Dr. Mohammad Mamunur Rashid.

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Appointments

March 2020 –February 2021	Postdoctoral Fellow, Dept. of Medicine, Dentistry and Biotechnology, Non coding RNA and Cancer Laboratory, Chieti University, Chieti, Italy
June 2015 –February 2020	Faculty, Dept. of Biochemistry, Primeasia University, Dhaka, Bangladesh
October 2007-August 2008	Officer, Quality Management and System Division The Acme Laboratories Limited, Dhaka, Bangladesh

Teaching Experience/Course Instructor

1. Introduction to Biochemistry 2. Human Physiology 3. Pharmaceutical Chemistry 4. Metabolism 5. Molecular Biology 6. Molecular Genetics 7. Biotechnology and Genetic Engineering 8. Endocrinology 9. Immunology 10. Cell Biology

Education

May 2011 – April 2015	Ph.D. in Molecular Medicine, University of Rome, La Sapienza, Italy
Nov. 2010 – April 2011	Research Intern, Islets Cell Exocytosis Lab, Lund University, Sweden.
Sept. 2008 – Oct. 2010	M.Sc. in Molecular Biology, Lund University, Sweden.
July 2004 – Aug. 2008	B.Sc. in Biochemistry and Molecular Biology, JU, Bangladesh

Publications

1. Russo MA, Sansone L, Polletta L, Runci A, **Rashid MM**, De Santis E, Vernucci E, Carnevale I, Tafani M[1] "Sirtuins and Resveratrol-Derived Compounds: A Model for Understanding the Beneficial Effects of the Mediterranean Diet". Endocr Metab Immune Disord Drug Targets. 2014 Jul 8.
2. **Rashid MM[1],** Runci A[1], Polletta L[1], Carnevale I[1], Morgante E[1], Foglio E[1], Arcangeli T[1], Sansone L[2], Russo MA[3], Tafani M[4] "Muscle LIM Protein/CSRP3: a mechanosensor with the role in autophagy." Cell Death Discov. 2015 Aug 3. doi: 10.1038/cddiscovery.2015.14
3. **Rashid MM**[1], Runci A[1], Russo MA[2], Tafani M[1,2] "Muscle Lim Protein (MLP)/CSRP3 at the crossroad between mechanotransduction and autophagy" Cell Death Dis. 2015 Oct 22. doi: 10.1038/cddis.2015.308.

4. **Mohammad Mamunur Rashid[1]**, Wondossen Sime[2], Anita Sjolander[1, 2] "Alteration of PPAR gamma expression in colorectal cancer" Lambert Publishing Group. 2014 April 2.

Conference Participation

1. **The Impact of Pro-inflammatory Mediators on Colorectal Cancer Stem Cells.** Kishan Bellamkonda[1], **Rashid Mohammad Mamunur[1]** , Wondossen Sime[2] and Anita Sjölander[1,2] Department of Laboratory Medicine, Division of Cell and Experimental Pathology, Clinical Research Centre, Skåne Univ. Hospital, Malmö, Lund University, Sweden.

2. **Factor based therapy for myocardial regeneration.** Eleonorna Foglio[1,] Alessandra Runci[1], **Mohammad Mamunur Rashid[1,]** Antonia Germani[2,] Federica Limana[2], Matteo Antonio Russo[1,2] [1]Dept. Experimental Medicine, Sapienza University, Rome, Italy; [2]Dept. Cellular and Molecular Pathology, IRCCS San Raffaele Pisana, Rome, Italy.

Research Experience

PhD study
Title: CSRP3 or MLP, a muscle LIM-domain protein, induces autophagy in skeletal muscle.
Dept. of Experimental Medicine, University of Rome, La Sapienza. Rome, Italy.

Summary of the Project:

Muscle LIM protein (MLP) is a microtubule-associated protein expressed in cardiac and muscle tissues that belongs to the cysteine-rich protein (CSRP/CRP) family. MLP has a central role during muscle development and for architectural maintenance of muscle cells. However, muscle cells rely on autophagy during differentiation and for structural maintenance. To study the role of MLP in autophagy, we have used C2C12 mouse myoblasts silenced or overexpressing MLP. Our results show that MLP contributes to the correct autophagosome formation and flux by interacting with LC3 as demonstrated by co-immunoprecipitation and PLA assay. In fact, MLP silencing results in decreased LC3-II staining and absent degradation of long-lived proteins. Moreover, MLP silencing impaired myoblasts differentiation as measured by decreased expression of MyoD1, MyoG1 and myosin heavy chain. Ultrastructural analysis revealed the presence of large empty autophagosomes in myoblasts and multimembranous structures in myotubes from MLP-silenced clones. Impaired autophagy in MLP-silenced cells resulted in increased susceptibility to apoptotic cell death. In fact, treatment of MLP-silenced C2C12 myoblasts and myotubes with staurosporine resulted in increased caspase-3 and PARP cleavage as well as increased percentage of cell death. In conclusion, we propose that MLP regulates autophagy during muscle cell differentiation or maintenance through a mechanism involving MLP/LC3-II interaction and correct autophagosome formation.

Post-graduate Project

Title- Differential expression of miRNAs in glucose-responsive and glucose unresponsive clonal beta cells. Department of Clinical Sciences, Lund University Diabetes Centre (LUDC), Malmo, Sweden.

Summary of the Project:

MicroRNAs are small RNAs that have been implicated in pancreatic beta cell functions. We aim to study the differences in the glucose regulation of four different miRNAs, miR-375, miR 124, miR-212 and miR132 in a glucose-responsive and glucose-unresponsive clonal beta cell lines. Two INS1-derived clonal beta cells, glucose-responsive INS1- 832/13 (Robust insulin secretor) and glucose-unresponsive INS1-832/2 were used. The cells were stimulated in varying glucose concentrations and insulin secretion was determined by RIA. The expression of all miRNAs from the different conditions was quantified using stem-loop RT-qPCR. Interestingly, miR-212 has also been found to be upregulated in the different diabetes disease models. although we found only significant difference in the miR-212 expression among the miRNAs assayed between the two cell lines, it is most likely that there are other miRNAs that are differentially-regulated. Such imbalance in the miRNA network in the INS1-832/2 cells may contribute to the impaired glucose stimulated insulin secretion (GSIS) characteristics.

MS Thesis

Title- Alteration of PPARγ expression in colorectal cancer patients and the effect of rosiglitazone in cell proliferation and survival of intestinal epithelial cell lines. Department of Laboratory Medicine, Lund University, Malmo, Sweden.

Summary of the Project:

Peroxisome proliferators-activated receptor-γ (PPARγ) is a member of the nuclear hormone superfamily, which is highly expressed in adipose tissues and intestinal epithelial cells. In my study, I examined the transcriptional levels of PPARγ in an invasive colon cancer cell line (SW480) and in normal epithelial cell-line (INT 407) where SW-480 exhibited lower levels of PPARγ mRNA compared to INT-407. Examination of human tissue array prepared from tumor and normal materials of colon cancer patients revealed that the expression of PPARγ was also higher in normal compared to colon cancer tissue samples. Correlation analysis of reduced PPARγ expression in cancer tissue has shown significant correlation with cell survival factor Bclxl whereas higher PPARγ expression in normal tissue showed significant correlation with pro-apoptotic protein Bax. That means low levels of PPARγ in cancer tissue might be associated with poor prognosis whereas higher levels of PPARγ expression in normal colon tissue might be associated with better prognosis. In short, the significant decrease in the level of PPARγ expression might be one possible risk factor for carcinogenesis. Overall, the present data suggests that PPARγ might act as tumor suppressor gene and its agonist might function as a promising drug for the treatment of colon cancer cells.

Laboratory skills

- Molecular Biology: plasmid purification, restriction digestion, molecular cloning, PCR, RT-qPCR, mutagenesis, protein purification, mammalian stable cell line construction, SiRNA transfection, RNA isolation. RNA Purification, Agarose Gel Electrophoresis

- Cell Biology: Immunohistochemistry, fluorescence microscopy, maintenance and culturing of mammalian cells, immunostaining of cells, tumor development in mouse, cell migration and invasion assays.

- Biochemistry: Western blot, ELISA, SDS-PAGE, Column Chromatography, MALDI- TOF.

Fellowship and Scholarships

Oct. 2011 – Oct. 2014	International PhD Fellowship for Non-italian Citizens funded By Italian Ministry of Education.
Dec. 2010 – May. 2011	Post-graduate Research Scholarship, Funded by Islets Exocytosis Group of Lund University, Sweden.
Mar. 2009 – Nov. 2009	MS Thesis Stipend Funded by Guest Scholarship, Swedish Research Council.
July 2008 – Feb. 2008	Undergraduate Merit Scholarship, Funded by University Grant Commission of Bangladesh for Jahangirnagar University, Bangladesh.

Language Skills

Fluent in English in writing, speaking, listening and reading. IELTS-7.5

Basic in Italian Language

Computer Skills

MS word, excel and Powerpoint, Photoshop, Statistical Analysis, SPSS, Internet.

Supervision Experience

Supervised six undergrad students in their undergrad project in different semesters. In addition to these, I have also worked as a batch advisor in different semesters.

Strengths

- Strong desires for leading an honest life with dignity.
- Dynamic and Self-motivated.
- Capable of adapting in any type of environment.
- Ambitious and Target oriented.
- Love to take challenging career.
- Like to work in a team.

Referee 1:

Wondossen Sime, PhD.
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Referee 2:

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Referee 3:

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Referee 4:

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