

**ALIMENTARY GLUCOSURIA
A RECONSIDERATION**

THESIS

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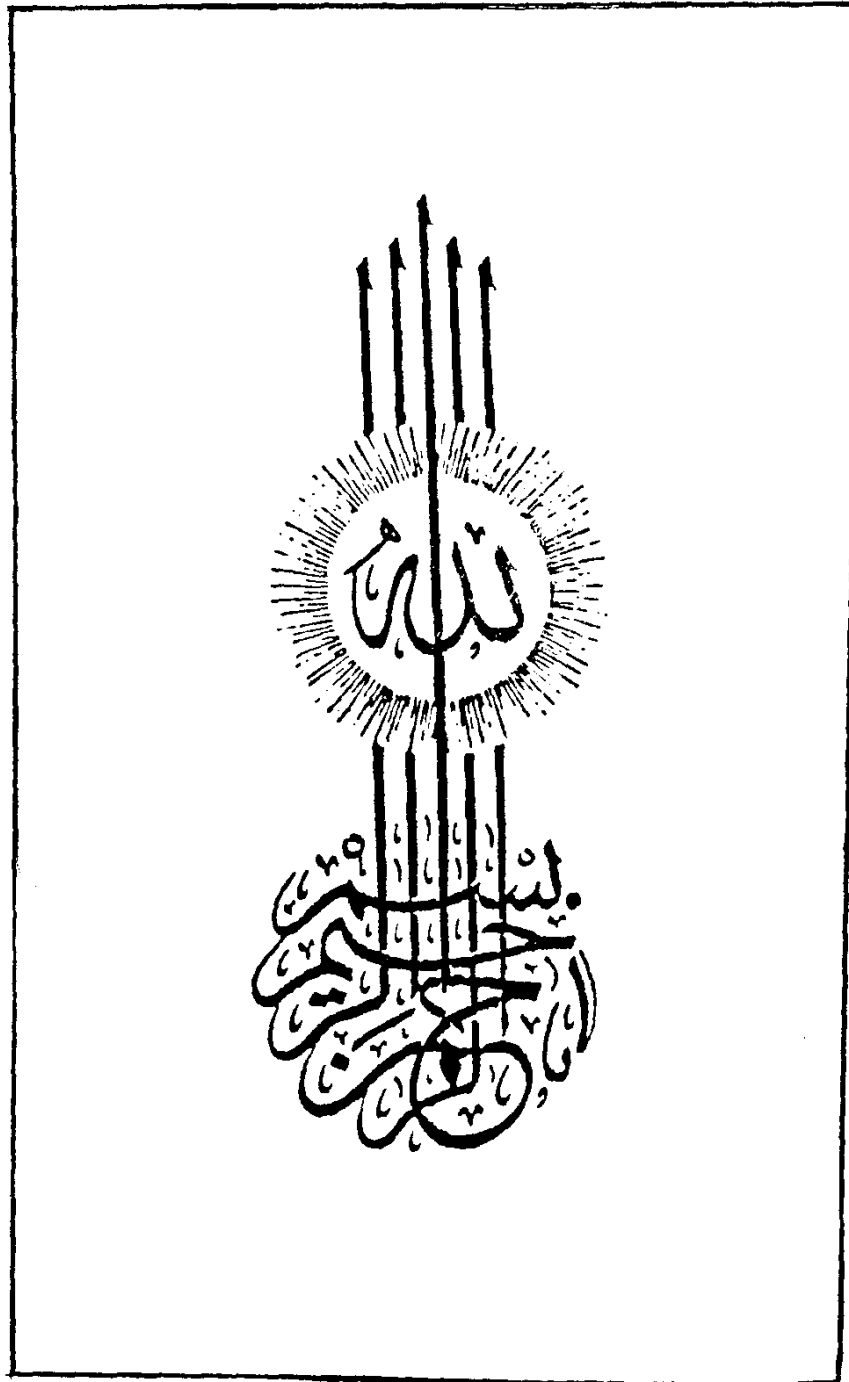
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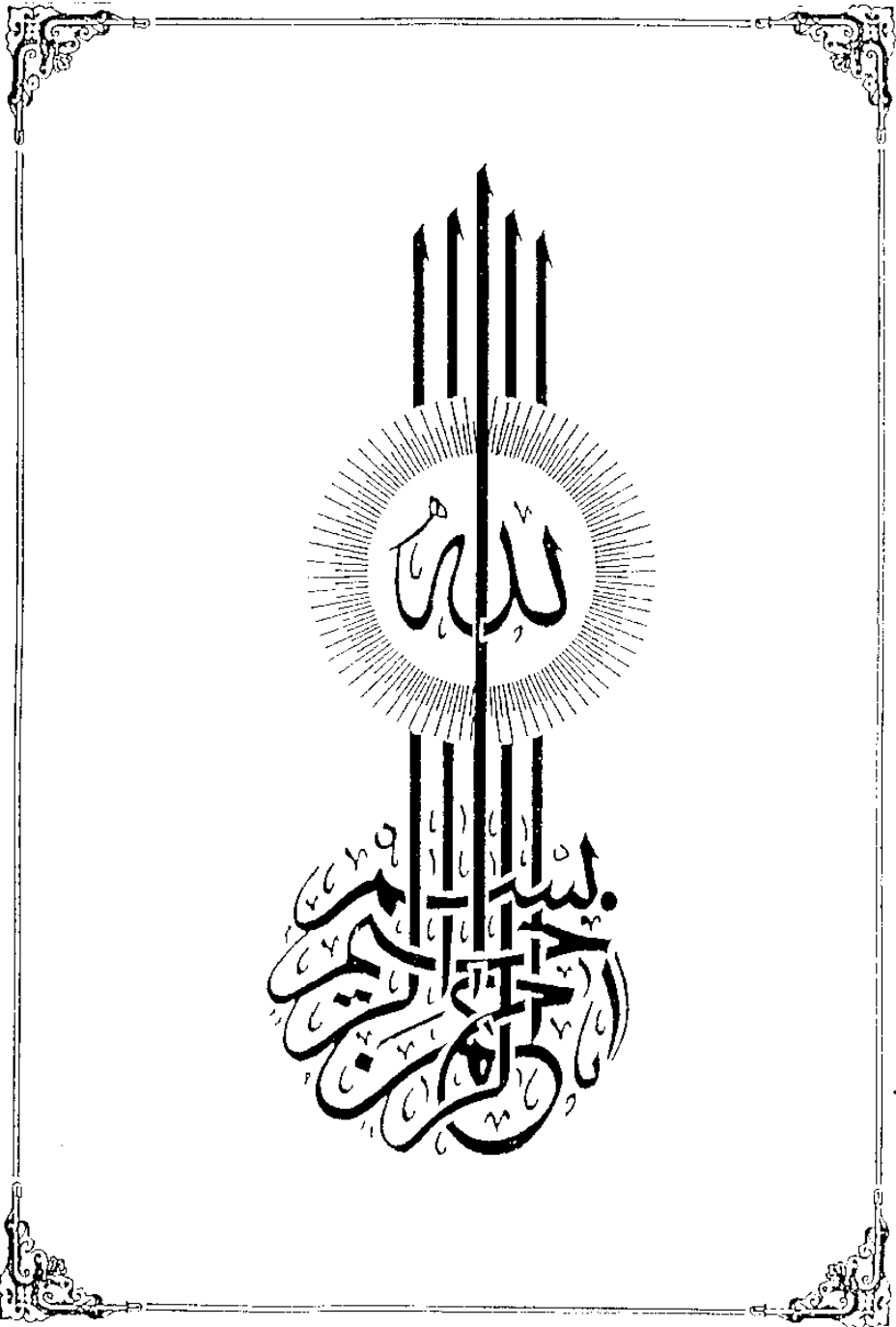
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ABBREVIATIONS

C.N.S.	: Central nervous system.
EF.A.	: Free fatty acid.
OGTT.	: Oral glucose tolerance test.
D.M.	: Diabetes mellitus.
C.A.M.P.	: Cyclic adenosine monophosphate.
GLF.	: Gastro intestinal factor.
M.W.	: Molecular weight.
NSILA.	: non suppressible insulin like activity.
IRI.	: Immuno reactive insulin.
NADPHA	: reduced Nicotinamide adenine dinucleotide phosphate.
ATP	: Adenosine trinucleotide phosphate.
α	: alpha.
B	: Beta.
M	: Molar
G-6-P	: glucose six phosphate.
T _m G	: Transfer maximum of glucose.
C	: Carbon.
B.S.L.	: Blood sugar level.

Aim of work:

The term alimentary glucosuria is traditionally employed to designate the urinary excretion of glucose by certain apparently normal individuals after the ingestion of excessive amounts of carbohydrates.

The significance of postprandial glucosuria , transient glucosuria and their relations to diabetes is still not well understood.

So we would like to study whether these phenomenae reflect a diabetic state or a different pattern of intestinal absorption, or any other disturbances in glucose homeostasis.

This thesis had not been submitted for any
degree at this or at any other University.

Magda Abd EL Meguid Amer.

INTRODUCTION

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It is well established that glucose homeostasis is elegantly preserved through the integration of many hormonal and neural signals. This applies both to the fasting state and with big carbohydrate meals. Failure of the blood glucose level to fall to 120 mg/100ml in two hours after oral ingestion of glucose is usually taken to indicate relative or absolute insulin deficiency i.e. diabetic state (William, 1981). It is normally considered that the peak blood glucose level should be below the renal threshold i.e. 180 mg/100ml with no glucosuria.

Along the course of glucose tolerance tests and in certain situations, the peak blood sugar level may be found higher than 180 mg/100 ml the two hours post prandial value is within normal or even low, this is usually taken to imply a delay in hepatic storage "lag" or to increased intestinal absorption. The former may be seen in liver disease and the latter is sometimes seen in some cases of thyrotoxicosis or after gastrectomy or gastrojejunostomy.

Such a curve may be even found in apparently normal individual "alimentary glucosuria". It is this last term that one really doubts and intended to reconsider with insulin assay in the face of increasing glucose loads in the same individual.

REVIEW OF LITERATURES

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Glucose Homeostasis

Glucose is the only nutrient that can be utilized by the brain, retina and germinal epithelium of the gonads. Therefore it is important to maintain a blood glucose concentration at an optimal level to provide this necessary nutrition. This depends on a balance of glucose entering and leaving the extracellular compartment (Levine, 1970).

The regulation of this balance depends upon the action of insulin anti-insulin hormones. (hyperglycemic hormones). for example, after a carbohydrate meal, the blood glucose level increases, stimulates the secretion of insulin which tends to decrease the blood glucose level.

On the other hand, during fasting, the blood glucose level decreases stimulating the secretion of anti-insulin hormones which increases the blood glucose level. So it is clear that there are factors adding glucose to blood and factors removing glucose from blood (opposing factors). The result is a condition of glucose equilibrium. This is called Homeostatic mechanism. (Guyton et al., 1978).

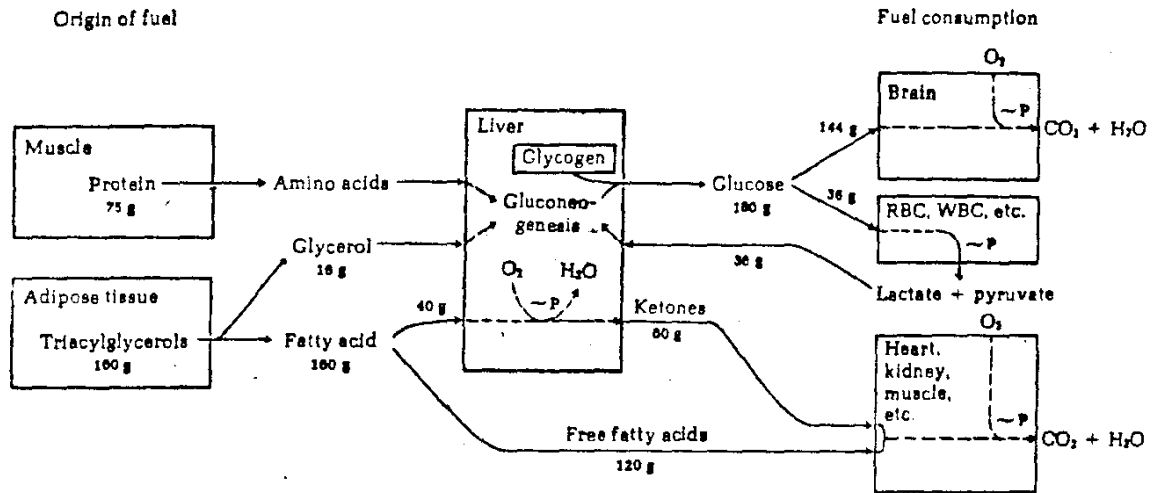
After glucose ingestion, the rate and magnitude of the rise in circulating glucose is determined by the

balance between the rate of glucose absorption from the gastro intestinal tract and the homeostatic mechanisms which serve to limit extracellular glucose accumulation. With respect to the latter, the elevation in plasma glucose caused by glucose ingestion is minimized by the prompt suppression of endogenous glucose production from the liver and more importantly by the uptake of exogenous glucose from the portal and systemic circulation. These processes are largely regulated by enhanced insulin secretion by the pancreatic islets and the release of gastro intestinal hormones (De Fronzo et al, 1978).

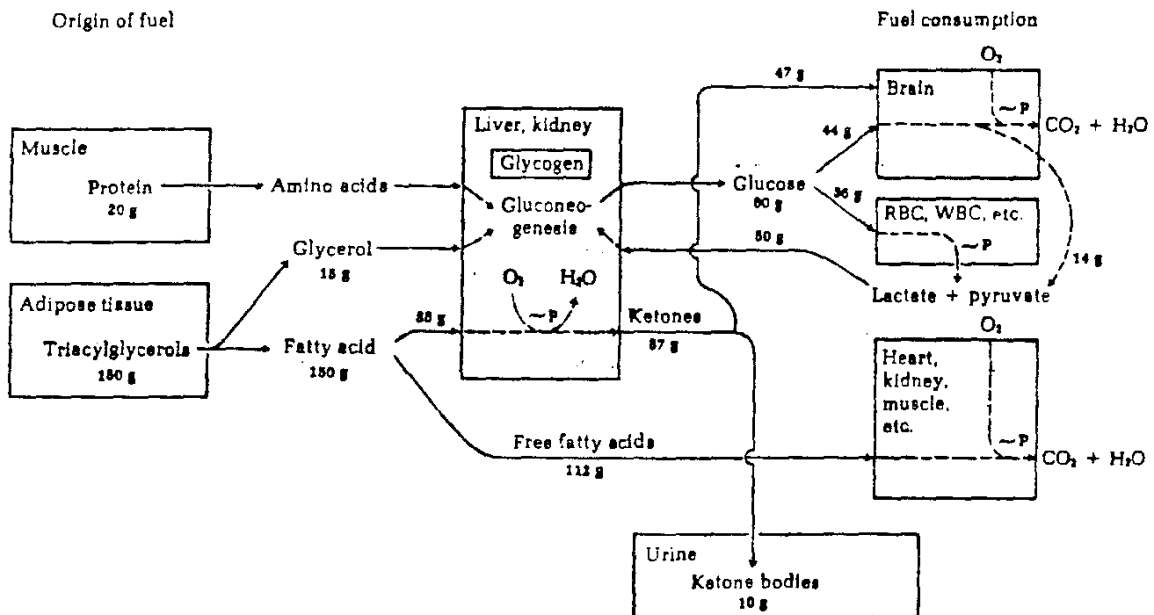
Homeostasis is preserved however during fasting by altering a number of metabolic pathways mediated by an integration of neural and hormonal signals to mobilize stores of glucose to insure proper functioning of the central nervous system (C.N.S.) mainly brain which oxidizes 100 gm of glucose/min to CO_2 and H_2O . In the average 70 Kg man, caloric reserves reside in adipose tissue triglycerides (85%), muscle protein (15 %) and muscle and liver glycogen (< 1%) (Fernstrom and Wurtman, 1974). Between meals and during emergent situations induced by stress or exercise, hepatic glycogen is the primary source for maintaining glucose concentration, with gluconeogenesis contributing less than 25%. The body attempts to conserve protein for mechanical catalytic processes within

the cell (Zak et al., 1979). Consequently free fatty acids and ketones from the liver are the major fuels used by most tissues. When food deprivation is extended muscle becomes almost entirely dependent upon free fatty acid and keto acids for its energy needs. These adaptive processes whereby that substitutes for glucose in muscle, are also applicable to the C.N.S. then all the fat reserve is depleted and muscle proteolysis and hepatic conversion of amino acids into glucose are augmented in order to maintain normal level of glucose necessary for the brain as shown in the following diagram.

Fasting man (36 h)



Fasting man, adapted (5-6 weeks)



The origin and consumption of fuel in man fasting for 36 hrs and 5-6 weeks-(Lenhinger, 1978).