شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم

بسم الله الرحمن الرحيم

HANAA ALY

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جامعة عين شمس

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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

HANAA ALY

Assessment of serum adropin level as a risk factor of ischaemic heart disease in type 2 diabetes mellitus cases

Thesis

Submitted for partial fulfillment of Master Degree in Endocrinology and Metabolism

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تقييم مستوى هرمون الادروبين كمعامل خطر لمرض نقص تروية القلب في حالات مرضى السكري من النوع الثاني

رسالة

توطئة للحصول علي درجة الماجستير في الغدد الصماء والايض

مقدمة من

الطبيب/ باهر إميل إبراهيم

بكالوريوس الطب و الجراحة

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List of Abbreviations

ADA	American diabetes association
Adr-Tg	Adropin-overexpressing transgenic mice
AGEs	Advanced glycation end products
AIDS	Acquired immunodeficiency syndrome
AMI	Acute myocardial infarction.
BMI	Body mass index
CAD	Coronary artery disease
CIMT	Carotid intima-media thickness
CPT1B	Carnitine palmitoyl transferase-1b
CSX	Cardiac syndrome x.
CT	Computed tomography
CVD	Cardiovascular disease
DAG	Diacylglycerol
DHAP	Dihydroxyacetone phosphate.
DIO	Diet-induced obesity
eNOS	Endothelial nitric oxide synthase
ERK1/2	Extracellular signal-regulated kinases 1/2
EPC	Endothilial progenitor cells
F-1,6-P	Fructose-1,6-bisphosphate
F-6-P	Fructose-6-phosphate
FAO	Fatty acid oxidation
G-3-P	Glycerol-3-phosphate
GA3P	Glyceraldehdye-3-phosphate
GDM	Gestational diabetes mellitus
GFAT	Glutamine: fructose-6-phosphate amidotransferase
GIP	Glucose-dependent insulinotropic polypeptide
GIPP	Gastric inhibitory polypeptide
GlcNAc	N-acetyl glucosamine
GLP-1	Glucagon like peptide-1
GPR19	G protein-coupled receptor
HDL	High density lipoprotein
HIV	Human immunodeficiency virus
HMGA1	High mobility group a1
HOMA-IR	The homeostatic model assessment of insulin resistance
IFG	Impaired fasting glucose
IGT	Impaired glucose tolerance
INSR	Insulin receptor gene
IRS1, IRS2	Insulin receptor substrate 1&2 ,

MAPK1	Mitogen-activated protein kinase 1
MESA	Multi-ethnic study of atherosclerosis
MetS	Metabolic syndrome
MGO	Methylglyoxal
NAFLD	Non alcoholic fatty liver disease
NASH	Non-alcoholic steatohepatitis
NICE	National institute for health and care excellence
NO	Nitric oxide.
OGTT	Oral glucose tolerance test
ORF	Open reading frame's
PAD	Peripheral arterial disease
PAI-1	Plasminogen activator inhibitor-1
PCOS	Polycystic ovarian syndrome ,
PDH	Pyruvate dehydrogenase ,
PDK4	Pyruvate dehydrogenase kinase 4.
PPAR γ	Peroxisome proliferator-activated receptor-γ
PI3K	Phosphoinoside-3 kinase
PKC	Protein kinase-c
QTL	Quantitative trait loci
ROS	Reactive oxygen species
RNS	Reactive nitrogen species
SA	Subclinical atherosclerosis
SAP	Stable angina pectoris
SNPs	Single-nucleotide polymorphisms
T2D	Type 2 diabetes
TGF-1	Transforming growth factor 1
TNF- α	Tumor necrosis factor alpha
UDP-cNAc	Uridine diphosphate n-acetyl glucosamine
UAP	Unstable angina pectoris
VEGF	Vascular endothelial growth factor
WHO	World health organization

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INTRODUCTION

Type 2 diabetes was referred to as "noninsulin-dependent diabetes" accounts for 90–95% of all diabetes. This form includes individuals who have relative insulin deficiency and have peripheral insulin resistance. At least initially, and usually throughout their lifetime, these individuals may not need insulin treatment to survive ***(ADA ., 2019)***

Diabetes Mellitus is the most frequent endocrine disease in developed countries estimated to have affected 366 million people worldwide and is expected to nearly double by 2030 owing to an increase in obesity, life span extension, and better detection of the disease. ***(Gupta et al., 2014)***.

It is widely accepted that in DM there is an impairment of endothelial nitric oxide synthase (eNOS) activity as well as enhancement of production of reactive oxygen species (ROS), resulting in diminished nitric oxide (NO) bioavailability and the consequent vascular alterations ***(Tousoulis et al., 2012)***

DM has many ways of leading to endothelial dysfunction. The increased oxidative stress, the alteration of lipogenesis, the reduction of nitric oxide, and the alteration of endothelial progenitor cells (EPC) function create what is called the diabetic state, in which the