Dr. Mateen A. Khan obtained his PhD degree in the field of Biotechnology from the Aligarh Muslim University, India, and later completed a postdoctoral fellowship in the Department of Biochemistry and Chemistry, Hunter College of the City University of New York, and School of Medicine, Stanford University, California, USA. Dr. Khan has published over 30 peer review journal articles, book and book chapter, and has been invited/attended over 30 national and international conferences. Dr. Khan is an active and productive member of the scientific community, serving as a reviewer for multiple scholarly journals including *Biochimica et Biophysica Acta-BBA, PLOS ONE, Journal of plant Pathology, Current Chemical Biology, Journal of Biochemistry and Modern Application, and the International Journal of Biochemistry, Biophysics & Molecular Biology*. He is the recipient of Faculty Award for Research Excellent 2022, Teaching Award 2022, and Outstanding Research Award 2019 by the Alfaisal University. He has been identified as one of the best young scientists at the City University of New York by Gene Centre foundation and his name has been published by News Review. Dr. Khan's research achievements have been identified by the faculty of 1000 biology scientist. He has supervised or mentored over 20 undergraduate and graduate students and has served on over 20 committees.

Dr. Khan research interests are directed toward understanding the mechanism of gene regulation of iron metabolism and how it impacts on disease process. Alzheimer disease is a neurodegenerative process that is the leading cause of death worldwide for people over the age of 65. Overexpression of Alzheimer amyloid precursor protein (APP) has been linked to Alzheimer's disease (AD). The fact that stem-loop structure of APP IRE mRNA has been linked to high level of iron and iron regulatory protein (IRP1) makes this an important model system for iron disorders research and is a potential therapeutic target. Protein aggregation and misfolding is directly associated with the neurodegenerative diseases. IRE-mRNA signaling pathway has been implicated in the modulation of amyloid, which is important to neurodegeneration in Alzheimer's disease. Therefore, the identification of small molecular APP mRNA chemical inhibitors to reduce amyloid protein aggregation can have therapeutic significance to Alzheimer's disease. IRE RNA inhibitors can decrease ferritin and transferrin receptor expression to alleviate the excess iron accumulation in AD brains cells. Furthermore, therapeutic IRE RNA inhibitors that down regulate APP protein translation and inhibit protein aggregation can promote neuronal survival. IRPs are key controllers of iron homeostasis and post-transcriptionally regulate expression of the major iron regulated genes. Despite considerable research efforts including identification of APP IRE mRNAs binding domain, binding proteins, and the specific mechanism through which IREs recruitment the IRP, initiation factors and ribosome, quantitative binding and structural studies are still unknown. The lack of these data is an important problem because the binding stability and highly ordered structure of APP/IRE complex plays a crucial role to find the start codon AUG for translation initiation. Therefore, it will be significant to determine the extent to which the IRP form complex with APP IRE, and the stability of the complex will be an important control point for the overall rate of protein synthesis. Lack of this knowledge makes it impossible to discriminate among proposed cellular regulatory mechanism between repressor IRP protein binding to IRE RNA in the gene regulation. Our major focus over the past several years has been involved the underlying mechanism based translational research of the iron mis-regulation and Alzheimer's disease by dissecting the signaling pathway which plays critical role in aggregation, misfolding of amyloid protein in brain as observed in neurodegenerative diseases like Alzheimer's and Parkinson's. These studies will provide the understanding of the mechanism of amyloid aggregation and provide new targets for therapeutic intervention in Alzheimer's disease.