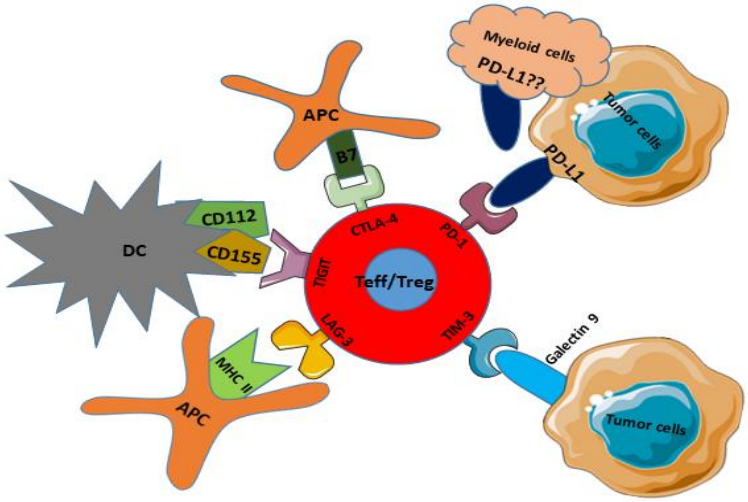
Summer Research Program – Projects

Project # 1

Title: Effect of inducing different immune checkpoints on behavior of cancer cells

Description: The focus of our research is to investigate the mechanistic role of multiple immune checkpoints in breast and colorectal cancers. We have recently reported that the expression of multiple immune checkpoints was higher in the circulation and TME of colorectal and breast cancer patients. In this project, we aim to investigate the mechanistic role of immune checkpoint after inducing their expression on breast and colorectal cancer cell lines. Here, we will transfect breast and colorectal cancer cell lines with GFP/Myc tagged pCMV constructs of immune checkpoints including PD-1, PD-L1, CTLA-4, TIM-3 and LAG-3 separately or in combination and study their effect on tumor cell proliferation, apoptosis, DNA damage and metastasis. To address this aim, we will utilize various laboratory techniques, such as cell culture, transfection, flow cytometry, quantitative real-time PCR, Co-immunoprecipitation and western blotting.

Mentors: Dr. Eyad Elkord, Principal Investigator. Email: eelkord@hbku.edu.qa, Dr. Varun Sasidharan Nair, Postdoc. Email: vsnair@hbku.edu.qa

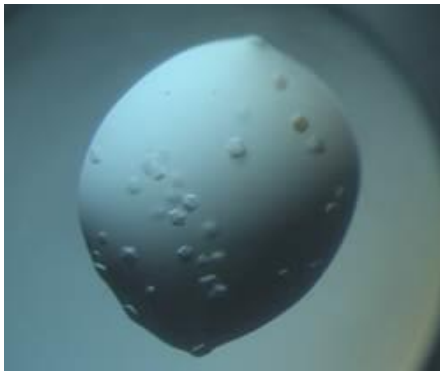


Project # 2

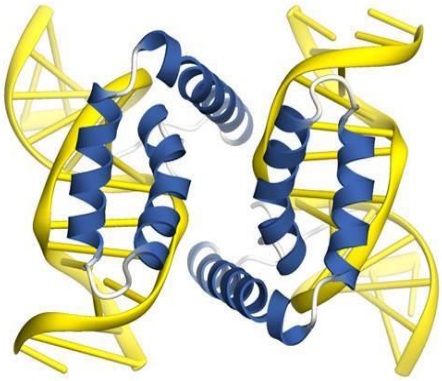
Title: Mechanisms in pancreatic development and breast cancer stemness mitigated by Sox2

Description: This proposal seeks to find detailed molecular mechanisms involved in two seemingly disparate areas of pancreatic development and breast cancer stemness. The common thread is Sox2, a transcription factor, implicated in pluripotency and other developmental roles but also shown to be a factor in various cancers. Normal Sox2 maintains stem cells in their pluripotent state but a double mutant was shown to have converted Sox2 into Sox17, an endodermal TF, shown to be critical for pancreatic β cell development. Wild type Sox2 has also been shown to be an important marker for many different cancers. This proposal seeks to shed light on the mechanisms driving pancreatic development and at the same time find mechanism involved in stemness of breast cancer. The proposal will use an integrative approach and make use of biochemistry and structural biology of in vitro systems and combine this with cell biology data from stem cell diabetes and cancer groups within QBRI.

Mentors: Dr. Prasanna R. Kolatkar, Senior Scientist. Email: pkolatkar@hbku.edu.qa, Dr. Zeyaul Islam, Postdoc. Email: zislam@hbku.edu.qa



Sox crystals.



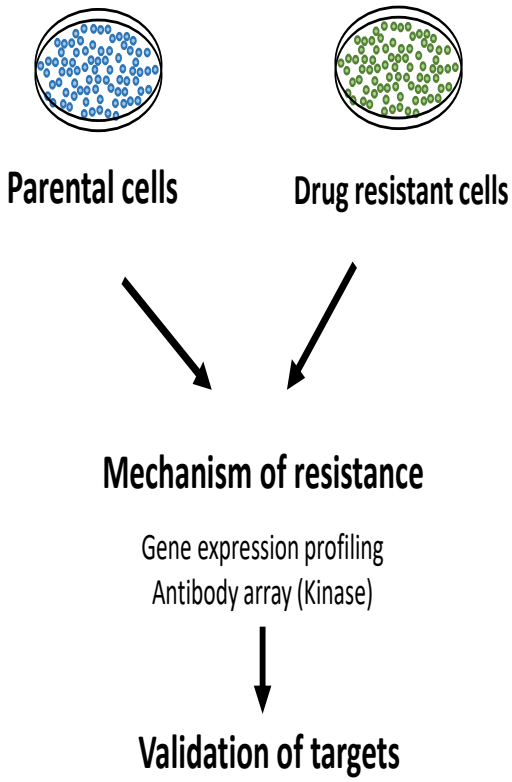
Structure of Sox17 bound to DNA

Project # 3

Title: Mechanisms of CDK4/6 inhibitor-resistance in breast cancer cells

Description: Although recent advance in early diagnosis and treatment has significantly improved survival rate of cancer patients, drug resistance is the major obstacle of successful treatment. Therefore, we will study the molecular mechanism underlying drug resistance for seeking the alternative strategies to overcome it. In this project, we will primarily focus on CDK4/6 inhibitors that are currently used for treatment of breast cancer. We will establish resistant cell lines and investigate their gene expression profiles and kinase activation/expression to determine the pathways altered in the resistant cells. Then, we will further study the effects of the inhibition of the altered pathways in the resistant cells on the CDK4/6 sensitivity.

Mentor: Dr. Hirohito Yamaguchi, Senior Scientist. Email: hyamaguchi@hbku.edu.qa

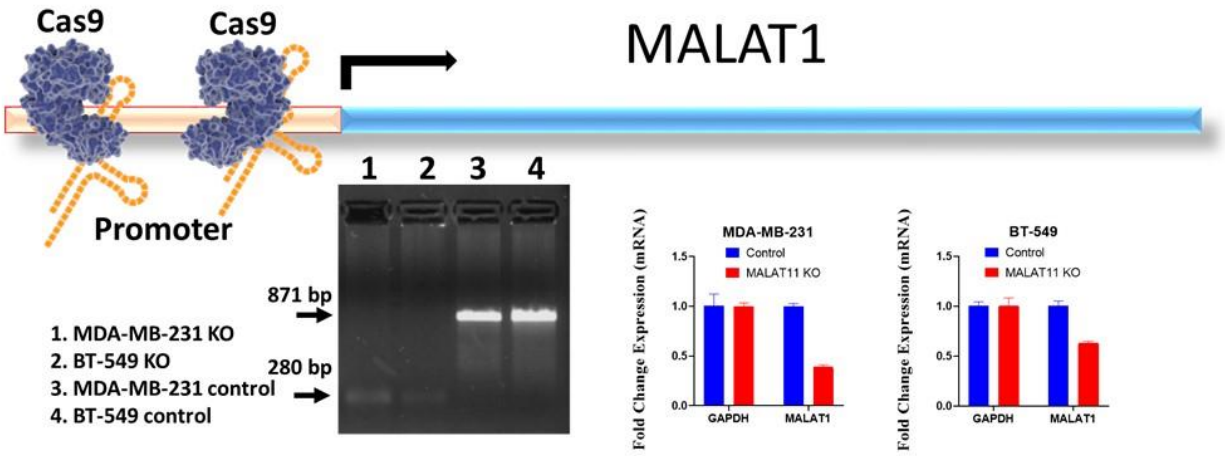


Project # 4

Title: Functional Study of Noncoding RNAs in Breast Cancer Using CRISPR/Cas9 Genome Editing

Description: Breast cancer is the second most common cancer type around the world underscoring a need for better understanding of breast cancer and the development of novel dlgnostic and therapeutic strategies. While only 2% of the human genome encodes for protein, almost 93% of the entire genome could actively be transcribed, suggesting a role for various noncoding RNAs in regulating various physiological and pathological processes. In current study, we aim to use CRISPR-Cas9 genome editing technology to understand the role of various noncoding RNAs in breast cancer and their potential utilization as disease biomarkers and therapeutic targets.

Mentor: Dr. Nehad Alajez, Senior Scientist. Email: nalajez@hbku.edu.qa



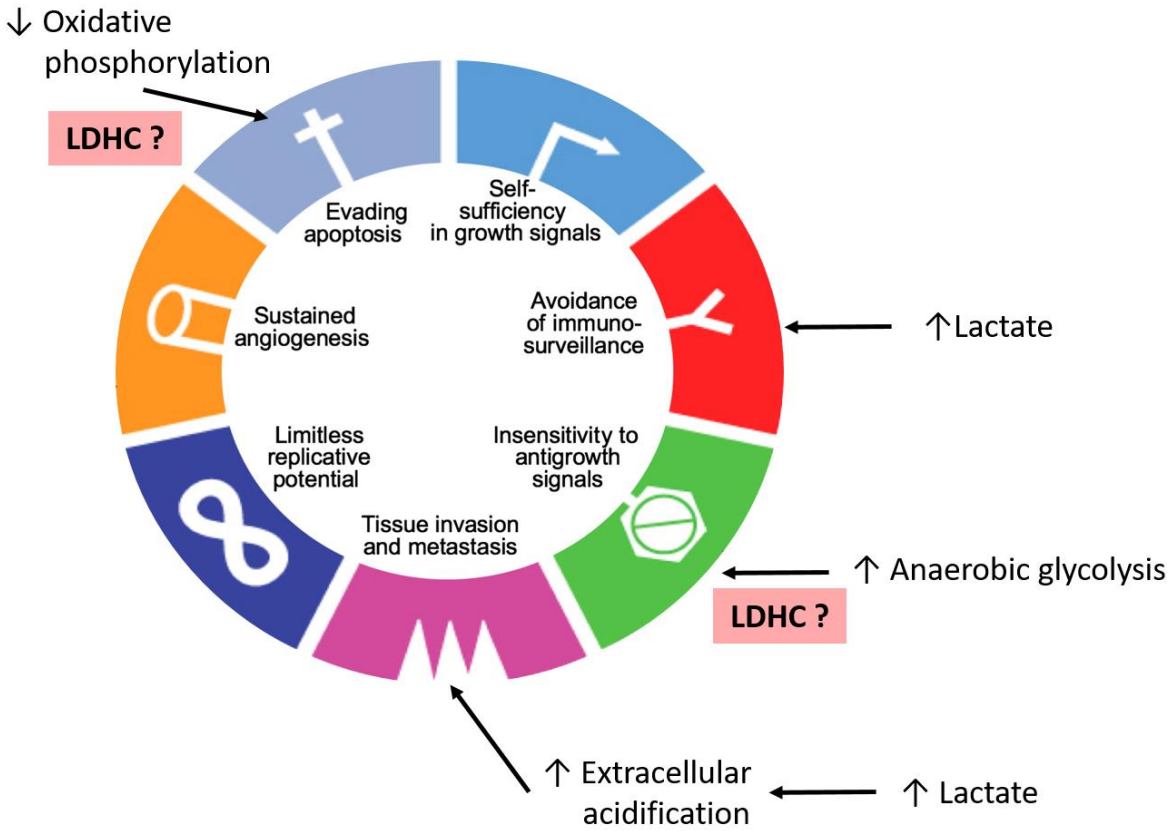
Project # 5

Title: Targeting cancer metabolism through Lactate Dehydrogenase C to improve breast cancer treatment response.

Description: Metabolic reprogramming is a common feature of cancer with tumors undergoing a metabolic switch from oxidative phosphorylation to aerobic glycolysis (Warburg effect), resulting in metabolic lactic acidosis of the tumor microenvironment. The associated extracellular acidification and intracellular alkalization have been reported to mediate resistance to anti-cancer drugs and inhibition of the anti-tumor immune response.
In this project, we will investigate the role of Lactate dehydrogenase C (LDHC) in lactic acidosis, mediated by monocarboxylic acid transporters (MCTs), and determine the effect of targeting this axis on treatment response to a DNA repair inhibiting agent in triple negative breast cancer. To this end, multiple techniques will be applied including cell culture, silencing, qRT-PCR, western blotting, flow cytometry, Seahorse and clonogenic assays.

Mentors: Dr. Julie Decock, Scientist. Email: jdecock@hbku.edu.qa, Dr. Adviti Naik Jana, Postdoc. Email: anaikijana@hbku.edu.qa

Lactic acidosis, treatment resistance and inhibition of anti-tumor immunity



Project # 6

Title: Proteomics of methylglyoxal-induced cytotoxicity in cancer chemotherapy

Description: Methylglyoxal (MG) is a reactive metabolite formed spontaneously in glycolysis. At high concentrations it induces apoptosis and cytotoxicity. MG is mainly metabolized by glyoxalase 1 (Glo1) of the glyoxalase system in the cytosol of all cells. Overexpression of Glo1 is associated with multidrug resistance in cancer chemotherapy, suggesting that anticancer drugs may induce cytotoxicity and antitumor effect, in part, by increase of cellular MG concentration to toxic levels. Herein we will study modification of proteins by MG and changes in protein abundance in extracts of human breast cancer MCF7 cell line to identify pathways of MG-induced tumor cell cytotoxicity.

Mentors: Dr. Paul J Thornalley, Scientific Director. Email: pthornalley@hbku.edu.qa, Dr. Patrick Wijten, Postdoc. Email: PWijten@hbku.edu.qa

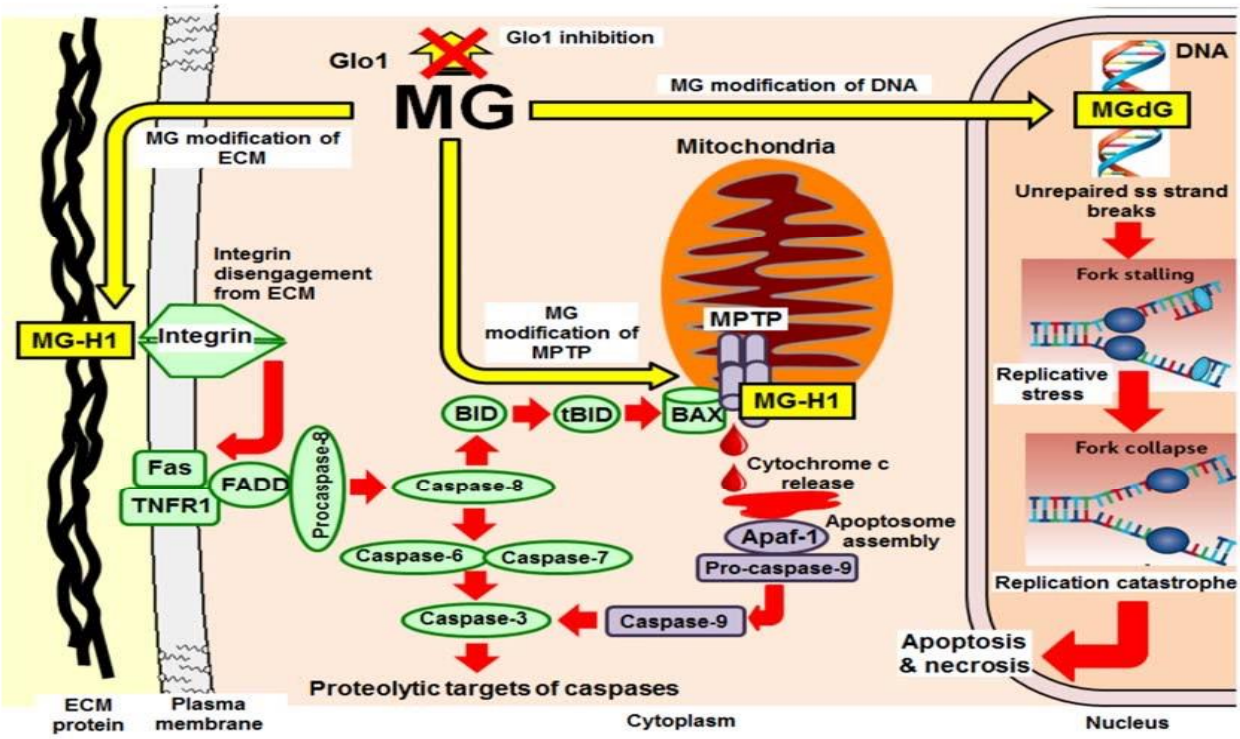


Figure 1. The mechanism of cytotoxicity of cell permeable Glo1 inhibitors through cellular accumulation of methylglyoxal accumulation in tumor cells. Yellow filled arrows, key reactions of MG. Red arrows: cell death response. Green elements: anoikis; purple elements, mitochondrial apoptotic pathway. See Background reading.

Project # 7

Title: Service Availability & Quality of Life for Grown-Up Individuals with Autism Spectrum Disorder in Qatar

Description: Quality of Life (QoL) is a critical measure of intervention outcome for individuals with cognitive and/or physical challenges. For those affected with Autism Spectrum Disorders (ASD), research continues to investigate treatment outcomes to guide service providers about the best intervention standards available. Limited studies have examined the QoL of individuals with ASD years after intervention. Previous research in the region investigated the QoL of the families of children with ASD, nonetheless, no study to date has explored the QoL in relation to grown-up individuals (16 years and above) with ASD within the Arab region. This study aims to explore the number of adolescent and adult cases with ASD, service availability such as vocational and pre-employment programs, QoL, and creating a registry for those cases within the context of Qatar. The findings of this study will provide information for policymakers on the state of service provision and its effects on the QoL of grown-up individuals with ASD living in Qatar.

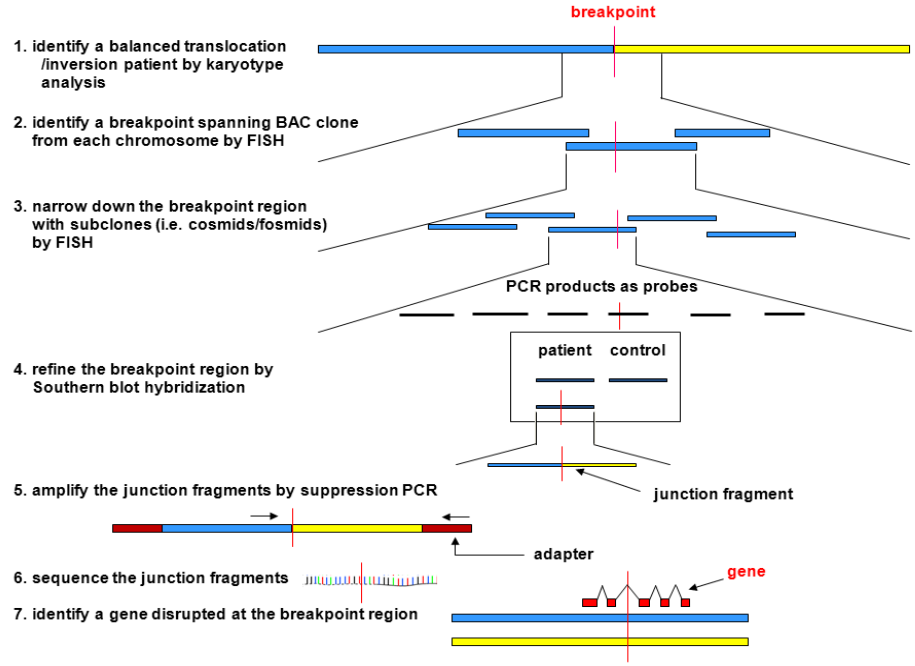
Mentor: Dr. Fouad A. Al-Shaban, Senior Scientist. Email: falshaban@hbku.edu.qa

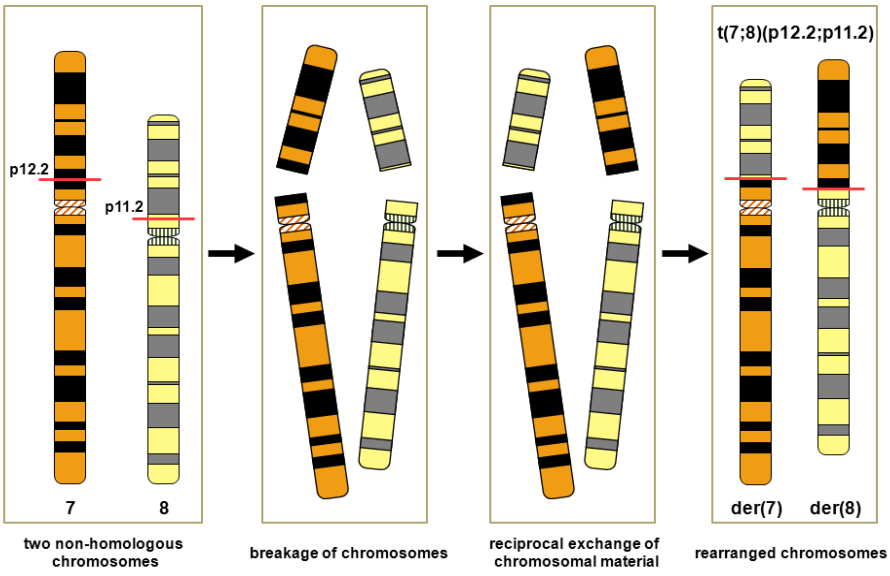
Project # 8

Title: Identifying autism gene at the breakpoint of balanced chromosome translocation

Description: Chromosome mutation as a form of balanced translocation is a good marker to locate an autism gene to a specific chromosome segment, because balanced translocations disrupt or dysregulate a gene at the breakpoint, thereby contributing to autism spectrum disorder. In this project, students will learn how to identify genes at the translocation breakpoint by using bioinformatics tools including human genome browser, gene expression pattern, interacting genes, copy number variation mapping, and interrogating the data from whole exome sequencing. Furthermore, they will learn how to amplify the junction fragment composed of DNA sequences of two different chromosomes by PCR.

Mentor: Dr. Hyung Goo Kim, Senior Scientist Email: hkim@hbku.edu.qa





Project # 9

Title: Functional assay for neuronal differentiation of human induced pluripotent stem cells (hiPSC) as a disease model.

Description: hiPSCs can be differentiated into multiple cell types, including neurons, which can be used as human disease models. Neuronal cells are required to study human degenerative diseases, for example Alzheimer's, Parkinson's, and neurodevelopmental disorders such as autism. Personalized medicine can be possible using patient-specific hiPSC, because hiPSCs offer the opportunity for development of therapeutics in model systems with patient-specific physiology. While the primary neurons of patients remain inaccessible for experimentation, hiPSC-derived neurons make it possible to study human neurons that carry the specific mutation or a neuropsychiatric disease.

The goal of this project is to carry out the functional assay to validate hiPSC-derived neurons using calcium imaging and patch clamping technique. The functional assay of neurons will be able to contribute to develop regenerative medicine and cell based therapeutics.

Mentor: Dr. Yongsoo Park, Scientist. Email: YPark@hbku.edu.qa

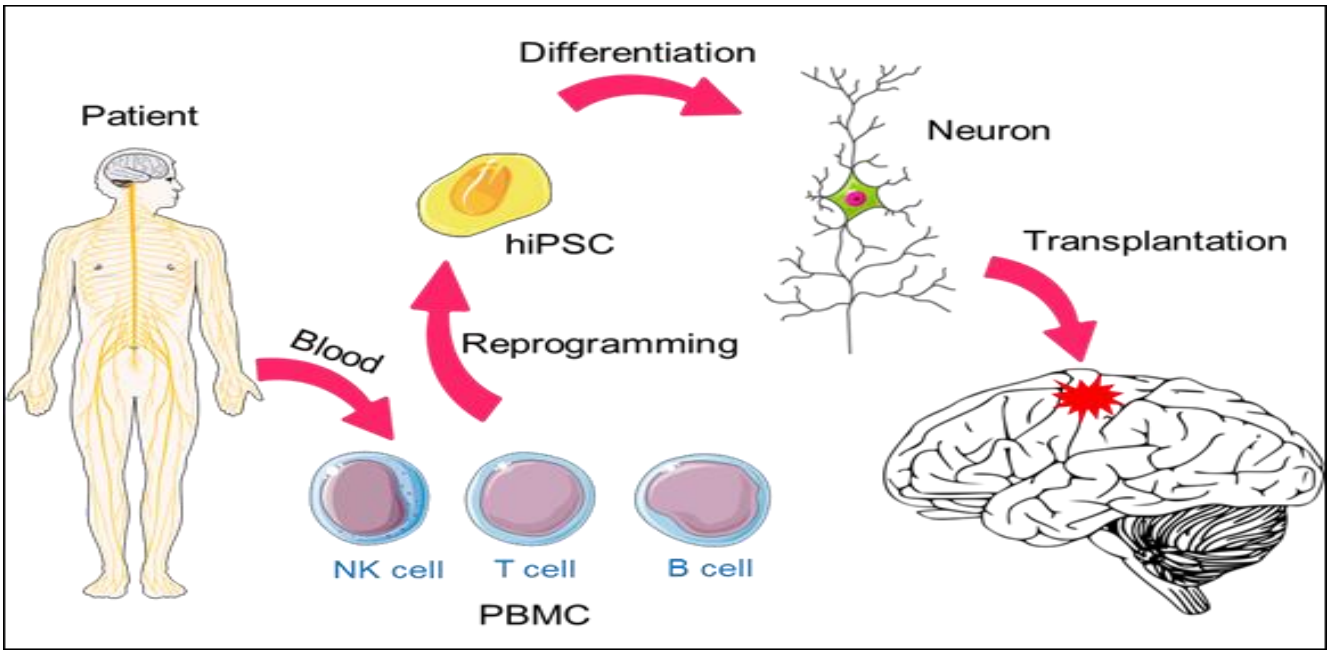


Figure 1. Schematic illustration of neuronal differentiation of human induced pluripotent stem cells (hiPSCs) as a disease model.

Project # 10

Title: Exosome biomarkers for autism spectrum disorder (ASD).

Description: ASD is a complex, multi-faceted neurodevelopmental disorder mainly characterized by core symptoms that include social interaction deficits, language difficulties and restricted, repetitive behavior. According to our QBRI study on ASD, the prevalence of ASD in Qatar is 1.14% (one in every 87 children), leading to the financial burden and stress on parent and family. Early intervention through medication, or behavioral therapy, can eliminate some of the ASD-related symptoms and significantly improve the life-quality of the affected individuals. Currently, early detection and intervention of ASD are highly limited, because ASD signs and symptoms including avoiding eye contact and repetitive movements are so subjective that ASD is usually diagnosed based on personal opinions.

Exosome RNA/proteins are considered as good biomarkers for different types of diseases including cancer, cardiovascular diseases, Alzheimer's disease and Parkinson's disease. Exosomes are small vesicles with 40–100 nm in diameter and are considered as the carriers of signaling macromolecules and RNAs for cell-cell communication, however the function of exosomes remain poorly understood. The goal of this project is to identify and validate a list of molecular biomarkers for early diagnosis of ASD.

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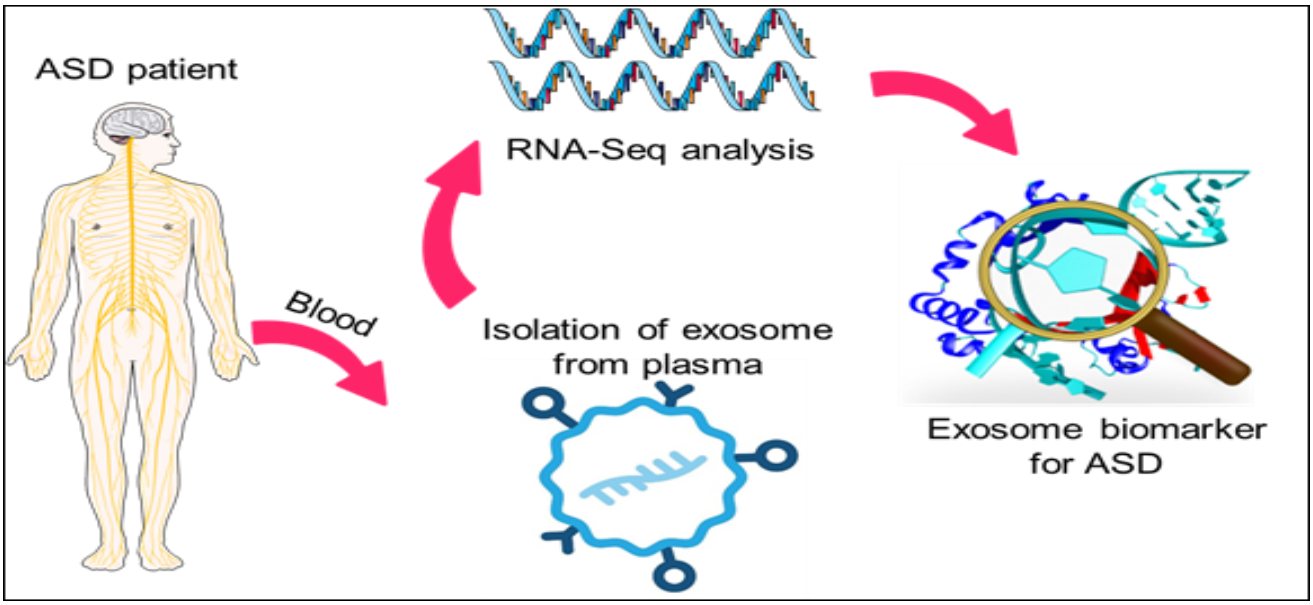

Figure 1. Schematic illustration of Exosome biomarkers for ASD.

Project # 11

Title: Small non coding RNA profiling in human biofluids of ASD individuals

Description: miRNAs are short RNA (22-25 nucleotides) that comprises about two-thirds of the human mRNAs. Each miRNA target a high number of potential mRNAs, indicating the essential role of miRNA regulation machinery in modulating gene networks. Although, miRNAs are expressed in different tissues, CNS expresses the highest percentage ~ 70%. These miRNAs are highly ubiquitous and varies dramatically through brain growth and development and within different regions in the brain. In recent studies using serum and saliva samples from ASD patients and healthy controls, the expression profile of circulating miRNA in ASD patients was significantly different than that of the control (Hicks et al., 2019; Kichukova et al., 2017; Mundalil et al., 2014). This highlights the possibility of using specific circulating miRNAs as potential biomarkers for ASD. We aim to track possible circulating miRNA as biomarkers in biofluids that will be identified by small RNA sequencing using Next Generation Sequencing (NGS).

Mentors: Dr. Sara A. Abdulla, Research Fellow. Email: saabdulla@hbku.edu.qa ,
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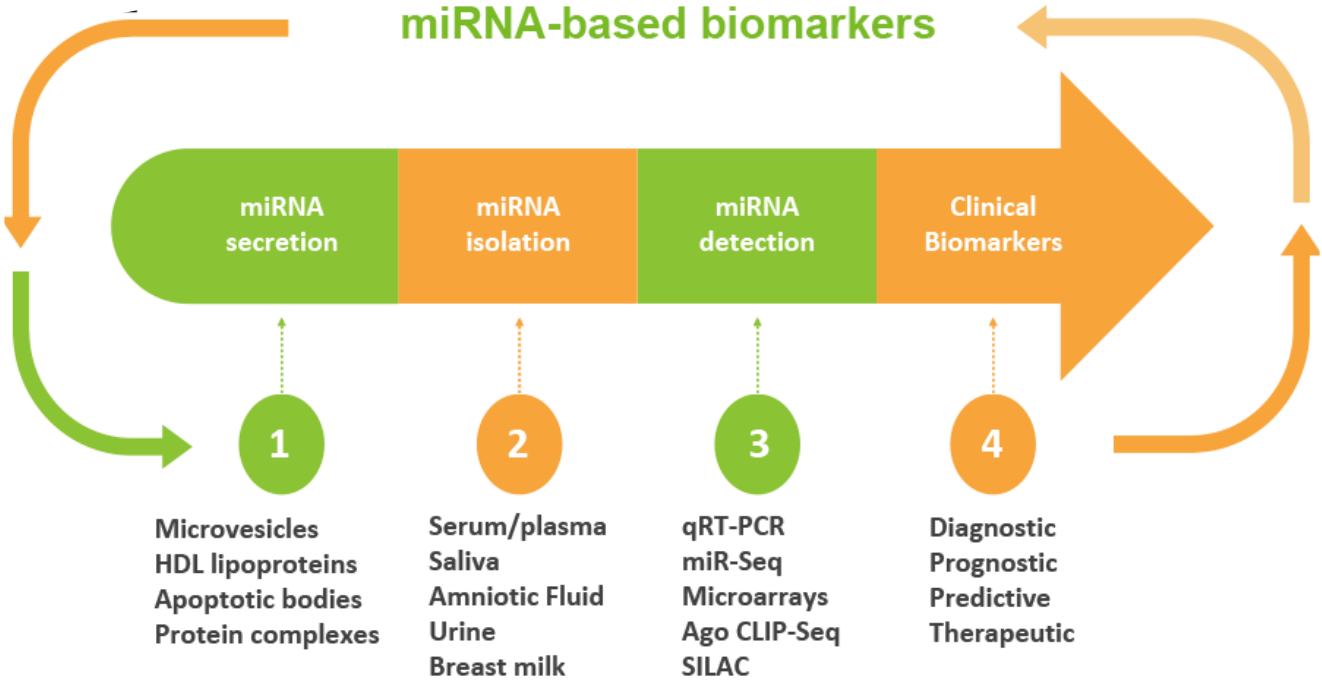

Project # 12

Title: Generation of brain organoids as an in vitro model of human brain development

Description: The human brain develops in a complex manner that makes it difficult to study brain disorders in model organisms. Lancaster et al. (2013) published a paper in Nature that developed a ground-breaking technology, termed cerebral organoids, that provides an in vitro model of human brain development in a three-dimensional culture system. These cerebral organoids can be derived from human induced-pluripotent stem cells (hiPSCs) and they undergo the full developmental processes from proliferation and migration into fully mature neurons and other brain cells such as astrocytes and oligodendrocytes. Importantly, various brain regions of dorso-ventral identities are developed together within one organoid tissue with spatial and temporal patterning specific to the human brain. Therefore, the cerebral organoid culture system recapitulates many of the unique features of human brain development and provides a promising approach for studying neurodevelopmental disorders such as Autism Spectrum Disorder (ASD). The cerebral organoids are also excellent alternatives to the traditional two-dimensional hiPSC cultures and other animal models that exhibit distinct developmental characteristics in comparison to the human brain. Eventually we would like to use this technology in order to study the underlying mechanisms of the abnormal brain development in ASD that might be unique to the Qatari population. This technology might also lead to the development of novel ASD biomarkers and therapeutic strategies that together provide a platform for personalized medicine in ASD.

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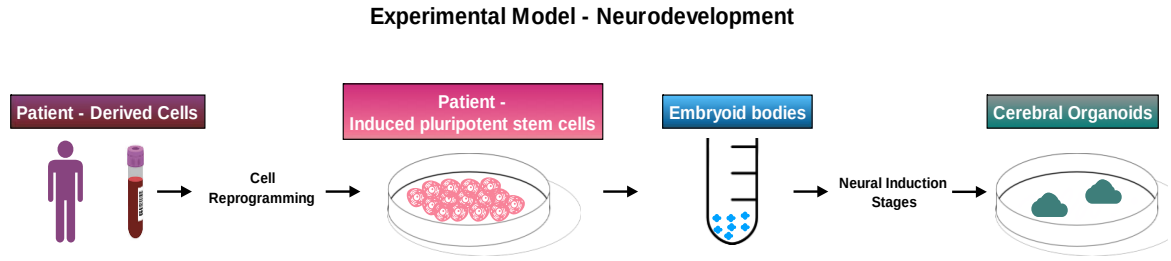
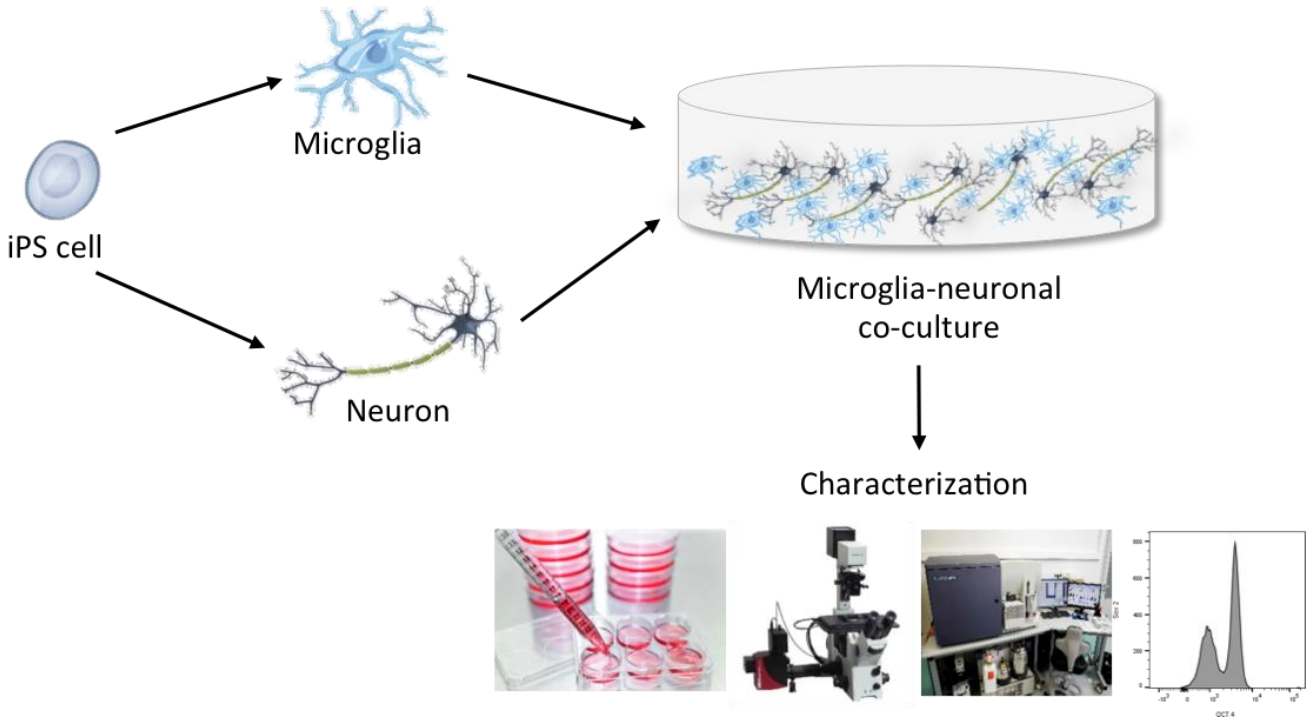


Project # 13

Title: Generation of brain microglia-neuronal co-culture from induced pluripotent stem cells

Description: Microglia are the brain resident immune cells, they maintain the immune homeostasis in the brain and provide supportive environment to neurons. Microglial dysfunction has been implicated in numerous neurological disorders. Previous human in vitro studies relied primarily on neuronal cultures derived from induced pluripotent stem cells (iPSCs) as a model for studying neurological disorders. However, recent progress in the field made it possible to generate microglia from iPSCs, and to co-culture these microglia with neurons as a more typical model of the in vivo brain environment. The microglia-neuronal co-culture provides an excellent model for studying neuro-immune interactions in neurological disorders.

Mentor: Dr. Abeer R. Al-Shammari, Research Fellow. Email: aalshammari@hbku.edu.qa



Project # 14

Title: Engineering antibodies for diagnostic and therapeutic approaches in neurodegenerative disease

Description: Common neurodegenerative diseases such as PD, Dementia with Lewy Bodies (DLB) and Multiple System Atrophy (MSA) are characterized by progressive deposition of α-synuclein (α-syn) protein within inclusions referred to as Lewy bodies and glial cytoplasmic inclusions respectively. Amongst the various approaches attempting to tackle the pathological features of synucleinopathies, immunotherapy holds much promise. α-Syn antibodies could potentially block processes leading to the pathogenesis of such neurodegenerative diseases. The limitation of such antibodies is their inefficiency in crossing the Blood-Brain Barrier. The aim of our project focuses on using a fusion protein engineered to include the FAb region of an existing α-Syn antibody. This single-chain-fragment-variable is designed to have increased BBB penetration by virtue of its smaller size and its conjugation with a carrier. It is envisaged that with enhanced penetration there will be superior brain targeting results compared to conventional α-syn antibodies.

Mentor: Dr. Vijay Gupta, Postdoc. Email: vgupta@hbku.edu.qa

Project # 15

Title: Assessing alpha synuclein neurotoxicity and its correlation to alpha synuclein S129 phosphorylation.

Description: Parkinson's disease is a neurodegenerative disorder that is characterized by neuronal inclusions known as Lewy bodies with phosphorylated α-syn at S129 being the major component. Identifying the toxic species of α-syn and understanding the seeding effect of these forms on the aggregation of the protein allow us to understand α-syn induced toxicity implicated in neurodegenerative diseases such as Parkinson's disease. Moreover, studying the impact of phosphorylation on α-syn toxicity allow us to identify whether α-syn phosphorylation promotes or inhibits toxicity of the protein.

Mentor: Dr. Simona Ghanem, Postdoc. Email: sghanem@hbku.edu.qa

Project # 16

Title: Detection of pathological alpha Synuclein aggregates in human specimen by RT- QuIC assay

Description: The real-time quacking induced conversion (RT-QuIC) assay is being applied increasingly as a potential diagnostic assay for amyloid disorders. This method is complex in nature and it is essential that it will be evaluated for both sensitivity and specificity. This will require an extensive assessment of the assay using tissue that is positive for alpha-synuclein aggregates, and matched samples. The project aims at:

1. Optimize the RT-QuIC protocol in order to reduce the fibrillization time while maintaining high specificity
2. Studying the effect of C-Terminal truncations on the aggregation propensity of a-synuclein in the RT-QuIC assay.

Mentor: Dr. Ilaria Poggiolini, Postdoc. Email: ipoggiolini@hbku.edu.qa

Project # 17

Title: Analyzing single-cell RNA sequencing (scRNAseq) data to identify cell-type, tissue and organ specific differences in sugar metabolism.

Description: Gene expression profiling of single cells by single-cell RNA sequencing (scRNAseq) has shown that different cell-types, tissues and organs can be distinguished based on their gene expression profiles using multivariate dimension reduction techniques. Rather than using all expressed genes in datasets (obtained from publicly available sources), this project will focus specifically on genes that encode enzymes in the Glyoxalase pathway - GLO1 and GLO2 - and sugar metabolism (glycolysis etc.) to investigate if different cell-types/tissues/organs can be distinguished based on these subsets of genes. This ability to distinguish could indicate that these pathways are differentially active in these different cells/tissues/organs.

Mentors: Dr. Paul J Thornalley, Scientific Director. Email: pthornalley@hbku.edu.qa,
Dr. Alberto de la Fuente, Postdoc. Email: adelafuente@hbku.edu.qa

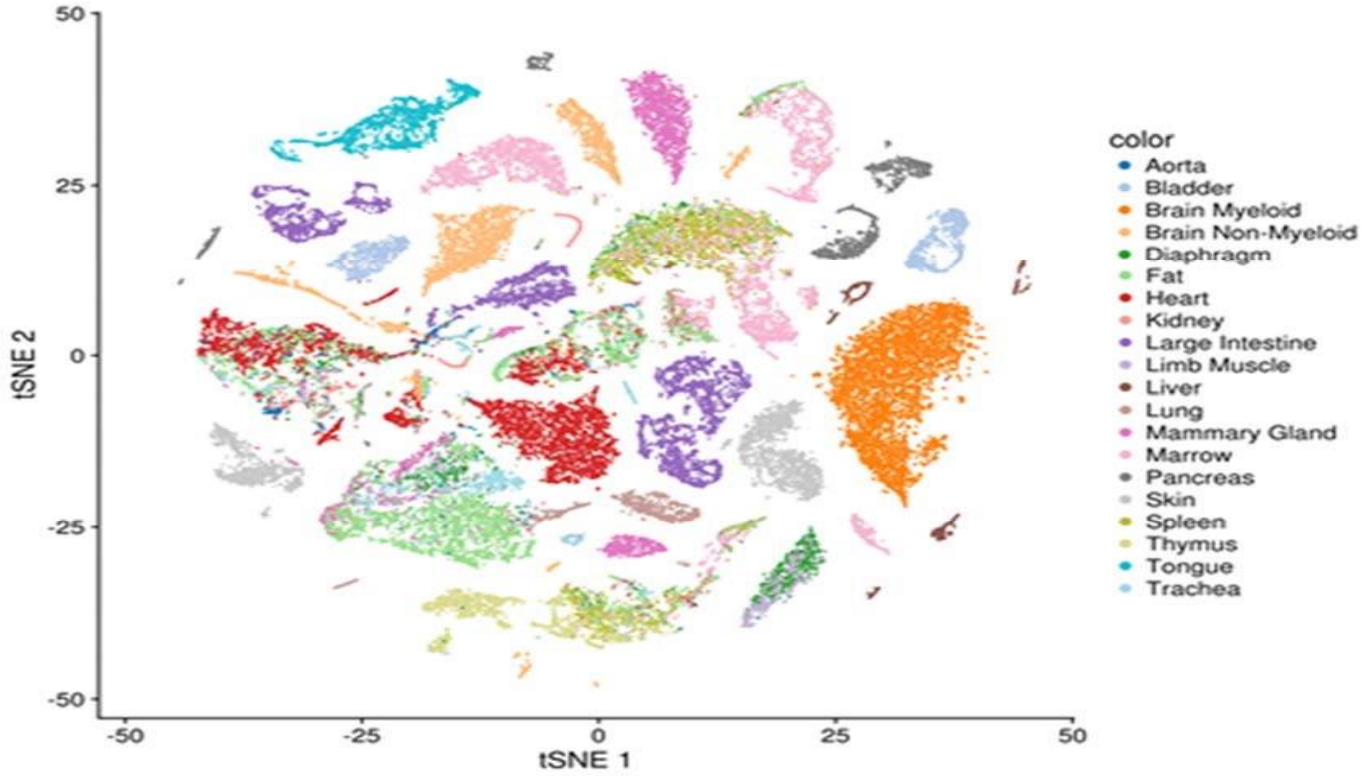


Figure. TSNE of all fluorescent activated cell sorting (FACS) from all organs. Common data analysis pipelines include a dimensionality reduction step for visualizing the data in two dimensions, most frequently performed using t-distributed stochastic neighbor embedding (TSNE).

Project # 18

Title: Involvement of dicarbonyl stress in dysfunctional epigenetic signaling in hyperglycemia

Description: Dysfunction of epigenetics in hyperglycemia have been linked to endothelial cell dysfunction in diabetes and increased risk of cardiovascular disease. Expression and activity of histone lysine N methyltransferase SET7 was increased by hyperglycemia. SET7 methylation of the gene promoter of protein p65 of the inflammatory signaling NF-kappaB system potentiates vascular inflammation. Interestingly, increased SET7 expression in hyperglycemia was corrected by overexpression of glyoxalase 1 (Glo1), suggesting that dicarbonyl stress may mediate SET7 induction and small molecule inducer of Glo1 expression, trans-resveratrol and hesperetin (tRES-HESP), may correct this. This may provide further support for use of tRES-HESP to treat diabetic vascular complications.

Mentors: Dr. Paul J Thornalley, Scientific Director. Email: pthornalley@hbku.edu.qa,
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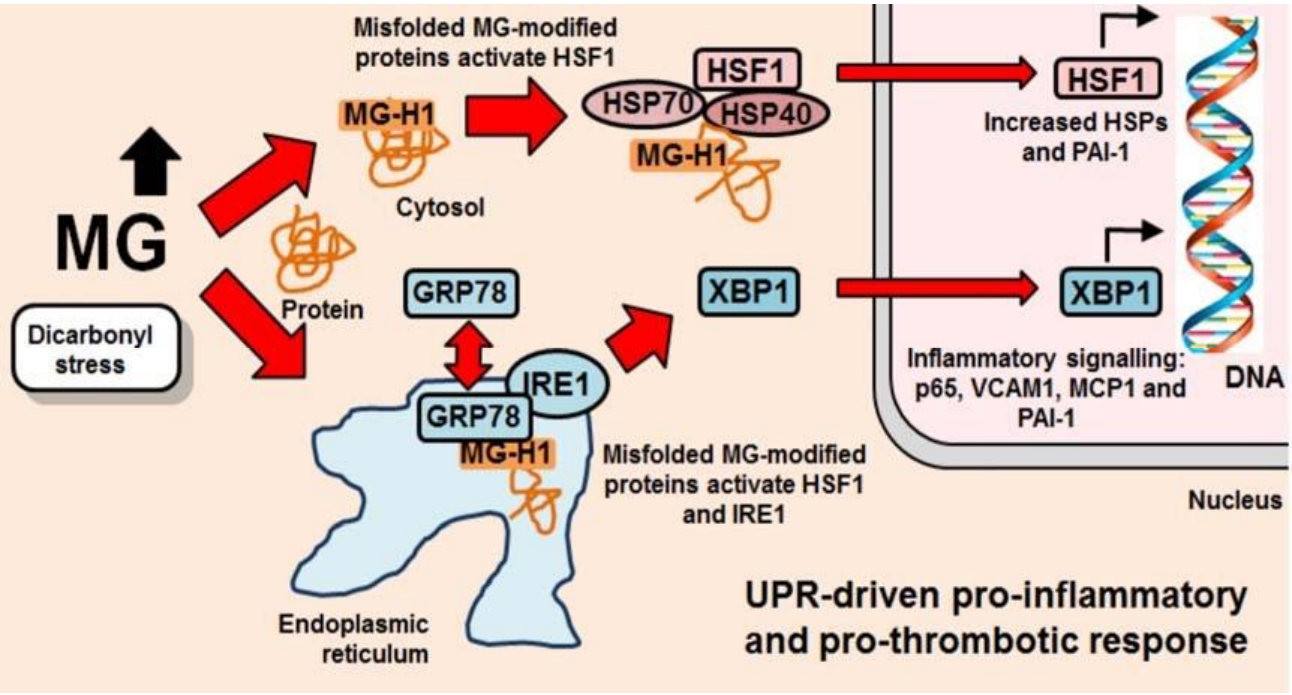


Figure. Activation of the cytosolic and endoplasmic reticulum unfolded response and inflammatory signalling by hyperglycemia-induced dicarbonyl stress. Schematic diagram of the mechanisms of activation of the unfolded protein responsive and pro-inflammatory response by dicarbonyl stress in endothelial cells in hyperglycemia. See Background Reading.

Project # 19

Title: Hypoxia-induced pre-conditioning of induced pluripotent stem cells

Description: Both type 1 diabetes (T1D) and type 2 diabetes (T2D) are caused by insufficient insulin secretion compared to insulin requirements. The underlying cause is near complete and relative loss of β-cells, respectively, with residual β-cells being dysfunctional. Our research aims to determine whether pre-conditioning of iPSC-derived beta cells and human islets with an hypoxic environment will facilitate their survival and function. We plan to characterize iPSC derived pancreatic progenitor cells in terms of the changes in their transcriptome and proteome in response to a range of hypoxic conditions, from mild to severe, in vitro. The ultimate goal of this project is to identify a novel mechanism by which an effective and viable cell therapy approach for the treatment of diabetes mellitus can be established.

Mentor: Dr. Alexandra Butler, Principal Investigator. Email: abutler@hbku.edu.qa

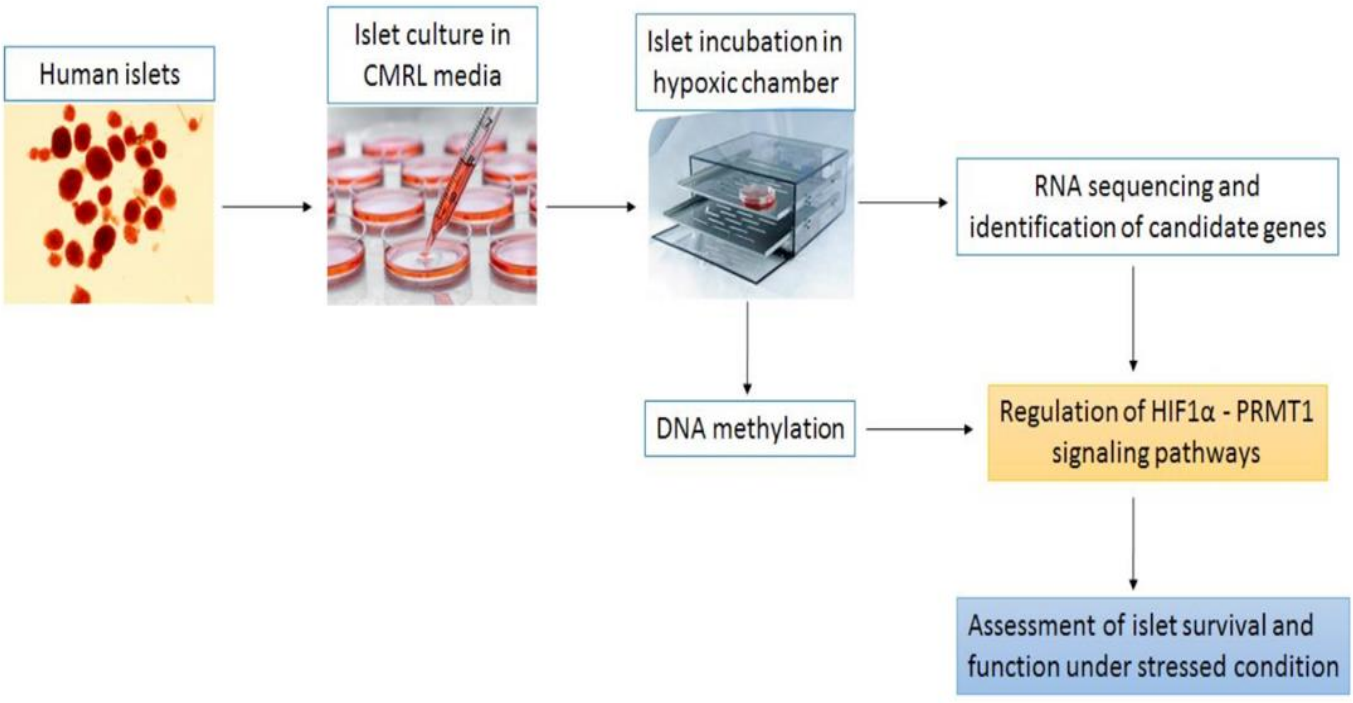


Figure 1: Schematic illustration of hypoxia pre-conditioning of iPSC-derived islets and mature human islets on β-cell survival and function

Project # 20

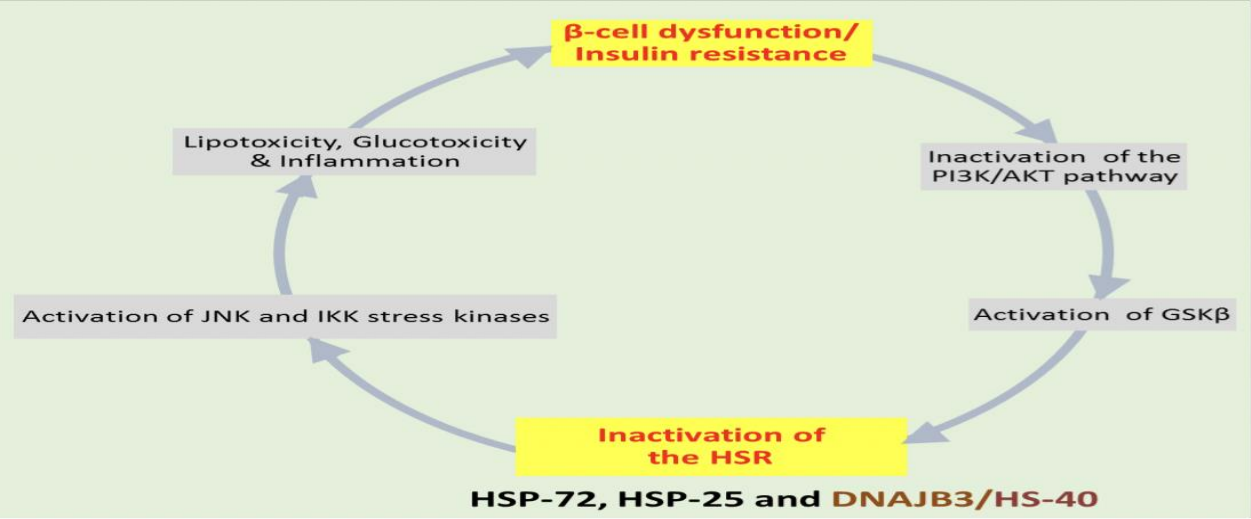
Title: Role of the heat shock response on pathophysiology of insulin resistance and type 2 Diabetes

Description: The overall focus of our research is to understand the role of DNAJB3, a component of the heat shock response in the pathophysiology of obesity and diabetes. We recently described that obese and diabetic humans displayed impaired expression of DNAJB3 with a concomitant increase in various forms of metabolic stress that are known to contribute to diabetes through the development of insulin resistance (i.e., inflammatory response, oxidative stress, endoplasmic reticulum stress and activation of JNK-stress kinase). We are currently pursing our research activity to elucidate the direct role of DNAJB3 in glucose homeostasis and insulin signaling both in vitro and in vivo. More specifically, we will investigate the effect of DNAJB3 on:

- Glucose uptake
- Protein translocation
- Insulin signaling
- Protein-protein purification
- Inflammatory response/Luciferase assay
- Metabolic stress

We will use an array of modern techniques used in molecular and cellular biology such as transient and stable expression of the clone of interest in transfected cells, transfection of silencing RNA, luciferase assay, glucose uptake, insulin signaling and apoptosis, western blot, RT-PCR

Mentor: Dr. Mohammed Dehbi, Principal Investigator. Email: mdehbi@hbku.edu.qa



Heat shock response and type 2 diabetes: The vicious metabolic cycle

Project # 21

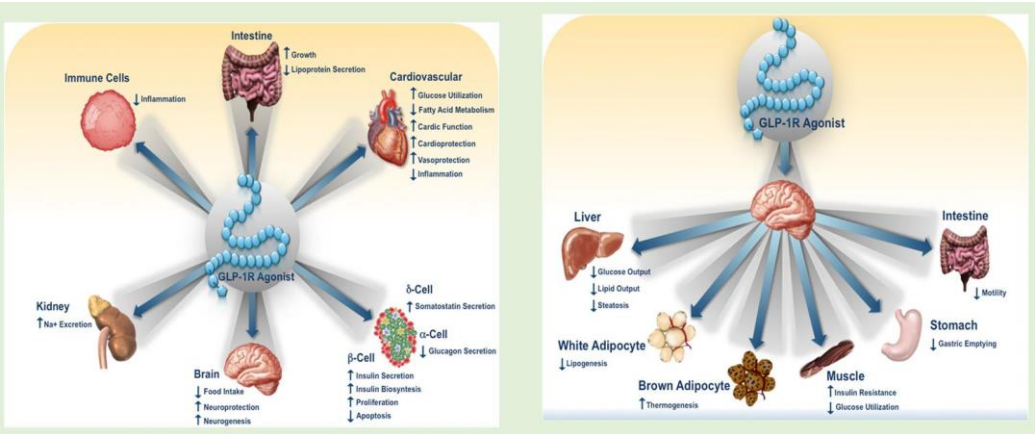
Title: Effect of Glucagon-like peptide-1analog on modulating metabolic stress: Possible role of heat shock response

Description: Insulin resistance (IR) and b-cell failure are the two core metabolic defects that lead to type 2 diabetes (T2D). These defects occur as a consequence of chronic metabolic stress that includes chronic low-grade inflammation, imbalance in the redox system, persistent ER stress. Failure of the heat shock response (HSR) to mitigate these various forms of metabolic stress is an early event that precedes IR as manifested by impaired expression of heat shock proteins (Hsps). Developing strategies that mitigate metabolic stress or restore the HSR hold the promise to improve insulin sensitivity and prevent b-cell failure in individuals at high risk, thereby, preventing the epidemic spread of T2D.

Although the current oral anti-diabetic drugs showed a clear beneficial effect to control and manage T2D, they have some undesirable side effects such as weight gain, digestive problem, CVD, risk of hypoglycemia & certain cancers, that may limit their use. In addition, they failed to show efficacy to preserve b-cell integrity and function. Recently, a new class of anti-diabetic drugs referred to as, Incretin hormones have become available and they showed efficacy with a higher therapeutic index. Incretin hormones are made by the gastrointestinal tract system and they consist of Glucagon-like peptide (GLP-1) and Gastric inhibitory polypeptide (GIP). They exert important actions that contribute to glucose homeostasis by stimulating insulin secretion by b-cell and improving its sensitivity at target tissues, reducing central satiety, promoting weight loss and mitigating metabolic stress. However, their effect on the heat shock response has never been investigated. In this investigation we will explore the in vitro effect of Exendin-4, a GLP-1 analog that mimics GLP-1 action on: 1) The expression of key components of the heat shock response (Hsp-40/DNAJB3, Hsp-25 and Hsp-72) in skeletal muscle, adipocyes, hepatocytes and pancretaic cells and 2- Investigate whether Exendin-4 effect is mediated by the activation of heat shock factor-1 “HSF-1”. The outcomes of this investigation will be related to glucose uptake and changes in the inflammation, oxidative stress and ER stress.

In this study, we will carry out a series of in vitro cell-based assays “western blot, transient gene transfer, luciferase activity, glucose uptake...”. If successful, this will be the first demonstration that GLP-1 analogs exert a beneficial effect by modulating the HSR. It will also complement us in vivo study that we plan to conduct on Qatari patients.

Mentor: Dr. Mohammed Dehbi, Principal Investigator. Email: mdehbi@hbku.edu.qa



Direct or indirect effects of GLP-1 analogs

Project # 22

Title: Generation of insulin-secreting beta cells from human induced pluripotent stem cells

Description: Generation of insulin-secreting beta cells from human pluripotent stem cells (hiPSCs/hESCs) has a great potential for treating diabetic patients. Also, those cells can be used for discovering new drugs and to understand the diabetes pathogenesis. Recently, we have established a highly efficient method to generate pancreatic beta cell precursors that could be differentiated into large number of insulin-secreting beta cells in vitro. This project is designed to provide participants with a solid understanding of the basic biology of hPSCs with a specific focus on pancreatic beta differentiation. It will equip participants with hands-on experience in the following areas:

- Culture and maintain hESCs/hiPSCs using feeder-free system.
- Examine the pluripotency and differentiation markers in undifferentiated and Differentiated hiPSCs/hESCs using different techniques.
- Differentiation of hiPSCs/hESCs into pancreatic beta cell precursors.
- Differentiation of hiPSCs/hESCs into insulin-secreting cells in vitro.

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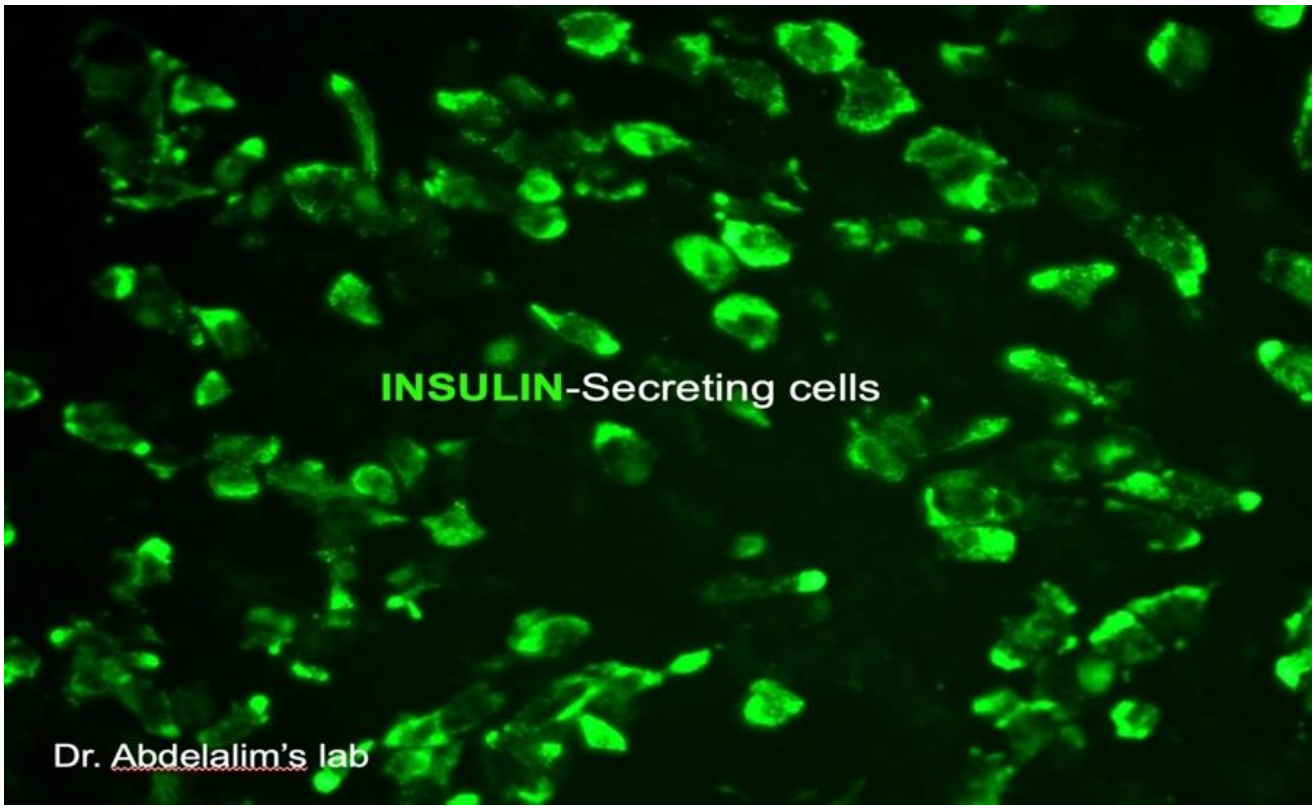

Project # 23

Title: Investigation of the relationship between salivary α-amylase activity and cardio-metabolic risk factors, cardiovascular and low grade inflammation markers in a high risk cohort of overweight/obese Qatari individuals: a cross-sectional study.

Description: The objective of this study is to examine the relationship between saliva and plasma sAA activity and cardiometabolic risk factors using gold-standard measure of adiposity (iDXA, bioimpedence) and other demographic, anthropometric and clinical data from adult participants of the Qatar biobank cohort. We will also investigate the association of sAA activity with cardiovascular parameters and markers of chronic low-grade inflammation. The ultimate goal of the study is to investigate the potential use of sAA activity for risk prediction and the target prevention of disorders related to obesity and inflammation such CVD.

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