"Prof. Mishra has established a new significant concept on selective E3 ubiquitin ligases that can serve as quality control first line of defense ameliorative measures against multifactorial proteostasis failures implicated in neurodegenerative diseases and imperfect aging."

Publications Citation Index:

- **Total Publications: 111**
- **Present: h-index: 31**
- **i10-index: 69**
- **Citations More than 15360**
- **Total Impact Factor of Publications: 667.42**
- **Average Impact Factor of each paper: 6.05**

A list of ten important publications:

No.	Paper details: All are Research Articles (*Corresponding Author-Work done in India)
1.	Vibhuti Joshi, Ribhav Mishra, Arun Upadhyay, Ayeman Amanullah, Krishna Mohan Poluri, Sarika Singh, Amit Kumar and **Amit Mishra*** (2019) Polyphenolic Flavonoid (Myricetin) Upregulated Proteasomal Degradation Mechanisms: Eliminates Neurodegenerative Proteins Aggregation. **Corresponding Author: Work from India* **Journal of Cellular Physiology** DOI: 10.1002/jcp.28695 Impact Factor: 6.51 *Key finding/Highlights of Work: This study signifies that Myricetin, a flavonoid, can eliminate various abnormal proteins from the cellular environment via modulating endogenous levels of Hsp70 chaperone and quality control (QC)-E3 ubiquitin ligase E6-AP. Taken together these findings suggested that new mechanistic and therapeutic insights based on small molecules mediated regulation of disturbed protein quality control mechanism, which may result in the maintenance of the state of proteostasis.*
2.	Ayeman Amanullah, Ribhav Mishra, Arun Upadhyay, P Purushotham Reddy, Ranabir Das and **Amit Mishra*** (2017) Indomethacin Elicits Proteasomal Dysfunctions Develops Apoptosis Through Mitochondrial Abnormalities. **Corresponding Author: Work from India* **Journal of Cellular Physiology** DOI: 10.1002/jcp.26081 Impact Factor: 6.51 *Key finding/Highlights of Work: We show that indomethacin, a well-known NSAID, induces proteasomal dysfunction that results in accumulation of unwanted proteins, mitochondrial abnormalities, and successively stimulate apoptosis in cells.Our results demonstrate how indomethacin affects normal proteasomal functions and induces mitochondrial apoptosis in cells. These findings also improve our current understanding of how NSAIDs can exhibit crucial anti-proliferative effects in cells. In near future, our findings may suggest a new possible strategy for the development of specific proteasome inhibitors in conjunction with other chemo-preventive anticancer agents.*
3.	Deepak Chhangani, Fumita Endo, Ayeman Amanullah, Arun Upadhyay, Seiji Watanabe, Ribhav Mishra, Koji Yamanaka* and **Amit Mishra*** (2016) Mahogunin ring finger 1 confers cytoprotection against mutant SOD1 aggresomes and is defective in an ALS mouse model. **Corresponding Author: Work from India* **Neurobiology of Disease** DOI: 10.1016/j.nbd.2015.11.017 Impact Factor: 7.04 *Key finding/Highlights of Work: Here, we found that the Mahogunin ring finger-1 (MGRN1) E3 ubiquitin ligase, which catalyzes mono-ubiquitination to the substrate, was dysregulated in the cellular and mouse models of ALS and that it preferentially interacted with various mutant forms of SOD1. Intriguingly, the motor neurons of presymptomatic ALS mice have diminished MGRN1 cytoplasmic distribution. MGRN1 was partially recruited to mutant SOD1 inclusions where they were positive for p62 and Lamp2. Furthermore, the present study identifies the MGRN1-mediated protein QC mechanism as a novel therapeutic target in neurodegenerative diseases.*

4.	Deepak Chhangani, Nobuyuki Nukina, Masaru Kurosawa, Ayeman Amanullah, Vibhuti Joshi, Arun Upadhyay and **Amit Mishra*** (2014) Mahogunin ring finger 1 Suppresses Misfolded Polyglutamine Aggregation and Cytotoxicity. **Corresponding Author: Work from India* **BBA Molecular Basis of Disease** DOI: 10.1016/j.bbadis.2014.04.014 Impact Factor: 6.63 *Key finding/Highlights of Work: We demonstrated that Mahogunin 21 ring finger 1 E3 ubiquitin protein ligase is depleted in cells that express expanded-polyglutamine proteins, we demonstrate that the partial depletion of MGRN1 increases the rate of aggregate formation and cell death, whereas the overexpression of MGRN1 reduces the frequency of aggregate formation and provides cytoprotection against polyglutamine-induced proteotoxicity. These observations suggest that stimulating the activity of MGRN1 ubiquitin ligase might be a potential therapeutic target to eliminate the cytotoxic threat in polyglutamine diseases.*
5.	**Amit Mishra***, Megha Maheshwari; Deepak Chhangani, Noriko Fujimori Tonou, Fumito Endo, Ajay P Joshi, Nihar R Jana and Koji Yamanaka* (2013) E6-AP association promotes SOD1 aggesomes degradation and suppresses toxicity. **Corresponding Author: Work from India* **Neurobiology of Aging** DOI: 10.1016/j.neurobiolaging.2012.08.016 Impact Factor: 5.13 *Key finding/Highlights of Work: Here for the first time, we report that E6-AP, a homologous to E6-AP C terminus-type E3 ubiquitin ligase depleted in ALS mouse models before neurodegeneration. We show that the overexpression of E6-AP suppresses the aggregation and cell death mediated by mutated SOD1 proteins and cellular protective effect is more prominent when E6-AP is overexpressed along with Hsp70. These data suggest that enhancing the activity of E6-AP ubiquitin ligase might be a viable therapeutic strategy to eliminate mutant SOD1-mediated toxicity in ALS.*
6.	Vibhuti Joshi, Arun Upadhyay, Deepak Chhangani, Rajesh N Sharan **Amit Mishra*** (2018) Gp78 Involvement In Cellular Proliferation: Can Act As A Promising Modulator For Cell Cycle Regulatory Proteins? **Corresponding Author: Work from India* **Journal of Cellular Physiology** DOI: 10.1002/jcp.26618 Impact Factor: 6.51 *Key finding/Highlights of Work: We demonstrate that cell surface glycoprotein Gp78, a putative E3 ubiquitin ligase, is involved in the stabilization of intracellular steady-state levels of p27. Transient overexpression of Gp78 increases the accumulation of p27 in cells in the form of massive inclusions like structures, which could be due to its cumulative increased stability in cells. We have also monitored how under stress condition, E3 ubiquitin ligase Gp78 regulates endogenous levels of p27 in cells. ER stress treatment generates a marginal increase in Gp78 endogenous levels, and this elevation effect was prominent for intracellular accumulation of p27 in cells. Taken together, our current findings suggest a valuable multifactorial regulatory mechanism and linkage of p27 with UPS pathway.*
7.	Arun Upadhyay, Ayeman Amanullah, Deepak Chhangani, Vibhuti Joshi, Ribhav Mishra and **Amit Mishra*** (2016) Ibuprofen Induces Mitochondrial-Mediated Apoptosis Through Proteasomal Dysfunction. **Corresponding Author: Work from India* **Molecular Neurobiology** DOI: 10.1007/s12035-015-9603-6 Impact Factor F: 5.68 *Key finding/Highlights of Work: In our present study, we have observed that ibuprofen reduces proteasome activity, enhances the aggregation of ubiquitylated abnormal proteins, and also elevates the accumulation of crucial proteasome substrates. Ibuprofen treatment causes mitochondrial abnormalities and releases cytochrome c into cytosol. Perhaps, the more detailed study is needed in the future to elucidate the molecular mechanisms of NSAIDs that can induce apoptosis without adverse effects and produce effective anti-tumor effects and consequently help in neurodegeneration and ageing.*

8.	Ayeman Amanullah, Arun Upadhyay, Deepak Chhangani, Vibhuti Joshi, Ribhav Mishra, Koji Yamanaka and **Amit Mishra*** (2017) Proteasomal Dysfunction Induced by Diclofenac Engenders Apoptosis Through Mitochondrial Pathway. **Corresponding Author: Work from India* **Journal of Cellular Biochemistry** DOI: 10.1002/jcb.25666 Impact Factor: 4.48 *Key finding/Highlights of Work: We have observed that diclofenac treatment induces proteasome malfunction and promotes accumulation of different critical proteasome substrates, including few pro-apoptotic proteins in cells. Exposure of diclofenac consequently elevates aggregation of various ubiquitylated misfolded proteins. This study suggests possible beneficial insights of NSAIDs-induced apoptosis that may improve our existing knowledge in anti-proliferative interspecific strategies development.*
9.	Arun Upadhyay, Ayeman Amanullah, Ribhav Mishra, Amit Kumar and **Amit Mishra*** (2018) Lanosterol Suppresses the Aggregation and Cytotoxicity of Misfolded Proteins Linked with Neurodegenerative Diseases. **Corresponding Author: Work from India* **Molecular Neurobiology** DOI: 10.1007/s12035-016-0377-2 Impact Factor: 5.68 *Key finding/Highlights of Work: Here, we demonstrate that treatment of lanosterol diminishes aberrant proteotoxic aggregation and mitigates their cytotoxicity via induced expression of co-chaperone CHIP and elevated autophagy. Finally, we observed that lanosterol mitigates cytotoxicity in cells, mediated by different stress-inducing agents. Taken together, our present results suggest that upregulation of cellular molecular chaperones, primarily using small molecules, can probably offer an efficient therapeutic approach in the future against misfolding of different disease-causing proteins and neurodegenerative disorders.*
10.	Ribhav Mishra, Ayeman Amanullah, Arun Upadhyay, Rohan Dhiman, Ankur Rakesh Dubey, Sarika Singh, Amit Prasad and **Amit Mishra*** (2020) Ubiquitin Ligase LRSAM1 Suppresses Neurodegenerative Diseases Linked Aberrant Proteins Induced Cell Death. **Corresponding Author: Work from India* **International Journal of Biochemistry & Cell Biology** DOI: 10.1016/j.biocel.2020.105697 Impact Factor: 5.65 *Key finding/Highlights of Work: Our study for the first time demonstrates that E3 ubiquitin Ligase LRSAM1 is a really interesting new gene (RING) class protein which suppresses the accumulation of misfolded protein aggregates and also alleviates their deleterious cytotoxic effects. This study will also allow us to better comprehend the problem of proteinopathies linked with aberrant protein accumulation and open new possibilities to better elucidate the molecular mechanisms involved in the pathologies of neurodegeneration and aging.*