Sri Lakshmi Narayana Institute of Medical Sciences

Date: 29.11.2018

From

Dr.Jansi Rani
Professor and Head,
Department of Biochemistry,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

To

The Dean,
Sri Lakshmi Narayana Institute of Medical College
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Permission to conduct value-added course: Molecular Basis of Gestation diabetes mellitus

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: **Molecular Basis of Gestation diabetes mellitus** for interns May to June 2019. We solicit your kind permission for the same.

Kind Regards

Dr. Jansi Rani

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean:

The HOD: Dr. Jansi Rani

The Expert: Kayalakshmy

The committee has discussed about the course and is approved.

Dean	Subject Expert	HOD
(Sign & Seal)	(Sign & Seal)	(Sign & Seal)

PROFESSOR & HOD
DEPARTMENT OF BIOCHEMISTRY
Sri Lakshmi Narayana Institute of Medical Sciences
PONDICHERRY 605 502

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.

[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affiliated to Bharath University, Chennai - TN]

Circular

18.01.2019

Sub: Organising Value-added Course: Molecular basis of Gestational diabetes mellitus. reg

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Molecular basis of Gestational diabetes mellitus"**. The course content and registration form is enclosed below."

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before February - March 2019. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

Encl: Copy of Course content

VALUE ADDED COURSE

1. Name of the programme & Code

Molecular basis of Gestational diabetes mellitus – BIO-10

2. Duration & Period

30 hrs & April and May -2019-2020

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled

Enclosed as Annexure- II

5. Assessment procedures:

Assessment closed

6. Certificate model

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 times April and May 2019

8. Year of discontinuation: 2019

9. Summary report of each program year-wise

Value Added Course- April –May -2019-20					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	BIO 10/ I Batch	**Molecular basis of Gestational diabetes mellitus**	Dr. Jansi Rani Dr.Kajalaksmy	MBBS Students	20 students MAY-JUNE 2019)

10. Course Feed Back

Enclosed as Annexure- V

RESOURCE PERSON

1. **Dr.Jansi Rani**
2. **Dr. Kajalakshmy**

COORDINATOR
Dr.Jansi Rani

PROFESSOR & HOD
DEPARTMENT OF BIOCHEMISTRY
Sri Lakshmi Narayana Institute of Medical Sciences

Course Proposal

Course Title: **Molecular Bases of Gestational diabetes Mellitus**

Course Objectives:

- GDM and T2DM share a common genetic background including glucose intolerance, insulin resistance, and insulin secretion. Respect similar risk factors and the genetic variants used to determine the risk of developing T2DM might also be associated with the prevalence of GDM.
- GDM is a major cause of prenatal morbidity and mortality, as well as maternal morbidity.

Course Outcome: The outcomes will help us to develop new therapeutic target which could be help us to manage to GDM as well as maternal it complications such as preeclampsia, hypoglycemia, respiratory distress, hyberbilirubinemia, and low birth weight.

Course Audience: MBBS students 2019 -2020 Batch

Course Coordinator: Dr.Jansi Rani

Course Faculties with Qualification and Designation:

1.Dr.Jansi Rani, Professor & HOD

2.Dr.Kajalakshmy, Assistant Professor

Course Curriculum/Topics with schedule (Min of 30 hours)

SlNo	Date	Topic	Time	Hours
1.	18-2-2019	Introduction of Gestational diabetes mellitus	2-3p.m	1
2.	19-2-2019	Prevalence of Gestational 1. Global Prevalence of GDM 2. Prevalence of GDM in India	4-6p.m	2

3.	20-2-2019	Screening guidelines of Gestational diabetes mellitus	4-6p.m	2
4.	21-2-2019	Complication of Gestational diabetes mellitus:	4-6p.m	2
5.	22-2-2019	Genetic variation and Gestational diabetes mellitus	4-6p.m	2
6.	23-2-2019	Genetic variation and Gestational diabetes mellitus	4-6	2
7.	24-2-2019	KCNJ11 gene polymorphism	4-6p.m	2
8.	25-2-2019	Relationship between KCNJ11 gene polymorphism and Biochemical	4-6p.m	2
9.	26-2-2019	KCNQ1 Gene polymorphism:	4-6p.m	2
10.	27-2-2019	Relationship between KCNQ1 gene polymorphism and Biochemical	4-6p.m	2
11.	28-2-2019	PPARgamma gene polymorphism and Gestational diabetes mellitus:	4-6p.m	2
12.	29-2-2019	Glucose kinase gene polymorphism	4-6p.m	2
13.	30-2-2019	Nutrition Therapy in Gestational Diabetes Mellitus	4-6P.m	2
14.	31-2-2019	Nutrition Therapy in Gestational Diabetes Mellitus	4-6P.m	2

15.	1-3-2019	**Nutritional Management of Gestational Diabetes and Nutritional Management of Women With a History of Gestational Diabetes**	4-6P.m	2
16.	3-3-2019	**Gestational diabetes mellitus and Pregnancy outcomes**	4-6P.M	2
		Total		31hrs

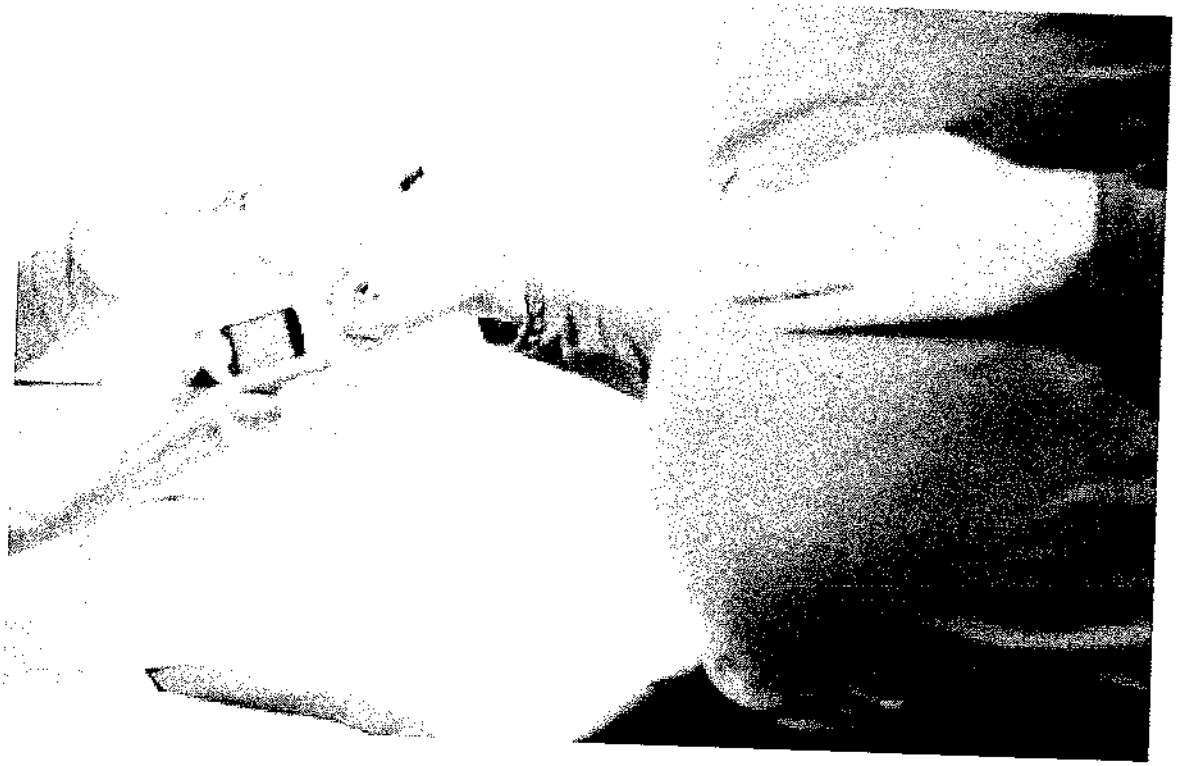
REFERENCE BOOKS:

Categories: 2020, Diabetes, Endocrinology, Endocrinology Research and Clinical Developments, Medicine & Health, Newly Published Books, Nova Medicine and Health

Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2006; 29 Suppl 1:S43–S48.

American College of Obstetricians and Gynecologists Committee on Practice Bulletins-. ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists. Number 30, September 2001 (replaces Technical Bulletin Number 200, December 1994). Gestational diabetes. *Obstetrics & Gynecology 98(3)*

Molecular Basis of Gestation diabetes mellitus



PARTICIPANT HAND BOOK

Annexure- I

COURSE DETAILS

Particulars	**Description**
Course Title	**Molecular Basis of Gestation diabetes mellitus**
Course Code	BIO-01
Topics and content of the course in the Hand book	1. Introduction of Gestational diabetes mellitus 2. Global Prevalence of GDM 3. Prevalence of GDM in India 4. Risk Factors in Gestational diabetes mellitus 5. Screening guidelines of Gestational diabetes mellitus 6. Complication of Gestational diabetes mellitus 7. Genetic variation and Gestational diabetes mellitus a. KCNJ11 gene polymorphism in Gestational diabetes mellitus b. KCNQ1 Gene polymorphism in Gestational diabetes mellitus c. PPARgamma gene polymorphism and Gestational diabetes mellitus d. Glucose kinase gene polymorphism in Gestational diabetes mellitus 8. IGF2B2 gene polymorphism and Gestational diabetes mellitus
Advantages of learning and evaluation	• To learn about biochemical and molecular basis of Gestational diabetes mellitus • TO identify the new protein and molecular markers of Gestational diabetes mellitus
Further learning	Protein targeting and protein expression in Gestational

Opportunities	diabetes mellitus
Key Competencies	
Target Student	MBBS and Nursing students
Duration	
Theory Session	31 hours
Assessment Procedure	

Introduction

Gestational diabetes mellitus (GDM) is the most common medical complication of pregnancy. It is associated with maternal and neonatal adverse outcomes. Maintaining adequate blood glucose levels in GDM reduces morbidity for both mother and baby. There is a lack of uniform strategies for screening and diagnosing GDM globally. This review covers the latest update in the diagnosis and management of GDM. The initial treatment of GDM consists of diet and exercise. If these measures fail to achieve glycemic goals, insulin should be initiated. Insulin analogs are more physiological than human insulin, and are associated with less risk of hypoglycemia, and may provide better glycemic control. Insulin lispro, aspart, and detemir are approved to be used in pregnancy. Insulin glargine is not approved in pregnancy, but the existing studies did not show any contraindications. The use of oral hypoglycemic agents; glyburide and metformin seems to be safe and effective in pregnancy.

KCNJ11 gene polymorphism

The KATP is a large macromolecule complex which have four rectifying potassium channel (Kir6.x) subunits and a central pore surrounded by four regulatory sulphonylurea receptors (SUR). Inward rectifying potassium channel kir6.x contains two subunit kir6.1 and kir6.2.

Sulphonylurea receptor has three isoforms:SURA, SURB. SUR1 is found in skeletal heart, and skeletal muscle, SURB is found in smoothmuscle and many neurons, SUR1 is expressed in neuro-endocrine cells, beta-cells and neurons. KATP protein has coding gene called as KCNJ11 (or kir6.2) subunit which is located on chromosome 11p15.1. The total length of KATP protein is 3418bp, containing only a 1173bp exon. KATP protein is mostly expressed in heart, skeletal muscle, vascular smooth muscle, nerves and various endocrine tissues in human. The KCNJ11 gene belongs to potassium channel gene family. The genetic variations of KCNJ11 causes replacement of glutamic acid by lysine at 23rd position, which inturn leads to reduced insulin secretion, which can ultimately leads to T2DM and Gestational diabetes mellitus.

KCNQ1 Gene polymorphism:

KCNQ1 (Potassium voltage-gated channel KQT1-like subfamily, member 1) and is located at chromosome 11p.15.5, which has family encodes a protein for a voltage-gated potassium channel. The KCNQ1 gene has total of 17exon, spans 404kb of chromosome and it has made of 676 amino acids with trans membrane regions and encodes the pore-forming alpha subunit of the voltage-gated K+ channel (KLQT1).

KCNQ1 gene polymorphism involved in both cardiac function and diabetes mellitus. IKS(slow delayed rectifier K+ channel), which has involved in the late repolarization phase of the cardiac action potential, maintained of vascular, smooth muscle tone, cell volume, regulation, leukocyte activation and proliferation and many other physiological function. KCNQ1 genes there are two genes are responsible for the regulation of IKS. One is KCNQ1 and another one is KCNE1, most important for KCNQ1 gene involved in IKS alpha subunit and KCNE1 gene responsible for beta-unit. Here alpha-subunit has positively charged and activated by depolarization. Therefore change to negative charge within cardiac cell. This repolarization involved in the late opening of the IKS channel then regulated by

phosphorylation of signalling cascade. Therefore polymorphism (or) mutation occurring in KCNQ1/KCNE1 gene, which can disrupt IKS channel. KCNQ1/KCNE1 gene might be inhibited or delayed to transport and assembly of the IKS subunit of the cell membrane from the rough endoplasmic reticulum.

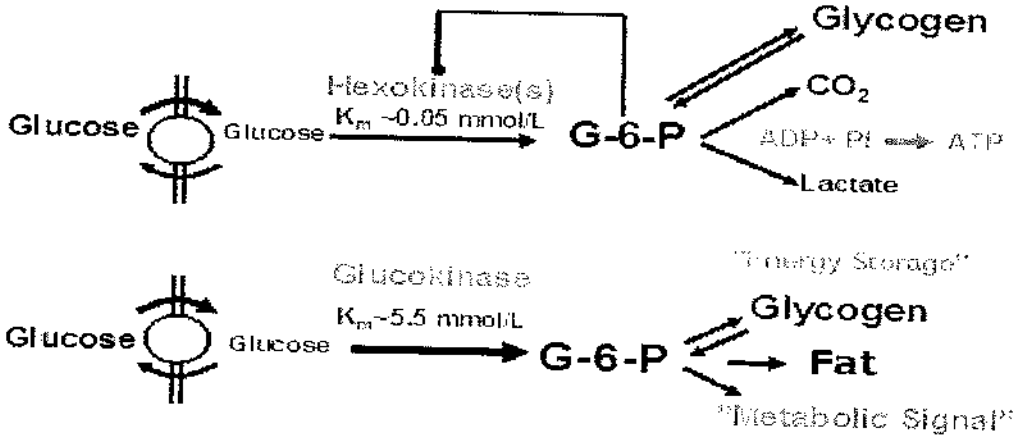
PPARgamma gene polymorphism and Gestational diabetes mellitus

Peroxisome proliferator-activated receptor contains three types namely as PPARγ, PPARβ, PPARδ. The PPARγ gene is located on chromosome 3 (10c.3p25=OMIM 601487)(191), which is contains 9 exon and spans more than 100kb. Peroxisome proliferator-activated receptor γ (PPARγ) is a nuclear hormone receptor. PPARγ has contains two isoform PPARγ1, PPARγ2 and it has 84 nucleotide and 28 aminoacids. PPARγ1 isoform expressed in adipocytes and PPARγ2 is expressed in adipocytes, vascular smooth muscle cells, macrophages, mesangial cells, and renal epithelial cells. PPARgama gene has been associated with energy metabolism, cell differentiation and the inflammatory response This Pro12Ala functional variation has been correlated with coronary heart disease (CHD), type 2 diabetes, obesity and lipid disorders, all of which are important risk for cerebrovascular disease. Some authors consider PPARγ2 gene variation to be associated with myocardial infarction, type 2 diabetes, and high insulin sensitivity, among other factors Peroxisome proliferator activity receptor gama(PPARγ) is a transcription factor with a key role in adipogenesis and insulin sensitization frequent mutations in the PPARγ gene have been described to be associated with obesity and diabetes related phenotypes. The common structural polymorphism with proline(pro) to alanine (Ala) substitution has been identified in Type2 Diabetes mellitus. An additional variant C1431T located in exon 6 of PPARγ is associated with susceptability to cardiovascular disease, leptin concentration and body mass index during pregnancy. Dynamic physiologic, metabolic and immunologic adaptations are

required to ensure fetal development and maternal wellbeing. Many of the metabolic adaptations are mediated by PPARs.

Glucose kinase gene polymorphism

The GCK gene is located at chromosome 7p15.3 and it has 12 exon splicing end and it contains 465 amino acid. This has three tissue-specific isoform present in glucosekinase gene. GCK is mainly expressed in pancreatic beta cell and hepatocyte. So it called as "pancreatic cell glucose sensor". This enzyme involved in first step of glycolysis pathway.



IGF2B2 gene polymorphism and Gestational diabetes mellitus

Insulin like growth factor 2 binding protein 2(IGF2B2) at located on the 3q27.2 chromosome and it is about 181,36 k which contains 16exon. Insulin like growth factor2 (IGF2) mRNA binding proteins (IMP1, IMP3).It has involved in RNA localization, stability, translation of RNA and mainly IGF2 plays a role in glucose homeostasis, mechanism of glucose uptake in different tissues, especially in hepatic gluconeogenesis and lipolysis pathway(201). *Dimitry A. Chistiakovet al.*, studies showed that IGF2BP2 gene involved in the regulation of IGF2 gene expression by binding of 5'-UTR of the IGF2 mRNA and it promotes to translation. *Kwok et al.*, reported that it is associated with increased risk of Gestational diabetes mellitus.

Chon et al., meta-analysis also observed IGF2BP2 gene polymorphism had increased risk of GDM. Meta-analysis studies of *Mao et al.*, report showed increased risk of diabetes mellitus and Gestational diabetes mellitus. *Chon et al.*, found polymorphism of PPARGγ2 and IGF2BP2 were associated with higher prevalence of Gestational diabetes mellitus. *Zhao et al.*, report found that rs4402960 polymorphism in IGF2BP2 is associated with and elevated risk of T2DM but these associations vary from ethnic population to other populations

SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES

DEPARTMENT OF BIOCHEMISTRY (2019-2020)

S.No	Register No.	Student List	Signature
1	U19MB331	NIRMAL KUMAR B	
2	U19MB332	NISHANTHI V	
3	U19MB338	PAVITHRA T	
4	U19MB336	PADMAJA T	
5	U19MB343	PRIYADHARSHINI M	
6	U19MB346	RAAJ SETHU VINAYACK R	
7	U19MB350	RAJAGOPAL R	
8	U19MB347	RABYA TABASUM	
9	U19MB345	PULAK ACHARYA	
10	U19MB334	NIVASINY P S	
11	U19MB340	PRADHEEP K	
12	U19MB347	RABYA TABASUM	
13	U19MB350	RAJAGOPAL R	
14	U19MB357	ROSHMASHREE S	
15	U19MB341	PRAKHAR GAUTAM	
16	U19MB346	RAAJ SETHU VINAYACK R	
17	U19MB347	RABYA TABASUM	
18	U19MB355	RIYA BRUNO	
19	U19MB356	ROLLAPATI TEJESH	
20	U19MB357	ROSHMASHREE S	

SRI LAKSHMI NARAYANA INSTITITE OF MEDICAL SCIECNES

GESTATIONAL DIABETES MELLITUS

Genetic factors and diet-related behaviours are two primary determinants of diabetes but the causes of ME/CFS _____.

a. are largely psychosomatic
b. are primarily due to poor diet
c. remain a complete mystery
d. none of these

1. In _____, blood glucose homeostasis ceases to function because the beta cells of the pancreatic islets are destroyed.

a. type 1 diabetes mellitus
b. type 2 diabetes mellitus
c. gestational diabetes
d. both type 2 diabetes mellitus and gestational diabetes

2. Myalgic encephalomyelitis or chronic fatigue syndrome has also been named _____.

a. post-viral fatigue syndrome
b. systematic exertion intolerance disease
c. chronic fatigue immune dysfunction syndrome
d. all of these

4. According to the _____, ME/CFS patients are alleged to have 'unhelpful cognitions' and 'dysfunctional beliefs' that their symptoms are caused by an organic disease.

a. Impaired Belief Theory
b. Dysfunctional Belief Theory
c. Maladjusted Belief Theory
d. Inhibited Belief Theory

5. The estimated number of people in the US that have diabetes (diagnosed or undiagnosed) is

a. 22 million

b.650,000

c. 16 million

d. 8.5 million

6. The new classification of diabetes is based on

a. etiology

b. type of treatment

C. type of insulin

d. age of onset

7. Type 2 diabetes is characterized by

a. insulin resistance

b. insulin lack

C. beta cell destruction

d. none of the above

8. The hormone that is secreted by the alpha cells of the pancreas that raises blood glucose when levels are low is:

a. glucagon

b. epinephrine

C. insulin

d. cortisol

9. Type 2 diabetes typically is diagnosed at a young age.

a. True

b. False

10. The renal threshhold for glucose is _____ mg/dl.

a. 180

b. 120

c. 200

d. 140

SRI LAKSHMI NARAYANA INSTITITE OF MEDICAL SCIECNES

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b. False

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d. 140

Annexure- V

Course feedback form

Course title: Date :

Course code:

Department: BIOCHEMISTRY

S.no	Design of the course	1	2	3	4	5
1	The objective of the course clear to you	✓				
2	The course contents met with your expectations	✓				
3	The lecture sequence were well planned	✓				
4	The lectures were clear and easy to understand	✓				
5	The audiovisual teaching aids were effectively used	✓	✓			
6	The instructor's encouraged interaction and was it helpful		✓			
7	The contents were illustrated with examples		✓			
8	Overall Rating of the course	✓				

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

✓ Excellent

Signature

Annexure- V

Course feedback form

Course title: Date :

Course code:

Department: BIOCHEMISTRY

S.no	Design of the course	1	2	3	4	5
1	The objective of the course clear to you		✓			
2	The course contents met with your expectations		✓			
3	The lecture sequence were well planned	✓				
4	The lectures were clear and easy to understand		✓			
5	The audiovisual teaching aids were effectively used		✓			
6	The instructor's encouraged interaction and was it helpful		✓			
7	The contents were illustrated with examples	✓				
8	Overall Rating of the course		✓			

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

— Excellent —

Signature

Sri Lakshmi Narayana Institute of Medical Sciences

CERTIFICATE OF MERIT

This is to certify that SHALINI R as actively participated in the Value Added Course on **OVER VIEW OF MOLECULAR MECHANISM OF GESTATIONAL DIABETES MELLITUS** held during April 2019 – May 2020 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. Kajalakshmy
RESOURCE PERSON

DEPARTMENT OF BIOCHEMISTRY
Sri Lakshmi Narayana Institute Of Medical Sciences
PONDICHERRY 605 502.

Dr. Jansi Rani
COORDINATOR

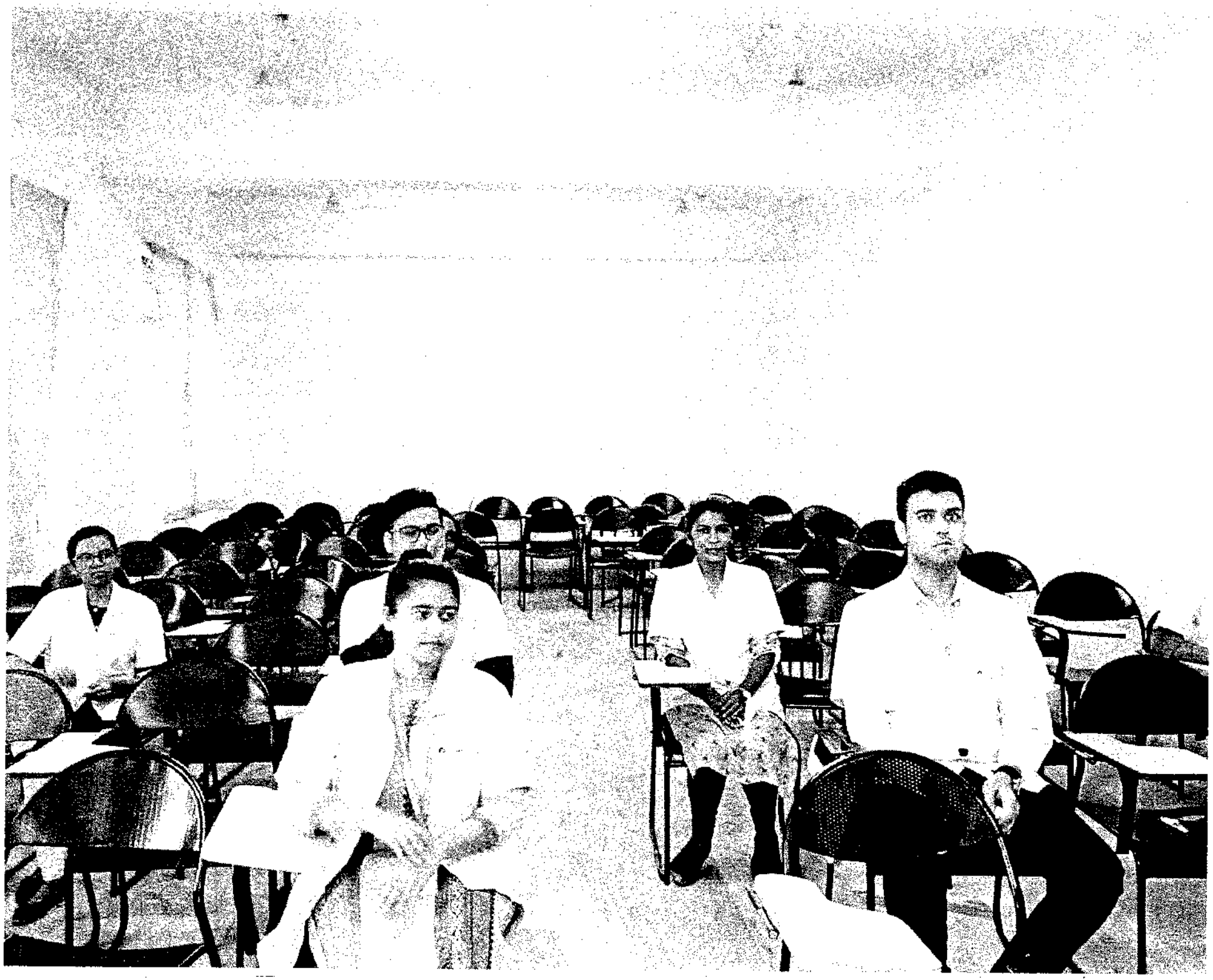
Sri Lakshmi Narayana Institute of Medical Sciences

CERTIFICATE OF MERIT

This is to certify that SAI RAKSHIKA J as actively participated in the Value Added Course on **OVER VIEW OF MOLECULAR MECHANISM OF GESTATIONAL DIABETES MELLITUS** held during April 2019 – May 2020 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. Kajalakshmy
RESOURCE PERSON
DEPARTMENT OF BIOCHEMISTRY

Dr. Jansi Rani
COORDINATOR



From

Date: 09.09.2019

Dr. Jansi Rani

Professor and Head,
Department of Microbiology,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To

The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Completion of value-added course:

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: Molecular basis of Gestational diabetes mellitus We solicit your kind action to send certificates for the participants, that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

Dr. Jansi Rani

Encl: Certificates

Photographs