and Professor, National Brain Research Centre, Manesar, India.

May 2013 – December 2018
SCIENTIST VI and Professor, National Brain Research Centre, Manesar, India.

October 2008 – April 2013
SCIENTIST V and Additional Professor, National Brain Research Centre, Manesar, India.

Sept 2003 – Sept 2008
SCIENTIST IV and Associate Professor, National Brain Research Center, Manesar, India.

Feb 2002 – Sept 2003
STAFF FELLOW, Laboratory of Molecular Medicine and Neuroscience, National Institute of Neurological Disorders & Stroke (NINDS), National Institutes of Health (NIH), Bethesda, USA

May 1999 – January 2002
RESEARCH INSTRUCTOR, Department of Pathology, Uniformed Services University of Health Sciences, Bethesda, USA.

June 1998 - April 1999

RESEARCH ASSOCIATE, Department of Pathology, Uniformed Services University of Health Sciences, Bethesda, USA.

July 1996 - May 1998

POST DOCTORAL FELLOW, Department of Pathology, Uniformed Services University of Health Sciences, Bethesda, USA.

February 1995 - June 1996

SENIOR RESEARCH FELLOW, Council of Scientific & Industrial Research New Delhi, at Central Drug Research Institute, Lucknow, India.

July 1994 - January 1995

SENIOR RESEARCH FELLOW, Department of Ocean Development, New Delhi, at Central Drug Research Institute, Lucknow, India.

July 1992 - June 1994

JUNIOR RESEARCH FELLOW, Department of Ocean Development, New Delhi, Central Drug Research Institute, Lucknow, India.

November 1991 - June 1992

RESEARCH ASSISTANT, Department of Ocean Development, New Delhi, at Central Drug Research Institute, Lucknow, India.

Academic Achievements, Honors and Awards, Scientific Recognition

1. **Meeting Director and Chair Local Organizing Secretary,** 26th Annual meeting Society of Neuroimmune Pharmacology, April 1-4, 2021, New Delhi, India.

2. **Keynote Speaker,** *How viruses affect human brain – finding answers*, at Recent Advances in Life Sciences (RALS 2020), DPG Degree College, Gurgaon, India February 22, 2020.

3. **Invited Speaker - TEDx talk,** *Decoding how viruses affect human brains* at Heritage Xperimental School, Gurgaon. September 28, 2019.

4. **Co-Chairperson** for session on role of the Gut-Brain axis in controlling CNS viral reservoirs at the International Society of Neurovirology and Society of NeuroImmuno Pharmacology joint meeting at Chicago, USA, April 10-14, 2018.

5. **Plenary Speaker,** *Human Neural Stem Cells as Models to Understand NeuroAIDS***,** at World NeuroCongress-2017, Aligarh Muslim University, Aligarh, India, Dec 9-10, 2017.

6. **Keynote Speaker,** ***Second International Conference of Public Mental Health and Neurosciences****, Bangalore, India, December 9-10, 2015.*

Scientific Bodies / Boards –

1. **Elected as Council Member** ***for Asia Pacific Society for Neurochemistry (APSN)****, an international society of countries in Asia Pacific,* 2020-2024.

2. **Elected as Fellow** of the Indian Academy of Neurosciences (IAN), the third largest academy of neuroscientists in South East Asia (2019).

3. **Elected Fellow** of the National Academy of Sciences India (NASI) - 2017.

4. **Elected Council Member (International),** for Society for NeuroImmune Pharmacology (SNIP), USA 2017- 2020, re-elected in 2020 for two more years.

5. **Elected as Member** of the Guha Research Conference (GRC) held on 5th December, 2017 in Lakesong Resort, Kumarakom, Kerala (2017).

6. **Elected Council Member, *Federation of Asia Oceanic Neuroscience Society (FAONS)*** *2012 - 2016.*

7. **Elected Board Member,** International Society of NeuroVirology (ISNV) USA, 2017.

8. **Research Highlight,** in Newsletter of International Society of Neurovirology (ISNV), in January 2010.

9. **Member**, National Advisory Committee, Centre of Neurosciences, Jiwaji Univ., Gwalior 2010.

10. **General Secretary,** Indian Academy of Neurosciences, India, 2011 -Till date.

11. **Executive Member**, Indian Academy of Neurosciences, India, 2004 – 2005 & 2008-2011.

12. **Joint Secretary**, Indian Academy of Neurosciences, India, 2005 - 2008.

13. **Session Speaker,** at annual meeting of Society of Neuroimmunopharmacology (USA) at Wuhan, China, April 2009.

14. **Convener and Chairperson,** Annual Meeting of Association of Clinical Biochemists of India. December 2007.

Awards -

1. **VASVIK Industrial Research Award** for contributions in Biological Sciences and Technology – 2018

2. **NINDS Group Merit Award** by Director NINDS, National Institutes of Health, USA, in Recognition of superior service and achievements while working in a group, at NINDS (2004).

3. **Special Act & Service Award** by United States Department of Health and Human Services (US DHHS), National Institutes of Health, USA, in Recognition and Appreciation of Sustained High Quality Work Performance for Services as a Staff Fellow during 2002-2003, Jan 2004.

4. **Sustained Superior Performance Award** by Henry M. Jackson Foundation for Advancement of Military Medicine, USA, for superior performance as a Post Doctoral Fellow, May 1998.

5. **Uvnas Prize** for the best paper published by member of the society in any journal on autocoids
   & biogenic amines, by the Indian Pharmacological Society, India, November 1995.

6. **Achari Prize** for the best oral presentation and quality of work XXVIII Annual meeting of Indian Pharmacological Society, Patiala, India, November 1995.

7. **Junior Investigator Award** for the International Symposia Organized by South Asian Society of Atherosclerosis and Thrombosis, December 17-18, 1994 at Bombay, India.

8. **Congress Award** at XII-International Congress of Pharmacol., Montreal, Canada, July 1994.

9. Qualified the **Council of Scientific & Industrial Research / University Grants Commission (National Eligibility Test) examination** Dec 1991 for the award of Junior Research Fellowship under UGC.

10. **Prof. P.S. Krishnan Gold Medal** in 1991.

11. Honored with **Smt. & Shri Rai Sahib Sheo Shanker Memorial Scholarship** in 1990-91, during M.Sc Biochemistry.

12. FIRST in the order of **merit** in both M.Sc. Part I and Part II examinations of Department of Biochemistry in 1990 & 1991.

Task Force / Scientific Committees

1. **Member,** ***Technical Expert Committee (TEC), Chronic Disease Biology,*** *Department of Biotechnology, New Delhi, India 2018 -Till date.*

2. **Special Invitee,** ***Scientific and Technical Appraisal and Advisory Groups (STAG), Medical Biotechnology,*** *Department of Biotechnology, New Delhi, India 2019.*

3. **Special Invitee,** ***Task Force of DBT-Boost to University Interdisciplinary Life Science Departments for Education and Research (DBT-BUILDER)*** *Programme 2019-20.*

4. **Member**, ***Task Force on Stem Cell Research and Regenerative Medicine****, constituted by Department of Biotechnology, India 2017-2018.*

5. **Member**, ***Special Expert Group on Neurosciences - Chronic Diseases Biology*** *constituted by Department of Biotechnology, India 2010 – 2014.*

6. **Member*****, Project Review Committee,*** *Neurological Sciences, Indian Council of Medical Research (ICMR), New Delhi, India 2019 – till date.*

7. **Member,** ***Expert group on Geriatrics and Biogerontology****, Indian Council of Medical Research, New Delhi, India, 2011.*

8. **Member,** ***Screening Committee for International Khorana Summer Scholarships, United States-India Education Foundation*** *2017.*

9. **Member,** ***Screening Committee for Neuroscience, 2014 and 2015 International Fulbright Science and Technology Award, United States-India Education Foundation.***

10. **Member,** ***Screening Committee for Science, 2010 International Fulbright Science and Technology Award****, United States-India Education Foundation.*

Institutional Ethics Committees

1. **Co-Chairperson,** *Institutional Committee on Stem Cell Research and Therapy (IC-SCRT), Sanjay Gandhi Post Graduate Institute of Medical Sciences (SGPGIMS), Lucknow, India 2019 - Till date.*

2. **Member,** *Institutional Committee on Stem Cell Research (IC-SCR), Institute of Liver and Biliary Sciences, New Delhi, India 2019 - Till date.*

3. **Member**, AIIMS Institutional Committee on Stem Cell Research and Therapy (IC-SCRT), India 2014-Till date

Contributions to academic committees

1. **Member, *Board of Studies***, *Interdisciplinary Brain Research Centre at Aligarh Muslim University, Aligarh, India, December 2016 – 2018.*

2. **Member, *Board of Studies***, *Department of Biochemistry, Jamia Hamdard University, New Delhi, India, 2016 - till date.*

3. **Member, *Academic Council,*** *National Brain Research Centre, Manesar, India, 2019-till date.*

4. **Member, *National Advisory Committee,*** *Centre of Neurosciences, Jiwaji Univ., Gwalior 2010.*

Executive Positions in Scientific Bodies

1. **General Secretary,** Indian Academy of Neurosciences, India, 2011 -Till date.

2. **Executive Member**, *Indian Academy of Neurosciences, India, 2004 – 2005 & 2008-2011.*

3. **Joint Secretary**, Indian Academy of Neurosciences, India, 2005 - 2008.

Editorial Boards

1. **Senior Editor,** *American Society of Neurochemistry (ASN Neuro), 2020 - till date.*

2. **Editor,** *Medical Virology, Virus Disease, March 2013 – 2020.*

3. **Associate Editor,** *NeuroInfectious Disease Section, Frontiers in Neurology, Switzerland, January 2020 – January 2022.*

4. **Review Editor,** *Frontiers in Neuropharmacology, Switzerland, 2014 – till date.*

5. **Guest Editor,** *Frontiers in Microbiology (Virology Section), Switzerland, 2015 – till date*

6. **Guest Editor,** Special Edition of Current HIV Research, USA 2015-2016.

7. **Member,** *Editorial Board, American Society of Neurochemistry, USA, 2018-2020.*

8. **Member,** Editorial Board, *Neurochemistry International, USA, 2012 - 2018.*

9. **Associate Editor,** Journal of Neurovirology, USA 2009 – till date.

10. **Associate Editor,** Annals of Neurosciences, India 2009 – till date.

Scientific / Academic Recognition

1. **Invited Speaker,** *How do viruses affect the human brain?* During the 150 years celebrations of the Rawenshaw University, Odhisha, Health and Disease: Contemporary concerns, September 12, 2020.

2. **Invited Speaker,** *SARS-CoV-2 is more than a respiratory virus -its potential in neuropathogenesis in COVID19 patients,* at the Bilateral Indo-US Webinar on COVID Biology jointly organized by IISER-Kolkata, IISC-Bangalore, University of Pennsylvania, USA and University of Colorado, USA, on August 17, 2020.

3. **Invited Speaker,** *Molecular Mechanisms used by viruses to affect human brain,* at Webinar which was part of the Lecture Series on contemporary issues in biosciences organized by

School of Life Sciences, Mahatma Gandhi Central University (MGCU), Motihari, Bihar, India June 15, 2020.

4. **Guest Faculty,** *Research methodologies for beginners* as a Webinar for an Online workshop organized by the Department of Biotechnology, Maharishi Dayanand University, Rohtak, Haryana, India May 5, 2020.

5. **Invited Speaker,** *Molecular mechanism of HIV-1 neuropathogenesis,* NCR Cluster Meeting at National Brain Research Centre, Manesar, India February 26, 2020.

6. **Guest Speaker,** *Molecular Mechanisms for virus induced neuronal damage,* at Workshop on Molecular Neurobiology from genes, Neurons to behavior in health and disease, at Regional Centre for Biotechnology (RCB), NCR Biotech Science Cluster, Faridabad, India February 24-29, 2020.

7. **Guest Faculty,** *What we know and what we need to know about how viruses affect human brain,* at Workshop on Animal Cell Culture Techniques and Applications at Department of Zoology, Miranda House, New Delhi, India, December 17-24, 2019.

8. **Invited Speaker,** *Novel insights into* Zika virus neuropathogenesis using human neural stem cells, at the 3rd Indo-US Symposium on "New Insights into the Inflammation, Immunity, and Pathobiology of Diseases", at Sinclairs Bayview, Portblair, Andaman Islands, India, December 3-8, 2019.

9. **Invited Speaker,** *Friends turn Foe* - Glia mediated neuronal damage in virus induced neuropathogenesis, at the 37th Annual meeting of Indian Academy of Neurosciences, at All India Institute of Medical Sciences, New Delhi, India, November 19-21, 2019.

10. **Invited Speaker,** *Molecular insights into zika virus induced neuropathogenesis.* NeuroCON 2019 at the Maharishi Markandeshwar Medical College and University, Mullana, India, November 15-18, 2019.

11. **Guest Faculty,** *Molecular mechanisms of zika virus induced microcephaly.* IBRO-APRC School, at Panjab University, Chandigarh, India, Nov 9-16, 2019.

12. **Guest Faculty,** *Novel insights into Molecular mechanisms of zika virus induced microcephaly.* IBRO-APRC School, at Banaras Hindu University, India, September 1-14, 2019.

13. **Guest Lecture,** *Molecular insights into virus induced damage to human brain cells.* Indian Institute of Science Education and Research (IISER) – Mohali, India, September 24, 2019.

14. **Invited Speaker,** *Molecular Mechanisms of Zika Virus Induced Microcephaly – some novel insights using human neural stem cell model.* Society of Neurochemistry (India), Jamia Hamdard University, New Delhi, India, October 10-12, 2019.

15. **Guest Faculty,** *Zika virus - what we know and want we need to know about its effects on human neural stem cells*, at the University extension lecture at Interdisciplinary Brain Research Centre (IBRC), J.N. Medical College, A.M.U., Aligarh, India on May 1, 2019.

16. **Guest Speaker,** *Novel insights into Zika Virus induced microcephaly* at IBRO-APRC School, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala, India on April 26, 2019.

17. **Invited Speaker,** *Zika Virus affects human neural stem cells*, at a Two day symposia "Exploring Mind, Brain and Behaviour" at Amity University NOIDA, India, during March 28-29, 2019.

18. **Invited Speaker,** *Understanding virus induced neurodegeneration with human neural stem cells*, national symposium on stem cell technologies in neurodegenerative diseases, at Era University, Lucknow, March 9, 2019.

19. Invited Lecture, *Cellular and Molecular mechanism of Zika virus induced alterations in properties of Zika virus human neural stem cells*, at Centre for Infectious Diseases, Indian Institute of Sciences (IISc), Bangalore, India December 13, 2018.

20. **Invited Lecture,** *Molecular mechanisms of Zika virus E protein induced alteration in human neural stem cells*, XXXVI annual meeting of Indian Academy of Neurosciences (IAN), at Varanasi, India, Oct 29-31, 2018.

21. **Guest Lecture,** *What we know and What we need to know about Zika Virus*, at Era University, Lucknow, India, October 18, 2018.

22. **Invited Speaker,** *Use of human neural stem cells as a model to understand neurodegenerative disorders*, during Luxembourg-German-India Alliance on Neurodegenerative diseases and Therapeutics (Lux-GIANT) under the Indo-German initiative, September 14-15, 2018.

23. **Invited Speaker,** *How do viruses affect human brain – some insights* Amity University – Haryana, Manesar, July 26, 2018.

24. **Invited Speaker,** *Zika viral protein alters human neural stem cell properties by altering miRNA circuitry,* at the Section of Infections of the Nervous System (SINS), National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, USA, April 30, 2018.

25. **Invited Speaker,** *Insights into HIV and Zika virus induced alterations in human progenitor cells,* at the University of Miami, Miller School of Medicine, Don Soffer Clinical Research Centre, Miami, USA, April 24, 2018.

26. **Invited Speaker,** *What we know and what we need to know about HIV-1 neuropathogenesis,* at the RUSH University Medical Centre, Department of Neurological Sciences, Chicago, USA, April 17, 2018.

27. **Invited Speaker,** *Friends turn foe during HIV-1 neuropathogenesis,* at the Alcohol Research Center, University of Chicago at Illinois, Chicago, USA, April 16, 2018.

28. **Co-Chairperson** for session on role of the Gut-Brain axis in controlling CNS viral reservoirs at the International Society of Neurovirology and Society of Neuro Immuno Pharmacology joint meeting at Chicago, USA, April 10-14, 2018.

29. **Invited Speaker,** *A novel model for understanding virus induced neurodegeneration* at the 10th NIPER Symposium on Nano-based Therapies for Neurodegenerative Diseases at National Institute of Pharmaceutical Education and Research (NIPER), Raebareli, March 27-28, 2018.

30. **Invited Speaker,** *What we know and what we need to know about virus induced neurodegeneration* at the Faculty Development Program of Delhi Technical University, New Delhi, India, March 14, 2018.

31. **Invited Speaker,** *"Role of glia mediated neuronal damage in HIV neuropathogenesis",* a meeting on Challenges in Clinical Neuroscience: from bench to bed side, at AIIMS-Bhubneshwar, Bhubneshwar, India, November 1, 2017.

32. **Invited Speaker,** *Cellular and Molecular Mechanisms of HIV-1 Neuropathogenesis,* at XXXV Annual meeting of Indian Academy of Neuroscience at Rawenshaw University, Cuttack, India, Oct 29-31, 2017.

33. **Guest Lecturer,** *Insights into mechanisms of neurodegeneration in HIV-1/AIDS*, at Era University, Lucknow, India, October 3, 3017.

34. **Invited Speaker,** Society of Neurochemistry Conference - 2017, *Astrocyte mediated neuronal damage in HIV-1 neuropathogenesis - how friends turn foe,* at Banaras Hindu University, Varanasi, India, September 20-22, 2017.

35. **Invited Faculty for IBRO/APRC Neuroscience School,** *Molecular insights into HIV-1 neuropathogenesis,* organized at National University of Singapore, Singapore. July 3-7, 2017.

36. **Invited Speaker, Central Inter-Disciplinary Research Facility,** Mahatma Gandhi Medical College and Research Institute, Pondicherry, India, June 20, 2017.

37. **Invited Speaker,** *Use of Human Neural Stem Cells as a Model to Understand Neurodegenerative Disorders,* hands-on workshop on "Molecular Biology Techniques & Stem Cells in Human Health and Diseases" at Amity Institute of Molecular Medicine and Stem Cell Research (AIMMSCR), Amity University Noida, India, June 12-16, 2017.

38. **Invited Speaker,** *An ill-fated relationship – HIV and Brain,* during International Symposium on Neurodegenerative Disorders (ISDN-2017) at National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore, India, March 29-30, 2017.

39. **Delivered 5th Sarath Chandran Memorial Lecture,** *Novel Insights into NeuroAIDS,* Sri Venkateshwara College, New Delhi, India, March 7, 2017.

40. **Guest Lecturer,** *Neural Stem Cells: the amazing cells in brain*, on National Science Day at Kendriya Vidyaylaya, NSG Campus, Manesar, India, February 28, 2017.

41. **Guest Lecturer,** *Unique model of neural stem cells for understanding NeuroAIDS.* Government Girls College, Gurgaon, India, February 13, 2017.

42. **Invited Speaker,** *Neural Stem cells as model system for understanding brain damage by HIV-* Delhi Technical University, New Delhi, India, February 11, 2017.

43. **Invited Speaker,** *Understanding neuron-glia crosstalk in HIV-1 Neuropathogenesis.* Brain Storming Session on DBT National Initiative-Glial Research on Health and Disease, Jiwaji University, Gwalior, India, February 2-3, 2017.

44. **Invited Speaker,** *HIV-1 Modulates Properties of Neural Stem Cells.* NeuroCON 2017, ICARE Institute of Medical Sciences and Research (IIMSR), Haldia, West Bengal, India, Jan 19-22, 2017.

45. **Invited Speaker,** *Consequences of HIV-1 Infection in Human Neural Stem Cells,* Indo-US symposium on Central Nervous System Viral Infections and its therapy, Doars, Chalsa Hilltop, India, November 14-17, 2016.

46. **Invited Speaker,** *Molecular insights into HIV-1 neuropathogenesis,* Department of Pathology Seminar Series, Uniformed Services University of Health Sciences, Bethesda, USA, November 1, 2016.

47. **Invited Speaker,** *Novel Molecular pathway of ATP perturbation of ATP release in human astrocytes contributes neuronal death in HAND,* 14th International Symposium of International Society of Neurovirology (ISNV) and HIV Endgame, Toronto, Canada, October 24-28, 2016.

48. **Invited Speaker,** *HIV-1 viral protein alters properties of human neural stem cells*, National University of Singapore, Singapore, September 1, 2016.

49. **Organizing Secretary,** XXXIV Annual meeting of Indian Academy of Neurosciences; *theme Molecules to Mind* at National Brain Research Centre, Manesar, India October 19-21, 2016.

50. **Invited Symposium Speaker and Chairperson**, *Neuron-Glia interactions: Friends turn foe in HIV-1 induced neurodegeneration, at* Asia Pacific Society of Neurochemistry (APSN 2016), Kuala Lampur, Malaysia, during August 26-30, 2016.

51. **Invited Speaker,** *Neural Stem cells as model for understanding healthy and diseased brain.* International Conference on Translation Medicine: Emerging Trends in Biomedicine, Biotechnology and Stem Cells Research – Present Status and Future Prospects. Amity University, Gurgaon, India, February 19-20, 2016.

52. **Keynote Speaker,** Second International Conference of Public Mental Health and Neurosciences, Bangalore, India, December 9-10, 2015.

53. **Session Speaker,** *Stem cell fate determinant TRIM32 mediates HIV-1 neuropathogenesis.* Annual Meeting of Indian Academy of Neurosciences, organized at Punjab University, Chandigarh, India, October 31 - November 2, 2015.

54. **Invited Faculty for IBRO/APRC Neuroscience School,** *Cellular and Molecular Pathways of HIV-1 Neuropathogenesis,* organized at National University of Singapore, Singapore. July 6-10, 2015.

55. **Invited Speaker**, *HIV Dementia,* 21st Annual Symposium – Neurodegeneration, Organized by Ranbaxy Science Foundation, ICGEB, New Delhi, India on March 9, 2015.

56. **Guest Faculty,** *Neural Stem Cells as a Tool to Understand NeuroAIDS*, Organized by Collaborative Undergraduate Biology Education (CUBE), HBCSE, Tata Institute of Fundamental Research, Mumbai, India on January 17, 2015.

57. **Resource Person for Lecture Workshop** Sponsored by Lecture Workshop sponsored by Science Academies' Education Panel *on "Building of an Organism" at* The Department of Life Sciences at Sophia College, Mumbai, India. November 21-22, 2014.

58. **Invited Speaker,** *Molecular Basis of HIV-1 Induced Neuronal Damage*, National Conference of Molecular Virology, Jamia Milia Islamia, New Delhi, India. November 17-18, 2014.

59. **Invited Faculty for IBRO/APRC Neuroscience School, "***Neural Stem cells as model for neurodegeneration"* organized at Panjab University, Chandigarh, India. November 3-8, 2014.

60. **Invited Speaker,** "*Friends turn foe - Role of astrocytes in HIV-1 neurodegeneration" at* 31st Annual Meeting of Indian Academy of Neurosciences at NIMHANS Bangalore, India, November 1-3, 2014.

61. **Invited Speaker,** *"Role of Purinergic Receptor P2X7 receptor in Astrocyte Mediated Neuronal Injury in NeuroAIDS".* Three Decades of Research in PML and Disorders Affecting the CNS. National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda USA, June 20, 2014.

62. **Invited Speaker,** "*HIV Induced Neurodegeneration"***.** National Seminar on Next Generation Sciences – Vision 2020 and Beyond, MD University, Rohtak, India, March 8, 2014.

63. **Session Speaker, "***What's on the other side of the coin? Glia in HIV-1 Pathogenesis".* Indo-US Symposium on Viral Infections on the Nervous System, Gurgaon, India, February 23-25, 2014.

64. **Invited Speaker, "***Neural Stem cells – a Tool to Understand Molecular and Cellular Basis of NeuroAIDS".* Workshop on Neural Staining Techniques, Department of Anatomy, Maulana Azad Medical College, New Delhi, India, March 7, 2014.

65. **Invited Speaker** "*Neuron-Glia Crosstalk - see how the masters do their job"* at the APSN-ISN School and Conference on Neurochemistry of Ageing Brain, CSIR-IICB Kolkata, India, January 29, 2014.

66. **Session Speaker,** *Molecular Mechanisms for Alterations in Human Neural Precursor Cell Proliferation by HIV-1 Tat and Morphine*. At the Annual Meeting of International Society of Neurovirology (ISNV), Washington DC, USA, October 29-November 1, 2013.

67. **Invited Speaker and Co-Chairperson,** *Astrocyte the Star Avatar-mediator of neuronal damage in HIV-1/AIDS*. At the 31st Annual meeting of Indian Academy of Neurosciences, Allahabad, India. October 25-27, 2013.

68. **Coordinator and Faculty** – Scientific Communication and Grant Writing Workshop, Sponsored by Department of Biotechnology, at XXXI Annual Meeting of Indian Academy of Neuroscience, Allahabad, Oct 24, 2013.

69. **Invited Speaker,** *Role of Glia in HAND*. At the Indo-US symposium at National AIDS Research Institute, Pune, India, in September 23-24, 2013.

70. **Invited Speaker,** *Understanding Healthy and Diseased Brain*. Brain Awareness Program, CSIR-Central Drug Research Institute, Lucknow, India, August 14, 2013.

71. **Invited Faculty,** *Stem Cells: Promise and Challenges*. Pankaj Seth, Government Girls College, Gurgaon, India, February 26, 2013.

72. **Invited Speaker and Co-Chairperson,** *Glia Mediated Neuronal Injury in HIV-1 Neuropathogenesis: "the other side of the coin"* in session at 27th Annual Meeting of Society of Neurochemistry, India. Feb 21-23, 2013.

73. **Invited Speaker and Co-Chairperson,** *In vitro neural stem cell system - An opportunity to "peep" inside the degenerating human brain?* at NeuroCon 2013 meeting at Indian Institute of Chemical Biology, Kolkatta, India, January 18-20, 2013.

74. **Invited Resource Person -** *Are we doing enough to cure HIV/AIDS in India?* Pankaj Seth, Invited Session Speaker, One Day Seminar on HIV/AIDS and Drug Abuse, Ukrul District, Manipur, Nov 3, 2012.

75. **Invited Session Speaker and Co-Chair** - *HIV-1 Induced Neuronal damage: Who is to be blamed?* Pankaj Seth at XXX Annual meeting of Indian academy of neurosciences, Guru Nanak Dev University, Amritsar, Oct 27-30, 2012.

76. **Coordinator and Faculty** – Scientific Communication and Grant Writing Workshop, Sponsored by Department of Biotechnology, at XXX Annual Meeting of Indian Academy of Neuroscience, Amritsar, Oct 27, 2012.

77. **Guest Speaker -** *Neural Stem Cells – an in vitro model to understand brain disorders*. At *Seminar Series on Stem Cell Application in health care at Amity University*, Haryana Oct 22, 2012.

78. **Invited Session Speaker** - Neural Stem Cells - *a window into neurodegenerative diseases.* Pankaj Seth, at Rajiv Gandhi Institute of Technology, Amethi, During National Seminar on "Stem Cell an emerging Healthcare Frontier" during August 20-21, 2012.

79. **Invited Seminar Speaker -** Neuron - Glia interactions during HIV Neuropathogenesis. Invited Seminar in *Calendar of Events of National Institutes of Health (NIH)*, Bethesda, USA, May 16, 2012.

80. **Invited Speaker -** Neural Stem Cells - a Tool for Basic Research. At Amity University, Haryana April 14, 2012.

81. **Session Co-Chairperson**, on AIDS and Substances of Abuse - A Global Scenario at 18th Society of Neuroimmune Pharmacology (SNIP) Scientific Meeting April 2012.

82. **Guest Faculty**, Indian Institute of Science Education and Research, IISER-Kolkata, Feb 8th, 2012.

83. **Invited Speaker**, Golden Jubilee Symposium, Department of Biochemistry, Panjab University, Chandigarh, February 10-11, 2012.

84. **Invited Speaker,** 5th Symposium on Molecular Medicine at Special Centre for Molecular Medicine, Jawaharlal Nehru University, New Delhi Feb 17-18, 2012.

85. **Invited Speaker,** National Seminar on Reactive Oxygen Species, Department of Biochemistry, Lucknow University, Lucknow, Dec 24, 2011.

86. **Invited Speaker,** Alzheimer's Disease Symposia, Department of Neurology, King George's Medical College, Lucknow, Dec 3, 2011.

87. **Invited Speaker,** 57th, Annual Meeting of Association of Physiologists and Pharmacologists of India, All India Institute of Medical Sciences, New Delhi, Dec 2011.

88. **Session Convener and Co-Chairperson,** on Neuron-Glia Biology at Annual Meeting of Indian Academy of Neurosciences, New Delhi, Oct 2011.

89. **Invited Speaker,** Department of Anesthesia and Critical Care Medicine, The Children's Hospital of Philadelphia, Philadelphia, USA, April 2011.

90. **Invited Speaker,** Department of Neuroscience, School of Medicine, Temple University, Philadelphia, USA, April 2011.

91. **Session Speaker,** Annual meeting of Society of Neuroimmunopharmacology (USA) at Florida, USA, April 2011.

92. **Session Speaker**, 98th Science Congress, SRM University, Chennai, India, Janurary 2011.

93. **Invited Speaker and Co-Chair**, Glia Symposia Health and Disease on Jiwaji University, Gwalior, India, Dec 2010.

94. **Convener, Session Speaker and Co-Chair,** Neural Stem Cell: Potential and Challenges, at 5th Congress of FAONS, Lucknow, Nov 2010.

95. **Invited Speaker,** Brain Awareness Week, Banaras Hindu University, Varanasi, March 2010.

96. **Guest Speaker,** National Science Day, Kendriya Vidyalaya, Manesar, India, February 2010.

97. **Guest Faculty,** Jawaharlal Nehru University Academic Staff College Lecture series for college teachers, January 2010.

98. **Research Highlight,** in Newsletter of International Society of Neurovirology (ISNV), in January 2010.

99. **Invited Speaker and Convener International Symposia**, in Annual Meeting of Indian Academy of Neurosciences at NIIMS University, Jaipur, December 2009.

100. **Invited Speaker**, Annual meeting of International Society of Neurovirology (USA), Miami, USA, June 2009.

101. **Invited Speaker**, International NeuroAIDS Research Meeting organized by HIV Neurobehavioral Research Center (HNRC), Miami, USA, May 2009.

102. **Invited Speaker,** Annual meeting of Society of Neuroimmunopharmacology (USA) at Wuhan, China, April 2009.

103. **Invited Speaker,** INDO-US Forum Meeting, Organized by US National Academy of Science, Agra, India, March 2009.

104. **Invited Speaker,** National Frontiers of Science Meeting, Indian National Science Academy, New Delhi, India, January 2009.

105. **Invited Speaker,** NeuroUpdate 2008 Kolkata, Calcutta National Medical College Hospital and Indian Institute of Chemical Biology, Kolkata, India, September 2008.

106. **Invited Speaker,** Science Popularization Series Lecture, Organized by Haryana State Council for Science and Technology, Gurgaon, India, August 2008.

107. **Guest Faculty,** "Stem cells: Good, Bad and Ugly." At Haryana Government Girls College, Gurgaon for refresher's course for degree college teachers, May 2008.

108. **Invited Speaker,** Organized by Uttar Pradesh Association of Science and Technology Advancement and National Academy of Sciences (Local Chapter), Lucknow, March 2008.

109. **Invited Speaker,** International Conference on Opportunistic Pathogens in AIDS, New Delhi, India, January 2008**.**

110. **Invited Speaker,** International Conference of Current Advances in Molecular Biochemistry, Lucknow, December 2007.

111. **Invited Speaker,** International Meeting of Association of Clinical Biochemists of India, New Delhi, December 2007.

112. **Invited Speaker,** Neural Stem Cells. Science Popularization Series Lecture, Organized by Haryana State Council for Science and Technology, Bhiwani, India, August 2007.

113. **Invited Speaker,** Indian Academy of Neuroscience, Varanasi, India, November 2007.

114. **Invited Speaker,** National Symposium on Glial Neurobiology, Jiwaji University, Gwalior Oct 2007, India.

115. **Invited Speaker,** NeuroAIDS in Asia and Pacific Rim, Sponsored by and National Institute of Neurological Disorders and Stroke, National Institute of Mental Health (National Institutes

of Health, USA), At Garvan Institute, Sydney, Australia July 2007.

116. **Invited Speaker,** Neurobiology and Neuroinformatics 2007 meeting, Cheju National University, Jeju City, South Korea, July 2007.

117. **Invited Speaker,** International Society of Neurochemistry (ISN) Symposia on Neuropathogenesis of HIV-1 Dementia, Silver Jubilee meeting of Indian Academy of Neuroscience, Lucknow, India, Dec 2006.

118. **Invited Speaker,** Symposia on Neurobiology of Infections, Indian Academy of Neuroscience, Bangalore, India, Dec 2005.

119. **Guest Faculty,** in CME-cum-Update on Stem Cell Biology and Regenerative Medicine, Organized by Moving Academy of Medicine and Biomedicine at Government Medical College Chandigarh, Chandigarh, India, April 2005.

120. **Invited Speaker,** Workshop on Stem Cells and Regenerative Medicine, Organized by Moving Academy of Medicine and Biomedicine at Patiala Medical College, Patiala, India, April 2005.

121. **Invited Speaker,** in Symposia on Neurodegeneration and Neuroregeneration, Indian Academy of Neuroscience, Gwalior, India, Jan 2005.

122. **Invited Speaker,** for World Alzheimer's Day The Alzheimer's Society and Related Disorders Society of India, New Delhi, India, September 2004.

123. **Guest Faculty,** Continuing Education Program at DIPAS, New Delhi, India, September 2004.

124. **Invited Speaker,** United States Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Edgewood, USA, January 2004.

125. **Invited Speaker,** International Symposium on Molecular Toxicology and Environmental Health, Lucknow, India, November 2003.

126. **Invited Participant** at Conference on Resuscitation Fluid Design and Resuscitation Protocols for Combat Casualties, Organized by National Academy of Sciences for Office of Naval Research, Washington D.C, USA, 1998.

Administrative Experiences

- Actively involved with setting up of the Translational Research Unit (TRU) of NBRC, currently scientist In-charge of TRU.

- Engaged in recruitment, assessment and promotion of scientific and non-scientific staff.

- Chairperson / Member of several committees of the institute that involved procurement of laboratory equipments, construction, tender processes for service providers like Housekeeping, transport, canteen etc.

- Actively involved in activities pertaining to national and international research collaborations and organization of scientific conferences both at National and International levels.

- Instrumental in visits of foreign dignitaries to the Centre with an aim to showcase research capabilities to place NBRC on global research map.

- Organizing Secretary of the first Indo-US symposium on Viral Infections of the Central Nervous System, February 2014, sponsored by Indo-US Science and Technology Forum.

- Scientist-In-charge for Brain Awareness Program in various academic and research institution throughout the country under NBRC banner to help in attaining a bigger footprint of NBRC.

- Scientist-In-charge for Open Day activities on Centre's Foundation Day to emphasize importance of neuroscience research for students and teachers from local school and colleges.

- Visioning Outreach Program for better visibility of the Institute.

- Liaison with Civil Hospital Gurgaon for clinical material, brain disorder patients and fetal brain tissue.

- Contributing immensely into Institutional Biosafety issues as Member Secretary of Institutional Biosafety Committee (IBSC) and holding annual Biosafety classes to make NBRC a safe working place.

- Other Institutional Responsibilities

    - **Chairman,** NBRC Library Committee, NBRC, 2018-2019.
    - **Coordinator,** Distributed Information Centre, NBRC, 2013 - 2019.
    - **Coordinator,** Molecular Biology Program (M.Sc and Ph.D), NBRC 2006-till date.
    - **Coordinator**, Clinical Neuroscience Program (M.Sc and Ph.D), NBRC 2004-05.
    - **Scientist-In-Charge,** Ph.D and Integrated Ph.D Entrance exam, NBRC 2007.
    - **Bio-safety Officer**, NBRC 2005-2010.
    - **Member Secretary,** Institutional Biosafety Committee, IBSC, NBRC 2010-till date.
    - **Member,** NBRC Academic Council, NBRC, 2005- till date.
    - **Member,** Board of Studies, NBRC 2005- 2019.
    - **Member,** Selection Committee for Post Doctoral Fellows and Graduate Students
    - **Assistant Warden,** NBRC Hostels, 2005 – 2010.
    - **Chief Warden,** NBRC Hostels, 2011-2014.

Technical Writing Experience

Grant Writing, Scientific writing for research publications, reviews and News Media.

Teaching/training

A. Currently teaching the M.Sc. and Ph.D Neuroscience course at National Brain Research Institute (NBRC), a Deemed University at Manesar (Gurgaon), India.

B. Conducted Hands on training on "Ribonuclease Protection Assay and its Applications in Modern Molecular Biology" at Central Drug Research Institute and Industrial Toxicology Research Center, India, 1999.

C. I have been actively involved in the initial training of all the graduate students, summer trainees and technicians who join the lab. I supervise their research activities by helping them initially to plan, execute and analyze their experiments.

Technical Skills

a) *Molecular Biology (Gene expression and regulation)* - Gene Silencing, cDNA Microarray, RT-PCR, RNase protection assays, Northern and Southern analysis, Nuclear run off Assay, Electrophoretic Mobility Shift Assay, Southern and Western blotting.

b) *Cell Biology and Immunology* – Human inducible pluripotent cells (hiPSCs) from blood cells, Isolation and characterization of Neural Stem cells and Neuronal tissue cultures, *in vitro* viral infection models, viral assay and transfection, Isolation of neutrohil and platelets from blood, cytokines assay by ELISA, Immunohistochemical staining, immunofluorescence, *In situ* hybridization, apoptosis.

c) Animal Models - Neuro-infection models, Thrombosis, Hemorrhage, Ischemia/reperfusion, Hypoxia, Tumor induction, Wound healing.

Research Funding

National

1. *Cellular and Molecular Basis of Neurobiology of HIV-1C in Human CNS cells*. PI - Pankaj Seth. Department of Biotechnology, Ministry of Science & Technology, Govt. of India. Rs. 3,489,000 for three years – No. BT/PR6838/Med/14/881/2005 (2006-2009).

2. *Characterization of Human Fetal Brain Derived Neural Stem Cells as a Model for studying neurodegenerative disease*. PI- Pankaj Seth. Department of Biotechnology, Ministry of Science and Technology, Govt. of India. Rs. 2,000,000 for three years – No. BT/PR6615/Med/14/857/2005. (2007-2012).

3. *Role of human umbilical cord blood stem cells and neural stem cells in neuronal regeneration and functional restoration: A comparative study in male adult rats with acute spinal cord injuries*. (PI- Dr. Sumit Sinha, AIIMS, New Delhi) Co-PI: Pankaj Seth (2011-2013).

4. "*Understanding Neuron-Glia Crosstalk in HIV Neuropathogenesis".* PI – Dr. Pankaj Seth. Funded under National Initiative of Glial Cell Research in Health and Disease, by DBT (March 2012-2016), India.

5. *"Insights into role of a dyslexia linked long non-coding RNA (lnc RNA) in human neural stem cell differentiation".* PI – Dr. Pankaj Seth, by Department of Science and Technology (DST) SR/CSRI/210/2016 (2017-2020), New Delhi, India.

6. *"Differentiation of fetal neural stem cells to oligodendrocytes – a disease model to model to decipher the pathogenesis and devise therapeutic strategies for cerebral palsy".* PI Pankaj Seth, by Department of Biotechnology, Rs 20,08000/- BT/PR17581/MED/31/333/2016 (March 2018-2020).

7. *Hypoxia induced changes in Blood Brain Barrier.* PI Pankaj Seth. Funded by Department of Biotechnology, BT/21413/MED/122/77/2017 (September 2018 – 2021).

8. *Effect of hypoxia on different neural cell types in vitro – a model to design therapeutic strategies against cerebral palasy*. PI Pankaj Seth. Funded by Department of Biotechnology, BT/21413/MED/122/40/2016 (October 2018 - 2021).

9. *Role of Ephrins/Eph receptor in HIV mediated neuropathogenesis.* PI Pankaj Seth. Funded by Department of Biotechnology, BT/21413/MED/122/40/2016 (June 2019 - 2022).

International

10. *Role of CNS Opportunistic Infections in Subsequent Development of HIV encephalitis*. Co-Investigator: Pankaj Seth. Funded by National Institutes of Health (NIH), USA, February 2008. 1R01 NS055628 (RO1/NIH grant) (2008-2013).

11. *"Role of Viral Proteins Interactions of JC Virus and HIV-1 in Viral Neuropathogenesis"* . Funded by Office of AIDS Research under the *Intramural NIH-to-India program, USA* June 2008; PI (Indian Side): Pankaj Seth (2008-2010).

12. *"Mollecular Mechanisms & Therapy for Cocaine Abuse in HIV Associated Neurocognitive Disorder (HAND)"* PI - Dr. Pankaj Seth. A joint grant funded by NIH (R21) and ICMR for collaborative research proposal under auspices of the U.S-India Bilateral Collaborative Research Partnerships (CRP) on the Prevention of HIV/AIDS and Co-morbidities (R21) Program (2011-2013).

Membership of Professional Bodies, Societies, Academics

- Board Member, International Society of Neurochemistry, USA.
- Elected Member and Fellow, The National Academy of Sciences, India.
- Elected Fellow, Indian Academy of Neurosciences, India.
- Member, International College of Neuropsychopharmacology, Austria.
- Member, Guha Research Conference, India.
- Member, Asia Pacific Society of Neurochemistry (APSN), Singapore.
- Member, International Society of Neurovirology, USA.
- Member, International Society of Neurochemistry, USA.
- Member, Society of Neuroimmune Pharmacology, USA.
- Member, International Brain Research Organization (IBRO).
- Life Member, Indian Academy of Neurosciences (IAN), India.
- Life Member, Society of Neurochemistry (India) (SCNI).
- Life Member, Indian Pharmacological Society (IPS), India.
- Life Member, Alumni Association of Biochemistry Department, Lucknow University, India
- Life Member, Uttar Pradesh Association for Science & Tech. Advancement, India.

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I. PAPERS/REVIEWS: 67 Cumulative Impact Factor: 248.281

1. Novel role of mortalin in attenuating HIV-1 Tat mediated astrogliosis. Priyanka R Wadhwa, R. Chaudhuri, TC Nag, and **P. Seth.** *Journal of Neuroinflammation* Sep 20;17(1):276. doi: 10.1186/s12974-020-01912-3. PMID: 32951595 ***Impact Factor: 5.793***

2. SARS-CoV-2 more than a respiratory virus: Its potential role in neuropathogenesis. CMS Singal, P. Jaiswal and **P. Seth.** *ACS Chem Neuroscience 2020* July 1;11(13):1887-1899. doi: 10.1021/acschemneuro.0c00251. Epub 2020 Jun 18. PMID: 32491829. ***Impact Factor: 4.486***

3. 'Primed' Mesenchymal Stem Cells: A Potential Novel Therapeutic for COVID19 Patients**.** SS. Raza, **P. Seth**, MA. Khan. *Stem Cell Reviews and Reports* June 2020: 1-10. doi:10.1007/s12015-020-09999-0 ; PMID: 32592163. ***Impact Factor: 5.316***

4. Genetic architecture of Parkinson's disease in the Indian population: Harnessing genetic diversity to address critical gaps in Parkinson's disease research. R Rajan, Divya Kp, RM Kandadai, VP. Satagopam, Madhusoodanan UK, P. Agarwal, N. Kumar, T. Ferreira, H. Kumar, S. Prasad Av, K. Shetty, S. Mehta, S. Desai, S. Kumar, Prashanth L K, M. Bhatt, P. Wadia, S. Ramalingam, G M Wali, S. Pandey, F. Bartusch, M. Hannussek, J. Krager, A. Kumar-Sreelatha, S. Grover, M. Sturm, P. Lichtner, J. Roeper, V. Busskamp, GR Chandak, JC. Schwamborn, **P. Seth**, T. Gasser, O. Riess, V. Goyal, PK. Pal, R. Borgohain, R. Krueger, A. Kishore and M. Sharma. *(*Article type: Study Protocol).
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5. Temporal transcriptome analysis of neuronal commitment reveals the preeminent role of the divergent lncRNA biotype and a critical candidate gene during differentiation. B Prajapati, M Fatima, M Fatma, P Maddhesiya, H Arora, **P. Seth** and S Sinha. *Cell Death Discovery* 2020. Apr 24; 6: 28. doi: 10.1038/s41420-020-0263-6. eCollection 2020. PMID: 32351715
   Impact Factor: 4.114

6. MiRNA-137-mediated modulation of mitochondrial dynamics regulates human neural stem cell fate. AS Channakkar, T Singh, B Pattnaik, K Gupta, **P. Seth**, and YK. Adlakha. *STEM* CELLS 2020, May;38(5):683-697. doi: 10.1002/stem.3155. ***Impact Factor: 6.022***

7. Sinomemine inhibits amyloid beta-induced astrocyte activation and protects neurons against indirect toxicity. D. Singh, A. Agrawal A, CMS Singal, HS Pandey, **P. Seth** and SK Sharma. *Molecular Brain* 2020, March; 13(1):1-10. doi: 10.1186/s13041-020-00569-6.
   Impact Factor: 4.902

8. Origin of a novel *CYP20A1* transcript isoform through multiple Alu exaptations creates a potential miRNA sponge. A Bhattacharya, V Jha, K Singhal, M. Fatima, D. Singh, G Chaturvedi, D. Dholakia, R Kutum, R Pandey, TE. Bakken, **P. Seth**, B Pillai, M Mukerji. *BioRxiv preprint* doi: https://doi.org/10.1101/618645. ***Impact Factor: --***

9. Identification and Classification of Hubs in microRNA Target Gene Networks in Human Neural Stem/Progenitor Cells following Japanese Encephalitis Virus Infection. S. Mukherjee, I. Akbar, R. Bhagat, B Hazra, A Bhattacharyya, **P. Seth**, D. Roy and A Basu. *mSphere* 2019 Oct 2;4(5). pii: e00588-19. doi: 10.1128/mSphere.00588-19. ***Impact Factor: 4.280***

10. Friends Turn Foe-Astrocytes Contribute to Neuronal Damage in NeuroAIDS. H. Pandey and **P. Seth.** Journal of Molecular Neuroscience 2019 Oct;69(2):286-297 doi: 10.1007/s12031-019-01357-1. Review. ***Impact Factor: 2.496***

11. Phosphoinositide-3-kinase inhibition elevates ferritin level resulting depletion of labile iron pool and blocking of glioma cell proliferation. P Gupta, P Singh, HS Pandey, **P. Seth** and CK Mukhopadhyay. *Biochim Biophys Acta Gen Subj*. 2019 Mar;1863(3):547-564. doi: 10.1016/j.bbagen.2018.12.013. Epub 2018 Dec 23. ***Impact Factor: 3.422***

12. Identification and epigenetic analysis of divergent long non-coding RNAs in multi-lineage differentiation of human Neural Progenitor Cells. B. Prajapati, M. Fatma, P. Maddhesiya, MK Sodhi, M. Fatima, T. Dargar, R. Bhagat, **P. Seth** and S. Sinha. *RNA Biology 2019* Jan;16(1):13-24. doi: 10.1080/15476286.2018.1553482. ***Impact Factor: 5.350***

13. Zika Virus E protein alters properties of human fetal neural stem cells by modulating microRNA circuitry. R. Bhagat, B. Prajapati, S. Narwal, N. Agnihotri, YK Adlakha, J. Sen, S. Mani and **P. Seth**. *Cell Death and Differentiation* Jul 26. doi:10.1038/s41418-018-0163-y 2018. ***Impact Factor: 8.000***

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15. *The Expanding Horizon of MicroRNAs in Cellular Reprogramming. Y. Adlakha and **P. Seth**. Progress in Neurobiology Jan* 148:21-39. doi: 10.1016/j.pneurobio.2016.11.003*, 2017.* ***Impact Factor: 13.217***

16. *Intra-generational protein malnutrition impairs temporal astrogenesis in rat brain.* A Ahmad Naik, N. Patro, **P. Seth** and IK Patro. *Biology Open* Jul 15;6(7):931-942. doi: 10.1242/bio.023432, 2017. ***Impact Factor:*** **3.286**

17. Japanese encephalitis virus induces human neural stem/progenitor cell death by elevating GRP78, PHB and hnRNPC through ER stress. S. Mukherjee, N. Singh, N. Sengupta, M. Fatima, **P. Seth**, A. Mahadevan, SK Shankar, A. Bhattacharyya and A. Basu. *Cell Death Disease* Jan 19;8(1):e2556. doi: 10.1038/cddis.2016.394, 2017. ***Impact Factor: 5.638***

18. Novel insights into role of miR-320a-VDAC1 axis in astrocyte-mediated neuronal damage in neuroAIDS. M. Fatima, B. Prajapati, K. Saleem, R. Kumari , C. Singal, Mohindar Singh Singal **P. Seth**. *Glia* 65(2):250-263. doi: 10.1002/glia.23089, 2017. ***Impact Factor: 5.846***

19. *NeuroAIDS: Past, Present and Future. **P. Seth**; EDITORIAL In: Current HIV Research 14 (5): 372, 2016.* ***Impact Factor: 1.612***

20. Cell Therapy for Neurological Disorders: The Elusive Goal: A Review. P.N Tandon and **P. Seth.** Neurology India 64: 612-623, 2016. ***Impact Factor: 1.758***

21. HIF-2<alpha> mediates a marked increase in migration and stemness characteristics in a subset of Glioma cells under hypoxia by activating an Oct-4/Sox-2- Mena (INV) axis. M. Bhagat, JK Palanichamy, P. Ramalingam, M Mudassir, K. Irshad, K. Chosdol, C. Sarkar, **P. Seth**, S.

Goswami, S. Sinha, P. Chattopadhyay. *Int J Biochemistry and Cell Biology* May; 74:60-71, 2016. doi: 10.1016/j.biocel.2016.02.017. ***Impact Factor: 3.505***

22. Tripartite Containing Motif 32 Modulates Proliferation of Human Neural Precursor Cells in HIV-1 Neurodegeneration M Fatima, R Kumari, JC. Schwamborn, A Mahadevan, SK. Shankar, R Raja, and **P. Seth**. *Cell Death and Differentiation* May;23(5):776-86 2016. doi: 10.1038/cdd.2015.138. Epub 2015 Nov 20. PMID: 26586575. ***Impact Factor: 8.218***

23. *Emerging Role of P2X7 Receptors in CNS Health and Disease.* M. Tewari and **P. Seth**. *Ageing Research Reviews* Nov 24 (Pt B):328-42. (doi: 10.1016/j.arr.2015.10.001) 2015. ***Impact Factor: 7.526***

24. Role of extracellular hydrogen peroxide on regulation of iron homeostasis genes in neuronal cell: Implication in iron accumulation. S. Dev, S. Kumari, N. Singh, SK Bal, **P. Seth**, CK. Mukhopadhyay. *Free Radical Biology and Medicine* 86:78-89, 2015. ***Impact Factor: 2.949***

25. Astrocytes mediate HIV-1 Tat-induced neuronal damage via ligand-gated ion channel, P2X7R. M. Tewari, Monika, R. Verghese, M. Menon and **P. Seth**. *Journal of Neurochemistry* 132: 464-476, 2015 (Featured on Cover page of the Journal). ***Impact Factor: 3.842***

26. Involvement of Extracellular signal-regulated kinase (ERK1/2)-p53-p21 axis in mediating neural stem/progenitor cell cycle arrest in co-morbid HIV-Drug abuse exposure. S. Malik, R. Saha and **P. Seth**. *Journal of Neuroimmune Pharmacology* 9:340-353, 2014. ***Impact Factor: 4.11***

27. HIV-1 Tat disrupts CX3CL1-CX3CR1 Axis in Microglia *via* the NF-κBYY1 Pathway. M. Duan, H. Yao, Y. Cai, K. Liao, **P. Seth** and S. Buch. *Current HIV Research* 12(3): 189-200, 2014. ***Impact Factor:* 1.757**

28. Cocaine and HIV-1 Interplay: Cellular and Molecular Mechanisms. S. Buch, H. Yao, M. Guo, T. Mori, **P. Seth**, J. Wang and T. Su. *Current HIV Research* 10: 425-428, 2012. ***Impact Factor:* 1.757**

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II CHAPTER IN BOOKS: 7

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2. Astrocytes in Neuroinflammation and Neuronal Disorders: *Shifting the Focus from Neurons.* M. Tewari and **P. Seth**. Book Chapter In: Inflammation: the Common Link in Brain Pathologies. Eds. Nihar R Jana and A. Basu. Springer Publishers, pgs 43-70, 2016.

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RESEARCH CONTRIBUTIONS:

Despite the fact that India has millions of HIV/AIDS patients, only 3-4 researchers are engaged in our country for studying molecular mechanisms of neurocognitive and motor deficits (neuroAIDS) in AIDS patients. This is mainly due to complexities of the disease, unavailability of AIDS brain, lack of apt experimental models and challenges in the field. Dr. Seth's laboratory is among the very few labs not only in India, but also in world, that has overcome these challenges to understand molecular mechanisms in the area of neuroAIDS and Zika virus (ZIKV), using primary cultures of human fetal brain derived neural stem cells (hNSCs). We are exploring underlying mechanisms for HIV-1 and Zika virus mediated neurodegeneration in hNSCs.

As documented in my bibliography, I have made several important contributions to the field of neurosciences particularly in understanding the molecular and cellular pathways of viruses that impact populations worldwide including India. Some of the major ones are listed below:

1. **Role of glia mediated neuronal injury in virus induced neurodegeneration:** We are focusing to understand how astrocytes mediate neuronal death using neuron glial co-cultures derived by differentiating human neural stem cells. We are investigating how HIV-1 Tat and Zika viral proteins modulate the neuron-glia interactions that culminate into neuronal damage. We use cell and molecular biology approaches that include - live cell calcium imaging in human brain cells, time lapse microscopy, miRNA sequencing based assays, gene expression studies and bioinformatics tools. Wherever possible, we also validate the *in vitro* findings in post-mortem brain sections of AIDS patients that are available through the NIMHANS human brain bank. In past, our laboratory has immensely contributed to study the role of astrocytes in mediating neuronal damage and effects of HIV-1 Tat on neural stem cell functions. The laboratory continues the in-depth research on understanding the cellular and molecular mechanisms how HIV-1 Tat protein causes glia mediated neuronal damage. We utilize a well characterized human fetal brain derived neural stem cell culture system established by our laboratory at the National Brain Research Centre.

In previous years, we reported astrocytes release excess ATP following exposure to HIV-1 Tat and that excess ATP was detrimental to neurons. We reported a novel molecular pathway of miRNA that regulates ATP release by controlling VDAC1 expression levels that subsequently modulates glia mediated neuronal damage. We demonstrated that astrocyte mediated neuronal damage by HIV-1 Tat could be rescued using VDAC1 siRNA or mimic miR-320a. This was also validated using autopsy tissues sections and *in vitro* culture system of human primary neurons and astrocytes. We provided novel insights into the possibility of the miRNA-320a and VDAC1 axis in HIV-1 neuropathogenesis and as possible target for ameliorating the neuronal damage **(Fatima et al GLIA 2017)**.

For deeper insights into HIV-1 neuropathogenesis, we continued our efforts into this important area of research. We identified that a HSP70 family of protein, mortalin offers protection against HIV-1 Tat by binding and then degrading it, hence making Tat unavailable for its neurotoxic effects in astrocytes. We could reach to this novel observation based on detailed and painstaking experiments and assays with primary cultures of human astrocytes and neurons. Our approach involved cellular and molecular assays, biochemical assays, mitochondrial bioenergetics, ultrastructure studies using electron microscopy, bioinformatics tools as well as validation in post-mortem brain samples from HIV/AIDS patients, from India's only brain bank at NIMHANS, Bangalore. To gain deeper understanding of the mechanism behind Tat degradation, we used a bioinformatics simulation tool to predict interaction between mortalin and HIV-1 Tat. We obtained Protein Data Bank (PDB) structures of mortalin (3N8E) and Tat (1TIV) and simulated in Clus-pro online protein docking tool, followed by visualization of the dock structure using pymol visualizing tool. This helped in prediction of the direct interaction of HIV-1 Tat and mortalin. *In silico* findings were validated with *in vitro* assays by performing coimmunoprecipitation assay.

In conclusion we discovered a novel role of mortalin in attenuating the glial mediated neuronal damage in presence of HIV-1 Tat. We strongly believe this could be employed as a critical molecular target for clinical intervention in eliminating deleterious effects of HIV-1 on neuronal survival and reducing the morbidity of HAND cases **(Priyanka et al J Neuroinflammation 2020)**.

2. **Zika Virus E protein alters human neural stem cell properties:** Zika Virus is spread through mosquito and is known to cause microcephaly, a condition in which baby is born with significantly smaller head size, due to abnormal brain development during pregnancy. Our laboratory carried out detailed investigations to understand the cellular and molecular mechanisms that may help to understand how Zika Virus causes microcephaly in babies. Since Zika viral infections lead to life crippling disorders or deaths in infants, Dr. Seth's laboratory performed experiments using human fetal neural stem cells to understand how proteins of this virus may affect properties of human fetal neural stem cells during development. Initially, they over-expressed several Zika Virus proteins in brain stem cells in culture, using expression vectors that were obtained from Dr. Shyamala Mani (a former NBRC and IISc scientist, who is currently at INSERM, France) and studied their effects on cell proliferation of human brain cells. Dr. Seth and his research team noticed that out of all the viral proteins studied by them, Zika Virus Envelope (E) protein caused maximum effects on proliferation of human brain stem cells. Dr. Seth's laboratory discovered that envelop (E) protein of Zika Virus affects proliferation rates of human neural stem cells and promotes premature but aberrant neurogenesis. In collaboration with Dr. Jonaki Sen at IIT-Kanpur we validated the findings in animal models and discovered that over-expression of E protein in developing rat pups by in utero electroporation resulted in significant decrease of

proliferation of brain stem cells. Global miRNA analysis using Next Generation Sequencing revealed 25 miRNAs that were significantly altered in response to E protein in brain stem cells. Dr. Seth's study provided novel insights for involvement of mir-1273g-3p and mir-204-3p in Zika Virus pathology as these miRNAs are significantly upregulated by overexpression of Zika Virus E protein. mir-1273g-3p and mir-204-3p directly target important developmental genes i.e. PAX3 and NOTCH2 respectively. Hence the study is of great clinical significance as it provides several novel insights into understanding how Zika Virus causes pathology. This study is a significant contribution of Indian scientists to the field of Zika Virus research community globally. This is one of the first detailed mechanism based study on Zika Virus from any Indian laboratory **(Bhagat et al Cell Death and Differentiation 2018 Jul 26. doi: 10.1038/s41418-018-0163-y and Featured by NATURE India, doi:10.1038/nindia.2018.93 Published online 27 July 2018).**

3. **HIV-1 viral protein Tat induces neural stem cell arrest:** Neurological complications in opioid abusing Human Immunodeficiency Virus-1 (HIV-1) patients suggest enhanced neurodegeneration as compared to non-drug abusing HIV-1 infected population. Neural precursor cells (NPCs), the multipotent cells of the mammalian brain, are susceptible to HIV-1 infection and as opiates also perturb their growth kinetics, detailed mechanistic studies for their co-morbid exposure are highly warranted. Using a well characterized *in vitro* model of human fetal brain-derived neural precursor cells, we investigated alterations in NPC properties at both acute and chronic durations. Chronic morphine and Tat treatment attenuated proliferation in NPCs, with cells stalled at G1-phase of the cell cycle. Furthermore HIV-Tat and morphine exposure increased activation of extracellular signal-regulated kinase-1/2 (ERK1/2), enhanced levels of p53 and p21, and decreased cyclin D1 and Akt levels in NPCs. Regulated by ERK1/2 and p53, p21 was found to be indispensible for Tat and morphine mediated cell cycle arrest. Tat and morphine co-exposure also augmented Interferon-gamma (IFN-gamma) levels in NPCs, leading to altered signal transducer and activator of transcription (Stat) levels and derangement of Stat-SRY (sex determining region-Y)-box2 (Sox-2) pathway. Our study elaborates on the cellular and molecular machinery in NPCs and provides significant mechanistic details into HIV- drug abuse co-morbidity that may have far reaching clinical consequences both in pediatric as well as adult neuroAIDS **(Malik et al; J Neuroimmune Pharmacology 2014)**. Furthermore, using HIV-1 infection of human neural precursor cells, transfection of hNPCs with HIV-1 Tat expressing vectors we have demonstrated importance of a Stem cell fate determinant Tripartite Containing Motif 32 (TRIM32) in mediating the effects of HIV-1 on stemness of human neural stem/precursor cells. Our detailed investigations have also proven the fact that modulation of microRNA-155 affects the proliferation of hNPCs. We have further validated these findings with autopsied brain sections from HIV-1/AIDS patients **(Fatima et al; Cell Death and Differentiation 2016)**.

4. **Enhanced neurotoxicity of HIV-1 Tat and drugs of abuse in human neural cells:** Our laboratory continues to actively pursue detailed investigations into neurotoxic effects of HIV-1 transactivating (Tat) protein on brain cells derived from human neural precursor cells (hNPCs). Feeling the need for better understanding of the co-morbid effects of HIV-1 Tat and drugs of abuse, we extended our investigations to look for cellular and molecular pathways that may play pivotal role in enhanced toxicity of these two agents, which are common among the HIV/AIDS population. Using human neurons differentiated from human neural precursor cells and human neuroblastoma cells, our research group first observed that co-exposure of neuronal cells to HIV-1 B-Tat and morphine resulted in a dose dependent increase in mortality of cells, confirming clinical observations made earlier by other investigators in the field. Detailed investigations into the apoptotic pathways led to the observation that

morphine exacerbated the HIV-1 Tat neurotoxicity by disturbing the delicate balance of pro- and anti-apoptotic genes leading to triggering the caspase cascade. The co-exposure of morphine with HIV-1 Tat resulted in enhanced reactive oxygen species (ROS) production which was NADPH oxidase dependent as apocynin pretreatment prevented ROS generation and subsequent neurotoxicity. The co-. exposure of these agents led to increased perturbation of mitochondrial membrane potential leading to depolarized mitochondrial membranes that also contributes to neuronal damage.

The widespread use of highly active anti-retroviral therapy (HAART) has modified the nature of neurocognitive impairments categorized in HIV/AIDS patients. Complications arising due to resistance and toxicities of highly active anti-retroviral therapy in HIV/AIDS patients necessitated search for neuroprotectants that could be used as adjuncts to the current HAART to lower their doses. In this context several anti-inflammatory compounds, anti-oxidants, NMDAR antagonists and calcium channel blockers had been tried with limited success. Neurotrophins such as Brain Derived Neurotrophic Factor (BDNF), Nerve Growth Factor (NGF) and Glial cell line-derived Growth Factor (GDNF) also offer protection against various neurotoxins and are speculated for their possible use as neuroprotective-adjuncts for HAART. On these lines, we explored the possibility of Platelet Derived Growth Factor-BB (PDGF-BB) as a neuroprotectant against co-morbid effect of HIV proteins Tat and morphine. In addition to cellular and biochemical pathways, we probed into the signaling mechanisms involved in morphine-induced exacerbation of HIV-Tat induced toxicity in human neurons and human neuroblastoma cells. Our data suggests involvement of MAPK ERK1/2 and JNK pathways in mediating the toxicity induced by Tat and morphine. It was confirmed by use of pharmacological inhibitors specific to these pathways that abrogated the co-morbidity of Tat and morphine on neuronal cells. Furthermore, we observed significant neuroprotective effects of PDGF-BB at various levels that were mediated by PI3K/Akt pathways (**Malik et al; PloS One 2011)**.

5. **<u>Clade Specific Differences in Neurotoxicity of HIV-1 Tat induced neurotoxicity of Human neurons:</u>** Damage to CNS cells occurs by HIV-1 virion itself as well as by HIV-1 proteins such as gp120 and Tat. Monocyte Chemoattractant Protein–1 (MCP-1)/CCL2 is an important factor and has been implicated in recruiting activated and potentially neuropathogenic immune cells (monocytes) into brain. Level of MCP-1/CCL2 has been correlated with degree of neurological deficits observed in AIDS patients. The low incidence of HAD in India calls for detailed studies into the HIV-1 Tat induced damage to brain cells and to investigate if there are any clade specific differences in neuropathogenesis. We studied clade specific differences in neurotoxicity following exposure to HIV-1 Tat protein in human fetal CNS progenitor derived astrocytes and neurons, and observed that HIV-1 Tat C is less neurotoxic than Tat B. Damage to neurons by HIV virus is by direct and indirect pathway, hence primary cultures of human astrocytes were transfected with expression vectors for Tat B and Tat C and supernatants collected from these cultures were added to primary cultures of human neurons in various formats to assess neuronal damage and disturbances in mitochondrial membrane potential. We observed that supernatants collected from astrocytes transfected with Tat B expression vectors were more neurotoxic as compared to those from Tat C transfected astrocytes, similar to our observations with Tat B protein treatments in human neuronal cultures. Using site directed mutants of Tat B and C protein, we have determined important motifs in the 101 amino acid protein that are responsible for the HIV-1 clade specific differences in the neurotoxicity of human neurons. We believe that these findings would provide important details of the neurodegenerative mechanisms by two HIV-1 clades (B and C) that affect the majority of AIDS population around the world (**Mishra et al and Seth, Annals of Neurology 2008; this work has been cited more than 150 times)**.

6. **Modulation of Human neural stem cells properties by HIV-1 Tat:** Neural stem/precursor cells replenish ageing or damaged brain cells till late in life by forming new neurons or neurogenesis. Neurogenesis occurs throughout adulthood in the dentate gyrus of hippocampus, the center for learning and memory, unfortunately HIV-1 has been reported to infect these important areas of brain. In addition to its presence in astrocytes and microglial cells, HIV-1 virus is reported in areas of neurogenesis in autopsy brain sections from pediatric AIDS patients, suggesting that HIV-1 can infect and thrive in human neural stem/precursor cells. Several investigators, including our group have shown that HIV-1 can affect neural stem/precursor cell functions in *in vitro* models. We used a well characterized cell culture model of human neural precursor cells established by us at NBRC. These neural precursors grow as undifferentiated, highly proliferative, adhered monolayer cultures. We cultured these cells in presence or absence of HIV-1 transactivating protein Tat. HIV-1 Tat attenuated the growth, proliferation and differentiation capabilities of human fetal brain derived neural stem/precursor cells. The major steps involved in process of regulation of neural precursor cells and neurogenesis are - proliferation, migration, differentiation and axonal guidance, so we used a pathway specific cDNA microarray comprising 263 genes specifically related to these functions rather than a global gene expression profile. Total RNA isolated from hNPCs undergoing differentiation/neurogenesis in presence and absence of HIV-1 Tat for 5 days were used for cDNA microarray. At Day 5 post differentiation, Tat resulted in down regulation of genes crucial for regulation of cell differentiation (ASCL1, JAG1), cell signaling genes involved in neurogenesis (JAG1), cell proliferation (JAG1, S100 beta, PTN, SPOCK1), regulation for cell motility and migration (KAL1, MTSS1), cell adhesion (KAL1, MTSS1, SPOCK1, PCDHB-2,-5,- 14,-15 and -16, ROBO2), synaptic function (S100 beta, PCDHB-2,-5,-14 and -16), regulation of transcription (ASCL1, CHD6 and PBX1), cell cycle regulation (MTSS, PTN, KAL-1 and SPOCK1) and growth factors and cytokines (CSPG5, FGF13, JAG1, NRG1 and PTN). These findings suggest that HIV-1 Tat affects at various levels of neural stem cell functions which affects its stemness as well as the neurogenesis related genes **(Mishra et al; J Neurovirology 2010)**.

7. **Molecular Pathogenesis of CNS Viral Infections using Human Fetal CNS Progenitor cells:** Understanding the cellular events that occur during the course of viral infections as well as the molecular regulation of virus–cell interactions is mandatory for defining the disease and its treatment. The human neurotropic viruses JC Virus (JCV) and HIV-1 are linked together for study in several ways. Both viruses are neuroinvasive, involve white matter diseases in the brain, and infect neuroglial cells as well as cells of the immune system. Hence the goals and experimental approaches used in studying JCV and HIV-1 are somewhat similar. The lytic JCV infection of oligodendrocytes results in the fatal demyelinating disease called Progressive Multifocal Leukoencephalopathy (PML) **(Reviewed by Seth et al., J Neurovirology 2003)**, while HIV-1 infection of microglial cells and perivascular macrophages results in encephalitis with a wide range of cognitive impairments and motor dysfunction, formerly known as AIDS dementia complex (ADC). PML occurs almost exclusively in immune compromised individuals, and in fact once rare, the incidence of this disease has increased nearly 1000-fold, primarily due to AIDS epidemic. We use multipotential human CNS progenitor cells, obtained from human fetal brain. These progenitor cells are maintained in an undifferentiated state or are selectively differentiated into highly purified populations of neurons or astrocytes to study the molecular mechanisms regulating cell type specific susceptibility to JCV or HIV-1 infection. Our recent observations have important implications not only for HIV pathogenesis but show that the virus is capable of entering and replicating in a wide variety of cell types. We have explored this cell culture system to convincingly demonstrate that human CNS progenitor cells express CXCR4 and are capable

of replicating HIV. This replication can be modulated by TNF. These observations are confirmed in autopsy tissue from pediatric brains from HIV infected patients as well. Our findings are likely to open up an entirely new area of research in HIV neuropathogenesis and hence are of utmost importance (Lawrence, D., others and **Seth et al J Virology 2004**). I am currently studying the various co-receptors for HIV-1 infection in these cell types, chemokine profiles during the course of differentiation of astrocytes and neurons from progenitor cells, as well as certain expression kinetics of certain DNA binding proteins such as nuclear factor-1 (NF-1), that are important for JC infection. We have recently defined that human CNS progenitor derived astrocytes support JC infection that leads to a non-apoptotic cell death (**Seth et al., J Virology 2004**).

8. **Mechanism(s) of Viral Neurodegeneration:** Arboviruses are a group of emerging pathogens that are striking communities with increasing frequency. Venezuelan equine encephalitis virus (VEE), a neurovirulent virus, is considered as a threat for population around the globe as an endemic pathogen as well as in case of a bio-terrorism attack. Research involving VEE requires vaccination against the virus and use of Bio-Safety Level-3 (BL-3) procedures, I have been formally trained for the use of BL-3 labs. VEE is a well characterized arbovirus for studying the effects of viral encephalitides on the central nervous system (CNS). The end-result of virus infection and invasion into the brain parenchyma is an acute meningoencephalopathy. Though the gross neuropathogenesis is somewhat understood, very little is known about the contributions of various neural cell types towards the neuropathology and the early immune response that occurs in the CNS following VEE infection. I have studied the mechanism(s) for neurodegeneration and neuropathogenesis following infection with a neurovirulent VEE. Precisely, my research efforts are directed towards studying the involvement of astrocytes and microglial cells in mediating neuronal damage by VEE and the pattern of cell death following VEE infection. We have so far observed that both astrocytes and microglial cells are affected following VEE exposure and virus tends to infect both cell types, finally resulting in cell death via apoptotic pathway. Our observations suggest that growth patterns are different for the V3000, V3032 and V3010 strains of VEE, in microglial cultures.

9. **Anti-Malarials and Certain Environmental Pollutants Enhance Virus Replication in Mice:** Earlier studies from this lab demonstrated for the first time that chloroquine enhanced Semliki Forest virus, encephalomyocarditis virus, and HSV-1 replication in mice. Similar enhancement in virus replication was seen in mice injected with other anti-malarials. Infected animals treated with the antimalarials had significantly shorter mean survival times and higher viral titers than untreated mice. There was concomitantly a greater degree of encephalomyelitis in these groups on histology. Cytokine mRNA levels were also differentially upregulated in SFV-infected mice treated with antimalarials, viz. Interleukin (IL)-1Ra, IL-12(p40), IL-6, & IL-1 in the brain; interferon- inducing factor, IL-1 , IL-1 , & IL-12(p35) in the spleen; and IL-1Ra in the liver, when compared to untreated infected mice (**Seth, et al., Am J Tropical Med. & Hygiene, 1999**). These results may have enormous significance in that they suggest that the wide-spread use of these anti-malarials may enhance/activate HIV infections, resulting in rapid spread of AIDS in malaria endemic areas. Pyridostigmine bromide (PB), a neuroprotective agent against nerve gas, was used by US troops in the Gulf War. I was entrusted with the responsibility to study the effect of this agent on virus replication in animals to better understand the causes for Gulf War syndrome. It was observed that PB enhanced the morbidity and mortality of virus infected animals. Our findings indicate that the phenomenon may be dependent on the differential expression of mRNA of certain cytokines, mainly IL-1Ra. Furthermore, we observed that following acute exposures to cadmium and manganese, there was a significant enhancement in the expression of

inflammatory cytokines at mRNA levels in mice brain **(Seth et al., Biometals 2003)**.

10. **Picroliv - A Natural Product Protects Cells and Regulates the Gene Expression during Hypoxia/Reoxygenation:** Cellular adaptation to hypoxia involves regulation of specific genes such as vascular endothelial growth factor (VEGF), erythropoietin (EPO) and hypoxia inducible factor (HIF)-1. We evaluated the protective effect of picroliv (a purified iridoid glycoside fraction from roots of *Picrorhiza kurrooa* with hepatoprotective, anti-inflammatory and antioxidant properties) against hypoxic injury by examining lactate dehydrogenase (LDH) release in Hep 3B and Glioma cells. The expression of hypoxia regulated genes, VEGF and HIF-1 was studied in human umbilical vein endothelial cells (HUVEC), Hep 3B and Glioma cells. Picroliv reduced the cellular damage caused by hypoxia as revealed by a significant reduction in LDH release compared to untreated control. The expression of VEGF and HIF-1 subunits (HIF-1 and HIF-1) was enhanced by treatment with picroliv during normoxia and hypoxia in HUVEC and Hep 3B cells and on reoxygenation the expression of these genes was reduced significantly as revealed by mRNA analysis using RT-PCR. Simultaneous treatment with picroliv during hypoxia inhibited VEGF and HIF-1 expression in Glioma cells whereas the expression was not reduced by picroliv treatment during reoxygenation as evidenced by both RT-PCR and Northern hybridization. VEGF expression as revealed by immunofluorescence sutdies correlates well with the regulations observed in the mRNA expression. We have also examined the kinase activity of tyrosine phosphorylated proteins and protein kinase C (PKC) in Glioma cells treated with picroliv during hypoxia/reoxygenation. A selective inhibition of protein tyrosine kinase activity leading to tyrosine dephosphorylation of several proteins including 80 kd protein, and a reduction in PKC was seen in cells treated with picroliv and hypoxia. These findings suggest that picroliv may act as a protective agent against hypoxia/reoxygenation induced injuries, and the underlying mechanism may involve a novel signal transduction pathway (Gaddipati, Madhavan, **Seth et al., Molecular and Cellular Biochemistry, 1999**).

11. **Preconditioning with Picroliv Helps Against Ischemia Reperfusion Injury (IRI) and hemorrhage resuscitation injury:** The protective potential of picroliv 'preconditioning' in modifying IRI was studied in various animal models. The models of injury studied were partial global cerebral ischemia, hemorrhagic shock, liver IRI and whole body hypoxia in Sprague Dawley (SD) rats, and global cerebral ischemia in gerbils. In all the models, animals were pretreated with picroliv (12mg/kg) by oral gavage once daily for 7 days. In both the models of cerebral ischemia, ischemia was induced by bilateral carotid artery ligation, and cerebral blood flow (CBF) patterns were monitored during the reperfusion period using a Laserflo monitor. In the liver IRI model, ischemia was induced by ligating the hepatic pedicle, and liver histology was evaluated at the end of varying periods of ischemia (30 & 60 min), with or without reperfusion (15-45 min). Hemorrhagic shock in adult SD rats was induced by bleeding them (28 cc/kg body weight) under anesthesia over a 10 min period. After 75 min, the rats were resuscitated with Ringer's lactate and survival noted at 72 hours. Picroliv had a protective effect against the extent of hemorrhage resuscitation induced injury to liver tissue and the protective mechanism involved the AP-1 transcription factor **(Seth et al., Shock 2003)**. Picroliv fed rats consistently showed a higher CBF during reperfusion after partial cerebral ischemia, when compared to saline fed controls, suggesting that picroliv may have a vasoactive effect. When gerbils were subjected to complete global ischemia, CBF peaks were attained to a similar degree in both groups. However, the rate of rise of CBF after 8 min of ischemia was lower in animals treated with picroliv. After hepatic ischemia, the picroliv fed animals appeared to have a lesser degree of perivenular hypoxic changes in paraffin embedded hematoxylin-eosin stained sections. TUNEL staining revealed scattered apoptotic hepatocytes in livers

subjected to reperfusion. Considerably less apoptosis was seen in the tissues of animals treated with picroliv. Ribonuclease protection assay of RNAs extracted from the livers showed a reduction in the transcript of IL-1 in the livers of picroliv fed rats, suggesting a modulation of cytokine expression during reperfusion injury by picroliv (Singh, Mani, **Seth et al., 2000**, **Eur. J Pharmacol**). The hemorrhagic shock model was used to look for differences in survival among picroliv fed and control groups. Survival at 72 h was 73% in the picroliv group as against 50% in the controls, suggesting a favorable trend of picroliv 'preconditioning'. Picroliv may have potential for use as a 'preconditioning' agent in reducing ischemia reperfusion injury. The effect of picroliv pretreatment was also assessed in a renal ischemia reperfusion (IRI) model where it was observed that picroliv protected against renal IRI by preventing apoptosis, modulating expression of adhesion molecules and maintaining better antioxidant status (**Seth et al., Biochemical Pharmacology, 2000**).

12. **Cellular interactions during thrombosis & nitric oxide mediated modulation of neutrophil free radical generation:** Thrombosis, an undesirable form of hemostasis, is a major cause of morbidity and mortality. Platelet, neutrophils and free radicals have been implicated in "setting in" of thrombosis. During my Ph.D. my research was focused better understanding of the mechanisms involved in thrombosis, so as to help in designing of better drugs for improved management of this vascular disease. Three well known specific platelet activating factor (PAF) antagonists SR-27417, BN-52021 and BN-50739 were tested in different models of thrombosis in mouse, rat and cat. My studies suggested that PAF plays an important role in arterial thrombosis but not in venous thrombosis, there by advocating the use of anti-PAF agents in cases of arterial thrombosis only, as they may not be that helpful in treating venous thrombosis (**Seth et al., Thrombosis Res., 1994**). Further our studies also suggested that therapeutic efficacy of nitric oxide (NO) donors or NO generating compounds can be augmented several folds if they are administered along with free radical scavengers (Dikshit & **Seth et al., Thrombosis Res., 1993**). Role of neutrophil derived nitric oxide had been always a topic of debate till, our studies provided substantial evidence that NO is an important regulator of rat neutrophil functions. My experimental findings clearly demonstrated that NO not only scavenged the neutrophil derived free radicals but also inhibited generation of free radicals from neutrophils and thereby plays a cytoprotective role (**Seth et al., Blood, 1994**). Augmentation of NO release and reduction in the free radical generation from neutrophils following thrombosis have been my most recent contribution, in this area of research (**Seth et al., Thrombosis Res., 1997**).