شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومـــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قَسَـــم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

يجب أن

تحفظ هذه الأفلام بعيداً عن الغبار

في درجة حرارة من 15 – 20 مئوية ورطوبة نسبية من 20-40 %

To be kept away from dust in dry cool place of
15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

B 1.920

Acknowledgment

I wish to express my sincere thanks and indebtedness to ***Prof. Dr. Zeinab Ahmad Ismail*** Professor and Head of Clinical Pathology Dept., Faculty of Medicine, El-Minia University for her kind support and continuos encouragement all through.

I am greatly indebted to ***Dr. Ahmad Abdel-Samie Omran***, Assistant Professor of Clinical pathology, Faculty of Medicine, El Minia University for his kind and indispensable guidance. He had provided me with valuable information and influenced the work with his personal experience.

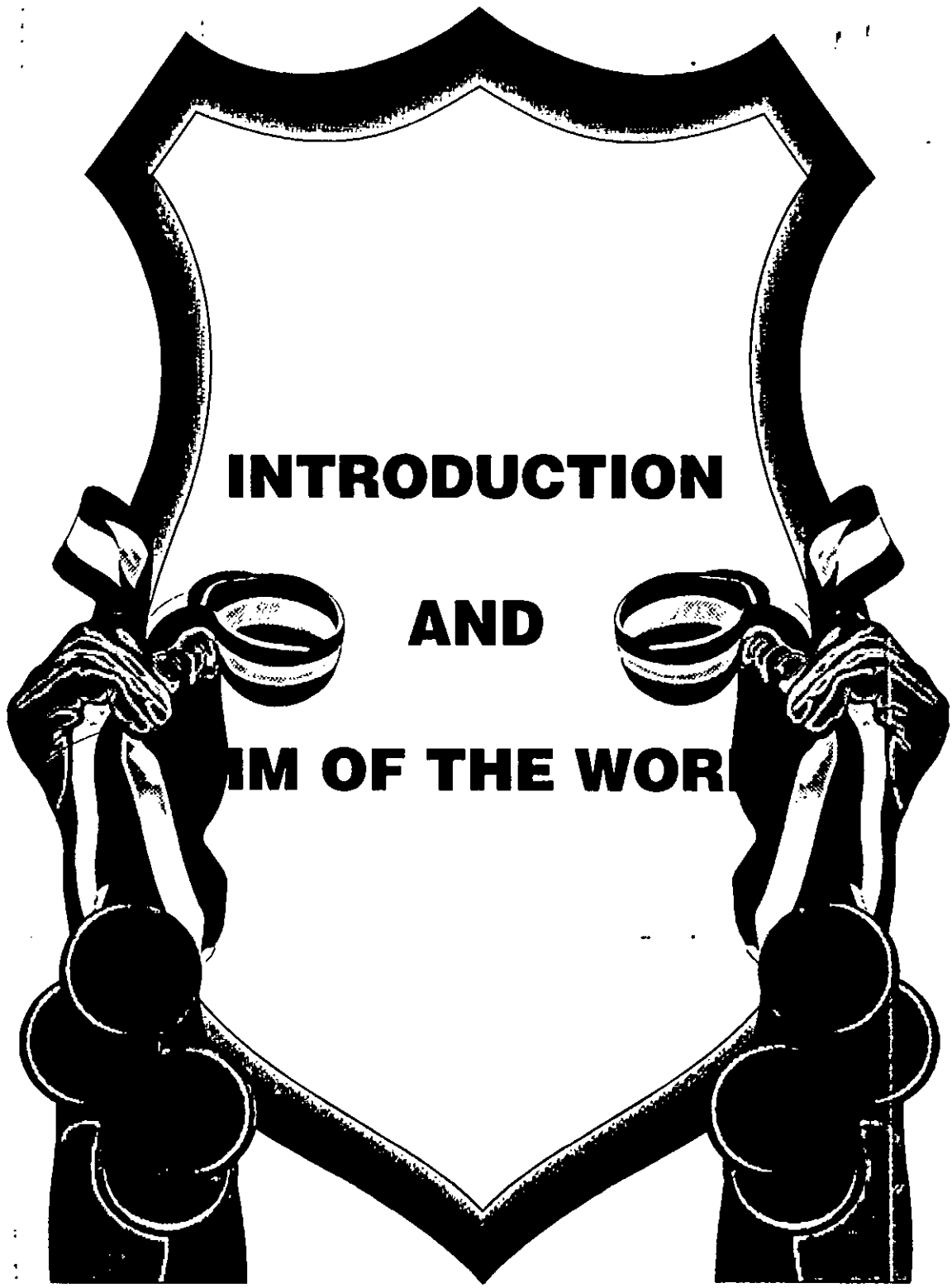
My deepest thanks, gratitude and indebtedness to ***Dr. Esmat Abdel-Aziz El-Sharkawi***, Lecturer of Clinical pathology, Faculty of Medicine, El-Minia University, for her advice, encouragement and great help throughout the research work. She was quite helpful and willing to guide me throughout this study.

Much thanks to ***Dr. Khaled Salah Osman,*** Lecturer of Clinical pathology, Faculty of Medicine, El-Minia University, for his great help and co-operation.

CONTENTS

LIST OF ABBREVIATIONS

Apo	: Apolipoprotein
BMI	: Body mass index
CM	: Chylomicrons
COHB	: Carboxy haemoglobin
ELISA	: Enzyme - linked immuno sorbent assay
FFA	: Free fatty acids
FH	: Familial hypercholesterolaemia
HbAIc	: Glycosylated haemoglobin
HDL-C	: High - density lipoprotein cholesterol
IDDM	: Insulin – dependent diabetes mellitus
IDL-C	: Intermidate - density lipoprotein cholesterol
LCAT	: Lecithin cholesterol acyl transferase
LDL-C	: Low - density lipoprotein cholesterol
Lp (a)	: Lipoprotein (a)
MCV	: Mean cell volume
PAI-1	: Plasminogen activator inhibitor
SI	: Smoking index
t-PA	: Tissue plasminogen activator
VLDL-C	: Very - low - density lipoprotein cholesterol



INTRODUCTION

AND

IM OF THE WOR

INTRODUCTION AND AIM OF THE WORK

Smoking is associated with increase in coronary heart disease risk that may partly be due to smoking-related changes in intermediate risk factors such as lipid levels, fibrinogen and blood pressure (Cullen et.al., 1998).

Lipids have an important role in virtually all aspects of biological life, serving as hormones or hormone precursors, aiding in digestion, providing energy storage and metabolic fuels, acting as functional and structural components in biomembranes and forming insulation to allow nerve conduction or to prevent heat loss. The causative relationship between plasma lipids, lipoproteins and atherosclerosis has been established conclusively in the past decade. (Evan and Gray, 1996).

Lipoprotein (a) [Lp (a)] is a plasma particle closely related to the low-density lipoprotein. It was firstly, described by Kare Berg in 1963, and its structure is now well characterized (Eaton et. al., 1987 & Scanu and Fless, 1990). Lp (a) contains a cholesterol-rich core that is covered by a complex of apoprotein B-100 covalently linked to apoprotein (a). Sequencing of apoprotein (a) has shown a high degree of homology with plasminogen, a key protein of the fibrinolytic system (Ramirez et. al., 1992).

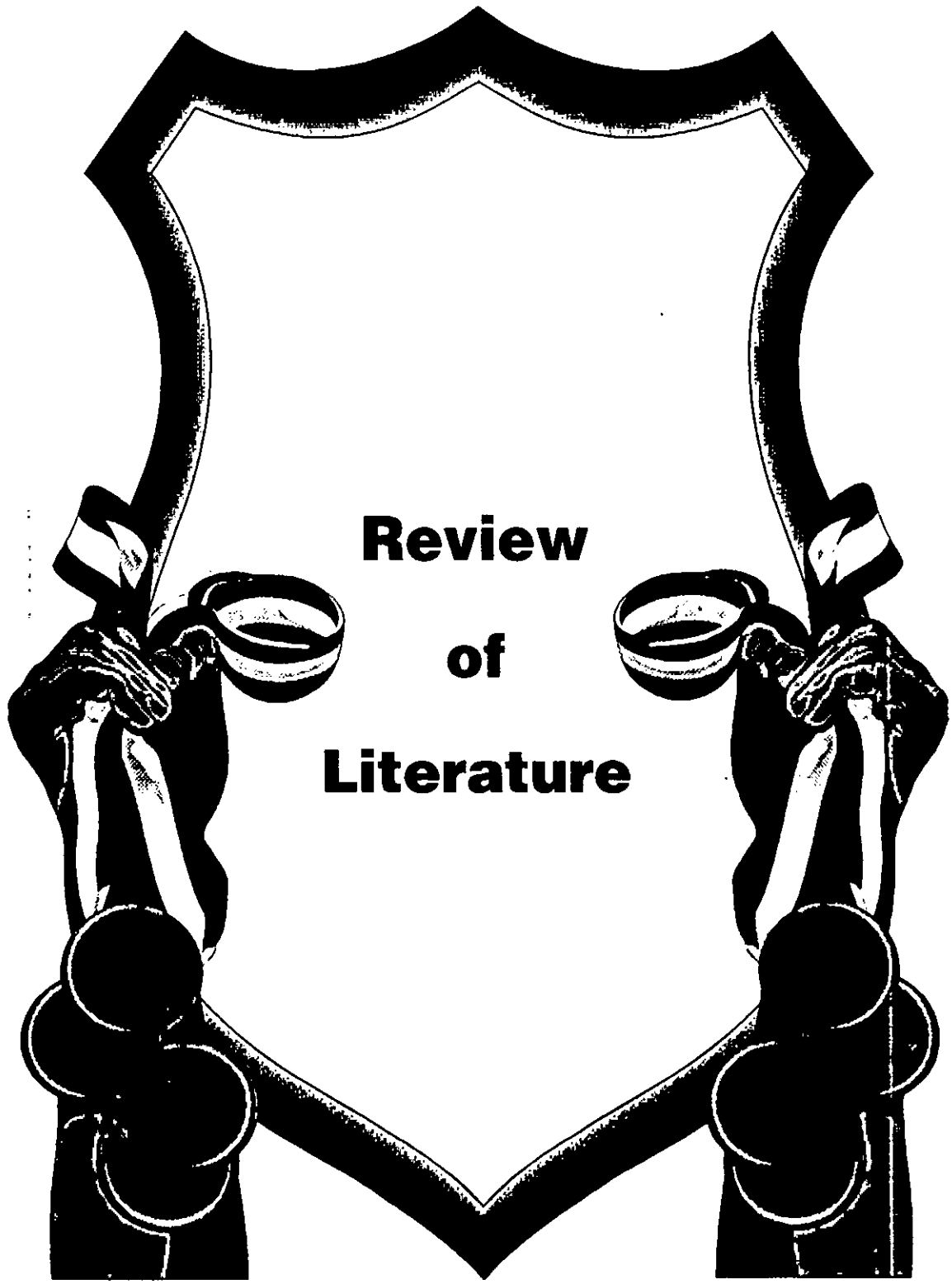
Synthesis of Lp (a) occurs in the liver. Its clearance from the circulation is as yet unknown (Stein and Myers, 1994).

Recently, a high plasma level of Lp (a) has been considered an independent risk factor for atherosclerosis and its sequelae, particularly myocardial infarction (Nakata et. al., 1993).

Serum Lp (a) level is a prominent lipidic predictor for ischaemic stroke in addition to the general risk factors such as history of hypertension , family history of stroke and cigarette smoking (Peng et.al., 1999)

High lipoprotein (a) has been observed in patients with ischaemic heart disease and cerebrovascular disease. Lp (a) is actually thought to be an independent risk factor for coronary disease (Prior et.al., 1995)

THE AIM OF THIS WORK is to study changes in serum lipids and lipoproteins particularly lipoprotein (a) in cigarette smokers compared to non-smokers.


Review
of
Literature

Smoking

Introduction

Whatever the origin of tobacco plant, it is likely that the particularly pleasurable consequence of inhaling its smoke were discovered more or less by chance. The history of western man's association with tobacco dates since 1492 (Stepney, 1980).

In the Middle East, local smoking patterns are not confined to cigarettes, goza & shisha are widely practiced (Salem et.al., 1973).

Model in 1985 defined what is called smoker's face as having one or more of the following:

1- Lines or wrinkles of the face.

2- A gauntness of facial features with prominence of the underlying bony contour.

3- An atrophic slightly pigmented grey appearance of the skin.

4- A plethoric slightly orange-purple and red complexion.

Such identification is important as smokers frequently require demonstration of the physical effects of smoking to assist them in stopping this habit.

Smoking behaviour

There is strong evidence that starting smoking is related to social factors, particularly the influence of parents and peer groups. Initiation of the

smoking habit is around puberty especially in those teenagers of smoking mothers. Having a smoking friend or an older brother or sister has got even more influence. The effect of the behaviour of parents, peers and siblings on childhood smoking is presumably mediated partly through increased exposure to cigarettes and partly through imitation and example. However, individual differences in personality-particularly in orientation towards the kind of psychological rewards offered by smoking must also be relevant. Taste and smell also influence smoking especially by a civilized man. Smoking is a mean of taking substance which excites sensory organs in the lips, mouth and throat and provides sensations of touch, taste and irritation. Freud in 1905 argued that smoking and drinking (together with thumb suckling in childhood) were primitive autoerotic activities found especially in individuals in whom there was a constitutional intensification of the erotogenic significance of the labial region. Some smokers explained their heavy smoking as insufficient weaning; they must have something to suck (Stepney, 1980).

Smoking index

Nitti in 1976 defined smoking index (SI) as the product of multiplying the number of cigarettes smoked per day by the number of years of smoking. He thus categorized smokers into:

- *Mild*: having (SI) less than 200.
- *Moderate*: having (SI) 200-600.
- *Heavy*: having (SI) more than 600.

On the other hand Franklin, (1961) defined heavy smokers as those who are smoking more than 30 pack-year and light smokers as smokers of less than 8 pack-year (Pack-year: number of packs smoked per day x number of years of smoking).

Composition of cigarette smoke

Cigarette smoke consists of gases, vapourized liquids and particles, many of which are minute droplets. If smoke is condensed by cooling or passed through a filter, a foul smelling dark brown material is obtained, the tar fraction. Over 1000 different substances have been identified in tobacco smoke, including nicotine, phenols, hydrocarbons, aldehydes, ketones, organic acids and alcohol in the particulate phase. The gas phase consists mainly of nitrogen, oxygen, carbon monoxide and carbon dioxide with traces of nitrogen oxide, ammonia, cyanides, aldehydes & ketones, acrolein and acids (Horsfield, 1981).

From the medical and physiological points of view the constituents of tobacco smoke may be divided into four groups:

I. Gases

The most important of these physiologically is carbon monoxide, other potentially toxic gases include cyanides and oxides of nitrogen

II. Nicotine

Nicotine has been consumed in the from of tobacco and other plants for many hundreds of years. The failure to quit smoking is attributed in large part to the addictive properties of nicotine.