



شبكة المعلومات الجامعية

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شبكة المعلومات الجامعية  
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# شبكة المعلومات الجامعية التوثيق الالكتروني والميكرو فيلم





شبكة المعلومات الجامعية

# جامعة عين شمس

التوثيق الالكتروني والميكرو فيلم

## قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها  
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# ENDOTHELIUM-DERIVED RELAXING FACTOR (EDRF/NO) AND GLYCATION OF ALBUMIN IN TYPE I-DIABETICS

*Thesis*

Submitted in Partial Fulfillment of the  
Master Degree in  
*Endocrinology and Metabolism*

B7653

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## LIST OF ABBREVIATIONS

ACE	Angiotensin converting enzyme.
ACEI	Antiotensin converting enzyme inhibitor.
cAMP	Cyclic adenosine monophosphate.
cGMP	Cyclic guanosine monophosphate.
DM	Diabetes mellitus.
EDCF	Endothelium-derived contracting factor.
EDCFs	Endothelium-derived contracting factors.
EDHF	Endothelium-derived hyperpolarizing factor.
EDRF	Endothelium-derived relaxing factor.
EDRF/NO	Endothelium-derived relaxing factor/nitric oxide.
EDRFs	Endothelium-derived relaxing factors.
ENOS	Endothelial nitric oxide synthase enzyme.
IFG	Impaired fasting glucose.
IGT	Impaired glucose tolerance.
LADA	Late autoimmune diabetes in the adult.
NO	Nitric oxide.
NO <sub>2</sub>	Nitrite.
NO <sub>3</sub>	Nitrate





## **ABSTRACT**

**Background:** NO produced by the endothelial cells of the blood vessels, goes abluminally to the smooth muscles in the vessels wall to exert its function as a relaxing factor for the vessel musculature. At the same time, it goes to the vessel lumen to prevent platelets adhesion and aggregation. This vital hormone is also carried to albumin and acts as a reservoir for the body. In diabetes mellitus, glycosylation of serum albumin, impairs its carrying capacity.

**Aim of the work:** The EDRF/NO production and its blood level in diabetes mellitus has been the subject of conflicting argument. It was the aim of the present study to find a genuine explanation, for the impaired endothelial function in type I diabetics.

**Subjects and Methodology:** 60 type I diabetic patients aged 4-15 years and 20 age matched normal controls have been selected; and their blood glucose level estimated together with assessment of their serum NO, fructosamine, albumin, creatinine, and both SGPT and SGOT enzymes. Correlation studies was done between blood glucose and serum fructosamine; blood glucose and serum NO; between serum fructosamine and serum NO.

**Results and Conclusion:** Estimation of serum albumin, creatinine and the liver enzymes SGPT, SGOT were all in the normal range, which points to

normal renal function and hepatic status in the diabetic patients (creatinine 1.22 mg/dl), SGPT (6.68 IU/L), SGOT (7.30 IU/L), while serum albumin was (4.59 gm/dl). However, there was a significant rise of blood glucose (374.53 mg/dl) and serum fructosamine (263.28 u mol/L); and a significant decrease in serum NO (27.26 u mol/L) in the diabetic group, compared with the normal controls. (glucose 84.40 mg/dl , fructosamine 153.15 u mol/L, and NO 46.65 u mol/L). Correlation study of blood glucose and serum NO showed a highly significant negative relationship between the two parameters in the diabetic group ( $r = -0.8469$ ). Again, correlation study between blood glucose and fructosamine showed a highly significant positive correlation ( $r = +0.7400$ ). At the same time, correlation study between serum fructosamine and serum NO showed a highly significant negative relationship ( $r = -0.7500$ ). This study came to the conclusion, that, the significant low NO production in type I diabetics, together with the low carrying capacity of glycated serum albumin, may result in low serum reservoir of this substance. This study recommends better control of blood glucose in all diabetics; And suggests the routine prescription of nitrates and/or ACE inhibitors to older diabetics to compensate for or to improve their endothelial dysfunction; and the future study of this suggestion.



# **Introduction & Aim of the Work**



