

11-2016

Glycemic Index of Foods, Adiposity and Metabolic Syndrome Risk in Emirati Young Adults

Maysm Nezar Mohamad

Follow this and additional works at: https://scholarworks.uaeu.ac.ae/all_theses

Part of the [Food Studies Commons](#)

Recommended Citation

Mohamad, Maysm Nezar, "Glycemic Index of Foods, Adiposity and Metabolic Syndrome Risk in Emirati Young Adults" (2016).
Theses. 687.

https://scholarworks.uaeu.ac.ae/all_theses/687

This Dissertation is brought to you for free and open access by the Electronic Theses and Dissertations at Scholarworks@UAEU. It has been accepted for inclusion in Theses by an authorized administrator of Scholarworks@UAEU. For more information, please contact fadl.musa@uaeu.ac.ae.



United Arab Emirates University

College of Food and Agriculture

GLYCEMIC INDEX OF FOODS, ADIPOSITY AND METABOLIC
SYNDROME RISK IN EMIRATI YOUNG ADULTS

Maysm Nezar Mohamad

This dissertation is submitted in partial fulfilment of the requirements for the degree
of Doctor of Philosophy

Under the Supervision of Dr. Ayesha Salem Al Dhaheri

November 2016

Declaration of Original Work

I, Maysm Nezar Mohamad, the undersigned, a graduate student at the United Arab Emirates University (UAEU), and the author of this dissertation entitled “*Glycemic Index of Foods, Adiposity and Metabolic Syndrome Risk in Emirati Young Adults*”, hereby, solemnly declare that this dissertation is my own original research work that has been done and prepared by me under the supervision of Dr. Ayesha Salem Al Dhaheri, in the College of Food and Agriculture at UAEU. This work has not previously been presented or published, or formed the basis for the award of any academic degree, diploma or a similar title at this or any other university. Any materials borrowed from other sources (whether published or unpublished) and relied upon or included in my dissertation have been properly cited and acknowledged in accordance with appropriate academic conventions. I further declare that there is no potential conflict of interest with respect to the research, data collection, authorship, presentation and/or publication of this dissertation.

Student's Signature:  _____

Date: 20-12-2016

Copyright © 2016 Maysm Nezar Mohamad
All Rights Reserved

Advisory Committee

1) Advisor: Ayesha Al Dhaheri

Title: Associate Professor

Department of Nutrition and Health

College of Food and Agriculture

2) Co-advisor: Antonios Zampelas

Title: Professor

Department of Nutrition and Health

College of Food and Agriculture

3) Member: Syed Mahboob Shah

Title: Associate Professor

Public Health Institute

College of Medicine and Health Sciences

4) Member: Christiani Jeya Henry

Title: Professor

Singapore Institute for Clinical Sciences

APPROVAL OF THE DOCTORATE DISSERTATION

This Doctorate Dissertation is approved by the following Examining Committee Members:

- 1) Advisor (Committee Chair): Ayesha Salem Al Dhaheri

Title: Associate Professor

Nutrition and Health Department

College of Food and Agriculture

Signature  _____

Date: 27th Nov, 2016

- 2) Member: Milos Ljubisavljevic

Title: Professor

Department of Physiology

College of Medicine and Health Sciences

Signature  _____

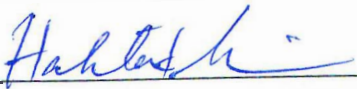
Date: 27th Nov, 2016

- 3) Member: Habiba Ali

Title: Associate Professor

Nutrition and Health Department

College of Food and Agriculture

Signature  _____

Date: 27th Nov, 2016

- 4) Member (External Examiner): Kees de Graaf

Title: Professor

Department of Agrotechnology and Food Sciences (Human Nutrition)

Institution: Wageningen University (Netherlands)

Signature  _____

Date: 27th Nov, 2016

This Doctorate Dissertation is accepted by:

for

Acting Dean of the College of Food and Agriculture: Professor Wissam Ibrahim

Signature  Date 20-12-2016

Dean of the College of ~~the~~ Graduate Studies: Professor Nagi T. Wakim

Signature  Date 20/12/2016

Abstract

This dissertation is concerned with determining the prevalence of metabolic syndrome (MetS) in Emirati females aged 17–25 years and its relation to overweight and obesity. It also aims to determine the glycemic index (GI) and glycemic load (GL) values for traditional Emirati foods that have not been tested yet.

In a cross-sectional study design, anthropometric measurements, blood pressure and biochemical measurements were collected from a total of 555 Emirati female college students and the prevalence of MetS was concluded. Furthermore, at least fifteen healthy subjects participated in the measurement of GI and GL values for each of the twenty-three Emirati test foods.

This study showed a high prevalence of MetS among college female young adults aged 17–25 years (6.8%). Of the 555 participants enrolled, 23.1% were overweight and 10.4% were classified as obese. MetS was significantly associated with obesity, waist-hip ratio, glycated hemoglobin and high sensitivity C-reactive protein. The current study also provides a comprehensive food composition table including proximate data, minerals, vitamins, lipids, and sugars contents, along with GI and GL values of twenty-three locally consumed foods in the UAE which could be utilized in offering better dietary recommendations for the Emirati population.

The results advocate the need for MetS identification and immediate intervention programs to improve the future health of this youthful group.

Keywords: Metabolic syndrome, Emirati, Young adults, Glycemic index, Glycemic load, Emirati foods, Diabetes, Obesity.

Title and Abstract (in Arabic)

مؤشر نسبة سكر الدم لبعض الأطعمة التقليدية الإماراتية، السمنة ومخاطر متلازمة الأيض عند الفتيات الشابات في الإمارات

الملخص

تهدف هذه الأطروحة لتحديد مدى انتشار متلازمة الأيض لدى الإناث اللواتي تتراوح أعمارهن بين 17 و25 عاماً في الإمارات وعلاقتها بزيادة الوزن والسمنة. كما أنها تُعنى بتحديد مؤشر نسبة سكر الدم لبعض الأطعمة التقليدية الإماراتية التي لم يتم اختبارها بعد.

في دراسة مستعرضة، تم جمع القياسات الجسمانية وقياس ضغط الدم والقياسات البيو كيميائية من 555 طالبة في جامعة الإمارات العربية المتحدة، واستخدمت البيانات لتحديد مدى انتشار متلازمة الأيض لديهن. كما شارك خمسة عشر شخصاً أصحاء على الأقل في قياس قيم مؤشر نسبة سكر الدم لكل من الأطعمة الإماراتية الثلاثة وعشرين التي تم اختيارها.

أظهرت هذه الدراسة ارتفاع معدل انتشار المتلازمة الأيضية بين الشابات اللواتي تتراوح أعمارهن بين 17 و25 عاماً (6.8%). كما تبين بأن 23.1% من الشابات المشاركات في الدراسة يعانون من زيادة الوزن و10.4% منهن يعانون من السمنة المفرطة. إضافة إلى ذلك، فقد كشفت الدراسة عن وجود ارتباط كبير بين المتلازمة الأيضية وكل من السمنة، ونسبة الخصر للورك، والهيموجلوبين السكري وبروتين سي التفاعلي عالي الحساسية. كما خلصت الدراسة إلى توفير جداول غذائية شاملة لثلاث وعشرين من الأطعمة التقليدية الإماراتية. بما في ذلك جداول عن نسب المركبات الغذائية والمعادن والفيتامينات والدهون، والسكريات، ومؤشر نسبة سكر الدم لهذه الأطعمة. وتعد هذه الجداول مرجعاً هاماً يمكن استخدامه لتقديم توصيات غذائية أفضل لسكان الإمارات.

وعكست نتائج هذه الدراسة عن الحاجة الملحة للكشف المبكر عن المتلازمة الأيضية وضرورة تطوير برامج للتدخل الفوري سعياً لتحسين الصحة المستقبلية لهذه الفئة العمرية من الشباب.

مفاهيم البحث الرئيسية: مؤشر نسبة سكر الدم، المتلازمة الأيضية، السمنة، الأطعمة التقليدية الإماراتية.

Acknowledgements

First and foremost, I wish to express my sincere gratitude to my advisor and mentor, Dr. Ayesha Al Dhaheeri. Thank you for having faith in me. Your time, expertise, support and teachings have been invaluable and dearly appreciated.

This study would not have been possible without the generous scholarship from the United Arab Emirates University. I would also like to thank my participants for their kindness and willingness to take part in this study, this study would not have been possible without them.

My thanks also go to the members of my committee, Professor Antonios Zampelas, Dr. Syed Shah, and Professor Jeya Henry for providing many valuable comments that improved the contents of this dissertation.

Special thanks also go to Mr. Amjad Jarar, Ms. Fatima Al Maqbaly and Mr. Usama Souka for their support, assistance and time. My gratitude goes to all of my friends who directly or indirectly helped me to complete this dissertation.

I wish to express my unqualified thanks to my Mom and dad who encouraged my academic interests from day one. They raised me to study hard and to give priority in my life to the quest for knowledge. I am also extremely grateful to my husband, sisters and brother for their unconditional love and support.

Dedication

I dedicate this dissertation to my beloved parents and family

Table of Contents

Title	i
Declaration of Original Work	ii
Copyright	ii
Advisory Committee	iv
Approval of the Doctorate Dissertation	v
Abstract	vii
Title and Abstract (in Arabic)	viii
Acknowledgements	ix
Dedication	x
Table of Contents	xi
List of Tables.....	xv
List of Figures	xviii
List of Abbreviations.....	xxi
Chapter 1: Introduction	1
Chapter 2: Review of Literature.....	5
2.1 Introduction to the Metabolic Syndrome	5
2.1.1 History of the Metabolic Syndrome	5
2.1.2 Definition of the Metabolic Syndrome	7
2.1.3 Diagnostic Criteria of the Metabolic Syndrome	8
2.2 Risk Factors of the Metabolic Syndrome.....	12
2.2.1 Genetics of the Metabolic Syndrome	12
2.2.2 Insulin Resistance.....	14
2.2.3 Abdominal Obesity	15
2.2.4 Glucose Intolerance.....	16
2.2.5 Elevated Blood Pressure	16
2.2.6 Atherogenic Dyslipidemia	17
2.2.7 Inflammation	18
2.3 Consequences of the Metabolic Syndrome.....	21
2.4 Management of the Metabolic Syndrome.....	26
2.4.1 AHA/NHLBI Guidelines	27
2.4.2 The ABCDE Approach	28
2.4.2.1 Assessment	29
2.4.2.2 Blood Pressure.....	29
2.4.2.3 Cholesterol	30
2.4.2.4 Diet/Diabetes Prevention.....	31
2.4.2.5 Exercise	35
2.4.3 Bariatric Surgery	36
2.5 Prevalence of the Metabolic Syndrome	38
2.6 Glycemic Index: History.....	44

2.6.1 Definition of the Glycemic Index and Glycemic Load.....	45
2.6.2 Methodology of the Glycemic Index	46
2.6.3 Factors Influencing the Glycemic Index	48
2.6.4 Glycemic Index in Health and Disease	49
2.6.4.1 Glycemic Index and Obesity	49
2.6.4.2 Glycemic Index and Diabetes	52
2.6.4.3 Glycemic Index and Cardiovascular Disease.....	53
2.7 Summary	55
Chapter 3: Materials and Methods	56
3.1 Introduction.....	56
3.2 Prevalence of the Metabolic Syndrome and its Component Factors among Female Students at United Arab Emirates University and their Association with Anthropometric Measurements.....	56
3.2.1 Study Population	56
3.2.2 Ethical Approval	58
3.2.3 Anthropometric Measurements	58
3.2.3.1 Height	60
3.2.3.2 Body Weight and Body Composition	60
3.2.3.3 Body Mass Index.....	61
3.2.3.4 Waist Circumference.....	62
3.2.3.5 Hip Circumference	63
3.2.3.6 Waist-Hip Circumference Ratio.....	64
3.2.3.7 Neck Circumference.....	64
3.2.3.8 Mid-Upper-Arm Circumference	65
3.2.3.9 Skinfold Thickness	65
3.2.4 Biochemical Measurements	67
3.2.4.1 Blood Collection	67
3.2.4.2 Analytical System	68
3.2.4.3 Lipid Assessment	70
3.2.4.4 Triglycerides.....	71
3.2.4.5 Total Cholesterol	71
3.2.4.6 Low Density Lipoprotein	72
3.2.4.7 High Density Lipoprotein.....	72
3.2.4.8 Fasting Plasma Glucose	73
3.2.4.9 High Sensitivity C-Reactive Protein	74
3.2.4.10 Hemoglobin and Glycated Hemoglobin.....	75
3.2.5 Blood Pressure Measurement.....	77
3.2.6 Power Analysis	79
3.2.7 Statistical Analysis	79
3.3 Glycemic Index (GI) Value of Commonly Consumed Foods in the UAE... 80	80
3.3.1 Study Population	80
3.3.2 Ethical Approval	81
3.3.3 Anthropometric Measurements	81
3.3.4 Blood Glucose	82
3.3.5 Test Foods	83
3.3.5.1 Chemical Analyses	85
3.3.5.2 Sample Preparation	86
3.3.5.3 Proximate Analysis	87
3.3.5.4 Exchange List Development	93

3.3.5.5 Determination of Minerals	94
3.3.5.6 Determination of Sugars.....	95
3.3.5.7 Determination of Lipids	96
3.3.5.8 Determination of Vitamins.....	98
3.3.5.9 Determination of Caffeine.....	100
3.3.6 Glycemic Index (GI) Procedure	100
3.3.6.1 Glycemic Index Calculation.....	103
3.3.6.2 Glycemic Load Calculation.....	104
3.3.7 Power Analysis	104
3.3.8 Statistical Analysis	105
Chapter 4: Prevalence of Metabolic Syndrome among Young Female Emirati Adults at United Arab Emirates University	106
4.1 Introduction.....	106
4.2 Results.....	109
4.2.1 Characteristics of Study Population	109
4.2.2 Prevalence of Metabolic Syndrome	111
4.2.3 Univariable and Multivariable Logistic Regression Analyses.....	114
4.2.4 High Sensitivity C-Reactive Protein and Metabolic Syndrome.....	119
4.2.5 Prevalence of Overweight/Obesity	120
4.2.6 Prevalence of Diabetes.....	122
4.3 Discussion	126
4.4 Conclusion	132
Chapter 5: Glycemic Index (GI) Value of Commonly Consumed Foods in the UAE.....	133
5.1 Introduction.....	133
5.2 Results and Discussion	137
5.2.1 Proximate Analysis	137
5.2.2 Minerals Analysis	142
5.2.3 Vitamins Analysis	150
5.2.4 Lipids Analysis	155
5.2.5 Sugars Analysis.....	158
5.2.6 Characteristics of Study Population	161
5.2.7 Glycemic Response.....	162
5.2.8 Glycemic Index and Glycemic Load Classifications	169
5.2.9 Exchange List.....	176
5.3 Conclusion	179
Chapter 6: Summary and Recommendations	182
6.1 Overall Summary	182
6.2 Recommendations.....	185
References	186
List of Publications	225
Appendices.....	226
Appendix 1: Information Sheet.....	226
Appendix 2: Consent Form.....	228
Appendix 3: Ethical Approvals.....	229
Appendix 4: Data Sheet	231
Appendix 5: Health Questionnaire.....	232

Appendix 6: Health Screening Questionnaire.....	234
Appendix 7: Information Sheet.....	236
Appendix 8: Data Sheet	238
Appendix 9: Ethical Approval	239
Appendix 10: Consent Form.....	240
Appendix 11: Graphs of the IAUC for all Test Foods.....	241
Appendix 12: Abstract for the 10th International Symposium on Body Composition.	253
Appendix 13: Abstract for the 12th Asian Congress of Nutrition.	254
Appendix 14: Research Paper 1	255
Appendix 15: Research Paper 2.....	263

List of Tables

Table 2.1: Brief History of the Metabolic Syndrome	6
Table 2.2: Summary of the Metabolic Syndrome’s Diagnostic Criteria according to the WHO, the EGIR, the NCEP ATPIII, and the AACE	9
Table 2.3: Summary of the Metabolic Syndrome’s Diagnostic Criteria according to the IDF, the AHA/NHLBI, and the harmonized definition IDF/AHA/NHLBI.....	11
Table 2.4: Summary of Systematic Reviews and Meta-analyses on the association of the MetS with CVD morbidity, CVD mortality, Diabetes, and All-Cause Mortality	25
Table 2.5: AHA/NHLBI Guidelines for Metabolic Syndrome Management	27
Table 2.6: AHA Guidelines for treating Atherogenic Dyslipidemia in the MetS.....	31
Table 2.7: Prevalence of the MetS among adults in GCC Countries.....	38
Table 2.8: Prevalence of the MetS in different countries.....	40
Table 2.9: Categories of Glycemic Index and Glycemic Load Values.....	46
Table 2.10: Main Factors Affecting the GI of Foods and Meals	49
Table 3.1: WHO Classification of Overweight in Adults According to Body Mass Index.....	62
Table 3.2: Cut-off Values of Waist Circumference Based on Ethnicity and Gender	63
Table 3.3: Recommended Cut Points for Lipid and Lipoprotein Level in Young Adults (20-24 years old).....	70
Table 3.4: Blood Test Levels for Diagnosis of Diabetes and Pre-diabetes.....	74
Table 3.5: High Sensitivity CRP Cut-off Points for CVD Risk Assessment.....	75
Table 3.6: Hemoglobin Levels for the Diagnosis of Anemia at Sea Level.....	76
Table 3.7: Classification of Blood Pressure	78
Table 3.8: Main ingredients used in the preparation of 23 traditional foods commonly consumed in the UAE.....	84
Table 3.9: Experimental portion size of test foods	102
Table 4.1: Demographic and Clinical characteristics of the study population by metabolic syndrome status	110

Table 4.2: Univariable analyses - Risk factors for MetS among young female adults aged 17 to 25 years (n=555), Al Ain, UAE.....	116
Table 4.3: Multivariable analyses - Risk factors for MetS among young female adults aged 17 to 25 years (n=555), Al Ain, UAE.....	117
Table 4.4: Subgroup multivariable analysis - Risk factors for MetS among young female adults aged 17 to 25 years (n=507), Al Ain, UAE – excluding 48 participants that had HbA1c \geq 6.5.....	118
Table 4.5: Distribution of the study population according to different measures of obesity	121
Table 4.6: Classification of the study population according to different standards for the diagnosis of diabetes and prediabetes	122
Table 4.7: Univariate logistic regression analysis model of the association between abnormal glycemic status based on fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) and selected variables among young Emirati female adults aged 17 to 25 years (n=555), Al Ain, UAE	124
Table 4.8: Adjusted analysis model of the association between abnormal glycemic status based on fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) and selected variables among young Emirati female adults aged 17 to 25 years (n=555), Al Ain, UAE	125
Table 5.1: Proximate Analysis of United Arab Emirates Traditional Dishes	141
Table 5.2: Micronutrient (Major Elements) Composition of United Arab Emirates Traditional Dishes	148
Table 5.3: Micronutrient (Trace Elements) Composition of United Arab Emirates Traditional Dishes	149
Table 5.4: Water-Soluble Vitamin Composition of United Arab Emirates Traditional Dishes	153
Table 5.5: Fat-Soluble Vitamin Composition of United Arab Emirates Traditional Dishes	154
Table 5.6: Lipid profile of United Arab Emirates Traditional Dishes	157
Table 5.7: Sugar Composition of United Arab Emirates Traditional Dishes	160
Table 5.8: Physical Characteristics of the Study Population	161
Table 5.9: Incremental Area Under the Curve (IAUC) for low GI test foods	164
Table 5.10: Incremental Area Under the Curve (IAUC) for medium GI test foods	166
Table 5.11: Incremental Area Under the Curve (IAUC) for high GI test foods	168

Table 5.12: Glycemic Index (GI) and Glycemic Load (GL) Values of United Arab Emirates Traditional Dishes.....	175
---	-----

Table 5.13: Meal Planning Exchange List of Twenty-Three United Arab Emirates Traditional Dishes.....	177
--	-----

List of Figures

Figure 2.1: Schematic Presentatin of the Metabolic Syndrome Risk Factors and Consequances.....	12
Figure 2.2: The Origins of the Metabolic Syndrome	13
Figure 2.3: Pro-inflammatory State of the Metabolic Syndrome.....	19
Figure 2.4: Hs-CRP and the Metabolic Syndrome	20
Figure 2.5: Prevalence of CHD Categorized by Presence of MetS and Diabetes.....	22
Figure 2.6: Summary of the ABCDE Approach with Recommendations	28
Figure 2.7: Change in the percentage of HbA1c during two years intervention of Medical Therapy, Gastric Bypass, or Biliopancreatic Diversion.....	37
Figure 2.8: Prevalence of the MetS in GCC Countries	39
Figure 2.9: The Incremental Area Under the Curve (IAUC) equals the sum of the areas A, B, C, D, E and F. Negative areas are not included.....	48
Figure 2.10: Changes in BMI during the follow-up for each intervention group.....	51
Figure 3.1: Study Participant’s Enrolment Process.....	57
Figure 3.2: Tanita Segmental Body Composition Analyzer BC-418	61
Figure 3.3: Lange Caliper.	66
Figure 3.4: Cobas® c111 Analyzer.....	69
Figure 3.5: HemoCue® Hemoglobin 201+ Analyzer	77
Figure 3.6: HemoCue® HbA1c 501 System.....	77
Figure 3.7: Omron HEM-907-E7 Digital Blood Pressure Monitor	79
Figure 3.8: HemoCue® Glucose 201+ Analyzer	82
Figure 3.9: Scaltec® SBA 31Analytical Electronic Balance	87
Figure 3.10: Mommert® Forced-Air Drying Oven	88
Figure 3.11: Muffle Furnace Oven and Crucibles	88
Figure 3.12: Foss 2300 Kjelttec Analyzer Unit	89
Figure 3.13: Foss Tecator 2020 Digester	89

Figure 3.14: Sotex System 2050	90
Figure 3.15: ANKOM 200 Fiber Analyzer	92
Figure 3.16: ANKOMTDF Dietary Fiber Analyzer	93
Figure 3.17: Varian ICP-Optical Emission Spectrometer model 710-ES.....	95
Figure 3.18: Waters High-Performance Liquid Chromatography (HPLC) System...	96
Figure 3.19: Young Lin 6500 Gas Chromatography System.....	97
Figure 3.20: Waters ACQUITY UPLC System.....	99
Figure 4.1: Number of Metabolic Syndrome (MetS) defining components present among UAEU young female adults 17-25 years (N = 555), Al Ain, UAE	111
Figure 4.2: Prevalence of metabolic syndrome components among UAEU young female adults 17-25 years (N = 555), Al Ain, UAE	112
Figure 4.3: Prevalence of metabolic syndrome by body mass index (BMI) category among young female adults aged 17 to 25 years (n = 555), Al Ain, UAE.....	113
Figure 4.4: Percentage of participants per number of metabolic syndrome components and BMI category among young female adults aged 17 to 25 years (n=555), Al Ain, UAE.....	113
Figure 4.5: Distribution of High Sensitivity C-reactive protein according to number of metabolic syndrome components present among young female adults aged 17 to 25 years (n=555), Al Ain, UAE. Box plots demonstrate minimum, maximum, median, 25th, and 75th percentile values for Hs-CRP.....	119
Figure 4.6: Distribution of Emirati young female adults aged 17 to 25 years (n=555) according to body mass index categories	120
Figure 4.7: Prevalence of abnormal glycaemic status based on fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) among Emirati young female adults aged 17 to 25 years (n=555), Al Ain, UAE	123
Figure 5.1: The incremental area under the blood glucose curves (IAUC) for low glycaemic index traditional meals. Standard errors of the mean values are represented by vertical bars.....	163
Figure 5.2: The incremental area under the blood glucose curves (IAUC) for medium glycaemic index traditional meals. Standard errors of the mean values are represented by vertical bars.....	165

Figure 5.3: The incremental area under the blood glucose curves (IAUC) for the high glycemic index traditional meals. Standard errors of the mean values are represented by vertical bars..... 168

List of Abbreviations

ADA	American Diabetes Association
AHA	American Heart Association
AOAC	Association of Official Analytical Chemists
Apo-B	Apo Lipoprotein B
ATP	Adult Treatment Panel
BMI	Body Mass Index
CDC	Centers for Disease Control and Prevention
CETP	Cholesterol Ester Transfer Protein
CHO	Carbohydrate
CRP	C - reactive protein
CVD	Cardiovascular Disease
DBP	Diastolic Blood Pressure
DXA	Dual X-ray absorptiometry
DM	Dry Matter
EGIR	European Group of Insulin Resistance

FAMEs	Fatty Acid Methyl Esters
FAO	Food and Agriculture Organization
FPG	Fasting Plasma Glucose
FRS	Framingham Risk Score
GC	Gas Chromatography
GCC	Gulf Cooperation Council
GI	Glycemic Index
GL	Glycemic Load
Hb	Hemoglobin
HbA1c	Glycated Hemoglobin
HC	Hip Circumference
HDL	High Density Lipoprotein
HPLC	High Performance Liquid Chromatography
Hs-CRP	High Sensitivity C - reactive protein
HTGL	Hepatic Triglyceride Lipase
HTN	Hypertension
IAS	International Atherosclerosis Society

IDF	International Diabetes Federation
IDL	Intermediate Density Lipoprotein
IL-6	Interleukin-6
LDL	Low Density Lipoprotein
MENA	Middle East and North Africa
MetS	Metabolic Syndrome
MUAC	Mid-Upper-Arm Circumference
NHLBI	National Heart, Lung, and Blood Institute
NICE	National Institute of Health and Clinical Excellence
NC	Neck Circumference
NCEP	National Cholesterol Education Program
NCD	Non-Communicable Disease
NDF	Neutral Detergent Fiber
NIH	National Institutes of Health
OGTT	Oral Glucose Tolerance Test
PAI 1	Plasminogen Activator Inhibitor 1
PCO	Polycystic Ovary Syndrome

RPM	Round Per-Minute
SBP	Systolic Blood Pressure
SCORE	Systematic Coronary Risk Evaluation
TC	Total Cholesterol
TG	Triglyceride
TNF- α	Tumor Necrosis Factor Alpha
T2D	Type 2 Diabetes
UAE	United Arab Emirates
UAEU	United Arab Emirates University
VLDL	Very Low Density Lipoprotein
WC	Waist Circumference
WHF	World Heart Federation
WHR	Waist-Hip Ratio
WHO	World Health Organization

Chapter 1: Introduction

Non-Communicable Diseases (NCDs) are the leading cause of deaths worldwide, with diabetes mellitus the fourth major cause of NCD deaths [1]. Diabetes is a chronic disease characterized by abnormalities in the metabolism of carbohydrate, fat, and protein, along with hyperglycemia. During the past three decades, the prevalence of diabetes mellitus in the world has doubled, which has presented a great challenge to public health in every country. The International Diabetes Federation (IDF) estimated the global prevalence for diabetes mellitus to be 8.3% in 2014, however, it is estimated that around 46% of people with diabetes remain undiagnosed [2]. Moreover, the IDF expects 205 million new cases of diabetes mellitus by the year 2035.

The United Arab Emirates (UAE) has been ranked as having the fifth highest prevalence in the Middle East and North Africa (MENA) region with 19%, coming after other Gulf Cooperation Council (GCC) countries, like Qatar, Saudi Arabia and Bahrain [2]. In 2010, a survey conducted in the UAE reported the prevalence of undiagnosed diabetes mellitus and pre-diabetes to be 14.6% and 31% respectively [3]. This indicates the great number of people living with undiagnosed diabetes mellitus and pre-diabetes in the country. The existence of pre-diabetes combined with dyslipidemia, elevated blood pressure, and obesity leads to the diagnosis of Metabolic Syndrome (MetS) [4]. The MetS has a major public health impact through high disease prevalence, significant downstream pathophysiological effects, and enormous financial responsibilities. Therefore, the diagnosis of the syndrome and its components at an early stage would assistance in preventing the development of type

2 diabetes mellitus (T2DM), hypertension, and cardiovascular disease (CVD), and may help reduce the risk of morbidity and mortality.

The prevalence of MetS was reported to be 32.9% among Emirati men, 45.9% among Emirati women, and the total prevalence was 40.5%, based on the IDF definition for MetS [5]. This alarming prevalence is thought to be emerging from the high prevalence of MetS among younger age groups (13% of Emirati adolescents suffered from MetS) [6]. The observed increasing incidence of MetS and pre-diabetes among children, adolescents, and younger adults caused an equal increase in the need for understanding the etiology of the disease, along with developing better screening practices, and improved prevention programs in the UAE. Anthropometric measures have been reported in several studies to assess in the diagnosis of MetS and its components [7-9]. Thus, identification of anthropometric measures of obesity that best identify MetS among the Emirati population is essential to improve screening and facilitate prevention.

The causes behind the epidemic of MetS and T2DM are part of a multifactorial genetic and social system, controlling behavioral and environmental influences [10]. However, there are several key risk factors believed to be contributing to the occurrence of the MetS. These include: weight gain and obesity[11]; sedentary lifestyle with low physical activity [12, 13]; ethnicity [14]; family history of diabetes and poor dietary habits [15]; while other factors could be cigarette smoking and alcohol consumption [16]. Accordingly, dietary management could have a great impact on the number of people affected by diabetes, or preventing it in those at high risk of developing the disease [17]. The American Diabetes Association recommended an effective ongoing support program targeting glycemic control,

weight loss of seven per cent of body weight and increasing physical activity to at least 150 min/week of moderate activity for people living with pre-diabetes [16]. Dietary management was found to be dependent on the quality and quantity of nutrient intake. Studies suggested that the total amount of carbohydrates or fat in the diet does not seem to be associated with T2DM risk, but specific forms of carbohydrates or fat were found to be associated with the disease [18, 19]. For example; the ability of carbohydrates to increase blood glucose and insulin levels after food ingestion differs, and that is known as the glycemic index (GI) of foods [20]. This concept was developed in 1981 as a tool for predicting the blood glucose response to various foods. Since the amount of carbohydrate in a food varies, researchers have also introduced the concept of glycemic load (GL), which is the amount of available carbohydrate in a serving size of a food item multiplied by its glycemic index [21]. Prospective studies investigating the relations between dietary carbohydrate intake and risk of T2DM using glycemic index (GI) and glycemic load (GL) supported the protective role of low GI and low GL diets against the development of T2DM [19]. However, a high GI food would cause a higher increase in the levels of blood glucose after its ingestion, it will in turn increase the demand of insulin. Regular consumption of high GI foods would therefore contribute to β cell distraction due to the elevated insulin demand and/or continuously raised blood glucose concentration [19].

A study in 2010 from the United Arab Emirates aimed to determine factors associated with poor glycemic control among patients with T2DM, stated the main factor behind poor glycemic control was not following a dietitian recommended eating plan and the negative attitude towards diabetes [22]. Another study from the UAE examining the differences in the prevalence of diabetes mellitus between different

ethnic groups in the UAE, reported a higher prevalence among UAE citizens (25%) compared to that of expatriates (13–19% depending upon country of origin) [23]. These results suggest the need for better counseling programs focusing on the UAE citizens, which in turn requires more information about the dietary practices of the UAE citizens, food composition tables of locally consumed foods, and the GI values of these food to facilitate the role of dietitians in developing better programs targeting glycemic control in people living with pre-diabetes.

This dissertation provides a series of studies aiming to investigate the prevalence of the MetS and its components among young Emirati adults; to identify the anthropometric measures of obesity that best identify MetS; and to develop a comprehensive food composition table with GI and GL values for locally consumed foods in the UAE. This was established in an effort to contribute to the understanding of these diseases, and to provide objective tools for nutrition therapy planning and dietary management in the UAE.

Chapter 2: Review of Literature

2.1 Introduction to the Metabolic Syndrome

2.1.1 History of the Metabolic Syndrome

The metabolic syndrome (MetS) can be described as a clustering of several risk factors, including central adiposity, impaired glucose tolerance, dyslipidemia, and hypertension.

The metabolic syndrome concept was first introduced in 1988 by Gerald Reaven. He called it the “Syndrome X” and defined it as a cluster of abnormalities (dyslipidemia, high blood pressure, and diabetes) arising from the underlying events of insulin resistance and hyperinsulinemia [24]. Ten years later, in 1998, the World Health Organization (WHO) first recognized the metabolic syndrome and developed diagnostic criteria that could officially be used for clinical diagnosis [25]. However, during these ten years, many other names were used to describe the co-occurrence of these abnormalities all together, such as: the Plurimetabolic Syndrome by Crepaldi and Nosadini in 1988 [26]; the Deadly Quartet by Kaplan in 1989 [27]; the Insulin Resistance Syndrome by DeFronzo and Ferrannini in 1991 [28]; and Diabesity by Shafir in 1993 [29]. Conversely, Sarafidis and Nilsson proposed that the recognition of the metabolic syndrome has evolved over about 90 years [30], starting in World War I, when Hitzemberger and Richter-Quittner identified a link between hypertension and diabetes [30]. The history of the metabolic syndrome is briefly described in Table 2.1, as adopted from Blaha and Tota-Maharaj with modifications [31].

Table 2.1: Brief History of the Metabolic Syndrome

Event	Year
Recognition of the co-existing of hyperglycemia and hypertension [30].	1920s
Describing fat distribution and emphasis on abdominal obesity [30].	1950s
Emphasis on hyperlipidemia/dyslipidemia as part of the cluster [30].	1960s
Metabolically-obese, normal weight phenotype defined.	1981
Gerald Reaven describes “Syndrome X”: clustering around insulin resistance [24].	1988
Kaplan describes “deadly quarter”: abdominal obesity, diabetes, hypertension, and high triglycerides [27].	1989
Metabolic syndrome first operationalized as clinical diagnosis, based on the WHO definition [25].	1998
NCEP ATP III definition: emphasis on abdominal obesity as surrogate for insulin resistance [32].	2001
American Academy of Clinical Endocrinologists defines “Insulin Resistance Syndrome”.	2003
The International Diabetes Federation definition - more emphasis on abdominal obesity [33]. AHA/NHLBI further refine the NCEP ATP III definition. American Diabetes Association denounces the concept of metabolic syndrome.	2005
ADA/Obesity Society/American Society for Nutrition question advantage of abdominal obesity over BMI.	2007
IDF/AHA/NHLBI/IAS: “Harmonized Definition” [4].	2009
WHO: metabolic syndrome of little utility for clinical practice or epidemiologic research.	2010

Source: Blaha and Tota-Maharaj, 2012 [31].

2.1.2 Definition of the Metabolic Syndrome

Over the past decade, the definition of the metabolic syndrome has been greatly argued. The argument lingers to doubt whether there is such a syndrome or not [34].

The first definition for “Syndrome X” proposed by Reaven in 1988 [24], was the clustering of dysglycemia, dyslipidemia, hypertension, and insulin resistance. The WHO proposed the addition of central obesity to the definition of the metabolic syndrome and established the first diagnostic criteria for it in 1998-1999 [25]. The WHO diagnostic criteria included the direct or indirect measure of insulin resistance. Insulin resistance measurement was also part of the diagnostic criteria proposed by the European Group for the Study of Insulin Resistance in 1999 [35]. Aiming to make the clinical diagnosis of the metabolic syndrome easier and more user friendly, the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP) III report established new guidelines in 2001 that do not include the measurement of insulin resistance [32]. The American Association of Clinical Endocrinologists then proposed an even more flexible definition in 2003 [36]. Other definitions were developed and used over the past period included the NCEP ATP definition in 2005 [37], and the International Diabetes Federation definition in 2006 [33]. The latest and most accepted diagnostic criteria for the metabolic syndrome is the harmonized definition of the International Diabetes Federation Task Force on Epidemiology and Prevention (IDF); the National Heart, Lung, and Blood Institute (NHLBI) ; the American Heart Association (AHA); the World Heart Federation (WHF); the International Atherosclerosis Society (IAS); and the International Association for the Study of Obesity [4], published in 2009.

2.1.3 Diagnostic Criteria of the Metabolic Syndrome

The diagnostic criteria of the metabolic syndrome that was published by the WHO 1998, and revised in 1999 [25], considered cardiovascular disease as the main outcome of the syndrome, and highlighted the importance of detecting insulin resistance along with the presence of at least two of the following criteria: elevated triglycerides; elevated blood pressure; central obesity; or reduced HDL [25]. The European Group for the study of Insulin Resistance (EGIR) in 1999, also required the measurement of insulin resistance [35]. This made the definition not very practical for use in epidemiological studies of large populations.

The NCEP ATP III diagnostic criteria proposed in 2001 [32] did not require the measurement of insulin resistance. However, it required the occurrence of at least three of the following five criteria: central obesity; reduced HDL; elevated triglycerides; elevated blood pressure; or elevated fasting glucose. The incidence and the number of these components was thought to contribute to increase the risk and progression of the syndrome [32].

The American Academy of Clinical Endocrinologists (AACE) proposed in 2003 a criteria for the diagnosis of the “Insulin Resistance Syndrome” [36]. It focused the discussion on insulin resistance and hyperinsulinemia as the underlying pathophysiology of the syndrome. Therefore, this definition was considered very broad as it included cardiovascular and non-cardiovascular consequences of insulin resistance, such as hypertension, polycystic ovarian syndrome (PCO), fatty liver disease, and acanthosis nigricans[36]. The purpose of this definition was to alert physicians to the metabolic state of the patient rather than determining the level of

cardiovascular risk. A summary of the four discussed criteria for the diagnosis of metabolic syndrome is presented in Table 2.2.

Table 2.2: Summary of the Metabolic Syndrome's Diagnostic Criteria according to the WHO, the EGIR, the NCEP ATP III, and the AACE

WHO [25] (1998 - 1999)	EGIR [35] (1999) for non- diabetics	NCEP ATP III [32] (2001)	AACE [36] (2003)
Insulin resistance: - Type 2 diabetes - Impaired fasting glucose - Impaired glucose tolerance (euglycemic clamp)	Insulin resistance OR Hyperinsulinemia	At least 3 of the following:	Impaired glucose tolerance: 2-hour postprandial glucose test >140 mg/dl
+ 2 of the following:	+ 2 of the following:	Fasting glucose: - ≥ 110 mg/dl	Fasting glucose: - between 110 and 126 mg/dl
Hypertension: - BP ($\geq 140/90$ mmHg)	Hypertension: $\geq 140/90$ mmHg OR - Anti-hypertensive medication.	Hypertension: $\geq 130/85$ mmHg OR - Anti-hypertensive medication.	Hypertension: $\geq 130/85$ mmHg
Dyslipidemia: - Triglyceride (≥ 150 mg/dl) OR - HDL (<35 mg/dl in men, <39 mg/dl in women)	Dyslipidemia: - Triglyceride (≥ 180 mg/dl) OR - HDL (<39 mg/dl) OR - Dyslipidemia medication	Elevated triglyceride: - (≥ 150 mg/dl)	Dyslipidemia: - Triglyceride (≥ 150 mg/dl) AND - HDL (<40 mg/dl in men, <50 mg/dl in women)
Obesity: - BMI > 30 kg/m ² OR - WHR >0.9 in men, >0.85 in women.	Central Obesity: - WC (Men ≥ 94 cm, women ≥ 80 cm)	Central Obesity: - WC (Men ≥ 102 cm, women ≥ 88 cm)	Overweight: - BMI > 25 kg/m ²
Microalbuminuria: - Urinary albumin excretion rate ≥ 20 μ g/min OR - Albumin: Creatinine ratio ≥ 30 mg/g	Impaired fasting glucose: - FPG ≥ 110 mg/dl	Reduced HDL: - <40 mg/dl in men, <50 mg/dl in women) OR - HDL medication	Other related factors: - Family history of T2DM, HTN, or CVD. - PCO. - Ethnicity. - Sedentary lifestyle. - Fatty liver disease. - Acanthosis nigricans.

WHO: World Health Organization; EGIR: European Group for the study of Insulin Resistance; NCEP ATP: National Cholesterol Education Program Adult Treatment Panel; BP: blood pressure; AACE: American Academy of Clinical Endocrinologists; HDL: high-density lipoprotein Cholesterol; BMI: body mass index, WHR: waist-to-hip ratio; WC: waist circumference; FPG: Fasting Plasma Glucose; T2DM: type 2 diabetes mellitus; CVD: cardiovascular disease; PCO: polycystic ovarian syndrome; HTN: hypertension.

In 2005, the International Diabetes Federation proposed a new diagnostic criteria emphasizing abdominal obesity. The IDF removed the WHO requirement for insulin resistance measurement, and made abdominal obesity required for the diagnosis, combined with four other factors (elevated triglycerides, elevated blood pressure, elevated FPG, and reduced HDL cholesterol) [33]. The central obesity was defined as waist circumference (according to an ethnic-specific value), which is considered a simple and easy screening tool; the rest of the criteria remained identical to the 2001 NCEP ATP III criteria.

The AHA/NHLBI also attempted to further refine and update the NCEP ATP III definition in 2005. They proposed changing impaired fasting plasma glucose to $FPG \geq 100$ mg/dl compared to the old $FPG \geq 110$ mg/dl used by the ATP III [37]. The AHA/NHLBI did not mandate central obesity for the diagnosis of the metabolic syndrome, and did not use ethnic-specific values for WC when defining central obesity. This lack of agreement on the definition of central obesity between the IDF and the AHA has resulted in confusion in the definition of metabolic syndrome among researchers and scientists.

In 2009, the International Diabetes Federation Task Force on Epidemiology and Prevention (IDF); the National Heart, Lung, and Blood Institute (NHLBI); the American Heart Association (AHA); the World Heart Federation (WHF); the International Atherosclerosis Society (IAS); and the International Association for the Study of Obesity have all agreed on a joint interim statement for a new harmonized definition for the metabolic syndrome [4]. The diagnostic criteria of the IDF (2005), the AHA/NHLBI (2005) and the harmonized definition (2009) are summarized in Table 2.3. The harmonized definition follows the ATP III definition, but central

obesity is no longer obligatory. Modifications might happen in the future, however, using single definition in the previous couple of years have allowed the ability for better comparisons among epidemiological studies.

Table 2.3: Summary of the Metabolic Syndrome's Diagnostic Criteria according to the IDF, the AHA/NHLBI, and the harmonized definition IDF/AHA/NHLBI.

IDF [33] (2005)	AHA/NHLBI [37] (2005)	The harmonized definition IDF/AHA/NHLBI [4] (2009)
Central Obesity: (ethnic-specific values) + 2 of the following:	At least 3 of the following: Central Obesity: - WC (Men \geq 102cm, women \geq 88 cm)	At least 3 of the following: Central Obesity: (population- and country- specific values)
Impaired fasting glucose: - FPG \geq 100 mg/dl OR - Medication for hyperglycemia	Impaired fasting glucose: - FPG \geq 100 mg/dl OR - Medication for hyperglycemia	Impaired fasting glucose: - FPG \geq 100 mg/dl OR - Medication for hyperglycemia
Hypertension: \geq 130/85 mmHg OR - Anti-hypertensive medication.	Hypertension: \geq 130/85 mmHg OR - Anti-hypertensive medication.	Hypertension: \geq 130/85 mmHg OR - Anti-hypertensive medication.
Elevated triglyceride: - (\geq 150 mg/dl) OR - Treatment of lipid abnormality	Elevated triglyceride: - (\geq 150 mg/dl)	Elevated triglyceride: - (\geq 150 mg/dl) OR - Treatment of lipid abnormality
Reduced HDL cholesterol: - Men $<$ 40 mg/dl - Women $<$ 50 mg/dl OR - HDL medication	Reduced HDL cholesterol: - Men $<$ 40 mg/dl - Women $<$ 50 mg/dl OR - HDL medication	Reduced HDL cholesterol: - Men $<$ 40 mg/dl - Women $<$ 50 mg/dl OR - HDL medication

IDF: International Diabetes Federation; AHA: American Heart Association; NHLBI: National Heart, Lung, and Blood Institute; HDL: high-density lipoprotein Cholesterol; WC: waist circumference; FPG: Fasting Plasma Glucose.

2.2 Risk Factors of the Metabolic Syndrome

There are many underlying risk factors discussed in the literature that could be related to the pathophysiology of the metabolic syndrome. Some are modifiable factors and others are non-modifiable. It has been proposed that all factors, whether they are lifestyle factors (excess nutrients and physical inactivity), hormonal factors, macronutrient factors or even aging factors, they are interlinked together in a very complicated manner. Some are simplified and summarized in Figure 2.1.

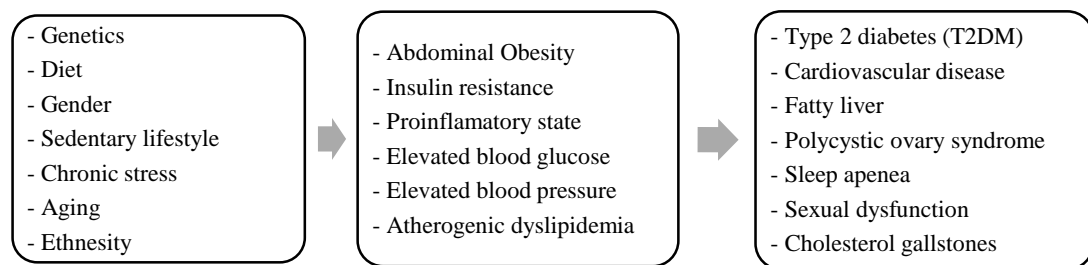


Figure 2.1: Schematic Presentation of the Metabolic Syndrome Risk Factors and Consequences

2.2.1 Genetics of the Metabolic Syndrome

Each component of the MetS could be explained by a complex set of interactions between the genes and the environment. Limited data is available about the genetic factors behind the prevalence of metabolic syndrome; however, the genetic susceptibility to the MetS can be organized in two groups: genes leading to insulin resistance, and genes related to obesity phenotypes. Several genes were identified to play a role in the development of MetS, yet the mechanisms of acting remained poorly understood. There is no doubt that the MetS is a polygenetic disease, along with a great number of influences from the environment. Moreover, even if the genetic-based risk was successfully assessed, it would be unlikely to play the main role in the MetS clinical management.

In 2001, the ATP III identified obesity as the main underlying risk factor of the MetS. The hypothesis emphasized the role of obesity in the development of MetS, explained by individuals having the genetic susceptibility of the syndrome but failing to manifest it because they never developed obesity [32]. In 2003 the WHO and the AACE identified insulin resistance as the main underlying risk factor of the MetS [36]. They agreed that physical inactivity, obesity, and aging all play a role in the development of the syndrome, but focused on insulin resistance as the key trigger.

The most accepted view on the origins of the metabolic syndrome suggests the importance of obesity and insulin resistance to engender the metabolic syndrome along with other environmental and genetic factors. This considers the independent effect of obesity and insulin resistance on the syndrome, and the effect of each of them on the other. This hypothesis is adopted from the Atlas of Atherosclerosis and Metabolic Syndrome by Grundy [38] and demonstrated in Figure 2.2.

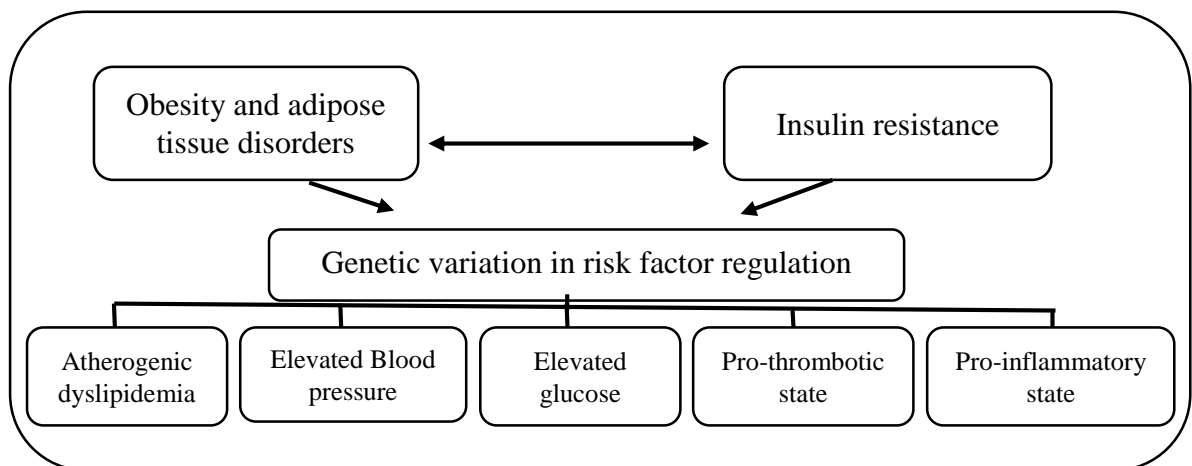


Figure 2.2: The Origins of the Metabolic Syndrome

Source: Grundy, 2011 [39].

Studies focusing on the MetS genetics have discussed two main genes in an attempt to explain the pathophysiology of the syndrome; these are the FTO (fat mass and obesity associated) gene and the PPARG (Peroxisome Proliferator-Activated

Receptor Gamma) gene [40, 41]. The FTO gene is thought to increase caloric consumption by upregulating the starvation signals of the hypothalamus resulting in increased BMI and overall adiposity [42, 43]. The PPARG gene transcribes the nuclear receptor family of the PPARs isotypes (PPAR α , PPAR δ , and PPAR γ) that are involved in the regulation of carbohydrate and lipid metabolism. Of these, the PPAR γ appears to play a principle role in the assimilation of lipids, insulin signaling, glucose metabolism, fatty acids storage, and inflammation in endothelial cells [40].

2.2.2 Insulin Resistance

The insulin resistance mechanism was used by many researchers to justify the occurrence of the MetS as a cluster and its individual components. Insulin resistance alone was not enough to provide an explanation for the 30% of individuals diagnosed with the MetS, yet had normal insulin sensitivity. Insulin resistance was suggested to result from ectopic fat deposition and lipotoxicity [44]. It is characterized by high concentrations of plasma insulin (hyperinsulinemia) that fail to maintain plasma glucose homeostasis resulting in hyperglycemia and other components of the MetS. The contributing factors to insulin resistance are very complex. Many hypothesis were developed trying to explain it, such as the adiponectin hypothesis [45], and the plasma copeptin hypothesis [46]; however, the obesity-induced insulin resistance hypothesis is the most accepted [47].

Insulin sensitivity could be measured in different ways at whole-body level. Traditional approaches use the hyperinsulinemic euglycemic clamp technique, which requires measuring the amount of glucose needed to match the rate of removal of different insulin concentrations. It could also be indirectly measured by measuring

fasting insulin value. An easier and faster way is to calculate the fasting insulin concentration to fasting plasma glucose (FPG) ratio by a mathematical formula known as HOMA-IR (homeostasis model assessment for insulin resistance) [48]. As the presence of insulin resistance is linked to the presence of central obesity, it is now proposed to use the “hypertriglyceridemic waist” as an easy and simple measure for the screening of insulin resistance [49].

2.2.3 Abdominal Obesity

Not all people living with obesity suffer from the MetS; similarly, not all normal weight people are healthy and free from all components of the syndrome. Modern medicine is still debating over the interaction between obesity, insulin resistance and the MetS. Obesity itself is a multifactorial condition, caused by a complex group of genetics and behavioral factors. Ectopic fat is the accumulation of fat in tissues other than the subcutaneous adipose tissue, such as the liver, epicardium of the heart, and the abdominal viscera. This is thought to happen when the subcutaneous adipose tissue becomes dysfunctional, insulin resistant, lipodystrophic (rare condition characterized by adipose tissue deficiency) or simply saturated with fat during excess nutrient intake [50]. This accumulation of fat in the liver leads to atherogenic dysglycemia, and the accumulation of fat in the muscles leads to insulin resistance [51]. Ectopic fat and visceral fat have shown greater correlations with the MetS when compared to the BMI [50]. Moreover, the role of the subcutaneous adipose tissue as an endocrine organ cannot be ignored. The adipose tissue can release a range of hormones (TNF-alpha, leptin, and resistin) pro-inflammatory, pro-thrombotic adipokines and cytokines that in turn induce a systemic pro-inflammatory state,

causing an increase the risk of metabolic disorders, such as diabetes, hypertension and CVDs [52]. The release of these cytokines into the blood circulation could also induce an inflammatory response in the epithelial arterial wall, leading to a disturbance in the atherosclerotic plaques, which triggers plaque rupture, generating acute coronary syndromes [53].

Simple anthropometric measures are used for the assessment of obesity and fat deposition, such as the body mass index (BMI), the waist-to-hip ratio (WHR), and the waist circumference (WC) [54]. Moreover, modern accurate computed imaging systems became available and allowed direct quantifying of body fat.

2.2.4 Glucose Intolerance

Glucose intolerance is caused by the effect of insulin resistance on different body tissue. Insulin resistance causes impairment in the uptake of insulin by cells, which effects the cascade of reactions induced by insulin, causing dysregulation of gluconeogenesis and glycogenolysis in the liver resulting in hyperglycemia. Detecting glucose intolerance (or prediabetes) can be done by oral glucose tolerance test (OGTT) 2-h PG in the 75-g OGTT 140 mg/dL (7.8 mmol/L) to 199 mg/dL (11.0 mmol/L) (IGT), or impaired fasting glucose (IFG) FPG 100 mg/dL (5.6mmol/L) to 125 mg/dL (6.9 mmol/L), or elevated HbA1c 5.7–6.4% [16].

2.2.5 Elevated Blood Pressure

The association between the MetS and the elevation in blood pressure has been identified, but the mechanisms have not been fully defined [4]. This elevation might only be high-normal (SBP 130-140/DBP 85-95 mmHg), or in some cases it increased

to the next category (>140/190 mmHg). Multiple factors were proposed to be involved, some of these are:

- Obesity → Oxidative stress → reduce bioavailability of nitric oxide → vasodilation → elevate blood pressure [55].
- Obesity → increase adipocytes tissue → adipocytes produce angiotensinogen → elevate blood pressure [56].
- Obesity → increase adipocytes tissue → adipocytes produce inflammatory cytokines → pro-inflammatory state → endothelial dysfunction → elevate blood pressure [56].
- Obesity → compresses the kidney with fat tissues → induce renal dysfunction → increase renin → elevate blood pressure [57].
- Insulin resistance → enhances the sympathetic nervous system → elevate blood pressure [58].
- Insulin resistance → increase sodium reabsorption → sodium retention → elevate blood pressure [58].

2.2.6 Atherogenic Dyslipidemia

Another underlying risk factor of the metabolic syndrome is atherogenic dyslipidemia. This condition is characterized by increased fasting and post-prandial triglycerides level, increased Apo-lipoprotein (Apo-B), increased very low-density lipoprotein (VLDL) cholesterol, increased small dense low-density lipoprotein (LDL) profile, low high-density lipoprotein (HDL) cholesterol and small HDL particles [59]. Atherogenic dyslipidemia is the core of the MetS, and it is defined according to the harmonized definition of the metabolic syndrome (IDF/ NHLBI/ AHA) [4] and the 2012 European guidelines on cardiovascular disease prevention [60] as an HDL-C level < 40 mg/dL in men and <50 mg/dL in women, with a triglyceride level ≥150 mg/dL. The underlying mechanisms for the development of atherogenic dyslipidemia

have been identified in the literature [59]. Due to insulin resistance, and/or fatty liver, and/or lipid overloading of the liver, the liver will overproduce Apo-B containing VLDL and VLDL triglycerides. VLDL is metabolized in the circulation into intermediate density lipoprotein (IDL) and LDL. The origins of the small dense LDL is not fully understood; however, many mechanisms were proposed to explain it. It could be the activity of cholesterol ester transfer protein (CETP), which catalyzes the exchange of cholesterol esters for triglyceride between LDL and VLDL, resulting in the depletion of cholesterol ester from the LDL, and the production of small LDL particles [59].

Other investigators believe that the increased production of Apo B-VLDL from the liver, would produce VLDLs that are already smaller in size, and when hydrolyzed they will produce smaller, denser LDL particles [61]. Lastly, it could be the higher activity of hepatic triglyceride lipase (HTGL) (existing in MetS patients), that is degrading the normal LDL into smaller denser LDL particles [62]. These mechanisms also account for the production of less overall HDL and smaller size HDL particles, causing less efficient reverse cholesterol transport in the circulation. Whereas, the small, dense LDL is more likely to get oxidized and has a great ability to transport cholesterol for the vessel.

2.2.7 Inflammation

People with the MetS were found to have a low-grade of systemic inflammation [47, 63]. Many biomarkers of inflammation were shown to have an association with the MetS, such as: increased high sensitivity C-reactive protein (Hs-CRP), increased interleukin 6 (IL-6), increased tumor necrosis factor alpha (TNF- α),

increased leptin, decreased adiponectin, and increased resistin [64]. Inflammation in the MetS is developed by the effect of insulin resistance on the liver and the dysregulation of the adipose tissue, along with the effect of insulin resistance on the sympathetic nervous system, causing an activation of inflammation and chemotaxis pathways. The pro-inflammatory state of the MetS is illustrated in Figure 2.3. Under the effect of insulin resistance, the liver produces higher levels of C-reactive protein (CRP), fibrinogen, and plasminogen activator inhibitor 1 (PAI-1). On the other hand, the adipose tissue produces a number of pro-inflammatory cytokines such as IL-6 and TNF- α , and reduce the production of adiponectin, which is considered an important anti-inflammatory agent.

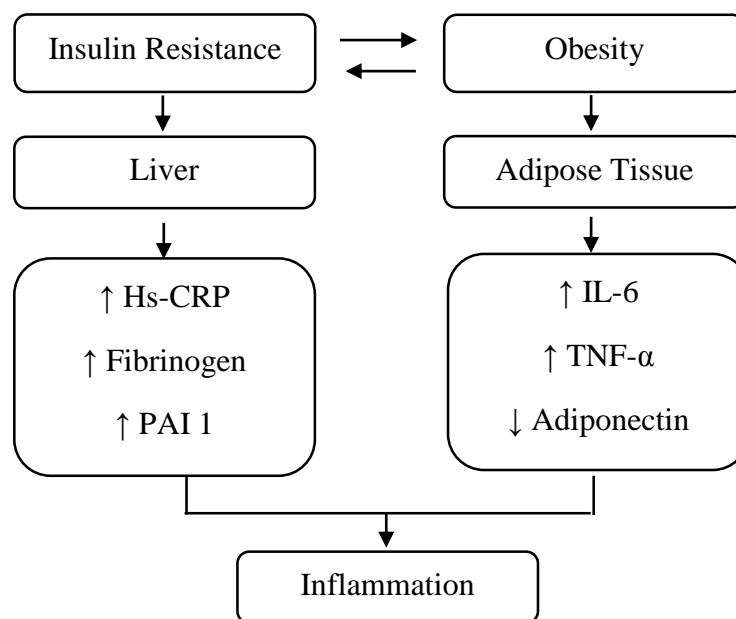


Figure 2.3: Pro-inflammatory State of the Metabolic Syndrome

Many inflammation markers were used through the years to predict cardiovascular events [63, 65], and a great number of investigators reported an association between pro-inflammatory state and MetS [65, 66]. Ridker and others showed evidence that the MetS represents an inflammatory state that could be seen by the progressive increase of Hs-CRP levels with the increased number of the MetS components according to the ATP III criteria (Figure 2.4) [67]. Up to date, the only well-standardized marker of inflammation and a predictor of the MetS and future cardiovascular events, is the Hs-CRP [68]. However, the measurement of Hs-CRP is not yet included in the diagnostic criteria of the MetS, although it has been recommended by a large number of investigators [67-71]. Inflammation is usually measured through the measurement of serum Hs-CRP, where a level above 3 mg/l is considered high according to the recommendations of the CDC and the AHA for cardiovascular disease (CVD) risk assessment [63]. Although levels of fibrinogen, IL-6, and adiponectin are also highly correlating with the prevalence of MetS in clinical research studies [38, 64], routine measurement of these inflammation markers is not yet recommended.

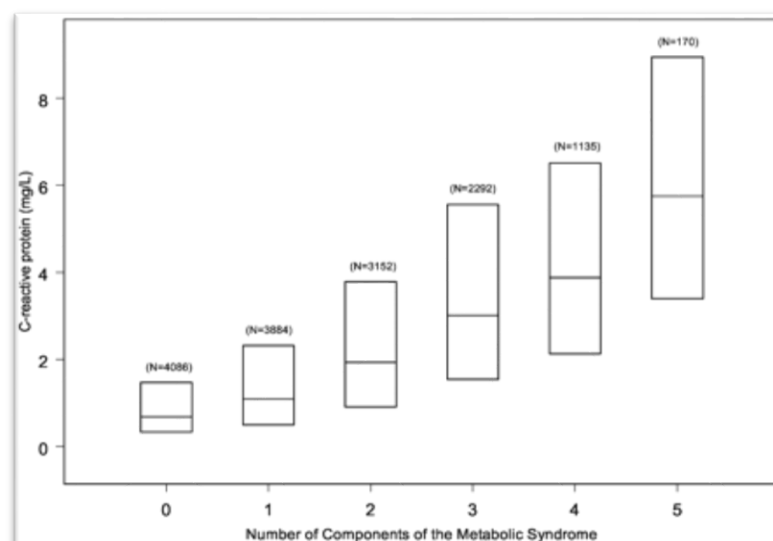


Figure 2.4: Hs-CRP and the Metabolic Syndrome

Source: Ridker and others, 2003 [67].

2.3 Consequences of the Metabolic Syndrome

The MetS is linked to the increased risk of a range of disease consequences including CVD, diabetes, polycystic ovary syndrome, fatty liver, asthma, cholesterol gallstones, sleep apnea, and sexual dysfunction, as shown earlier in Figure 2.1. Moreover, people living with diabetes, CVD, hypertension, and coronary heart disease were reported to have a much higher prevalence of the MetS when compared to the general population.

The Syndrome X was proposed in 1988 by Gerald Reaven [24] to explain the grouping of CVD risk factors in individuals with insulin resistance. However, the current understanding of the MetS includes increased risk of atrial fibrillation, coronary heart disease (CHD), ischemic stroke, myocardial infarction, aortic valve disease, and cardiomyopathy [72, 73].

The first epidemiological study associating the MetS with CVD morbidity and mortality was the Botnia study in 2001 [74]. It included 4,483 subjects, aged between 35 and 70 years in Finland and Sweden. The study used the WHO 1998 definition for the MetS and reported a three-fold increase in the risk for CHD and stroke in subjects with the MetS ($P < 0.001$).

A year later (2002), the Kuopio Ischemic Heart Disease Risk Factor Study was published [75]. This prospective cohort study included 1,209 Finnish men, between the age of 42 and 60 years, who were studied from 1984 till 1998. The study concluded an increase in CVD and all-cause mortality in men with the MetS, even in the absence of diabetes and CVD at the baseline.

A meta-analysis and a systematic review published in 2010, investigated the association between cardiovascular risk and the ATP III definitions of the MetS. It reported data from 87 studies including 951,083 patients, and showed that the MetS is associated with an increased risk of CVD mortality (RR: 2.40), CVD (RR: 2.35), stroke (RR: 2.27), all-cause mortality (RR: 1.58), and myocardial infarction (RR: 1.99) [76]. Moreover, the prevalence of the MetS among people living with diabetes from various ethnic groups and different populations was reported to be between 76% - 92% [77-79].

It is important to highlight that the risk of the MetS occurs even when diabetes is absent. In 2003, Alexander and others [80] showed that patients with the MetS but without diabetes had a higher prevalence of CHD (13.9%) than patients with diabetes but without the MetS (7.5%), as presented in Figure 2.5.

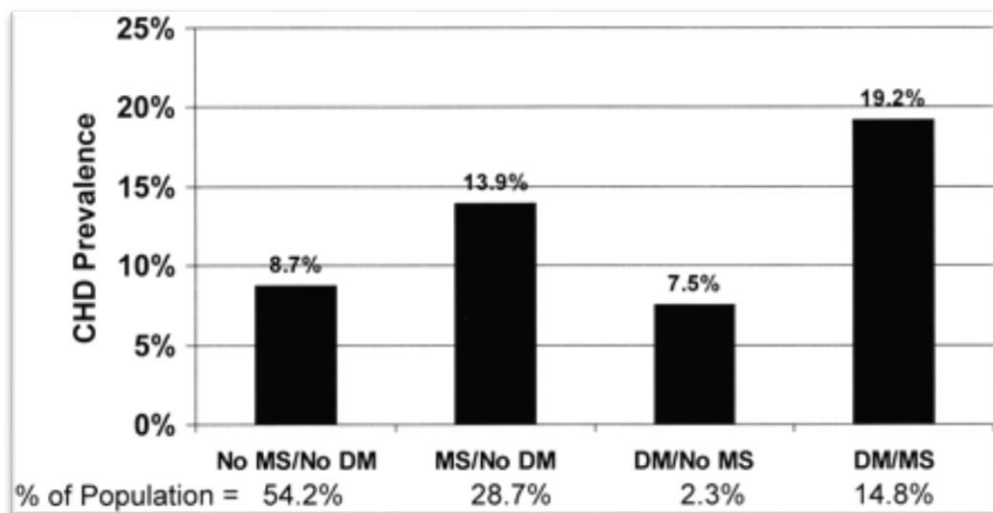


Figure 2.5: Prevalence of CHD Categorized by Presence of MetS and Diabetes

Source: Alexander and others, 2003 [80].

This does not propose that there is no relation between the MetS and diabetes. In fact, the MetS is an important underlying risk factor for the development of

diabetes. Additionally, the relative risk of diabetes was reported to be three-fold higher among patients with the MetS compared to those without it [81].

The pathophysiology of T2DM is usually explained by a two-step hypothesis. First, individuals develop insulin resistance partly caused by obesity as explained earlier in Section 2.2.2. Second, pancreatic β -cells get damaged or/and their function decline gradually with time, which might happen even before the clinical diagnosis of hyperglycemia. Considering the MetS is a state of pre-diabetes and insulin resistance, it should not be surprising that it has the ability to predict new cases of T2DM.

The San Antonio Heart Study was published in 2003 [82]. It included 1,734 participants, between the age of 25 and 64 years, who completed eight years of follow-up. The study concluded that the MetS predicts T2DM independent of impaired fasting glucose. It also showed that the MetS was associated with a five-fold higher risk of diabetes over the eight years follow-up even with normal fasting plasma glucose. This finding would suggest the need for a measure of diabetes that could have a greater prediction power for diabetes and the metabolic syndrome than fasting plasma glucose.

The Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial [83] showed an association between higher HbA1c levels and increased risk of mortality over a 3.4 years follow-up. Glycated hemoglobin (HbA1c) is a simple, practical measure, and has the ability to reflect glucose levels in the blood from the previous three months. As a matter of fact, the new recommendation by the ADA on the Standards of Medical Care in Diabetes-2015 [16], have added HbA1c test for the diagnostic criteria of diabetes (HbA1c > 6.5%) and pre-diabetes (HbA1c 5.7%-6.4%),

and did not require the confirmation of the test with other tests like fasting plasma glucose or glucose tolerance test if the HbA1c has been confirmed by replication.

In 2007, two studies were conducted to study the association of glycated hemoglobin (HbA1c) and the MetS. The first is the Chennai Urban Rural Epidemiology Study (CURES) [84], which included 1,644 Asian Indian participants with normal glucose tolerance (FPG <100 mg/dl and 2-h post-prandial plasma glucose <140 mg/dl). It used the ATP III guidelines for the diagnosis of MetS. The study showed strong association between HbA1c and the MetS (OR: 2.9 (95% CI 2.08–4.00); $P < 0.001$).

The second study was conducted among 22,465 middle aged non-diabetic Korean participants [85]. The study concluded that the prediction of MetS could be achieved at 5.45% of HbA1c and it recommended the use of HbA1c as a predictive measure of impaired fasting glucose and MetS among the Koreans.

To conclude the consequences of the MetS and show the relative risk factor between the MetS and CVD morbidity, CVD mortality, diabetes, and all-cause mortality, a summary of all systematic reviews and meta-analyses published to describe these associations is presented in Table 2.4.

Table 2.4: Summary of Systematic Reviews and Meta-analyses on the association of the MetS with CVD morbidity, CVD mortality, Diabetes, and All-Cause Mortality

Author and year of publication	Selection criteria	Definition of the MetS	Outcome and follow-up	No. of studies/ participants	Relative risk (95% CI)
Ford, 2005 [86]	Articles published between 1998 and August 2004	NCEP 2001	All-cause mortality Cardiovascular disease	23,247 participants 43,054 participants	1.27 (0.90–1.78) 1.65 (1.38–1.99)
Galassi <i>et al.</i> , 2006 [87]	Articles describing prospective studies published between 1966 and April 2005	WHO 1999, NCEP 2001 and modifications	All-cause mortality Cardiovascular disease mortality Incident cardiovascular disease	21 studies	1.35 (1.17–1.56) 1.74 (1.29–2.35) 1.53 (1.26–1.87)
Gami <i>et al.</i> , 2007 [88]	Articles describing prospective studies published between 1966 and March 2005	WHO 1999, NCEP 2001 and modifications	Cardiovascular events or death	37 studies, 43 cohorts and 172,573 participants	1.78 (1.58–2.00)
Ford <i>et al.</i> , 2008 [81]	Articles published between 1998 and April 2008 describing population-based cohort studies	WHO 1999 EGIR 1999 NCEP 2001 NHLBI/AHA 2004 AHA/NHLBI 2005 IDF 2005	Incident diabetes over 2.3–20 years	42,419 participants (total – subgroups for different definitions)	5.17 (3.99–6.69) 4.45 (2.41–8.22) 3.53 (2.84–4.39) 5.16 (4.43–6.00) 5.12 (3.26–8.05) 4.42 (3.30–5.92)
Li <i>et al.</i> , 2008 [89]	Articles published up to July 2007	WHO 1999, NCEP 2001	Incident stroke	13 studies and 92,732 participants	1.6 (1.48–1.75)
Hui <i>et al.</i> , 2010 [90]	Articles published between 2001 and 2009	WHO 1999, NCEP 2001 and modifications	All-cause mortality over mean 11.5-year follow-up	21 studies and 372,411 participants	1.46 (1.35–1.57)
Mottillo <i>et al.</i> , 2010 [76]	Articles published up to June 2009	NCEP 2001 and revised NCEP 2004	Cardiovascular disease Cardiovascular disease mortality All-cause mortality Myocardial infarction Stroke	87 studies and 951,083 participants	2.35 (2.02–2.73) 2.40 (1.87–3.08) 1.58 (1.39–1.78) 1.99 (1.61–2.46) 2.27 (1.80–2.85)

Source: Wild and others, 2011 [91]

2.4 Management of the Metabolic Syndrome

The management of the MetS is achieved by treating underlying risk factors of the syndrome. Therefore, the first-line therapy usually targets lifestyle modifications including weight reduction for people living with obesity and introduction to regular workout for inactive individuals [4]. Therapeutic lifestyle changes such as introduction of the Mediterranean diet and teaching patients about the low glycemic index diet along with recommending a certain intensity and duration of physical activity (for example 10,000 steps/day) will help in decreasing insulin resistance, which in turn will facilitate the treatment of the syndrome.

Drugs were also used in certain conditions to treat or even prevent the MetS. Some of these are used for the management of diabetes (Metformin), hypertension (ACE-inhibitor), CVD (aspirin) [92] and atherogenic dyslipidemia (statin, fibrate, and niacin). However, some drugs have adverse side effects that include raising insulin resistance; which should be stopped (if possible) and replaced with agents that do not increase insulin resistance. Bariatric surgery could also be a treatment option, especially for those patients living with morbid obesity. It has shown to resolve all components of the MetS by a single procedure, eliminating the necessity for daily drugs consumption.

Many guidelines are available from different organizations to help in the management and prevention of the MetS. However, most lack simplicity, or not specific for MetS, or treat each component separately and are difficult to use in clinical settings.

2.4.1 AHA/NHLBI Guidelines

In 2005, the American Heart Association (AHA) and the National Heart, Lung, and Blood Institute (NHLBI) published detailed guidelines for the diagnosis and treatment of MetS [37]. These guidelines are a great tool for health care professionals to use during clinical settings, or for research purposes.

According to the AHA/NHLBI guidelines, the primary goal of clinical management in patients with MetS is to decrease the risk of atherosclerosis and to delay progression of T2DM, hypertension and CVD. A summary of these guidelines and recommendations is presented in Table 2.5.

Table 2.5: AHA/NHLBI Guidelines for Metabolic Syndrome Management

Metabolic risk factors	Goals of Therapy
Atherogenic dyslipidemia	Primary target: reduce LDL-C Secondary target: reduce non-HDL-C Tertiary target: increase HDL-C
Elevated BP	Reduce BP to <140/90 mm Hg And to <130/80 mm Hg if diabetes present.
Elevated glucose	IFG → delay progression to T2DM Diabetes → HbA1c < 7.0%
Pro-thrombotic state	Reduce thrombotic and fibrinolytic risk factors
Pro-inflammatory state	Lifestyle therapies
Lifestyle risk factors	
Abdominal obesity	Reduce body weight by 7% to 10% during the first year of therapy. Continue weight loss till achieving desirable weight (BMI <25 kg/m ²)
Physical inactivity	Regular moderate-intensity physical activity (30 min/day 5 days/week (and preferably ≥60 min, every day).
Atherogenic diet	Reduced intake of saturated fat (<7% of total calories), trans fat, cholesterol (<200 mg/dL), and total fat (25% to 35% of total calories).

Source: Grundy and others, 2005 [37].

2.4.2 The ABCDE Approach

Five requirements were suggested for effective MetS management guidelines, these include being simple, comprehensive, easy to remember, easy to update and does not focus much on the details [31]. In 2008, Blaha and others [93] developed the first practical and comprehensive approach tailored especially for patients with MetS, that is known as the “ABCDE” approach. This approach was established at Johns Hopkins Hospital and peer-reviewed in the literature. It was then updated in 2010 to take into consideration new, and novel, research in the field of prevention and management of the MetS [94]. The ABCDE approach was derived, in large, from the existing 2005 AHA/NHLBI guidelines. Using this approach allows health care professionals to provide the same attention and care to all their patients in an equal manner. The “ABCDE” letters are easy to memorize as they are the first five letters of the alphabet, and each of them represents an aspect in the management of the syndrome. This is presented in Figure. 2.6.

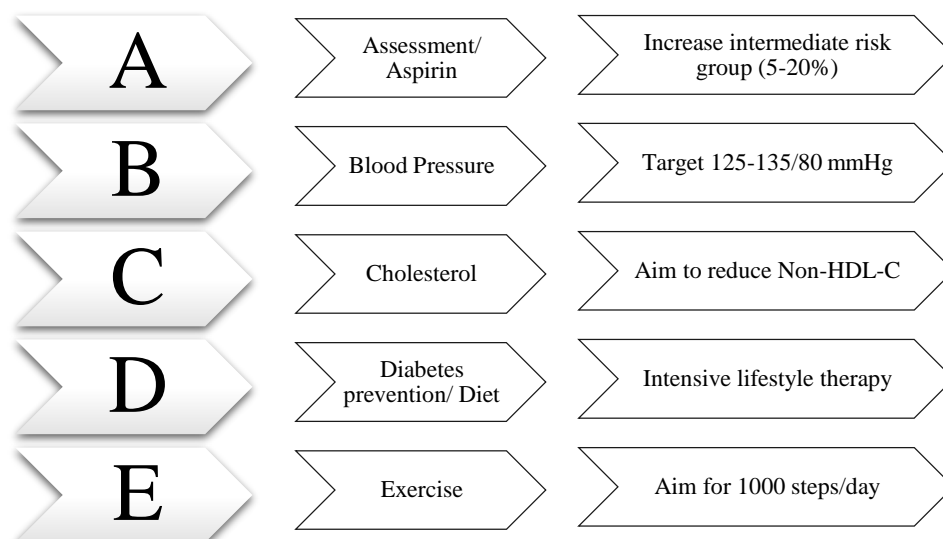


Figure 2.6: Summary of the ABCDE Approach with Recommendations

Source: Blaha and others, 2012 [31].

2.4.2.1 Assessment

Assessment is the most important step of any management program. If assessment was accurately performed, then diagnosis would be easily determined, intervention will be more effective, and evaluating the intervention can be effortless and more beneficial. The Framingham Risk Score (FRS) [95] in the United States and the Systematic Coronary Risk Evaluation (SCORE) project [96] in Europe are widely used for the assessment and prediction of CVD risk. These scores could predict the risk of developing CVD within a ten-year period through the Ten-Year Risk model. The model is divided into three risk groups, low (0-10%), intermediate (10-20%) and high risk group (>20%). However, evidence from the DECODE study [97], showed that patients classified in the low-risk group and had the MetS, presented around three-fold more fatal CVD events compared to those from the same low-risk group but without the syndrome. These findings lead to a novel risk detection strategy by Blaha, suggesting the expansion of the intermediate-risk group to 5-20% in the presence of the MetS. That would allow health care providers to initiate earlier intervention strategies.

In addition to assessment, aspirin therapy has been added to the management approach because evidence from the JPAD trial showed that a low dose of aspirin (81-100 mg) can lessen thrombotic complications linked to CVD [92].

2.4.2.2 Blood Pressure

Elevated blood pressure is a key underlying risk factor of the syndrome. According to the 2011 guidelines of the National Institute of Health and Clinical Excellence (NICE), a measure $\geq 140/90$ mmHg is the cutoff point for the diagnosis of

hypertension [98]. The target of the MetS management is to achieve < 125-135/80 mmHg based on the recommendations of the ACCORD trial [99], as the results of this study found no difference between SBP of 134 mmHg and SBP of 119 mmHg in decreasing CVD events after a 4.7 years follow-up. The targeted blood pressure level could be achieved by several approaches from lifestyle therapies including diet and physical activity to pharmacologic agents (if needed) such as ACE-inhibitor, beta-blockers, thiazide diuretics, and calcium channel blockers.

2.4.2.3 Cholesterol

The “C” letter refers to “Cholesterol” and could also stand for “Cigarettes”. However, smoking cessation is not often highlighted in the management of the syndrome, because smoking is a risk factor for atherosclerotic cardiovascular disease, but not for the MetS.

Cholesterol is a key measure of atherogenic dyslipidemia, one of the main underlying risk factors of the MetS (see section 2.2.6). Under cholesterol, the focus should be on measuring non-HDL-C. Non-HDL-Cholesterol includes the concentrations of Apo-B lipoproteins, LDL, VLDL, IDL, and chylomicron remnants. It can predict CVD better than LDL-C, especially when the levels of triglycerides are high [59]. It can be calculated by measuring total cholesterol and HDL-C during routine lipid profile testing.

Lifestyle therapies are the first approach in the management of atherogenic dyslipidemia. A meta-analysis published in 2008 [100] included 37 prospective cohort studies on the association of glycemic index and chronic disease risk concluded that

diets with high fiber content and low glycemic index were preferable because of their favorable effects on serum glucose, serum triglycerides, and insulin levels.

Regular exercise is also recommended for the management of atherogenic dyslipidemia. The HERITAGE Family Study showed that people with elevated triglycerides levels and reduced HDL levels, acquired the greatest advantage from a regular aerobic exercise training program [101].

If dyslipidemia was still existing even after therapeutic lifestyle changes were implemented, then certain drugs could be used to assist patients in achieving their target lipid profile. The use of statins, fibrates and niacin has been recommended by the AHA [37], which is presented in Table 2.5 based on the risk group of the patient.

Table 2.6: AHA Guidelines for treating Atherogenic Dyslipidemia in the MetS

Risk Group	LDL-C goal (mg/dl)	Non-HDL-C goal (mg/dl)	Recommended treatment
Low	160	190	Therapeutic lifestyle changes
Intermediate	130	160	Therapeutic lifestyle changes
Intermediate high	(130-100)	(160-130)	Use statin to achieve goals.
High	(100-70)	(130-100)	To achieve LDL goal → use statin. To achieve non-HDL goal → intensify statin, and if the goal was not met, add fibrate or niacin.

Source: Grundy and others, 2005 [37].

2.4.2.4 Diet/Diabetes Prevention

“D” refers to diet and diabetes prevention. However, many therapies are now available for diabetes prevention, thus it is treated as a separate aspect in the management approach. Diet is supposed to feature weight reduction goals and recommended macronutrient profile of food.

Weight reduction is the primary goal of treatment for patients with central obesity. It was proven that a reduction of 7%-10% from the initial weight will improve all MetS components through reducing underlying risk factors like insulin resistance, inflammation and ectopic fat storage [102].

Many diets were suggested and used over the years to induce weight loss. Very-low-caloric diet (800 Kcal/day) was able to reduce 6.5% of weight during four weeks, resulting in a significant reduction of blood pressure, serum triglyceride, serum glucose, and serum cholesterol [103]. However, these kinds of diet are usually deficient in many essential nutrient and have poor compliance on the long run. Over the period of one year a hypocaloric high-carbohydrate, low-fat diet (HCLF) was compared with a hypocaloric low-carbohydrate, high-fat (LCHF), or high-protein diet, also known as the Atkins diet. The study found significant weight reduction, improvement in insulin sensitivity, and reduction in blood pressure were associated with both diets, and no significant difference was found between the two [104].

A randomized control trail studied the effectiveness of four different diets for reducing weight and MetS risk factors. One hundred and sixty obese/overweight adult participants (22-72 years old) with hypertension, impaired fasting glucose, or dyslipidemia were equally divided into the four following groups, Atkins diet (low-carbohydrate), Zone diet (macronutrient balance), Ornish diet (low-fat), and Weight Watchers diet (calorie restriction) [105]. After a year of dietary intervention, all four diets triggered a modest reduction in body weight and a significant reduction in the LDL/HDL ratio. Greater dietary adherence rates were associated with greater weight reduction and improved cardiac risk factors for all groups; however, all groups had low adherence after one year.

The Mediterranean diet characteristics matches that of the diet recommended by the AHA in 2005 [37]. It is rich in omega-3 fatty acids, natural antioxidants and fiber from fruits, vegetables, nuts and whole grains. A recent meta-analysis of 50 studies including 534,906 participants, investigated the association between the Mediterranean diet and the MetS. Adherence to the diet revealed a protective role against MetS components like waist circumference (-0.42 cm; 95%CI -0.82 to -0.02), triglycerides (-6.14mg/dl; 95%CI -10.35 to -1.93), systolic BP (-2.35 mmHg; 95% CI -3.51 to -1.18), and plasma glucose (-3.89mg/dl; 95%CI -5.84 to -1.95) [106]. These findings are very important from a public health perspective, since this diet is easy to follow and it is highly efficient for prevention of the MetS and its components.

The low glycemic index diet is another great dietary approach for the management of the MetS and prevention of T2DM and CVD [107, 108]. It is easy to adopt by various populations and cultures because cultural or traditional diets can be tailored to follow it. It is simply about choosing low glycemic index foods and avoiding those with high glycemic index, regardless of the cuisine. The aim of the diet is to cause a slow increase in plasma glucose after food ingestion. This diet is recommended by the ADA for the prevention and management of diabetes [16]. A randomized control trial included 73 obese young adults compared the metabolic benefits of a low glycemic index diet and a low-fat diet for a total of 18 months (six months dietary intervention and 12 months follow-up). After the six months of intervention, the low glycemic index group had a greater reduction in triglyceride ($P=0.02$) and greater increase in HDL-C ($P=0.002$). At 18 months, the low glycemic index diet group had significantly more reduction in weight (-5.8 vs -1.2 kg; $P=.004$) than the low-fat diet [107]. These findings demonstrate how beneficial a low glycemic index diet could be, especially for the patients with the MetS.

All discussed diets had the ability to reduce weight; however, weight reduction is not enough. Maintenance of weight reduction is the key to prevention of metabolic risk factors. Before recommending a certain diet for a patient, the dietitian should think about the possibility of adopting that diet for life. Short-term (maximum of two years) dietary intervention studies are only available, and they all show partial weight regain and poor compliance on the long run [105, 107, 109]. Therefore, long-term studies are required to confirm the ability of patients to lose weight using dietary interventions without regaining it.

Patients should also be advised about improving their sleeping patterns. Recent meta-analyses and systematic reviews provided evidence that quality and quantity (very-long or short) of sleep are highly associated with weight gain and increase incidence of diabetes, they could also predict cardiovascular and metabolic outcomes [110-112].

Diabetes prevention programs usually use lifestyle therapies to reduce the incidence of diabetes by reducing weight and increasing physical activity levels [113]. Moreover, other approaches like pharmacologic therapies including metformin [114], α -glucosidase inhibitors [115], and thiazolidinedione [116] were investigated for prevention and management of diabetes. Even bariatric surgery is considered one of the treatment options, especially for people living with morbid obesity and other comorbidities.

A randomized control trail entitled “The Diabetes Prevention Program” included 3,234 non-diabetic adults, with elevated fasting plasma glucose and/or elevated two-hour post prandial plasma glucose concentrations. The study included two intervention groups, an intensive lifestyle therapy group and a metformin (850mg

twice/day) group, and one control group receiving placebo. The mean follow-up period of the study was 2.8 years. The metformin group reduced the risk of diabetes development by 31%, whereas the intensive lifestyle intervention group decrease it by 58%, in comparison with the placebo group [114].

The ADA's recommendations in 2015 included the use of intensive lifestyle modifications as a first line therapy for diabetes prevention and management that embraces reducing the weight by 7% to 10% and increasing physical activity to 150 minutes per week (moderate-intensity). The use of metformin could be considered for patients with higher risk of developing diabetes (HbA1c > 6%, metabolic syndrome, or family history of diabetes). The use of other oral drugs has currently no specific recommendations, and could be used as a tertiary line of therapy [16].

2.4.2.5 Exercise

Physical activity was shown to promote weight reduction, improve insulin sensitivity, reduce ectopic adipose tissue, and decrease inflammatory markers. A systematic review and meta-analysis compared the effectiveness of dietary interventions versus dietary plus exercise interventions on the long-term (two years), confirmed the significant effect of diet plus exercise interventions in reducing weight, compared to the diet only interventions [117].

Many studies from around the world confirmed the beneficial effect of exercise in preventing and delaying the onset of MetS [118-121]. In the USA, 8,570 adult men participated in a cross-sectional fitness study. The study concluded high inverse association between cardiorespiratory fitness, muscular strength and the prevalence of MetS [122].

In Mexico, 5,118 adult participants were enrolled in a cohort study investigating the association of MetS risk factors and level of physical activity. The cohort noted a great reduction in the risk of developing MetS among men (OR 0.72; 95% CI 0.57-0.95) and women (OR 0.72; 95% 0.57-0.95) who reported exercising for more than or equal to 30 minutes per day during their leisure time [123].

Current recommendations of the IDF/AHA/NHLBI advocate working out for a minimum of 30 minutes five times per week. However, it is preferred to do moderate-intensity physical activity for ≥ 60 min/day, every day [4]. Patients should be advised to aim for weight reduction and weight maintenance when choosing the type and intensity of their work out. Pedometers and other activity tracker could be used for motivation [120]. Cardiac rehabilitation is highly recommended for patients with a high risk of CVD or who recently experienced coronary heart failure[124].

2.4.3 Bariatric Surgery

Treatment options for patients with obesity could also include bariatric surgery. Bariatric surgery could be considered as a treatment option for patients with morbid obesity (BMI >40 kg/m²) or those with obesity (BMI >35 kg/m²) plus serious obesity related comorbidities [125]. A meta-analysis published in 2013 included 11 studies and 796 participants (BMI mean 30-52), and compared non-surgical methods of treating obesity with surgical methods. The study found significantly higher reduction in weight induced by bariatric surgery [126]. A randomized control trail included 60 patients (30 and 60 years) with a BMI ≥ 35 , a five-year history of diabetes, and an HbA1c $\geq 7.0\%$, randomly divided into three groups. The first group received medical therapy, the second and third groups underwent two different types of bariatric surgery,

gastric bypass or biliopancreatic diversion. After two years of follow-up, the study found no reduction in diabetes in patients from the medical therapy group, a 75% reduction in the gastric bypass group and a 95% reduction in the biliopancreatic diversion group ($P<0.001$, Figure 2.7) [127]. The same results were obtained with similar trails investigating other surgical techniques like Roux-en-Y gastric bypass and Sleeve gastrectomy [128, 129].

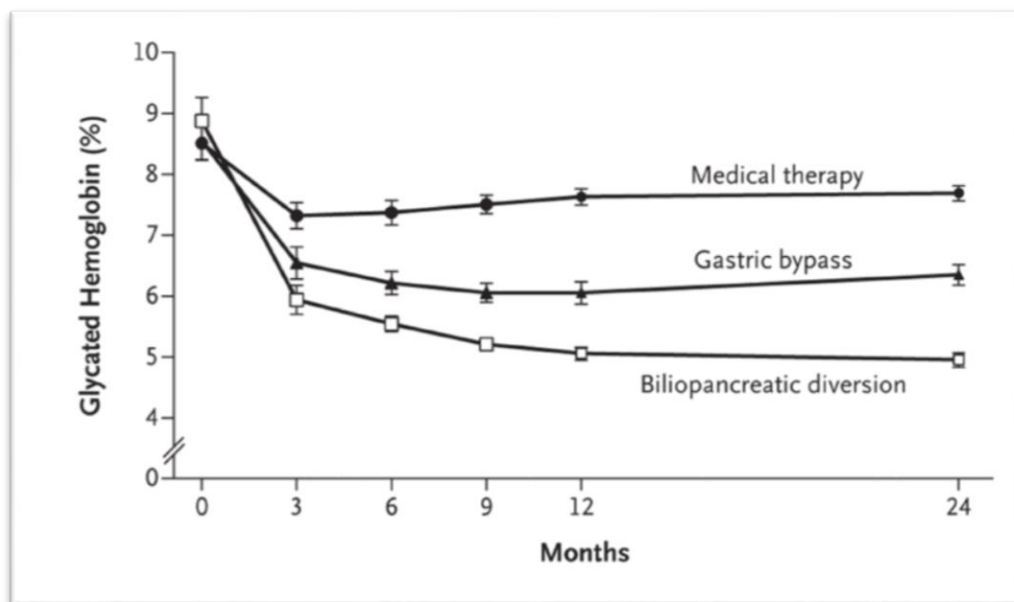


Figure 2.7: Change in the percentage of HbA1c during two years intervention of Medical Therapy, Gastric Bypass, or Biliopancreatic Diversion
Source: Mingrone and others, 2012 [127].

2.5 Prevalence of the Metabolic Syndrome

Prevalence of the MetS differs between nations, genders, age groups, ethnic groups, and also according to the definition used for diagnosing. Moreover, the prevalence of the syndrome is increasing among many populations around the world, this is mainly due to the decrease in physical activity, increase in rates of obesity and overweight, along with other environmental factors [130]. The total prevalence of the MetS among Emirati adults according to the IDF and ATPIII definitions was 40.5% and 39.6%, respectively in 2008[5]. This is higher than all other Gulf Cooperation Council (GCC) countries (Table 2.7, Figure 2.8), and amongst the highest worldwide (Table 2.8).

Table 2.7: Prevalence of the MetS among adults in GCC Countries

Country	Prevalence	Sample Size	MetS Definition	Reference
Oman	Men: 22.8% Women: 24.4% Total: 23.6%	3,137	WHO	El-Aty <i>et al.</i> , 2014 [131]
Qatar	Men: 20.7% Women: 32.1% Total: 26.5%	1,204	ATPIII	Bener <i>et al.</i> , 2009 [132]
	Men: 29.6% Women: 37.7% Total: 33.7%	1,204	IDF	Bener <i>et al.</i> , 2009 [132]
Kuwait	Men: 36.2% Women: 36.1% Total: 36.2%	2,280	IDF	Al Rashdan <i>et al.</i> , 2010 [133]
UAE	Men: 35.1% Women: 42.7% Total: 39.6%	4,097	ATPIII	Malik <i>et al.</i> , 2008 [5]
	Men: 32.9% Women: 45.9% Total: 40.5%	4,097	IDF	Malik <i>et al.</i> , 2008 [5]
KSA	Men: 36.6% Women: 34.1% Total: 35.3%	2,850	ATPIII	Al Daghri, <i>et al.</i> , 2010 [134]

WHO: World Health Organization; ATP: National Cholesterol Education Program Adult Treatment Panel; IDF: International Diabetes Federation; UAE: United Arab Emirates; KSA: Kingdom of Saudi Arabia.

It was noted that the MetS is more prevalent among adult women. The difference between men and women ranged from 0.1% in Kuwait to 13% in the UAE. This could be explained by the lower physical activity levels reported among Emirati females due to cultural and weather restrictions [135].

The prevalence of the syndrome was reported to be significantly associated with older age, in the same population. For example, in Saudi Arabia the prevalence was 12.8% in the 18-29 years old group, increased to 32% in those aged 30-39, 53.1% among adults aged 40-49 and 67.1% among older people (50-55 years) [134]. A greater association was also observed between obesity level and the prevalence of the syndrome in both genders. In China, for example, the prevalence of MetS increased from 9.8% in normal weight adult men to 26.9% in overweight adult men, and from 17.8% in normal weight adult females to 31.1% in overweight adult females [136]. The MetS prevalence is generally increasing significantly over time. For instance, the Korean population witnessed a great increase from 24.9% in 1998 to 31.3% in 2007 [137]. The prevalence of the MetS in different countries around the world is presented in Table 2.8.

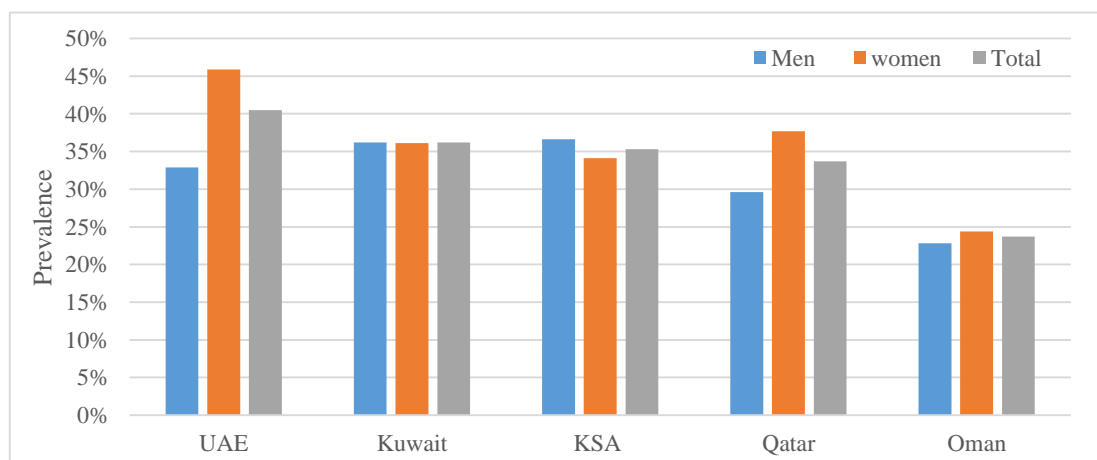


Figure 2.8: Prevalence of the MetS in GCC Countries
Source: From data presented in Table 2.7

Table 2.8: Prevalence of the MetS in different countries

Region	Country	Prevalence of MetS	Sample Size (age/years)	MetS definition	Reference
MENA	Turkey	33.9%	4,259 (20-90)	ATPIII	Kozan <i>et al.</i> , 2007 [138].
	Jordan	36.3%	1,121 (>25)	ATPIII	Khader <i>et al.</i> , 2007 [139].
	Lebanon	31.2%	499 (18-65)	IDF	Sibai <i>et al.</i> , 2008 [140].
	Tunisia	45.5%	836 (>40)	IDF	Harzallah <i>et al.</i> , 2006 [141].
	Iran	30.1%	10,368 (≥ 20)	ATPIII	Azizi <i>et al.</i> , 2003 [142].
Europe	Portugal	23.9%	1,436 (>18)	ATPIII	Santos <i>et al.</i> , 2004 [143].
	Spain	22.7%	11,149 (>18)	Harmonized definition	Guallar <i>et al.</i> , 2014 [144].
	Canary Islands	24.4%	578 (>18)	ATPIII	Alvarez <i>et al.</i> , 2003 [145].
	Italy	17%	2,100 (>19)	ATPIII	Miccoli <i>et al.</i> , 2005 [146].
Asia	Japan	7.8%	3,264 (20-79)	Japanese criteria	Arai <i>et al.</i> , 2006 [147].
	India	31.6%	1,123 (>20)	ATPIII	Gupta <i>et al.</i> , 2004 [148].
	China	21.3% 18.2% 10.5%	15,540 (≥ 18)	ATPIII IDF CDS	Xi <i>et al.</i> , 2013 [149].
	Korea	31.3%	2,890 (≥ 20)	ATPIII	Lim <i>et al.</i> , 2011 [137].
	Russia	19.3% 17.6% 18.9%	3,705 (18-90)	AHA ATPIII IDF	Sidorenkov <i>et al.</i> , 2010 [150].
America	Brazil	21.6%	251 (20-88)	ATPIII	Velásquez <i>et al.</i> , 2007 [151].
	U.S.A	34%	13,635 (>20)	ATPIII	Ervin, 2009 [152].
	Canada	19.1%	1,800 (≥ 18)	ATPIII	Riediger <i>et al.</i> , 2011 [153].
Australia	Australia	22.1% 21.7% 30.7% 13.4%	11,247 (≥ 25)	ATPIII WHO IDF EGIR	Cameron <i>et al.</i> , 2007 [154].
	New Zealand	Māori: 32% Pacific 39% Europeans 16%	1,006 996 2,020 (35-74)	ATPIII	Gentles <i>et al.</i> , 2007 [155].

MENA: Middle East and North Africa; USA: United States of America; ATP: National Cholesterol Education Program Adult Treatment Panel; IDF: International Diabetes Federation; CDS: Chinese Diabetes Society; WHO: World Health Organization; EGIR: European Group of Insulin Resistance.

This high prevalence of the syndrome was not only observed among adults, many studies reported high numbers in adolescents as well. In the US, the prevalence of the MetS among adolescents (12-19 years) was 4.2% [156]. Six point five percent of Mexican children and adolescents (10-18 years) met the ATPIII definition of the syndrome [157]. In Saudi Arabia, the prevalence among 10-18 years old boys and girls was 9.4% [158]. In Kuwaiti female adolescents (10-19 year), 14.8% met the IDF defining criteria of the MetS [159]. Moreover, 13% of Emirati adolescents were reported to have the MetS [6]. These numbers are extremely high and action is urgently needed at the national and global level to reduce the prevalence of MetS, and its components, especially obesity. The obesity epidemic is usually blamed on physical inactivity and poor dietary habits [160].

Studies from the UAE reported very low physical activity levels between adults and adolescents, especially females [135, 161, 162]. In 2009, Belal reported that the prevalence of physical inactivity is almost 37.9% among men and 56.7% among women. Moreover, 38.4% of adolescent males and 42.6% of adolescent females spend ≥ 3 hours/day sitting [163]. In a qualitative study among Emirati female adults, the first barrier to being physically active was stated to be the lack of social support from friends and families [164]. Another study claimed that low physical activity levels between Emirati female adolescents is mainly due to cultural and climate restrictions [135]. As a matter of fact, there is a lack of national-level studies investigating the level of physical activity in the country, barriers to being active, and transition of physical activity trends overtime. This type of information is highly important as it is the basis for developing future interventions, policy making decisions, and assessing these actions in the long term.

Dietary habits in the UAE are better documented in the literature. In 1998, Musaiger and others reported very low consumption of vegetables, fruits and dairy products (yogurt, cheese and milk) among Emirati adults [165]. In 2009, Bin Zaal and others, indicated high consumption of fast food and high caloric snacks among Emirati adolescents, and found a significant association between the consumption of chocolates, sweets, fast foods, soft drinks, and rates of obesity in Emirati adolescent girls [166]. In the same year, Bilal stated that 77.5% of men and 75.7% of women eat less than five servings of fruits and vegetables per day [163].

Recently, a study was published about nutrition transition in the UAE. The study presented data about the high prevalence of obesity and factors behind it. Positive energy balance was seen among all age groups, with around 40% of boys and girls (6-10 years) consuming more calories than their requirements. More than 20% of calories consumed were coming from calorie-dense snacks and 14% from empty-calorie drinks [167]. Other than the low consumption of fruits and vegetables, many studies blamed high rates of obesity on the replacement of traditional Emirati foods by westernized high caloric fast foods, and the replacement of water with empty-calorie soft drinks [165-168]. A very recent study investigated the nutritional knowledge about Emirati traditional foods among Emirati adults. About 50% of females and 70% of males stated that they consider traditional foods healthy; however, only 18.2% of males and 14% of females reported daily consumption of traditional foods [169]. Conversely, other studies from the Gulf region reported considering traditional food as an unhealthy option, and it might be the reason behind the epidemic of obesity and T2DM. These studies build their conclusions on food composition data available in their countries, showing high carbohydrates and fat content in traditional foods [160, 170-173].

Unfortunately, very little information is available about the food composition and glycemic index of Emirati traditional foods. Food composition databases offer comprehensive information on the concentrations of nutrients in foods; this information is considered the base of any quantitative study of nutrition. It is also useful in clinical practice, food manufacturing, designing health promotions, regulation of nutrition and health claims, epidemiological studies, and policy decision making [174].

Recently, ten traditional dishes commonly consumed in the UAE were chemically analyzed for proximate composition and mineral content [175]. However, some information is still needed to make better nutritional recommendations. For instant, lipid profile, such as monounsaturated fatty acids, polyunsaturated fatty acids, saturated fatty acids and trans-fatty acids content is considered crucial for planning a diet like the Mediterranean diet. Glycemic index and glycemic load are also very necessary for meal planning, especially in a population with high prevalence of MetS, diabetes and CVD. Planning low glycemic diets using traditional foods could be the key for better glycemic management and might prevent the progression of the MetS into T2DM and CVD as discussed earlier in Section 2.4.2.4.

2.6 Glycemic Index: History

The dietary fiber hypothesis developed in the 1970s by Trowell was the first step into the establishment of the glycemic index concept [176]. Trowell observed that the fiber part of carbohydrates was not absorbed in the gut, which influenced certain actions within the lumen of the gastrointestinal tract. He linked this observation to the risk of some diseases like diabetes and ischemic heart disease [177, 178].

In 1981, David Jenkins and his colleagues invented the term “glycemic index” [20]. The concept was developed from observing the dramatic variation in blood glucose response after the ingestion of carbohydrate rich foods. Ever since, many studies started investigating the possible health benefits of low glycemic index diets. The GI concept was first used for the prevention and management of diabetes [21]. Afterwards, studies also investigated other important implications of the GI for the prevention and treatment of obesity and coronary heart disease [179, 180]. Recent evidence indicates the role of low glycemic index diets in the treatment and prevention of diabetes [181], hyperlipidemia and CVD [182]. Moreover, low GI diets were associated with improved insulin sensitivity [183] and regulating appetite [184].

In 1995 the first International Tables of Glycemic Index were published. The aim of these tables was to gather all published data on the GI from different studies and produce a convenient tool to be used in research and clinical practice [185]. About 600 separate entries were included in the first edition of the GI tables. Furthermore, in 2002 [186] and in 2008 [187] an updated version of the GI tables was published and included GL values as well. The 2008 International Table of Glycemic Index and Glycemic Load Values includes more than 2,480 individual food items [187].

The use of GI for the classification of carbohydrate-containing foods has been validated by the Food and Agriculture Organization of the United Nations and the World Health Organization in 1998 [188]. They recommended the use of GI of foods along with food composition tables as a guide for food choices. The American Diabetes Association also recommends the use of low GI diets for diabetes management and prevention [16]. Since many studies reported a reduction in HbA1c levels from around 3% to about 19% when low GI diet was implemented [181, 189, 190].

2.6.1 Definition of the Glycemic Index and Glycemic Load

The glycemic index is a measure of how fast a certain food could increase blood glucose after ingestion compared to that of pure glucose. It is defined as “the incremental area under the blood glucose response curve of a 50g carbohydrate portion of a tested food expressed as a percent of the response to the same amount of carbohydrate from a standard food taken by the same subject (either white bread or glucose), on a different day” [188]. The area under the curve (AUC) of the blood glucose response after consumption of pure glucose is given the value of 100. The AUC of blood glucose curve after ingestion of a carbohydrate-containing food is compared to that of pure glucose and classified as low (induces a slow raise of plasma glucose), intermediate, or high (causes a quick and sharp increase of plasma glucose) [20]. The categories of the GI values are presented in Table 2.9. Foods like legumes and non-starchy vegetables tend to have low glycemic index, while foods like rice, white bread, and honey cause larger post prandial elevation in plasma glucose concentrations and therefore considered high in glycemic index [182].

In 1997, the concept of “glycemic load” was introduced [21, 191]. Glycemic load is a way to measure the glycemic index of foods depending on a standard portion size. It is typically calculated as the glycemic index of the food multiplied by the amount of available carbohydrate in standard serving, divided by 100 [21]. It is regularly essential to consider the GL in conjunction with GI values, particularly when the carbohydrate content of the test food is relatively small. For instance, broad beans have a high GI but because of their low carbohydrates content, they are classified as a low GL food [186] . The categories of the GL values are presented in Table 2.9.

Table 2.9: Categories of Glycemic Index and Glycemic Load Values

Category	Glycemic Index Value	Glycemic Load Value
Low	≤ 55	≤ 10
Medium	56-69	11-19
High	≥ 70	≥ 20

Source: Brand-Miller and others, 2005 [192], Salmeron and others, 1997 [191].

2.6.2 Methodology of the Glycemic Index

The first protocol of measuring GI with clinical utility and without methodological controversy was described by Wolever and others in 1991 [193]. This protocol was later adopted by the FAO/WHO in 1998 with minor modifications [188].

The sample size for testing glycemic index with sufficient statistical power should be at least 10 test subjects [188]. Fifty grams of available carbohydrates is used to calculate the portion size of test foods, which is equivalent to 50 g of standard food (glucose). However, for low carbohydrate foods, 25 g could be used [194]. Tests should be randomized in blocks and the duration of testing should not exceed four months [194]. The standard food should be measured twice and once for each test food,

with one day gap between tests [188]. The evening before a test, volunteers should restrict caffeine-containing drinks, avoid vigorous physical activity and fast overnight [195]. During the test, subjects should consume foods with 250 ml of water within 10-15 min. Capillary finger-prick blood samples are collected while fasting and at 15, 30, 45, 60, 90, and 120 min after ingesting the test meal. The area under the glucose-response curve is calculated based on incremental AUC, by ignoring the area under the baseline. Incremental area under the curve (IAUC) for each food is calculated as a percent of the mean response to the IAUC of standard food taken by the same subject. The values of all subjects are then averaged to obtain the GI value for the food [193].

According to the FAO/WHO expert report on the GI methodology, The GI is calculated using the following equation:

$$GI = \frac{IAUC \text{ for the test food containing } (X)g \text{ of available CHO}}{IAUC \text{ of a reference food with an equal CHO portion}} \times 100$$

The IAUC under each blood glucose response curve (for standard food or test food) is calculated as illustrated in Figure 2.9, by adding areas A+B+C+C+D+E+F all together and ignoring the negative areas [188]. The individual IAUC values for each test food in each subject are presented as a percentage of the mean IAUC value for the repeated standard food tests taken by the same subject. The GI of each test food is calculated as the mean of the resulting GI values from different subjects for each food [194].

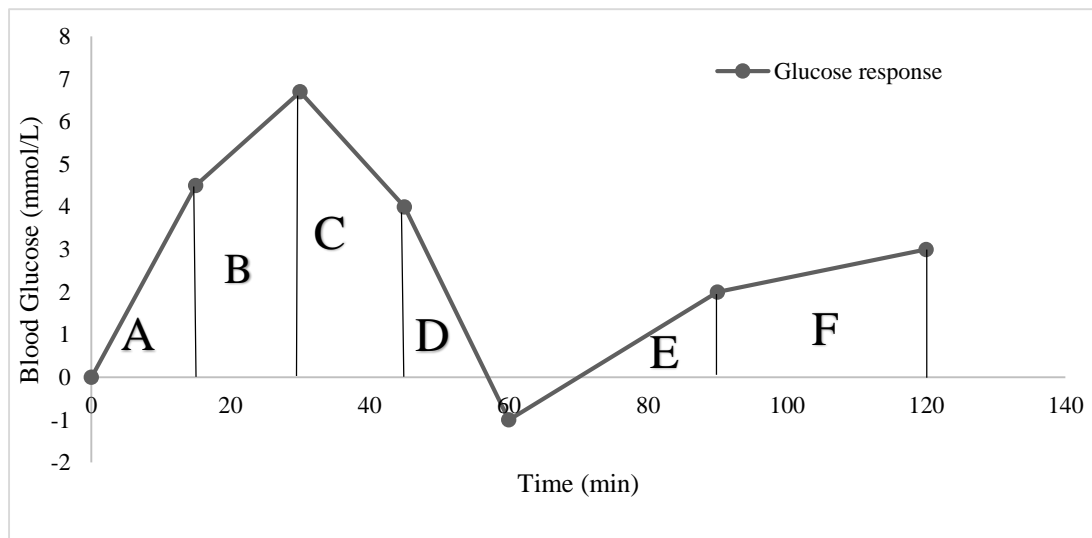


Figure 2.9: The Incremental Area Under the Curve (IAUC) equals the sum of the areas A, B, C, D, E and F. Negative areas are not included

Source: FAO/WHO, 1998 [188]

2.6.3 Factors influencing the Glycemic Index

The GI of foods varies significantly due to many factors such as cellular structure (e.g. degree of ripening), gross structure (e.g. grinding and particle size), granular starch structure (e.g. heat treatment), amount of carbohydrates, type of sugar (i.e. glucose, sucrose, lactose, or fructose), co-existence of nutrients (i.e. dietary fiber, fat, or protein), starch nature (amylose and amylopectin), cooking method, and food processing [194]. Other factors could include the addition of organic acids, gelling fibers, amylase inhibitor or sugars to the food [196]. Therefore, there is usually a considerable variation in the GI of the same food produced by different manufacturers or in a different country. A summary of these factors is presented in Table 2.10.

Table 2.10: Main Factors Affecting the GI of Foods and Meals

Food Factor	Example	Effect on GI
Starch type	Amylopectin (branched)	Higher GI
	Amylose (unbranched)	Lower GI
Starch granular structure	Heating	Higher GI when gelatinized
Degree of ripeness	Higher ripeness	Higher GI
Processing	Grinding, milling, or rolling	Higher GI
Acids	Added	Reduce GI
Gelling fibers	Added	Reduce GI
Amylase inhibitor	Added	Reduce GI
Added sugars	Fructose	Reduce GI
Protein	Added	Reduce GI
Fat	Added	Reduce GI

Source: Arvidsson-Lenner and others, 2004 [196].

2.6.4 Glycemic Index in Health and Disease

Recently, the glycemic index became not only popular as a useful tool in planning meals for patients with diabetes, but also as an important intervention for the prevention and management of dyslipidemia, cardiovascular disease, obesity, and other chronic diseases in the general population [189]. The hypothesis suggests that a higher increase in the postprandial glycaemia, is the triggering mechanism for disease progression [19].

2.6.4.1 Glycemic Index and Obesity

Epidemiological studies show a great increase in the epidemic of obesity worldwide. In the United States, the prevalence of overweight adults (≥ 20 years) was 33.9% in 2012 [197]. In the same report, 35.1% of the US population was found obese, and 6.4% met the criteria of extreme obesity (BMI greater than or equal to 40) [197]. In the UAE, the prevalence of overweight and obesity among female adults increased

from 36% in 2000 to reach 48% in 2010 [167]. This included an increase in extreme obesity among Emirati women from 7% in 2000 to 17% in 2010 [167]. The reason behind this epidemic is blamed on positive energy balance, characterized by low energy expenditure due to physical inactivity, and high energy intake due to over consumption of calorie dense foods [198]. Yet the reality is that it is more complicated because many genetic and environmental factors are involved. In an attempt to solve the issue, many diets were developed over the years to promote weight loss and maintenance, such as the low fat diet, high protein diet, low calorie diet and very low calorie diet [104, 199]. These diets have shown poor compliance in the long run, and were all associated with weight regain after a period of time [105]. This is probably due to the effect of these diets on reducing basal metabolic rate, therefore, compensating for caloric reduction [200].

The low glycemic index diet does not restrict caloric intake nor limit the intake from any of the food groups that are essential for a balanced diet. It simply advises increasing the consumption of foods from the low glycemic index category and avoid those from the high glycemic index category. Moreover, recent evidence suggest that low glycemic index diets may help prevent weight regain and promote weight reduction maintenance after weight loss, by reducing the usual decrease in energy expenditure and basal metabolic rate associated with weight reduction [201].

There are two suggested mechanisms by which a low GI diet is suggested to promote weight loss. First, by reducing appetite and food intake owed to differences in plasma glucose and insulin concentrations. The second hypothesis is that low GI diet reduces fat storage and lessens fat oxidation due to reduced plasma insulin responses [179].

Many studies investigated the effect of a low glycemic index diet on body weight. In 2014, a randomized control trial of 104 obese adolescents (15-18 years), evaluated the effect of a low GI diet versus a conventional Chinese diet on BMI over six months. This intervention trial reported an association between low GI diet and decreased calorie intake, healthier dietary composition (increased fiber intake and reduced fat intake), and reduced BMI in obese adolescents [202]. Another randomized control trial published in 2014 included 122 individuals with overweight and obesity. Participants were randomly assigned to a high-GI diet, a low-GI diet, or a low-fat diet. After six months of intervention, the trial reported a significant reduction in the BMI (Figure 2.10) and improvement in insulin sensitivity in the low GI diet group compared to the low-fat [203].

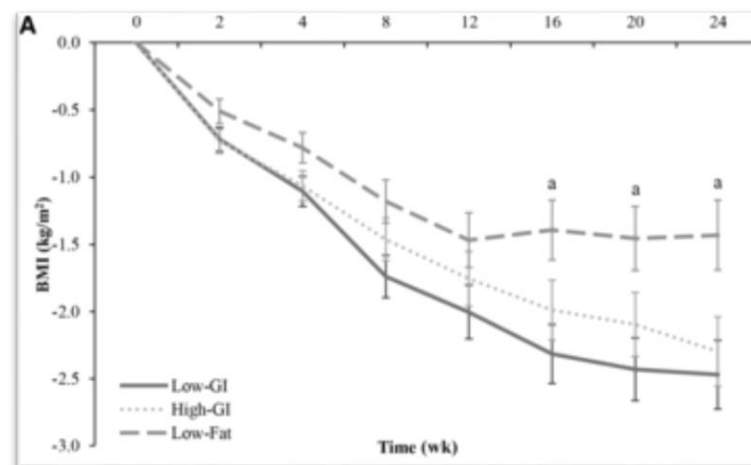


Figure 2.10: Changes in BMI during the follow-up for each intervention group.

Source: Juanola-Falgarona and others, 2014 [203].

In contrary, another randomized clinical trial entitled “DIOGENES” investigated the effect of a high dietary protein diet and GI diet on weight loss maintenance in people living with overweight and obesity in two centers across Europe. The one-year results of this intervention reported no significant difference

between the two diets in reducing weight; however, the high protein diet was associated with less weight regain after the year of intervention [204].

It is important to keep in mind that a high GI diet is not the only factor contributing to the epidemic of obesity. In fact, many genetic and environmental factors are involved in a very complex manner, not only at the individual level, but also at the family, community, country and even global level [179]. Low GI diet has been shown to reduce weight and help in maintaining lost weight, however, controversy is still present because there are no long-term and big-scale studies available on GI and body weight [100].

2.6.4.2 Glycemic Index and Diabetes

Diabetes is a chronic disease characterized by abnormalities in the metabolism of carbohydrate, fat, and protein along with hyperglycemia. Complications of the disease include macrovascular and microvascular damage in the eyes (retinopathy), kidneys (nephropathy) and nerves (neuropathy). If left untreated, these complications could progress into blindness, kidney failure, amputation and even death [205].

During the past three decades, the prevalence of diabetes mellitus in the world has doubled, which presented a great challenge to the public health of all nations. According to the recent regional fact sheets of the International Diabetes Federation [2] there are 382 million people living with diabetes in the world with a prevalence of 8.3%. The IDF expected an increase by 205 million by 2035. The prevalence of diabetes in the Middle East and North Africa (MENA) region is even higher than the world average with 9.7%. Based on the same fact sheets, United Arab Emirates was ranked as having the fifth highest prevalence in the MENA region (19%), coming after

other GCC countries, Saudi Arabia, Kuwait, Bahrain and Qatar having the prevalence of 23.9%, 23.1%, 21.9% and 19.8% respectively [2].

Diet and lifestyle modifications are considered the basis for preventing and managing diabetes. The main aim of therapeutic lifestyle changes for people living with diabetes are weight reduction, improving glycemic control and reducing the risk of cardiovascular comorbidities [16]. Postprandial glycaemia could be controlled by controlling the amount of carbohydrates in a diet (carbohydrate counting and exchange list) or the nature of carbohydrates in food (glycemic index). The use of glycemic index for managing diabetes is still controversial. The American Diabetes Association (ADA) reviewed the evidence on the use of GI as an intervention for diabetes prevention and management in the “2015-Standards of Medical Care in Diabetes” publication [16]. They stated that the use of glycemic index and glycemic load in individuals with diabetes is recommended as studies have demonstrated a reduction in HbA1c by -0.2% to -0.5% when a low GI diet was followed [16]. The European Association for the Study of Diabetes (EASD) also recommended people with diabetes to choose low GI foods for better postprandial glycemic management [206]. However, some researchers have criticized the use of the GI system for people with diabetes, claiming that it is very complex and it might limit food choices if adopted as a lifestyle [182].

2.6.4.3 Glycemic Index and Cardiovascular disease

According to the World Health Organization, cardiovascular diseases are the leading cause of death globally. In 2012, about 17.5 million people died from cardiovascular diseases, representing 31% of total global deaths [1]. Among these deaths, coronary heart disease was behind 7.4 million and 6.7 million were due to

stroke [1]. Cardiovascular diseases (CVDs) are a cluster of disorders occurring in the heart and blood vessels, such as coronary heart disease, ischemic heart disease, stroke, cerebrovascular disease, peripheral arterial disease, rheumatic heart disease, congenital heart disease and others. Most of the CVDs are preventable through improving dietary habits, cigarettes cessation, physical activity and reduction in alcohol consumption. Diets targeting prevention of CVDs mainly focus on the type and amount of fats present in a diet, such as the Mediterranean diet [207]. However, epidemiological studies have reported an association between high glycemic index and glycemic load diets and increased risk of CVDs [100, 108].

The 2011 DIOGENES trial, a controlled dietary intervention study from across Europe that included 932 overweight adults who were randomized to one of the five ad libitum diets for 6.5 months. The diets were either high protein, low protein, high GI, low GI, or a control. The study resulted in a greater reduction in high-sensitivity C-reactive protein blood levels (-0.46 mg/L; 95% CI -0.79 to -0.13) among the groups assigned to GI diet, compared to the high GI diet group ($P < 0.001$). This reduction in low-grade inflammation suggests a positive effect of GI diet with cardiovascular risk [208]. A randomized control trial among 73 people living with obesity (18-35 years) studied the difference between low GI diet and low-fat diet for six-months intervention and a one-year follow-up. The study indicated a beneficial effects of the low GI on high-density lipoprotein cholesterol (HDL-C) and triglyceride concentrations even after the intervention was over [107].

Available evidence supports the role of glycemic index and glycemic load in cardiovascular disease risk management and prevention; however, further studies

investigating risk of CVD in relation to GI and GL are needed to provide the required power for future meta-analyses on this topic [209].

2.7 Summary

The epidemic of metabolic syndrome was thoroughly discussed in this literature review. The importance of addressing this cluster of diseases in the UAE population was highlighted, and recommendations for prevention and management mainly through dietary practices were discussed.

Chapter 3: Materials and Methods

3.1 Introduction

This chapter outlines and discusses the research design, methodology and analyses followed in each of the two experiments presented in this dissertation; therefore, it is divided into two sections:

- Section I: prevalence of the metabolic syndrome and its component factors among female students at United Arab Emirates University and their association with anthropometric measurements;
- Section II: Glycemic Index (GI) values of commonly consumed foods in the UAE.

For both sections, the participants' recruitment, research design schemes, data collection and data analysis are discussed.

3.2 Prevalence of the Metabolic Syndrome and its Component Factors among Female Students at United Arab Emirates University and their Association with Anthropometric Measurements

3.2.1 Study Population

This study is a cross-sectional population-based epidemiological study. It was conducted during the academic year 2013/ 2014 at United Arab Emirates University (UAEU) in Al-Ain, United Arab Emirates. The study population included students from all eight colleges of the university except the College of Medicine and Health Sciences, as they are located in a different campus. The sampling method was stratified random sampling [210]. All female students were divided into strata by college, then a random subsample consisting of 10% of the students from each college were proportionally selected. Those were then contacted through e-mail to voluntarily participate in the study.

The total number of females registered in the university during that academic year was 8846 students. 885 students received an invitation to participate. 654 students showed interest in the study with a response rate of 74%, and out of these 99 students were excluded from the study for the following reasons: 56 students did not show up for the data collection appointment, 25 students refused to give blood, 7 students were pregnant, 5 students were breastfeeding, 4 students were on long-term medication and 2 students did not have good blood flow. Therefore, the final number of participants was 555 female students between the age of 17 and 25 years old (Figure 3.1).

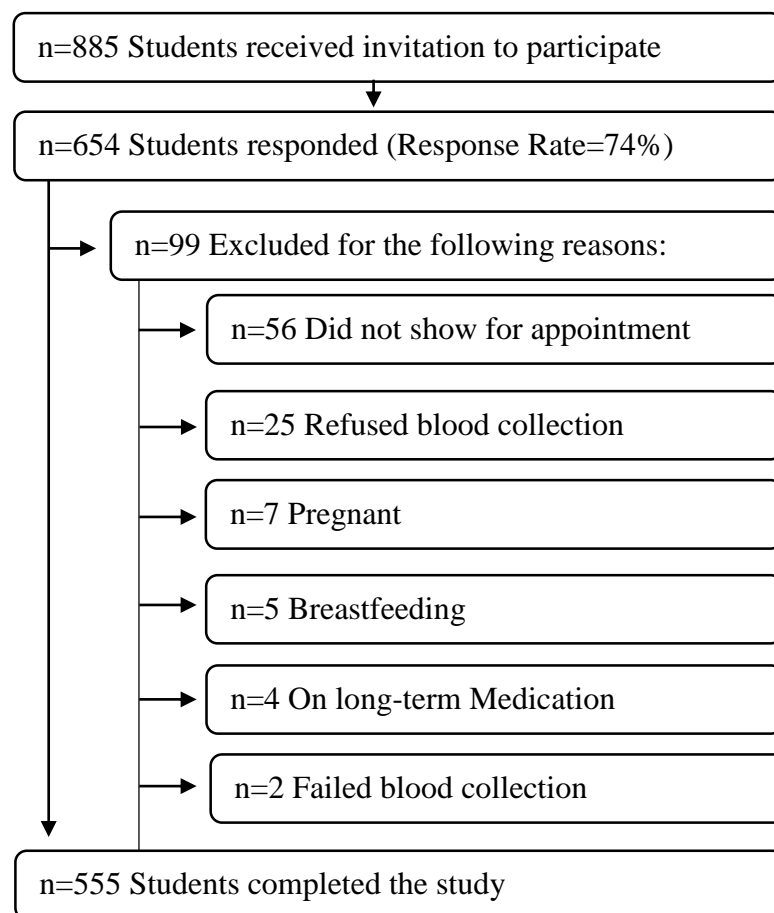


Figure 3.1: Study Participant's Enrolment Process

Participants were asked to read the study information sheet carefully and were given the chance to ask questions related to the study before taking part (Appendix 1).

Participants were excluded if they did not meet the following inclusion criteria: had a disease or was on medication; was pregnant or breastfeeding; older than 25 years old or younger than 17 years old; and if she refused to provide blood sample. A written informed consent to participate in the study has been obtained from all participants before taking part (Appendix 2). An identification number was assigned to each participant to maintain anonymity, which confirmed confidentiality and helped link the participant to their clinical measurements and blood samples. Only cumulative data was reported to protect the privacy of all participants.

3.2.2 Ethical Approval

Ethical approval for the study was obtained from the United Arab Emirates University Scientific Research Ethics Committee (UAEU, Reference Number DVCRGS/370/2014) and from Al Ain Medical District Human Research Ethics Committee (Number 14/48) (Appendix 3). All participants who agreed to join the study provided a written informed consent before taking part, as attached in Appendix 2.

3.2.3 Anthropometric Measurements

The use of anthropometric measures for the assessments of the nutritional status of individuals and societies was first introduced by Brožek in 1956 [211]. It has been found that anthropometric measures have a great advantage as they can be related to past exposures, present practices or health related events in the future. Anthropometric measurements are normally divided into two groups, the first group contains measures for the assessment of body size, such as body weight, height and

head circumference. The second group determines body composition, which includes body fat and body fat-free mass [10, 212]. Both groups are highly important and their use has been recently increased in clinical settings, assessing interventions' outcomes and conducting surveillance at the population level [213].

Body size and composition measures including: height, weight, body fat composition, waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR), neck circumference (NC), mid-upper arm circumference (MUAC) and skin-fold thickness at four sites (biceps, triceps, subscapular and suprailiac) were all carried out by the researcher (Mohamad, M.) in the nutrition clinic of the Nutrition and Health Department at UAEU. All measurements were completed during a single fifty-minute session with the participants reporting to the clinic at fasting state and have restricted fluid (excluding water intake) for 12-14 hours prior to testing.

Results were recorded immediately after the measurements on a data sheet (Appendix 4) and were then checked by another skilled researcher. Each measurement was taken three times and averaged. As the participants of this study were all females, they were asked not to visit the clinic during their menstrual cycle. Measures were taken while the participants wore minimal clothing (as local culture permits) and no shoes. Participants were asked to rest for 15 minutes to allow climate adjustments and relaxation before any measurements were taken. During rest time, they were asked to complete a brief health questionnaire (Appendix 5). It included questions about demographic data, supplements and medications use, tobacco use, diet, physical activity, sleeping patterns, perceptions about obesity, personal history of NCDs, and family history (first-degree relatives) of NCDs. Participants completed the questionnaire under the supervision of the research team to respond to any clarification

needed on any aspects of the questionnaire or the study as a whole. All devices used for measurements were calibrated on a daily basis throughout the study.

3.2.3.1 Height

Height was measured using a portable stadiometer (Seca Stadiometer, Seca Ltd, Birmingham, UK), and was measured to the nearest millimeter. Participants were asked to take a deep breath and stand upright, looking straight ahead horizontal with the Frankfurt plane, with shoulders relaxed, legs straight, and knees placed together. They were also asked to keep their feet flat with heels close together, and their shoulder blades, buttocks, and heels touching the measurement board [10].

3.2.3.2 Body Weight and Body Composition

Body weight and body composition were measured using the Tanita Segmental Body Composition Analyzer (Tanita BC-418 Ltd, Tanita UK, Figure 3.2). The analyzer was placed on a hard, flat surface, and body weight was recorded to the nearest 0.1 kg.

The Tanita segmental body composition analyzer uses the BIA (bio-electrical impedance analysis) method for analysis. It measures impedance by introducing a safe known amount of constant current source with a high frequency (50 kHz, 500 μ A) into the body. It then calculates body fat percentage, fat mass, fat-free mass, and predicted muscle mass through a regression formula using height, weight, age, and impedance between right hand and foot as variables[214].



Figure 3.2: Tanita Segmental Body Composition Analyzer BC-418

Participants were asked to step bare foot on the weighing platform unassisted, looking straight ahead, making sure that heels were placed on the posterior electrodes, and the front part of the feet were in contact with the anterior electrodes. Participants were asked to stand still for few seconds to allow weight measurement to appear on the screen, then were asked to grasp the grips with both hand and wait for a few more seconds to allow measurement of the impedance.

3.2.3.3 Body Mass Index

The most commonly used measure for the classification of adult population according to their weight status is the ratio of weight to height which is known as Body Mass Index (BMI) or (Quetelet's Index). BMI is calculated by dividing the weight in kilograms by the height squared in meters (kg/m^2) [215]. The latest publication of the World Health Organization (Table 3.1) was used as a guideline for the classification of adults according to BMI [198].

Table 3.1: WHO Classification of Overweight in Adults According to Body Mass Index

Classification	BMI (kg/m²)	Risk of comorbidities
Underweight	< 18.50	Low
Normal range	18.50 – 24.99	Average
Overweight	≥ 25.00	
Pre-obese	25.00 – 29.99	Increased
Obese class I	30.00 – 34.99	Moderate
Obese class II	35.00 – 39.99	Severe
Obese class III	≥ 40.00	Very severe

Source: WHO, 2000 [198].

3.2.3.4 Waist Circumference

Waist circumference (WC) showed high correlation with abdominal fat content, measured by computer tomography or by dual X-ray absorptiometry (DXA), in several studies [54, 216]. Measurement was performed using a plastic tape touching the skin, with a width of 0.7cm, held in a horizontal plane, midway between the inferior margin of the ribs and the superior border of the iliac crest or at umbilicus level for obese participants [217]. The participants were asked to stand erect, relaxing the abdomen, with arms placed on the side, and weight equally divided over both legs. WC was recorded to the nearest millimeter. The IDF and the American Heart Association/the National Heart, Lung, and Blood Institute (AHA/NHLBI) cut-offs [4] (Table 3.2) were used according to ethnicity and gender to identify the increased risks associated with excess abdominal fat like that of diabetes and metabolic syndrome.

Table 3.2: Cut-off Values of Waist Circumference Based on Ethnicity and Gender

Country/ ethnic group	Gender	Waist circumference
Europids	Male	≥ 94 cm
	Female	≥ 80 cm
South Asians	Male	≥ 90 cm
	Female	≥ 80 cm
Chinese	Male	≥ 90 cm
	Female	≥ 80 cm
Japanese	Male	≥ 85 cm
	Female	≥ 90 cm
Ethnic South and Central Americans	Use South Asian recommendations until more specific data are available	
Sub-Saharan Africans	Use European data until more specific data are available	
Eastern Mediterranean and Middle East	Use European data until more specific data are available (Arab) populations	

Source: Alberti, 2009 [4].

3.2.3.5 Hip Circumference

Hip circumference (HC) is usually not used for assessing body composition and the distribution of body fat as a single measure. It is rather used along with the WC to calculate the waist-hip circumference ratio (WHR). HC was measured using a plastic tape, with a width of 0.7cm, held in a horizontal plane by touching the skin without pressing the soft tissue, at the level of maximum posterior extension of the buttocks [218]. Participants were standing erect, relaxing the abdomen, feet together and arms placed on the side [219], HC was recorded to the nearest millimeter.

3.2.3.6 Waist-Hip Circumference Ratio

Waist-Hip circumference ratio (WHR) is a simple and easy method which allows distinguishing between fatness in hips and buttocks areas and fatness in the abdomen and waist areas. It is calculated by dividing waist circumference by hip circumference. Several studies reported strong association between elevated WHR and increased risk of developing type 2 diabetes (T2D), ischemic stroke and coronary heart disease [220, 221]. The cutoffs values used for waist-hip ratio were (0.90) and (0.80) for men and women, respectively, as suggested for Middle Eastern populations in 2011 by the World Health Organization [222].

3.2.3.7 Neck Circumference

Neck circumference (NC) is used as an indicator for upper-body subcutaneous adipose tissue distribution. Several population-based studies have shown an elevation of cardiovascular and metabolic syndrome (MetS) with the increase of neck circumference values [9, 223]. NC was measured at mid-neck height, between midcervical spine and midanterior neck and recorded to the nearest 1 millimeter, using plastic tape, calibrated weekly, with the tape not being too tight or too loose, and just lying flat on the skin. Participants were asked to keep the head up, relax the shoulders and look straight ahead [224]. The cutoffs for neck circumference are not well established yet; however, in the Chinese population the values of ≥ 38 cm and ≥ 35 cm for men and women respectively, were reported to be the best cutoffs to identify overweight participants. However, the same study suggested that the values of ≥ 39 cm and ≥ 35 cm for men and women respectively, were the best cutoffs to determine metabolic syndrome (MetS) participants [9].

3.2.3.8 Mid-Upper-Arm Circumference

Mid-upper-arm circumference (MUAC) is usually used for the determination of nutritional status in population-based studies, as it is useful for the diagnosis of protein-energy malnutrition and participants' starvation, especially in children and adolescents [225]. Participants were asked to remove their sleeves and stand erect, and bend their right arm at the elbow in a perpendicular angle. The edge of the shoulder (acromion process on shoulder blade) and elbow (olecranon process of the ulna) were located and marked. Plastic non-stretchable tape was used to measure the distance between the two marks and the middle point was located and marked. The MUAC was then measured using the same measuring tape, wrapped around the mark, while the arm is hanging down the side of the body and relaxed. MUAC was recorded to the nearest millimeter [226]. MUAC values of 23.0 cm and 22.0 cm for men and women respectively, were suggested to be useful cut-off points for simple screening of nutritional state in adults of the third world [227].

3.2.3.9 Skinfold Thickness

Skinfold thickness measurements are usually used for the estimation of total body fat; a single skinfold measure or multiple skinfolds could be used. Many studies have suggested the use of skinfolds for assessing subcutaneous fat considering the low cost, simplicity (when performed by trained examiner) and effectiveness in comparison with ultrasound [228, 229].

A Lange Caliper (Cambridge Scientific Industries, Cambridge, MD, Figure 3.3) was used for the measurement of skinfold thickness at four sites: triceps, biceps, subscapular and suprailiac sites as described by Lohman and others in 1988 [230].

During all skinfold measures, participants were asked to stand erect, with shoulders relaxed and arms hanging freely at their sides. The right side of the body has been used for taking all measures, as it is the current practice by the National Health and Nutrition Examination Survey (NHANES). The middle point in the upper right arm was already marked for the measurement of the MUAC. This point was used for the measurement of triceps (from the back side of the arm) and biceps (from the inner side of the arm). The skin and underlying fat were grasped at a vertical angle, about 2 cm above the mark, using the thumb and the forefinger of the left hand of the examiner. The caliper was then placed on the mark and the value was recorded to the nearest 0.5 millimeter. Measures were repeated three times and averaged [231]. The subscapular skinfold was measured by grasping the skin and underlying fat at the inner border of the scapula at an angle of 45° from horizontal. The suprailiac was taken from the midaxillary line directly above the iliac crest by picking up the skin and underlying fat at an angle of 45° following the natural folding of the skin [10].

The logarithmic transformation of the sum of means from all four sites of skinfold thickness was used for the calculation of equivalent fat content as a percentage of total body weight. Fat content was calculated using specific linear regression equation for females between 17-29 years old, as described by Durnin and Womersley in 1974 [232].



Figure 3.3: Lange Caliper

3.2.4 Biochemical Measurements

Biochemical measurements are usually used to confirm a clinical diagnosis or to detect abnormalities in the levels of lipid profile. Unlike clinical, dietary and anthropometric measures, biochemical measures provide an objective method for the assessment of nutritional status [10]. Static biochemical measures were used in this study, with the biological fluid being blood, and the parameters of interest being: fasting plasma glucose (FPG); total cholesterol (TC); high density lipoprotein (HDL); low density lipoprotein (LDL); triglycerides (TG); high sensitivity C-reactive protein (Hs-CRP); hemoglobin (Hb); and glycated hemoglobin (HbA1c). Wet blood chemistry method using venous blood samples was used for the analysis of all previously mentioned tests. Hemoglobin and glycated hemoglobin tests were completed using whole blood, while serum was used for other tests.

3.2.4.1 Blood Collection

Blood collection was performed by a registered phlebotomist nurse. All equipment needed for blood collection was prepared and organized at the blood collection station ahead of time, including: gel separator in Sekurplast test tubes with clot activator (5 ml) (VACUTEST KIMA, Arzergrande, Italy); vacutainer system with butterfly needle attachment (Greiner Bio-One, GmbH, Austria); alcohol swabs; cotton swabs; tourniquet; plastic gloves; sharp container and safe-box containing dry ice to transport blood samples into the laboratory for analysis, which was adjacent to the nutrition clinic in our case. Labels with the identification number of each participant were placed on the test tube by the researcher [233].

The World Health Organization guidelines published in 2010 for drawing blood were followed [234]. The nurse identified a good size vein in the antecubital fossa or forearm and used a vacutainer with a butterfly needle to collect blood into the test tube. Two drops (8 μ L) of whole blood from the vacuum tube were utilized for the analysis of hemoglobin and glycated hemoglobin. All used butterfly needles were disposed in the sharp container and all other contaminated supplies were properly disposed according to proper biohazards waste disposal. Test tubes were inverted gently six to seven times and allowed to clot for two hours in ice. Tubes were then centrifuged at 1500 RPM for 15 minutes. Serum was transferred (1 ml * 2 aliquots) into 2 ml Eppendorf safe-lock tubes, properly labeled and immediately stored using labeled Eppendorf containers at - 80°C until the time of analysis. The date of withdrawal, centrifugation and storage were recorded. At the time of analysis, samples were thawed on ice for 30 minutes with proper handling during thawing and storage [235].

3.2.4.2 Analytical System

An in vitro diagnostic test for the quantitative determination of total cholesterol, triglyceride, LDL-cholesterol, HDL-cholesterol, high sensitivity C-Reactive protein and fasting plasma glucose concentrations in human serum was performed using an automated biochemical analyzer (Cobas® C111, Roche Diagnostics, Indianapolis, IN, Figure 3.4), which has been evaluated and recommended for such use [236]. All tests were run in triplicate and averaged.

The researcher (Mohamad. M) attended the training held by the Roche Company on the proper operation methods of the Cobas® c111 analyzer and was certified to run the device. The Cobas® C111 reagents, calibrators, and controls were

used according to the manufacturers' recommendations. The reagents needed to perform lipid, glucose and hs-CRP tests were loaded into the reagent disk and calibrated using appropriate calibrators provided by the company. Calibrator f.a.s® was used for the calibration of glucose, triglyceride and total cholesterol. Calibrator f.a.s Lipids® was used for the calibration of LDL and HDL cholesterol, and Calibrator f.a.s.Proteins® was used for the calibration of high sensitivity C-reactive protein. The daily operation included routine tasks that are needed to prepare and monitor the system and to analyze samples. When the system is switched on it performs several checks to make sure that all preconditions are met, and the screen displays the current status of the system. The system asks for daily maintenance orders that include: checking external fluid containers; loading the reagent disk; checking the cuvettes; performing calibration and performing quality control using PreciControl ClinChem Multi 1® and PreciControl ClinChem Multi 2®, as required. Once preparation was done, the samples were identified, required tests were selected and samples were placed.



Figure 3.4: Cobas® c111 Analyzer

3.2.4.3 Lipid Assessment

Cobas® C111 uses the homogeneous enzymatic colorimetric principle for testing a lipid profile. A lipid profile is a group of blood tests used as a screening tool to identify abnormalities in cholesterol, HDL, LDL and triglyceride blood levels. This could help in the diagnosis of a certain disease, such as a genetic disorder, a cardiovascular disease or any other diseases. It is also important for monitoring the efficiency of certain drugs.

Dyslipidemia is one of the risk factors for many diseases like diabetes, ischemic heart disease and hypertension [237-239]. The clinical interpretation of lipid values in this study was according to the Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents (Table: 3.3) set in 2011 by the National Heart, Lung, and Blood Institute (NIH)[240].

Table 3.3: Recommended Cut Points for Lipid and Lipoprotein Level in Young Adults (20-24 years old)

Category	Low, mg/dL	Borderline-Low, mg/dL	Acceptable, mg/dL	Borderline-High, mg/dL	High, mg/dL
Total Cholesterol	-	-	<190	190-224	≥225
LDL-Cholesterol	-	-	<120	120-159	≥160
Non-HDL Cholesterol	-	-	<150	150-189	≥190
Triglycerides	-	-	<115	115-149	≥150
HDL Cholesterol	<40	40-44	>45	-	-

Source: NIH, 2011[240].

3.2.4.4 Triglycerides

The determination of triglycerides is useful for the diagnosis and treatment of many diseases like diabetes mellitus and nephrotic syndrome [241, 242]. Determination of triglycerides using Cobas® C111 follows the principle of enzymatic colorimetric test, as described by Siedel and others in 1993 [243]. The triglyceride reagent was handled as described by the manufacturer. Excessive foaming was removed from the surface of the reagent prior to loading into the analyzer. Human serum was collected and prepared according to Tietz, 1995 [244] as described in section 3.2.4.1. 2 μ L of serum and 120 μ L of the reagent were required for each run of the test. The measuring range of the test was 8.85-885 mg/dL. The expected value for the normal range is <115 mg/dL and the cut-off value used for defining metabolic syndrome was \geq 150 mg/dL [4].

3.2.4.5 Total Cholesterol

Cholesterol analyses are used for screening against atherosclerotic risk and in the diagnosis and treatment of disorders involving elevated cholesterol values, such as heart diseases [245]. Cobas® C111 follows the enzymatic colorimetric method for the analysis of cholesterol as described by Allain and others (1974) [246]. The Roche cholesterol assay meets the National Institute of Health 1992 goals of less than or equal to 3% for both precision and bias [247]. The Cholesterol reagent was handled as recommended by the manufacturer. 2 μ L of serum and 47 μ L of the reagent were required for each run of the test. The measuring range of the test was 9.7-800 mg/dL. The acceptable value for normal range is <190 mg/dL [240].

3.2.4.6 Low Density Lipoprotein

Low Density Lipoproteins (LDL cholesterol) play an important role in causing and influencing the progression of atherosclerosis especially coronary sclerosis [248]. Various methods are available for the determination of LDL cholesterol such as: ultracentrifugation as the reference method; precipitation methods; and lipoprotein electrophoresis. Cobas® C111 follows the homogeneous enzymatic colorimetric method for the analysis of LDL cholesterol as described by Bachorik and Ross, 1995 [249]. This direct LDL cholesterol assay meets the National Cholesterol Education Program 1995 [249] goals of < 4% total Coefficient of Variation, bias < 4% versus reference method, and 12% total analytical error [250]. The LDL cholesterol reagent was handled as recommended by the manufacturer. Human serum was collected, stored and prepared as described in section 3.2.4.1. 2µL of serum and 200µL of the reagent were required for each run of the test. The measuring range of the test was 3.86-548 mg/dL. The acceptable value for normal range is <120 mg/dL [240].

3.2.4.7 High Density Lipoprotein

Monitoring HDL cholesterol in human serum is of clinical importance, as an inverse correlation exists between HDL cholesterol concentrations and the risk of atherosclerotic disease. Elevated HDL cholesterol concentrations are protective against coronary heart disease, whereas lower concentrations of HDL cholesterol coexisting with elevated triglyceride levels, increase the risk for cardiovascular disease [251]. A variety of methods are available to determine HDL cholesterol, including: ultracentrifugation; electrophoresis; precipitation-based methods; high performance liquid chromatography (HPLC); and direct methods. Cobas® C111 follows the homogeneous enzymatic colorimetric method for the direct analysis of HDL

cholesterol as described by Sugiuchi and others in 1995 [252]. This direct HDL cholesterol assay meets the 1998 National Institutes of Health (NIH)/ National Cholesterol Education Program (NCEP) goals for acceptable performance [253]. The HDL cholesterol reagent was handled as recommended by the manufacturer. Human serum was collected, stored and prepared as described in section 3.2.4.1. 2.5 μ L of serum and 200 μ L of the reagent were required for each run of the test. The measuring range of the test was 3-120 mg/dL. The normal reference range of the HDL-Cholesterol for females is >45 mg/dL [240]. The cut-off value used for defining metabolic syndrome for females is HDL cholesterol of <50 mg/dL [4].

3.2.4.8 Fasting Plasma Glucose

The concentration of glucose in blood is controlled within narrow limits by many hormones, the most important of which are produced by the pancreas. An increase in blood glucose concentration is usually caused by a deficiency in the insulin hormone secretion or action, which is referred to as diabetes mellitus [17]. Measuring fasting plasma glucose has been recommended by the ADA for the diagnosis of diabetes and screening of high risk populations [16]. It is also part of the harmonized joint statement (IDF/AHA/NHLBI) diagnostic criteria of MetS [4]. Cobas® C111 follows the enzymatic reference method with hexokinase UV test for the analysis of glucose as described by Tietz, 2006 [254]. The glucose reagent was handled as recommended by the manufacturer. Human serum was collected, stored and prepared as described in section 3.2.4.1. 2 μ L of serum and 180 μ L of the reagent were required for each run of the test. The measuring range of the test was 1.98-720 mg/dL. The cut-off value used for defining metabolic syndrome is FPG ≥ 100 mg/dL [4]. The cut-off

values for fasting plasma glucose are presented in Table 3.4 according to the standards of medical care in diabetes set by the American Diabetes Association, 2016 [255].

Table 3.4: Blood Test Levels for Diagnosis of Diabetes and Pre-diabetes

	FPG mg/dL	OGTT mg/dL	HbA1c %
Normal	≤99	≤139	About 5
Pre-Diabetes	100-125	140-199	5.7–6.4
Diabetes	≥126	≥200	≥6.5

Source: American Diabetes Association, 2016 [255].

3.2.4.9 High sensitivity C-Reactive Protein

High sensitivity C-reactive protein (Hs-CRP) is an acute phase protein in inflammatory reactions. Measurement of CRP is used for the detection of infection, inflammatory disorders and associated diseases. Highly sensitive measurement of CRP may also be used for the assessment of risk for future coronary heart disease [256]. Several studies have suggested the addition of Hs-CRP for metabolic syndrome diagnostic criteria and for the assessment of global cardiovascular risk [69, 71]. A variety of methods are available to determine Hs-CRP, including nephelometry and turbidimetry. The Cobas® C111 Hs-CRP assay is based on the principle of particle-enhanced immunological agglutination, where human CRP agglutinates with latex particles and coated with monoclonal anti-CRP antibodies. The precipitate is then determined turbidimetrically as described by Price and others in 1987 [257]. The Hs-CRP reagent was handled as recommended by the manufacturer. Human serum was collected, stored and prepared as described in section 3.2.4.1. 6µL of serum and 110 µL of the reagent were required for each run of the test. The measuring range of the

test as recommended by the Centers of Disease Control and Prevention (CDC) and the American Heart Association (AHA) [63, 258] for cardiovascular disease (CVD) risk assessment is shown in Table 3.5.

Table 3.5: High Sensitivity CRP Cut-off Points for CVD Risk Assessment

Relative risk for CVD	Hs-CRP Level mg/L
Low	<1.0
Average	1.0–3.0
High	>3.0

Source: Pearson and others, 2003 [63].

3.2.4.10 Hemoglobin and Glycated Hemoglobin

Hemoglobin (Hb) is a protein in the red blood cells responsible for carrying oxygen from the lungs to all body tissues and returning carbon dioxide from tissues into the lungs. Measurement of hemoglobin concentration in whole blood is the most commonly used test for iron deficiency anemia [10]. HemoCue® Hb 201⁺ portable photometer system (HemoCue® Ltd, UK, Figure: 3.5) was used for the assessment of hemoglobin concentration using 4µL of venous whole blood according to the blood collection method described in section 3.2.4.1. The precision and accuracy of hemoglobin values obtained using HemoCue® Hb 201⁺ are comparable to those obtained using the cyanmethemoglobin method [259]. Standardized procedures for measuring hemoglobin concentration using HemoCue® Hb 201⁺ were followed to enhance the accuracy and reliability of the test, as described by Burger and Pierre-Louis, 2003 [260]. The interpretive criteria used for defining anemia as recommended by the World Health Organization [261] is presented in Table 3.6.

Table 3.6: Hemoglobin Levels for the Diagnosis of Anemia at Sea Level

Population	Non-Anemia	Mild Anemia	Moderate Anemia	Severe Anemia
Non-pregnant women (15 years of age and above)	≥ 120 g/L	110-119 g/L	80-109 g/L	< 80 g/L

Source: WHO, 2011 [261].

Glycated Hemoglobin (HbA1c) is a form of hemoglobin that was first identified as an unusual hemoglobin found in patients with diabetes. It has been correlated to glucose measurements and was clinically used for monitoring diabetic patients, as it reflects average blood glucose over the previous eight to twelve weeks [262]. HbA1c is recommended to be used as a diagnostic test for diabetes mellitus by the World Health Organization [263].

The HemoCue® HbA1c 501 system (HemoCue® Ltd, UK, Figure: 3.6) has been used for assessing glycated hemoglobin percentage using 4 μ L of venous whole blood. Blood collection method is described in section 3.2.4.1. The HemoCue® HbA1c 501 system uses a boronate affinity assay to separate the glycated hemoglobin fraction from the nonglycated fraction. The setup, operating procedures and storage instructions for the analyzer, reagent cartilage and check cartilage were performed as described in the operating manual. Quality control of the system was performed on daily and monthly bases using the appropriate check cartilage (HemoCue® HbA1c 501 Daily Check Cartilage and HemoCue® HbA1c 501 Monthly Check Cartilage). Good correlation has been found when the HemoCue® HbA1c 501 system was compared to the KESLAB laboratory method, Variant II (BioRad), using venous blood patient samples [264]. The Cut-off values of HbA1c for the diagnosis of diabetes and

pre-diabetes are presented in Table 3.4, which follows the 2015 recommendations of the American Diabetes Association [16].



Figure 3.5: HemoCue® Hemoglobin 201+ Analyzer



Figure 3.6: HemoCue® HbA1c 501 System

3.2.5 Blood Pressure Measurement

Blood pressure is the pressure that blood applies on the wall of the arteries in the circulatory system. It is measured per unit area on the walls of arteries (mm Hg). The cardiac cycle consists of two phases, the contraction phase causing systolic blood pressure (SBP) and the relaxation phase causing diastolic blood pressure (DBP). Blood pressure is used for the diagnosis and monitoring of hypertension, which is characterized by an increase of systolic or diastolic blood pressure. Hypertension is found to be associate with increased incidence of renal disease and cardiovascular disease (CVD) [265]. Systolic and Diastolic blood pressures were measured using a calibrated digital automated blood pressure monitor (Omron HEM-907-E7 Digital

Blood Pressure Monitor, Omron Healthcare Europe, Mie, Japan, Figure 3.7). Blood pressure was measured using the participants' right upper arm, after removing clothing covering the cuff placement. Appropriate cuff size was determined according to the MUAC measurements of the participant. They were seated on a comfortable chair, asked to uncross their legs with the back of the right arm supported on a table to keep it at the level of the right atrium. They were then instructed to relax and avoid talking during the measurement procedure. The measurement was taken twice with 5 minutes break between measures, and the average measurement was recorded [266].

The Omron HEM-907 device has been validated and evaluated in several studies, having passed the two phases of the International Protocol, and therefore becoming eligible for use in clinical measurement of blood pressure [267, 268]. The use of such an automated device for measuring blood pressure requires no expensive training, reduces observer errors and minimizes the white coat effect [266]. The cut-off values used for the interpretation of blood pressure measures are presented in Table 3.7, as published in the Seventh Report of the Joint National Committee (JNC7) on prevention, detection, evaluation and treatment of high blood pressure in 2003 [269].

Table 3.7: Classification of Blood Pressure

Category	SBP mm Hg		DBP mm Hg
Normal	<120	and	<80
Prehypertension	120-139	or	80-89
Hypertension, Stage 1	140-159	or	90-99
Hypertension, Stage 2	≥160	or	≥100

Source: Chobanian and others, 2003 [269].



Figure 3.7: Omron HEM-907-E7 Digital Blood Pressure Monitor

3.2.6 Power Analysis

A school-based study conducted by Mehairi and others in 2013 reported the prevalence of MetS among adolescents (12-18 years old) according to the IDF criteria to be 22% and 4% among boys and girls respectively [6]. Minitab software (version 16, Minitab Inc., PA) was used to calculate sample size. With a planned proportion estimate of 4% at 95% confidence level, a sample size of 555 would achieve a 1.58% margin of error for the survey of the female student population. With a planned proportion of 50%, generally used when there is no prior information, at 95% confidence level the sample size of 555 would achieve a 4.04% margin of error.

3.2.7 Statistical Analysis

Data analyses were carried out using Stata version 13 (Stata Corp, College Station, TX). Descriptive statistics were computed; continuous variables were summarized by means and standard deviations (SD) and proportions for categorical variables. The Student's *t*-test was used to compare means for continuous variables between participants with and without MetS. Univariable and multivariable logistic regression analysis was used to study the association between anthropometric and chemical measures and the presence or absence of metabolic syndrome as the outcome

variable. To account for perfect prediction of MetS by BMI categories and the small sample size across BMI class, we applied the Firth logistic regression to obtain reasonable and robust estimates. All statistical significance was assessed at the 5% significance level.

3.3 Glycemic Index (GI) Value of Commonly Consumed Foods in the UAE

3.3.1 Study Population

A total of 112 healthy participants from UAEU students and staff were recruited voluntarily to participate in this study. This was done through posters distributed around the campus buildings, e-mail invitations and word of mouth. Participants did not take part if they had any of the exclusion criteria, defined as: age being less than 18 or more than 55 years old; body mass index (BMI) value more than or equal to 25 kg/m² or less than 18.5 kg/m²; fasting plasma glucose (FPG) value of more than 6.1 mmol/l, and having a known history of impaired glucose tolerance or diabetes mellitus. Participants were asked to complete a health-screening questionnaire before taking part in the study to make sure that they met the inclusion criteria (Appendix 6). Participants who met the inclusion criteria were given complete details about the study protocol (Information sheet, Appendix 7) and the chance to ask questions. They were asked to fast for 12 hours the night before the test, while water consumption was allowed in moderation. The day before the GI test, they were requested to limit their intake of caffeine-containing drinks and to minimize their participation in any intense physical activity, such as: long periods of swimming or running, gym workout, lifting weights and aerobics.

3.3.2 Ethical Approval

Ethical approval for the study was obtained from the United Arab Emirates University Scientific Research Ethics Committee (UAEU, Reference Number (516/09) (Appendix 8)). All participants provided written informed consent before taking part. An example of the consent form used in this study is attached in Appendix 9.

3.3.3 Anthropometric Measurements

Anthropometric measurements were carried once for each participant, prior to recruitment, to confirm the participant fit the inclusion criteria. All measurements were completed in the fasting state from 7-10 a.m., while wearing minimal clothing (as local culture permits) and no shoes, after resting for 15 minutes to allow climate adjustments and relaxation. All devices used for measurements were calibrated on a daily basis. Results were recorded immediately after measurement on a data sheet (Appendix 7) and were checked by another skilled researcher. Each measurement was taken three times and averaged.

Height, weight, body composition, waist circumference, and hip circumference were measured as described earlier sections 3.2.2.1, 3.2.3.2, 3.2.3.4, and 3.2.3.5. Body mass index and waist-hip ratio were calculated using equations described in sections 3.2.3.3 and 3.2.3.6, respectively.

3.3.4 Blood Glucose

Blood samples were obtained using the OneTouch® UltraSoft™ Adjustable Blood Sampler (Johnson and Johnson, Middle East, Inc) which uses the OneTouch® UltraSoft™ pen and the OneTouch® FinePoint™ lancets with a thin tip for less painful penetration. Capillary blood was obtained from the third finger on the left hand. Several reports suggested the use of capillary blood rather than venous blood sampling for reliable glycemic index (GI) testing [194, 270]. Before the finger-pricking process, all the equipment and supplies that were needed were assembled and hand hygiene was performed. Participants were asked to warm their hand to increase blood flow. The WHO 2010 guidelines for withdrawing blood were followed [234]. The site of the finger-pricking was selected, and 70% isopropyl alcohol was applied and allowed to air dry. Then, the skin was punctured and the first drop of blood was wiped away using dry tissue. Squeezing the finger was avoided to minimize plasma dilution. A 5 μ L blood sample was collected in the microcuvette by capillary action. All sharps, and waste materials were disposed of appropriately. Blood glucose was measured using the HemoCue Glucose 201⁺ portable system (HemoCue® Ltd, UK, Figure 3.8). This machine uses the principle of modified glucose dehydrogenase in which the total amount of glucose is measured at the end point photometrically. It is factory calibrated and traceable to the ID GC-MS method; therefore, it needs no further calibration. It has been previously used in a number of investigations [195, 271].



Figure 3.8: HemoCue® Glucose 201+ Analyzer

3.3.5 Test Foods

Twenty-three different foods commonly consumed in the United Arab Emirates were tested, including breads (n=6), entrée dishes (n=2), main dishes (n=7) and sweet dishes (n=8) as shown in Table 3.8. The test foods were purchased from three popular restaurants in the UAE that specialize in Emirati cuisine and have standardized recipes (see Table 3.8 for major ingredients). These restaurants were selected based on a questionnaire conducted on 315 participants, to investigate the most popular Emirati cuisine restaurants in the area. Three samples from each dish were obtained at three different occasions for the chemical analyses, and each food sample was analyzed in triplicate. The average is reported as the mean \pm SD for all chemical tests.

Table 3.8: Main ingredients used in the preparation of 23 traditional foods commonly consumed in the UAE

Test Food	Description	Major ingredients
Breads		
Arabic bread	Baked bread	Wheat flour, salt, yeast and water
Regag bread	Thin crispy crepe	Wheat flour, salt and water
Chebab bread	Emirati pancake	Wheat flour, egg, yeast, salt, sugar, milk powder, saffron and water
Muhalla bread	Emirati crepe	Wheat flour, egg, sugar, milk powder, saffron, cardamom and water.
Khameer bread	Baked bread	Wheat flour, egg ,yeast, salt, sugar, milk powder, saffron and water
Gurus	Fried bread	Wheat flour, vegetable oil, egg, sugar, salt and water
Entrée Dishes		
Fendal	Boiled sweet potato	Sweet potato and water
Chami	Cottage cheese	Buttermilk, salt and white cumin seeds
Main dishes		
Harees, beef	Crushed wheat with meat	Crushed wheat, meat, water, ghee, salt and cardamom
Thareed, beef	Bread with meat stew	Wheat flour, meat, potatoes, onions, tomatoes, zucchini, tomato paste, vegetable oil, spices and water
Thareed, chicken	Bread with chicken stew	Wheat flour, chicken, potatoes, onions, tomatoes, zucchini, tomato paste, vegetable oil, spices and water
Biryani, chicken	Rice with chicken	Rice, salt, Ghee, spices, chicken, garlic and onion
Machbous, fish	Rice with fish	Rice, tomato, onion, water, salt, spices and fish
Arseyah	Rice with chicken	Rice, salt, chicken, water, cardamom and cinnamon
Marqoqa	Bread with chicken stew	Wheat flour, chicken, potatoes, onions, tomatoes, carrots, tomato paste, vegetable oil, garlic, spices and water
Dessert Dishes		
Khabisa	Cardamom pudding	Wheat flour, Water, Ghee, sugar and cardamom
Leqemat	Doughnut cake	White flour, vegetable oil, salt, sugar, egg and yeast
Batheetha	Date paste	Date fruit, wheat flour, Ghee, sugar, cardamom and cinnamon
Kanfarooosh	Doughnut cake	Wheat flour, yeast, sugar, egg, vegetable oil, saffron, cardamom and backing powder
Sago	Sago seed with sugar	Sago seeds, sugar, saffron and water
Asida	Flour with sugar	Wheat flour, sugar, salt and water
Habba Hamra	Red seed drink	Evaporated milk, red seed, cardamom, saffron, black pepper and sugar
Balalet	Sweet vermicelli	Vermicelli, water, sugar, ghee and cardamom

3.3.5.1 Chemical Analyses

Food composition databases offer comprehensive information on the concentrations of nutrients in foods, this information is considered the base of any quantitative study of nutrition. It is also useful in clinical practice, food manufacturing, designing health promotions, regulation of nutrition and health claims, epidemiological studies and policy decision making [174]. Awareness about the importance of establishing food composition tables in the UAE and other Gulf Cooperation Council (GCC) countries has increased during the last twenty to thirty years. In Saudi Arabia, twenty Saudi dishes were analyzed for their cholesterol and fatty acid contents [272], and six dishes were investigated by Al-Jebrin and others in 1985 for chemical composition and nutritional quality [170]. In Qatar, seventeen traditional Qatari dishes were chemically analyzed in the year 1994 [273]. In a 1998 study, conducted by Musaiger and others, where they studied the physical, proximate and mineral composition of four types of fermented dairy products in Bahrain [171]. In Oman, the proximate, mineral, fatty acid and cholesterol compositions of twenty dishes were analyzed [172]. In Kuwait, thirty-two Kuwaiti composite dishes were analyzed for their proximate composition and phytate content [274]. Recently, ten traditional dishes commonly consumed in the United Arab Emirates (UAE) were chemically analyzed for proximate composition and mineral content [175]. Although nutritional-composition data of traditional dishes in the U.A.E. and other Arab Gulf countries exist, it does not cover all test foods selected in this study. Also, available carbohydrate content should be known to perform glycemic index testing for any food. It was not calculated in other studies, and one could not even calculate it by subtracting fiber content from total carbohydrates content, because fiber content was not even reported [275].

Proximate composition, mineral content, vitamin content, lipids and sugars analyses for test foods were carried out in the Nutrition and Health Department laboratory at UAEU. The standard procedures of the Association of Official Analytical Chemists (AOAC) 2003 were followed [276]. Mineral content was determined by Inductively Coupled Plasma Optical Emission Spectroscopy ICP-OES [277]. Sugar, vitamin, caffeine, and cholesterol analysis were conducted using the HPLC technique. Total carbohydrate and available carbohydrate content were estimated by difference [274]. The energy content was calculated by multiplying the protein, carbohydrate, and fat by factors of 4, 4, and 9 respectively [273]. Each test was done in triplicate and averaged to minimize possible errors, and to increase reliability and accuracy.

3.3.5.2 Sample Preparation

Each test food was purchased and analyzed on three different occasions (beginning, middle and end of the month) to ensure consistency of restaurants in preparing and standardizing the recipe. Upon purchase, each food was transported into the laboratory in a cool-box. Extraneous matter such as bones from chicken and meat, was removed. The main components of each meal were then individually weighed. Test foods were thoroughly homogenized using a mechanical high-speed blender and then sampled for moisture analysis. The residual homogenized samples were dried in the oven at 65°C for 16 hours, ground into fine powder and stored in air-tight containers at -80°C for further analysis.

3.3.5.3 Proximate Analysis

a) Determination of Moisture

Moisture analysis follows the simple principle of evaporation of water from the sample through oven drying. Dry matter is then determined as the residual after drying [278]. Aluminum dishes were dried at 105°C for two hours and then allowed to cool down to room temperature in the desiccator. Dishes were then weighed using Scaltec® SBA 31 analytical electronic balance sensitive to 0.01 mg (Scaltec® Instruments, Heiligenstadt, Germany, Figure 3.9). Approximately 1 gram of sample food was weighed and spread uniformly across the aluminum dish. Mommert® forced-air drying oven (Schutzart DIN 400-50-IP20, Figure: 2.10) was used to dry the samples for 16 hours at 105°C ± 3°C. Samples were placed uniformly in the oven to allow air circulation, then moved to the desiccator to cool down to room temperature and weighed to the nearest 0.01 mg. The percentage of total dry matter and total moisture were calculated using the following equations [276]:

$$\% \text{ Total Dry matter} = \frac{(\text{weight of dry sample and dish in grams} - \text{weight of dish in grams})}{\text{Initial weight of sample in grams}} \times 100$$

$$\% \text{ Total Moisture} = 100 - \% \text{ Total DM}$$

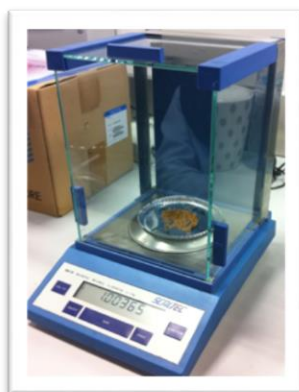


Figure 3.9: Scaltec® SBA 31 Analytical Electronic Balance



Figure 3.10: Mommert® Forced-Air Drying Oven

b) Determination of Ash Content

Crucibles were dried in the drying oven for at least two hours at 105 °C, and moved into a desiccator using tongs. They were allowed to cool down to room temperature, weighed, and recorded to the nearest 0.01 mg. Approximately one gram of the sample was weighed and ashed in a muffle furnace oven (Carbolite EML 11/6, Figure 3.11) at 500 °C for four hours. Crucibles were then allowed to cool in the furnace to less than 200°C, and then weighed with the ash to the nearest 0.01 mg. The percentage of ash was calculated using the following equation [279]:

$$\% \text{ Ash} = \frac{(\text{Weight of crucible and ash in grams} - \text{Weight of crucible in grams})}{\text{Weight of sample in grams}} \times 100$$



Figure 3.11: Muffle Furnace Oven and Crucibles

c) Determination of Protein Content

The Kjeldahl method was used for nitrogen determination in food samples and a factor of 6.25 was adopted for protein-content estimation [280]. The first step in the Kjeldahl method is the digestion of samples. Around 0.5 gram of sample was weighed into the digestion tube and was digested by sulfuric acid (96%) and a catalyst (Kjeldahl catalyst selenium tablets) using Foss Tecator 2020 digester (Foss Tecator, Höganäs, Sweden, Figure 3.12) at 410°C for about 45 minutes. This process resulted in the conversion of nitrogen to ammonia. A Foss 2300 Kjeltec Analyzer Unit (Foss Technologies Co., Ltd., Höganäs, Sweden, Figures 3.13) was then used to determine ammonia and protein content [276].



Figure 3.12: Foss 2300 Kjeltec Analyzer Unit



Figure 3.13: Foss Tecator 2020 Digester

d) Determination of Fat Content

The fat content was determined by Soxhlet extraction according to the AOAC and Horwitz, 2003 [276]. The extraction cups were dried at 105°C, for 2 hours then weighed to the nearest 0.1 mg. 2 grams of sample were placed in a 33 mm × 80 mm extraction thimble (supplied by the manufacture), and extracted with 50 ml n-hexane/acetone (1:1, v/v) in boiling solvent for 60 minutes using the Soxhlet extraction in a Sotex system 2050 (Foss, Hillerød, Denmark, Figure: 2.14). Thimbles were then raised to the rinse position for another 60 minutes to allow evaporation of as much solvent as possible. The extraction cups were removed from the extractor and placed in an operating fume hood to finish evaporating the solvent at low temperature, then dried at 105°C for 30 minutes, allowed to cool down in the desiccator and weighed to the nearest 0.1 mg [281]. The percentage of fat content was then calculated using the following equation [276]:

$$\% \text{ Crude Fat} = \frac{(\text{weight of cup} + \text{fat residue in grams}) - (\text{weight of empty cup in grams})}{\text{Initial sample weight in grams}} \times 100$$



Figure 3.14: Sotex System 2050

e) Determination of Fiber Content

The Neutral Detergent Fiber (NDF) method was used to determine fiber content in food samples, which measures fiber residue (hemicellulose, cellulose, and lignin) remaining after digestion in a detergent solution [282]. NDF analyses was performed using ANKOM 200 fiber analyzer (ANKOM²⁰⁰, 65 rpm agitation; ANKOM Technology, Macedon, New York, USA, Figure: 3.15). The ANKOM Technology NDF method was followed [283]. The ANKOM filter bags (F57 and F58, ANKOM Technology) were weighed. Subsequently 0.50 grams of sample was placed in each bag, weighed, sealed using heat sealer (1915, ANKOM Technology) and marked using a solvent and acid-resistant marking pen (F08, ANKOM Technology). A bag was left empty and used as a blank. Afterwards, the bags were soaked in acetone for 10 minutes, and placed on a wire screen to dry. Samples inside the bags were spread uniformly within them by shaking and flicking the bags to eliminate clumping. The bags were then placed in the ANKOM 200 fiber analyzer (Figure 3.15) with Neutral Detergent Solution, alpha-amylase, and sodium sulfate. When the NDF extraction was over, the bags were rinsed, soaked in acetone again, and allowed to dry. Then they were placed in an oven at 105°C for 3 hours, allowed to cool in the desiccator, and then weighed. The percentage of Neutral Detergent Fiber content was then calculated according to the following formula [283]:

$$\% NDF = \frac{(\text{Dried weight of bag with fiber process} - (\text{Bag tare weight} \times \text{Blank bag correction}))}{\text{Sample Weight}} \times 100$$



Figure 3.15: ANKOM 200 Fiber Analyzer

The Total Dietary Fiber (TDF) was also analyzed in this study. According to the FAO/WHO in 1998[188], it is defined as the edible part of plant and animal material that is not hydrolyzed by the digestive enzymes in humans. It was determined using the AOAC 991.43 method[276]. TDF analyses was performed using the ANKOM^{TDF} dietary fiber analyzer (ANKOM Technology, Macedon, New York USA, Figure 3.16). The ANKOM filter bags (DF-S, DF-FT, ANKOM Technology) were labeled, and weighed using the bag weighing holder (TDF52, ANKOM Technology). The chemicals and the enzymes containers were filled before each use of the analyzer as recommended by the manufacturer. After completing the automated processes, the SDF bags were rinsed with acetone twice. Once the acetone has evaporated, the bags were sealed with a heat sealer (1915, ANKOM Technology). They were then placed in the oven to dry for 90 minutes at 105°C. Afterwards, their weight was recorded. One bag of each sample was then sent for the determination of the protein content and the other one is used for the determination of the ash content. The Percentage of Total Dietary Fiber Value was then calculated using the dietary fiber data spreadsheets available on the ANKOM Technology website [284].

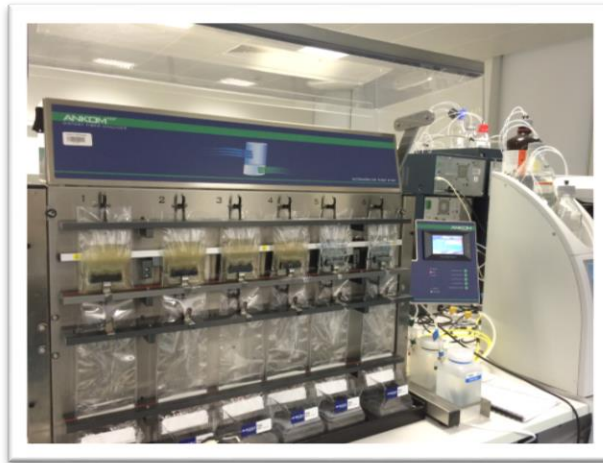


Figure 3.16: ANKOMTDF Dietary Fiber Analyzer

3.3.5.4 Exchange List Development

Exchange list was first used as a method of meal planning for patients with diabetes and for those on weight loss regimens [285]. The food exchange list helps people to monitor food portion sizes, and energy intake. Basically it translates scientific nutrition knowledge into a practical tool. Foods from the same list can be used interchangeably without changing estimated amounts of carbohydrates, fat, protein, and total energy obtainable in a meal [286].

The rounding-off method described by wheeler and colleagues [285] was used to fit food items into exchanges as follows:

For carbohydrate exchange: if a food portion had 1g to 5g of carbohydrates, it was not counted as a serving. If it had 6g to 10g of carbohydrates, it was counted as a half serving. When food had 11g to 20g of carbohydrates, it was counted as one serving.

For fat exchange: if a food portion had 0g to 2g of fat, it was not counted as a serving. Yet, if it had 3 g of fat, it was counted as a half serving. Food portion was counted as one serving if it had 4g to 7g of fat.

For protein exchange: if a food portion had 0g to 3g of protein from the meat and meat substitutes list, it was not counted as a serving. However, if it had 4g to 10g of protein, it was counted as one serving.

3.3.5.5 Determination of Minerals

Major minerals and trace metals (Ca, K, Na, Mg, P, S, Co, Cu, Fe, Mn and Zn) were simultaneously determined in foods by Inductively Coupled Plasma Optical Emission Spectrometer (ICP-OES) on a Varian ICP-OES model 710-ES (Varian, Palo Alto, CA, USA). Simultaneous axially viewed plasma with full computer control of instrument settings and compatible accessories was used for the analysis as outlined in the manufacturers' manual. Food samples were prepared for mineral determination by CEM Mars 5 microwave digestion system (Mars 5, CEM Corporation, Matthews, USA, Figure 3.17). The process of preparing the samples was performed based upon the recommendation in US EPA method 3015A guidelines [287] as described by Heckman in 1971 [288].

Homogenized food samples were weighed into portions of 0.5 grams and placed into the microwave digestion vessels; then 10 ml of concentrated nitric acid and 2 ml hydrochloric acid were added into the sample. The vessels were capped and placed in the microwave digestion system [289]. After cooling, de-ionized water was added, and the sample solution was aspirated through a nebulizer. The resulting aerosol was transported to the plasma torch where excitation happened. Element-specific

emission spectra were created by radio-frequency inductively coupled plasma, which was dispersed by a grating spectrometer; concentrations of the line spectra were observed at specific wavelengths by a charged coupled detector. A fitted background correction was used to offset the blank signal and the matrix effect.



Figure 3.17: Varian ICP-Optical Emission Spectrometer model 710-ES

3.3.5.6 Determination of Sugars

Various types of sugars including monosaccharides (glucose, and fructose), disaccharides (sucrose, maltose, and lactose) and trisaccharide (raffinose) were determined in foods by the High-Performance Liquid Chromatography (HPLC) method.

Determination of sugars was performed using a Waters HPLC system (Waters, Milford, MA, USA, Figure 3.18). The system is composed of a Waters 717 Plus Autosampler, a Waters 1525 Binary HPLC pump, and a Waters 2414 Refractive Index Detector, operated with the Breeze software. Sugars were simultaneously analyzed onto a μ Bondapak® NH₂ 10 μ m 125Å column, 3.9 mm inner diameter by 300 mm (Waters Associates, Milford, MA, USA). The temperature was kept at 35 °C. The

mobile phase consisted of acetonitrile and water (83:17, v/v), and the flow rate was 1.5 mL/minute. Sample preparation and analysis procedures were adopted with modification from Yuan and others (1999) [290] and from Smith and others (1986) [291].



Figure 3.18: Waters High-Performance Liquid Chromatography (HPLC) System

3.3.5.7 Determination of Lipids

The fatty acid composition of foods has recently become mandatory to be listed on food labels, which helps the consumer make healthier choices. However, the fatty acid composition is a complex combination of saturated, monounsaturated, and polyunsaturated fatty acids with a diversity of carbon chain lengths.

The identification of key fatty acids requires several standards and capillary columns. The fatty acid composition analysis was performed using a Young Lin 6500 gas chromatograph (YL-6500 GC, Gyeonggi-do, South Korea, Figure 3.19), fitted with a SP-2380 Fused Silica Capillary Column (30 m × 0.25 mm I.D × 0.20 μm film (2-4110), Sigma Aldrich, St. Louis, MO).

The analytical column was heated at 50°C for 2 minutes, afterwards it was raised to 250°C at 4°C/min, and then held for 15 min. The carrier gas used for the analysis was helium, (20 cm/second) at 150°C. The Supelco 37 Component FAME

Mix standard was used for the identification of key fatty acids in the tested food samples. The Fatty Acid Methyl Esters (FAMES) were prepared following AOAC Method 969.33 [276]. The analysis procedure followed was as described in the application notes of the manufacture (Sigma Aldrich) [292].

The determination of the cholesterol content in food is also very important. However, the possible relationship between dietary cholesterol and atherosclerosis is still debatable and lacks homogenous scientific evidence [293]. The determination of cholesterol was performed using a Waters HPLC system (Waters, Milford, MA, USA, Figure 3.18). The system is composed of a Waters 717 Plus Autosampler, a Waters 1525 Binary HPLC pump, and a Waters 2487 Dual λ Absorbance Detector, operated with the Breeze software. Cholesterol was simultaneously analyzed onto a reversed phase XTerra C18 column, 4.6 mm inner diameter by 150 mm, 5 μm (Waters Associates, Milford, MA, USA). The oven temperature was kept at 25 $^{\circ}\text{C}$. The mobile phase consisted of methanol and 2-propanol (70:30, v/v), and the flow rate was 1.0 mL/minute. Cholesterol components were detected by the UV detector that was set at the wavelength of 212 nm. Sample preparation and analysis procedures were adopted from Indyk (1990) and Essaka (2007) with modification [294, 295].



Figure 3.19: Young Lin 6500 Gas Chromatography System

3.3.5.8 Determination of Vitamins

Vitamins are a wide-ranging group of organic compounds that are essential for the functioning of human bodies. They are classified in two main groups: water-soluble and fat-soluble vitamins. The determination of vitamins in the food we consume is important when it comes to adopting good eating habits in humans, understanding possible loss in vitamins through food storage and preparation, and developing food labels.

Seven water-soluble and all fat-soluble vitamins (Vitamin C, Thiamin, Riboflavin, Niacin, Vitamin B-6, Folate, Vitamin B-12, Vitamin A, Vitamin E (alpha-tocopherol), Vitamin D (D2+D3), and Vitamin K (phylloquinone)) were simultaneously determined in foods by the High-Performance Liquid Chromatography (HPLC) method.

The determination of water-soluble vitamins was performed using a Waters HPLC system (Waters, Milford, MA, USA, Figure 3.18) [296]. The system is composed of a Waters 717 Plus Autosampler, a Waters 1525 Binary HPLC pump and a Waters 2487 Dual λ Absorbance Detector, operated with the Breeze software. Water-soluble vitamins were simultaneously analyzed onto a reversed-phase XTeera C18 column (4.6 mm inner diameter by 150 mm, 5 μ m) from Waters (Waters Associates, Milford, MA, USA). For water-soluble vitamins, the mobile phase consisted of 50 mM K_2HPO_4 (pH7): methanol, gradient: 1% methanol for 5 minutes, 1-30 % methanol (linear gradient) over 15 minutes, 30 % methanol for 5 minutes and the flow rate was 1.0 mL/minute. The column temperature was kept at 35 °C. The injection volume was 10 μ l. The analytical column effluents were monitored at $\lambda=220$ nm for all the water-

soluble vitamins. Supelco Application Note 148, was used as a reference for sample preparation and analysis procedures [297].

The determination of fat-soluble vitamins was performed using Waters ACQUITY UPLC system (Waters, Milford, MA, USA, Figure 3.20). The system is composed of a Waters ACQUITY sample manager, a Waters ACQUITY Binary solvent manager, and a Waters ACQUITY PDA e λ Detector, operated on an Empower software. Fat-soluble vitamins were simultaneously analyzed onto a reversed-phase ACQUITY BEH C18 column (2.1 mm inner diameter by 100 mm, 1.7 μ m) from Waters (Waters Associates, Milford, MA, USA). For fat-soluble vitamins, the mobile phase consisted of Solvent A: Water: Acetonitrile (90:10), and solvent B: methanol and Acetonitrile (50:50, v/v), and the flow rate was 0.7 mL/minute. The temperature was kept at 35 °C. The injection volume was 5 μ l. The analytical column effluents were monitored at λ =285nm for vitamin E, at λ =265nm for vitamin K1, K2, D2, D3 and at λ = 325 for vitamin A acetate. Sample preparation and analysis procedures were adopted from Moreno and Salvado (2000) with modification [298].



Figure 3.20: Waters ACQUITY UPLC System

3.3.5.9 Determination of Caffeine

Caffeine content should be listed on the food label according to food labelling legislations. Although none of the foods investigated in this study have caffeine-containing ingredients; caffeine analysis was still required to examine the possible addition of caffeine during preparation.

The determination of caffeine was performed using a Waters HPLC system (Waters, Milford, MA, USA, Figure 3.18). The system is composed of a Waters 717 Plus Auto-sampler, a Waters 1525 Binary HPLC pump, and a Waters 2487 Dual λ Absorbance Detector, operated with the Breeze software. Caffeine was simultaneously analyzed onto a reversed-phase XTeera C18 column (4.6 mm inner diameter by 150 mm, 5 μ m) from Waters (Waters Associates, Milford, MA, USA). The mobile phase consisted of acetonitrile and water (10:90, v/v), and the flow rate was 1.0 mL/minute. Caffeine was detected by the UV detector that was set at the wavelength of 265 nm. Samples preparation procedures were adopted with modification from Srdjenovic and others (2008) [299]. Whereas the analysis procedures were adopted from Erickson (2011) with modification [300].

3.3.6 Glycemic Index (GI) Procedure

The GI measurement procedure followed was adapted from Wolever et al. (1991) [193] and Brouns et al. (2005) [194]. This same protocol is also recommended by the FAO/WHO (1998) [188]. Testing was repeated in at least fifteen participants for each test food. Prior to the test day, participants were requested to limit their intake of caffeinated drinks and avoid involvement in intense exercise, such as: long periods of swimming or running, gym workouts, lifting weights and aerobics. Participants

were asked to fast for 12 hours (overnight) the night before each test, though drinking water was allowed in moderation.

Using the randomised crossover design, participants tested the reference food three times and each test food for one time only. Food testing was carried out on separate occasions with at least one-day gap between measurements to minimize any carry-over effects. The reference food provided was glucose powder (glucose dextrose monohydrate) dissolved in 200ml of water. Test foods were tested in equivalent available carbohydrate amounts (25 or 50 g) as per the reference food and were also served with 200 ml water. Test foods were purchased one day before the test, then heated in the morning of the test. Participants were encouraged to consume the reference or test foods within 15 minutes and to minimise physical activity during the testing time. Available carbohydrate content was used to determine the experimental portion (g) that would provide 50g or 25g of available carbohydrates from each test food. The amount of available carbohydrate was calculated by subtracting dietary fiber content from total carbohydrate content [227]. The majority of test foods were tested against 50 grams of available carbohydrate. Nevertheless, if the serving size was found too large to ingest comfortably, this test food was tested against 25 grams of available carbohydrate [194]. In this study, only Chami (Cottage cheese) was tested against 25 grams of available carbohydrate due to its very low carbohydrates content (5.44g/100g). The experimental portion size of each test food, shown in Table 3.9, could vary according to the quantity of carbohydrate available in that food.

Table 3.9: Experimental portion size of test foods

Food	Available Carbohydrate in test Food (g/100g) *	Reference Available Carbohydrates (g)	Experimental portion (g)
Arabic bread	63.47 ± 0.32	50	79
Regag bread	44.37 ± 0.49	50	113
Chebab bread	45.88 ± 1.17	50	109
Muhalla bread	67.66 ± 4.79	50	74
Khameer bread	54.93 ± 3.47	50	91
Gurus	54.45 ± 2.60	50	92
Fendal	31.64 ± 0.24	50	158
Chami	5.31 ± 0.58	25	471
Harees (beef)	7.74 ± 1.15	50	323
Thareed (beef)	10.87 ± 0.24	50	460
Thareed (chicken)	12.73 ± 2.65	50	393
Biryani (chicken)	19.69 ± 2.05	50	254
Machbous (fish)	18.00 ± 0.82	50	278
Arseyah	9.85 ± 0.37	50	508
Marqoqa	16.00 ± 0.52	50	313
Khabisah	56.13 ± 4.65	50	89
Leqemat	44.19 ± 1.35	50	113
Batheetha	38.24 ± 0.97	50	131
Kanfarroosh	39.62 ± 0.92	50	126
Saqo	23.43 ± 3.54	50	213
Assidah	21.09 ± 1.14	50	237
Habba Hamra	15.96 ± 1.20	50	313
Balalet	27.89 ± 2.19	50	179

*Data are expressed as Mean ± SD

As recommended by the FAO/WHO in 1998 [270], each participant was tested for the reference food twice and once for each test food. Tests were done in random order and on separate days, with a minimum of a one-day gap between measurements, to lessen the carry-over effect. On the day of the test, participants were asked to report to the clinic in the morning, having fasted for 12 hours. They were served a test food or the reference food and were asked to consume it within 15 minutes. All the test and standard foods were served with 200 mL water.

Capillary blood samples were obtained by finger-prick as described earlier in section 3.3.4. Blood glucose was measured using the HemoCue Glucose 201+ portable system (HemoCue® Ltd, UK, Figure 3.8). A fasting blood sample was obtained at 0 minutes and the reference or a test food was consumed directly afterwards. Additional blood samples were obtained at 15, 30, 45, 60, 90 and 120 minutes after the participant had begun to eat. During the time of the measurements, participants were asked to stay in the testing room and reduce physical activity to the minimum [270].

3.3.6.1 Glycemic Index Calculation

Blood glucose response is usually expressed as the area under the curve (AUC). The total AUC includes the area underneath the curve down to a blood glucose measure of zero, and it is a measure of the average blood glucose concentration during the testing period; However, the incremental AUC is a measure of the change of blood glucose from the fasting plasma glucose measurement; therefore, the GI calculation is based on the incremental area under the blood glucose response curve (IAUC), and above the fasting level only. Thus if blood glucose level falls below the baseline, the area beneath it was ignored. Accordingly, the IAUC cannot be less than zero [270]. The IAUC for each test food consumed by each subject was expressed as a percentage of the mean IAUC for the reference food consumed by the same subject, as follows:

$$GI = \frac{IAUC \text{ for the test food containing } (X)g \text{ of available CHO}}{IAUC \text{ of a reference food with an equal CHO portion}} \times 100$$

The GI of each test food was calculated as the mean for the whole group.

3.3.6.2 Glycemic Load Calculation

The glycemic load is a measure of the overall glycemic impact of the meal. Studies have shown that diets with a high glycemic load increase risk of type 2 diabetes mellitus [19, 21]. Epidemiologic studies also suggest that a high dietary GL increases the risk of coronary heart diseases (CHD) in manner independent of known CHD risk factors [108]. Glycemic load was calculated by the following formula [301]:

$$GL = \frac{GI \text{ of test food} \times \text{amount of available CHO in a serving of test food}(g)}{100}$$

Serving size of test foods was not available from manufacturer, therefore it was adopted [195] from the Photographic Atlas of Food Portions for the Emirate of Abu Dhabi [302]. The amount that provides the best fit in the exchange system was then chosen to be a serving [286].

3.3.7 Power Analysis

The number of participants enrolled in the study will determine the width of the confidence interval (CI) and the power of the study to detect differences in glycemic index. It has been suggested that inclusion of 10 participants provides a reasonable degree of power and precision for most purposes of measuring GI [194]. In order to increase the power of the study to detect small differences in GI as well as to increase precision, a minimum of 15 participants were enrolled for each test food.

A recent study by Al Dhaheri and colleagues [303] reported a GI difference of 1.4 for glucose response over time, and 1.6 for glucose response between the foods tested. A difference of 1.4 was also reported by Wolever in 2003 [301]. A power analysis was performed using Minitab software for windows version 16 (Minitab Inc.,

PA). The power of the study was calculated to estimate the number of participants needed to detect 1.4 unit differences in GI with an alpha of 0.05 using a paired t-test. A sample size of 15 participants for each test food was considered sufficient to detect difference in GI with 88% power.

3.3.8 Statistical Analysis

Data entry and analysis were carried out using Statistical Package for Social Sciences (SPSS) for windows, version 21.0 (SPSS Inc., Chicago, Ill., USA). Data was analyzed using Kruskal-Wallis to compare medians of measurements of nutrients for the various foods, because these measurements did not satisfy the normality assumption of ANOVA. The Paired t-test was used for the comparison of the mean glucose responses of the reference food with each one of the test foods. The Kruskal-Wallis test was used to find the significant differences between the IAUC of the 23 meals and Mann-Whitney test was used to follow these differences. Statistical significance was set at P-value of < 0.05 . Values of different parameters were expressed as the mean \pm standard deviation.

Chapter 4: Prevalence of Metabolic Syndrome among Young Female Emirati Adults at United Arab Emirates University

4.1 Introduction

Non-communicable diseases (NCDs) are the leading cause of deaths worldwide, and diabetes mellitus (DM) is the fourth major cause of NCD deaths [1]. DM is a complex, chronic illness that occurs either when pancreatic cells do not produce enough insulin or when the body is resistance to the insulin it produces [255]. The International Diabetes Federation (IDF) estimated the global prevalence for DM to be 8.3% in 2014. However; 46% of people with diabetes remained undiagnosed. Moreover, the IDF expects 205 million new cases of DM by the year 2035 [304]. With a DM prevalence of 19%, the United Arab Emirates (UAE) was ranked as having the fifth highest prevalence in the Middle East and North Africa (MENA) region, coming after other Gulf Cooperation Council (GCC) countries, like Saudi Arabia, Kuwait, Bahrain and Qatar [304]. In 2010, a survey conducted in the UAE reported the prevalence of undiagnosed DM and pre-diabetes (prediabetes refers to individuals with impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT) [255]) to be 14.6% and 31% respectively [3]. This indicates the great number of people living with undiagnosed DM and pre-diabetes in the country.

The diagnosis of Metabolic Syndrome (MetS) is based on the existence of pre-diabetes combined with dyslipidemia (elevated levels of total or low-density lipoprotein (LDL) cholesterol, or low high-density lipoprotein (HDL) cholesterol levels), elevated blood pressure, and obesity [33]. A study conducted on Emirati adults (+20 years old) by Malik and others in 2008, reported the prevalence of MetS to be 32.9% among men, 45.9% among women, and the overall prevalence of MetS was 40.5%, based on the IDF definition of MetS [5]. This alarming prevalence is thought

to be emerging from the high incidence of MetS among younger age groups (adolescents 12-18 years old) as suggested by Mehairi and others in 2013 [6]. The latter study reported that 13% of adolescents suffered from MetS, and this prevalence showed a positive correlation with higher body mass index (BMI) values, as it reached 59% in obese boys. This dramatic increase in the incidence of MetS including pre-diabetes, emphasizes the need to apply the current screening recommendations by the American Diabetes Association (ADA) for type 2 diabetes; hence, this endorses the case for testing overweight children and adolescents to detect pre-diabetes [16].

The UAE is located in the Arabian Gulf region, and has a population with a median age of 30 years. Only 1% of the population is aged over 60 years, and about 15% of the population is aged under 15 years of old [305]. The country has undergone numerous social and economic changes since oil was discovered forty years ago. The lifestyle of the Emirati population has changed considerably over the past 40 years due to the rapid improvement in socioeconomic status. This transition has led to less physical activity and altered eating habits. These changes, in addition to the adoption of a western lifestyle and diet, have led to the rise in the prevalence of overweight and obesity in the UAE, particularly among females [167].

The United Arab Emirates University (UAEU) was founded in 1976, and it is the first and oldest comprehensive national university in the UAE. It is located in Al Ain city in the Abu Dhabi Emirate (capital of the UAE). The University admits UAE nationals from all seven Emirates and is currently enrolling around 14,000 students.

The prevalence of MetS among Emirati females has been reported to be higher than that for Emirati males in the adult population (32.9% among men, 45.9% among women); however, in adolescents the prevalence among females was found to be much

lower than it is for males (21% among boys, and 4% among girls) [5, 6]. Several studies from the UAE have reported the sedentary lifestyle of adolescent females [135, 306]. This highlights the importance of investigating MetS among young female adults, in order to facilitate the understanding of its prevalence and risk factors.

High prevalence of MetS is a significant contributor to the increased cardiovascular morbidity and mortality associated with DM [74]. Primary prevention of the syndrome should address its risk factors including obesity, prediabetes, and prehypertension [121]. Aiming efforts toward reducing obesity and physical inactivity, across young populations is a start, especially among those at high risk. Therefore, identification of high risk population coupled with risk reduction strategies, could reduce the prevalence of MetS and may prevent its development.

There is a paucity of data available about the prevalence of MetS and its relation with overweight and obesity among young female adults in the UAE. Furthermore, this research aimed to determine the prevalence of MetS in Emirati females aged 17–25 years as this age range has not been studied previously, and its relation to overweight and obesity in the UAE. Therefore, the purpose of this study was to answer two main questions:

- What is the prevalence of MetS in Emirati females aged 17–25 years?
- What are the risk factors of MetS in Emirati females aged 17–25 years?

4.2 Results

4.2.1 Characteristics of Study Population

The demographic and clinical characteristics of the study population by metabolic syndrome status are presented as mean \pm standard deviation, in Table 4.1. The mean age of the study population was 20.4 ± 1.7 years, ranging from 17 to 25 years. The average age of participants with MetS was not significantly different from those without MetS (20.9 vs. 20.4 years, $P = 0.057$). However, participants with MetS had a significantly higher weight; height; hip circumference; neck circumference; body mass index; body fat percentage; and high-sensitivity C-reactive protein ($P < 0.001$). MetS was also associated with higher glycated hemoglobin (6.3 ± 1.1 vs. 5.5 ± 0.8 %; $P < 0.001$), and LDL-C levels (102.5 ± 30.9 vs. 91.0 ± 24.7 , $P = 0.006$).

Table 4.1: Demographic and Clinical characteristics of the study population by metabolic syndrome status

	With Metabolic Syndrome (N=38)	Without Metabolic Syndrome (N= 517)	<i>Student's t-test P-value *</i>
	Mean ± SD	Mean ± SD	
Age (Year)	20.9 ± 1.7	20.4 ± 1.7	0.057
Weight (kg)	82.1 ± 17.1	58.9 ± 12.2	< 0.001
Height (cm)	161.3 ± 5.2	158.9 ± 5.8	0.013
Hip Circumference (cm)	115.42 ± 12.35	98.67 ± 9.71	<0.001
Neck Circumference (cm)	33.51 ± 5.47	30.88 ± 3.62	<0.001
Body Mass Index (kg/m²)	31.5 ± 6.3	23.2 ± 4.6	<0.001
Body Fat (%)	40.3 ± 3.9	32.9 ± 5.3	<0.001
Serum Low Density Lipoprotein (mg/dL)	102.5 ± 30.9	91.0 ± 24.7	0.006
Serum Total Cholesterol (mg/dL)	165.7 ± 37.9	155.6 ± 31.9	0.063
Glycated Hemoglobin (%)	6.3 ± 1.1	5.5 ± 0.8	<0.001
Hemoglobin (g/dL)	12.00 ± 1.6	11.8 ± 1.5	0.482
High sensitivity C-reactive protein (mg/L)	5.25 ± 2.21	1.06 ± 1.56	<0.001

**P* < 0.05.

4.2.2 Prevalence of Metabolic Syndrome

The overall prevalence of the MetS using the IDF harmonized joint scientific statement criteria was 6.8 % (95% CI: 5% to 9%) (N = 38). Moreover, no MetS defining components were found in 242 (43.6%) participants. At least one MetS component was found in 213 participants (38.4%); two MetS components were present in 62 participants (11.2%); three MetS components were found in 27 participants (4.9%); four components of MetS were present in 10 participants (1.8%); and all five MetS components were found in only one participant (0.2%). (See Figure 4.1)

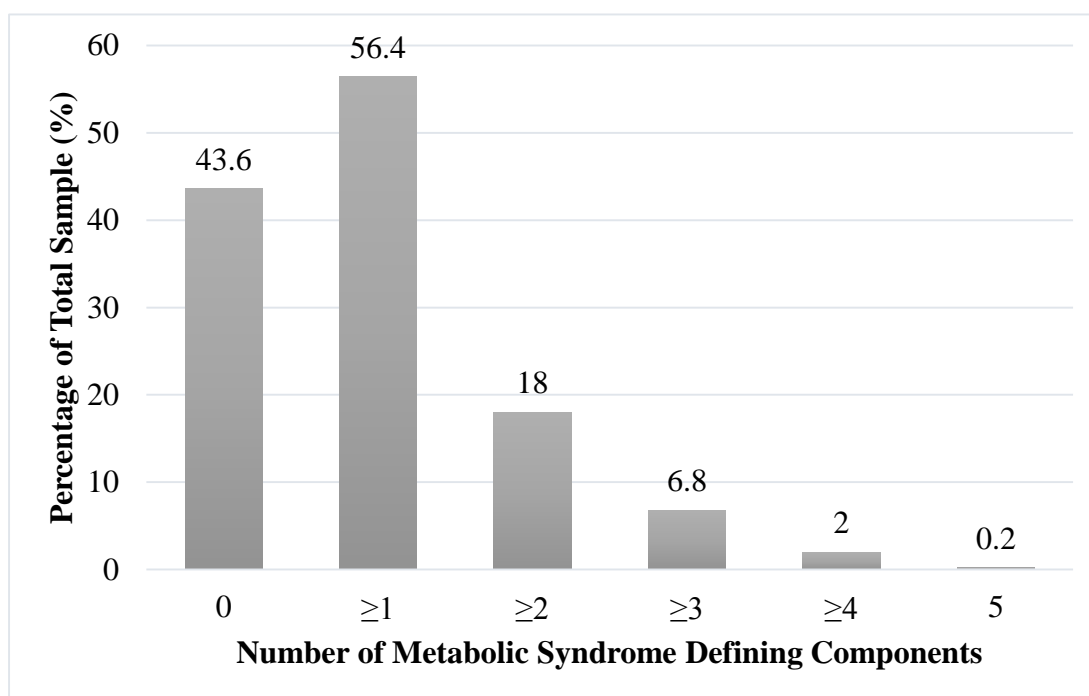


Figure 4.1: Number of Metabolic Syndrome (MetS) defining components present among UAEU young female adults 17-25 years (N = 555), Al Ain, UAE

The prevalence of MetS components among the study population are presented in Figure 4.2. The most frequent component of MetS among UAEU young female adults was reduced HDL-C levels (48.8%); followed by central obesity (18.2%); impaired fasting glucose (9.7%); hypertension (5.45); and hypertriglyceridemia (1.4%).

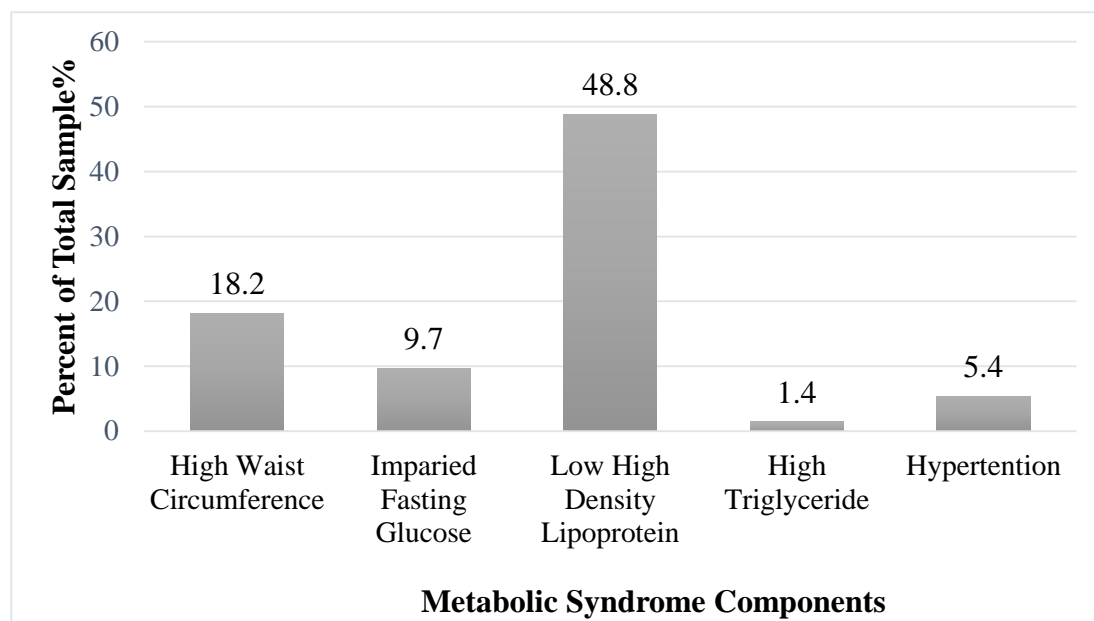


Figure 4.2: Prevalence of metabolic syndrome components among UAEU young female adults 17-25 years (N = 555), Al Ain, UAE

A Chi-square test of association between MetS and BMI categories among young female adults showed a statistically significant association ($P < 0.001$), and was particularly high among participants living with obesity (34.5%) compared to 10.1% overweight, and 1.7% normal-weight, as shown in Figure 4.3. None of the five MetS components were observed in 69% of the normal-weight participants whereas all obese participants had at least one MetS component. Obese participants were more likely to have three or more MetS components (52.6%) than overweight (34.2%) and normal-weight (13.2%) participants. Furthermore, none of the underweight participants had three or more MetS components (Figure 4.4).

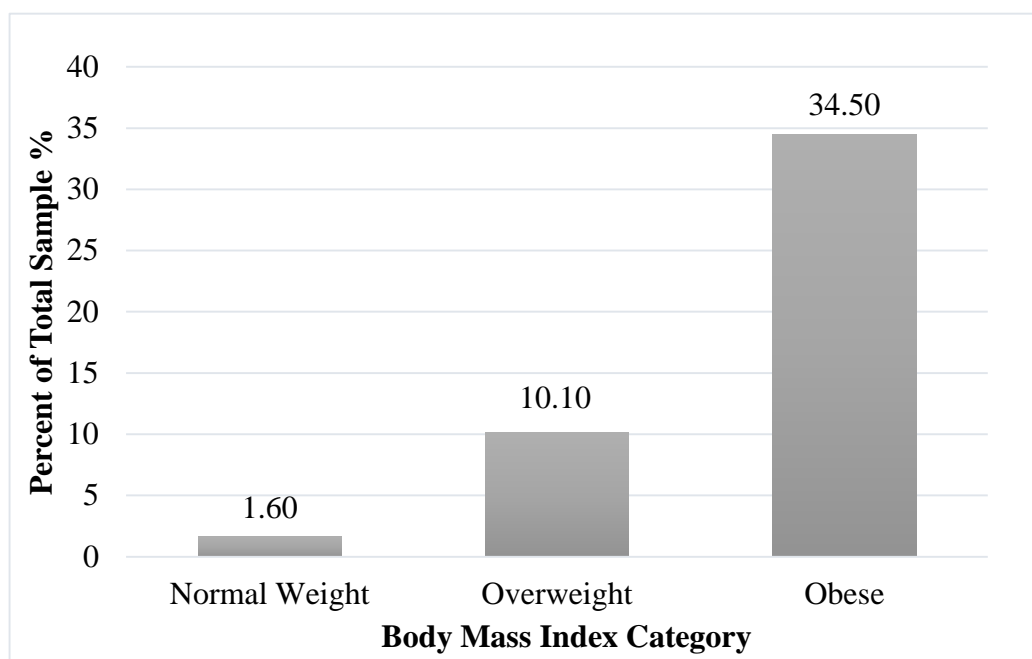


Figure 4.3: Prevalence of metabolic syndrome by body mass index (BMI) category among young female adults aged 17 to 25 years (n = 555), Al Ain, UAE

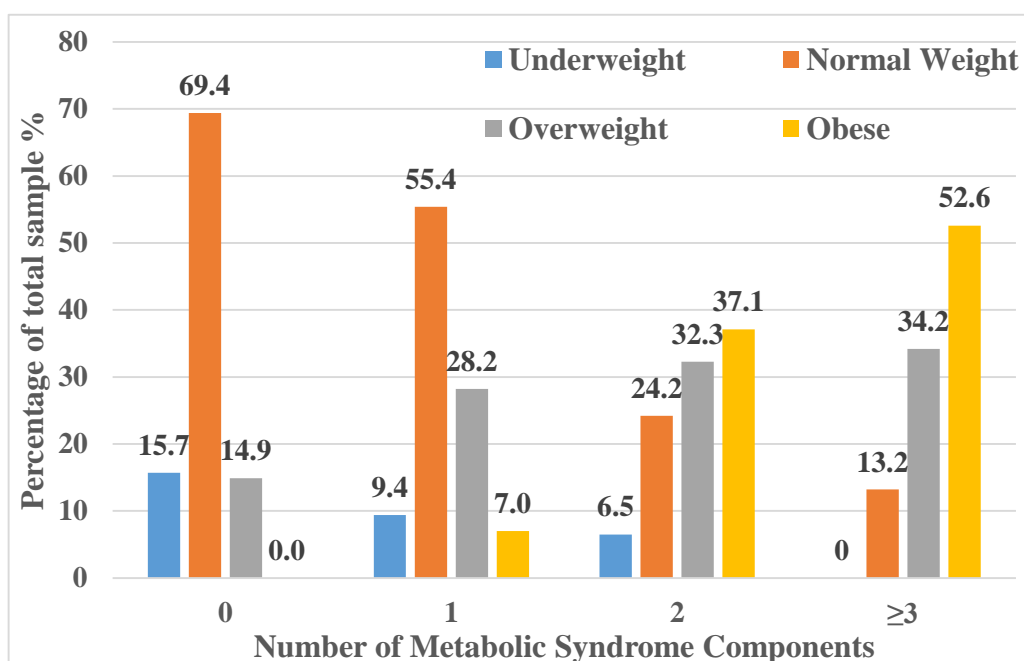


Figure 4.4: Percentage of participants per number of metabolic syndrome components and BMI category among young female adults aged 17 to 25 years (n=555), Al Ain, UAE

4.2.3 Univariable and Multivariable Logistic Regression Analyses

Logistic regression was used to determine the association between independent variables such as age, family history, anthropometric measures and biochemical measures, and the presence of metabolic syndrome defined according to the IDF harmonized criteria. Table 4.2 and Table 4.3 show the univariable and multivariable logistic regression results for the odds of MetS by potential risk factors, respectively. In the univariable analyses, older participants (23–25 years) were three times more likely to have MetS (odds ratio [OR] = 2.96; 95% CI: 1.03 to 8.52) than younger participants (17–19 years) but this effect was not significant in the multivariable analyses (adjusted OR [aOR]= 1.14; 95% CI: 0.30 to 4.30).

Participants who reported having a family history of diabetes or hypertension (n = 305) had a 2.8 times elevated risk of MetS (OR = 2.81; 95% CI: 1.31 to 6.06) compared with participants without a family history of diabetes or hypertension (n=250) in the univariable analyses but not in the adjusted analyses (aOR=1.85; 95% CI: 0.73 to 4.65).

Participants who were overweight or obese were, respectively, 6.4 (95% CI:2.3 to 17.5) and 29 (95% CI; 29.2 to 79.3) times more likely to have MetS than those of normal-weight in the univariable analysis ($P<0.001$). These findings remained significant even after adjusting for other potential confounders (i.e. aOR = 3.8; 95% CI: 1.15–12.52 for overweight; and aOR = 11.2; 95% CI: 3.06–40.86 for obese participants). Participants with percentage body fat $\geq 35\%$ (n = 227) showed a significantly higher risk for the development of MetS in the univariable analyses (OR = 14.27; 95% CI: 4.99 to 40.83; $P<0.001$), but the difference was not significant after adjusting for other factors (aOR = 3.12; 95% CI: 0.91–10.68).

A waist-hip ratio (WHR) of more than 0.8 was significantly associated with at least three times increased risk of MetS ($P < 0.001$) in the adjusted analyses when compared with those with a WHR < 0.8 (aOR = 3.04; 95% CI: 1.10–8.44).

Elevated glycated hemoglobin (HbA1c) ($\geq 6.5\%$) showed a high significant association with the presence of MetS (OR = 14.15; 95% CI: 4.78 to 41.86; $P < 0.001$) in univariable analyses and remained significant in the adjusted analyses (aOR = 22.49, 95% CI: 6.37 to 79.42; $P < 0.001$).

Total cholesterol ≥ 200 mg/dL and low density lipoprotein (LDL) ≥ 130 mg/dL conferred a greater likelihood for MetS: OR = 3.34 (95% CI: 1.48 to 7.49), and OR = 1.86 (95% CI: 0.96 to 3.63), respectively, in the univariable analyses but not in the adjusted analyses aOR = 1.04 (95% CI: 0.46 to 2.35), and aOR = 1.71 (95% CI: 0.53 to 5.55), respectively. However, the odds of MetS were significantly higher in those with high sensitivity C-reactive protein > 3 mg/L (OR = 339.92; 95% CI: 77.6 to 1489.5), when compared to high sensitivity C-reactive protein ≤ 3 mg/L in univariable analyses and remained significant in the adjusted analyses (aOR = 217.89; 95% CI: 36.87 to 1287.81; $P < 0.001$).

A subgroup analysis was conducted for the study population excluding all females with HbA1c ≥ 6.5 resulting in a total sample size of 507 participants. Results of the subgroup analysis remain largely unchanged based on the magnitude of the effect sizes, direction of significance and overall conclusions. However, in the multivariate analysis, only waist hip-ratio was no longer significant in the subgroup analysis (OR = 2.12; 95% CI: 0.65–6.87; $P = 0.211$) (Table 4.4)

Table 4.2: Univariable analyses - Risk factors for MetS among young female adults aged 17 to 25 years (n=555), Al Ain, UAE

Variable	N	With MetS			
		n (%)	Crude Odds Ratio (95%CI)	P-value	P-value trend
Age, Year (%)					0.103
17-19	194	8 (4.1)	Reference		
20-22	299	23 (7.7)	1.94 (0.85 - 4.42)	0.116	
23-25	62	7 (11.3)	2.96 (1.03 - 8.52)	0.044	
Family history of diabetes or hypertension (%)					0.005
No	250	9 (3.6)	Reference		
Yes	305	29 (9.5)	2.81 (1.31 - 6.06)	0.008	
Body Mass Index (kg/m²)					<0.001
Underweight (<18.5)	62	0 (0.0)	0.44 (0.02 - 8.03)	0.58	
Normal weight (18.5 - <25)	306	5 (1.6)	Reference		
Overweight (25-29.9)	129	13 (10.1)	6.35 (2.30 - 17.51)	<0.001	
Obese (≥30.0)	58	20 (34.5)	29.19 (10.75 - 79.28)	<0.001	
Body Fat (%)*					<0.001
<35%	328	4 (1.2)	Reference		
≥35%	227	34 (14.9)	14.27 (4.99 - 40.83)	<0.001	
Waist-Hip Ratio**					0.001
<0.8	509	28 (5.5)	Reference		
≥0.8	46	10 (21.7)	4.77 (2.15 - 10.59)	<0.001	
Anemia***					0.246
No	271	22 (8.1)	Reference		
Yes	284	16 (5.6)	0.67 (0.35 - 1.32)	0.249	
Total Cholesterol (mg/dL)					0.007
< 200	502	29 (5.8)	Reference		
≥200	53	9 (16.9)	3.34 (1.48 - 7.49)	0.004	
Low Density Lipoprotein (mg/dL)					0.072
<130	380	21 (5.5)	Reference		
≥ 130	173	17 (9.8)	1.86 (0.96 - 3.63)	0.067	
High sensitivity C-reactive protein (mg/L)					<0.001
≤3	493	2 (0.4)	Reference		
>3	62	36 (58.1)	339.92 (77.6 - 1489.5)	<0.001	
Glycated Hemoglobin (%)					<0.001
< 5.6	374	6 (1.6)	Reference		
5.6 - 6.4	133	23 (17.3)	12.82 (5.09 - 32.29)	<0.001	
> 6.5	48	9 (18.8)	14.15 (4.78 - 41.86)	<0.001	

* Physical status: the use and interpretation of anthropometry 1995 [213]

** Waist circumference and waist-hip ratio: Report of a WHO expert consultation 2011 [222]

*** Hemoglobin concentrations for the diagnosis of Anemia according to the WHO 2011 [261]

Table 4.3: Multivariable analyses - Risk factors for MetS among young female adults aged 17 to 25 years (n=555), Al Ain, UAE

Characteristics	N	n (%)	With MetS	
			Adjusted Odds Ratio (95%CI)	P-value
Age (Year)				
17-19	194	8 (4.1)	Reference	
20-22	299	23 (7.7)	1.89 (0.72 – 4.94)	0.19
23-25	62	7 (11.3)	1.14 (0.30 – 4.30)	0.85
Family history of diabetes or hypertension (%)				
No	250	9 (3.6)	Reference	
Yes	305	29 (9.5)	1.85 (0.73 – 4.65)	0.19
Body Mass Index (kg/m²)				
Underweight (<18.5)	62	0 (0.0)	0.85 (0.04 – 17.26)	0.92
Normal weight (18.5 - <25)	306	5 (1.6)	Reference	
Overweight (25-29.9)	129	13 (10.1)	3.80 (1.15 – 12.52)	0.028
Obese (≥30.0)	58	20 (34.5)	11.19 (3.06 – 40.86)	<0.001
Body Fat (%)*				
<35%	328	4 (1.2)	Reference	
≥35%	227	34 (14.9)	3.12 (0.91 – 10.68)	0.07
Waist-Hip Ratio**				
<0.8	509	28 (5.5)	Reference	
≥0.8	46	10 (21.7)	3.04 (1.10 – 8.44)	0.033
Anaemia***				
No	271	22 (8.1)	Reference	
Yes	284	16 (5.6)	1.04 (0.46 – 2.35)	0.92
Total Cholesterol (mg/dL)				
< 200	502	29 (5.8)	Reference	
≥200	53	9 (16.9)	1.71 (0.53 – 5.55)	0.37
Low Density Lipoprotein (mg/dL)				
<130	380	21 (5.5)	Reference	
≥ 130	173	17 (9.8)	0.92 (0.37 – 2.32)	0.86
High sensitivity C-reactive protein (mg/L)				
≤3	493	2 (0.4)	Reference	
>3	62	36 (58.1)	217.89 (36.87 – 1287.81)	<0.001
Glycated Haemoglobin (%)				
< 5.6	374	6 (1.6)	Reference	
5.6 - 6.4	133	23 (17.3)	8.92 (3.39 - 23.48)	<0.001
≥ 6.5	48	9 (18.8)	22.49 (6.37 – 79.42)	<0.001

* Physical status: the use and interpretation of anthropometry 1995 [213]

** Waist circumference and waist-hip ratio: Report of a WHO expert consultation 2011 [222]

*** Hemoglobin concentrations for the diagnosis of Anemia according to the WHO 2011 [261]

Table 4.4: Subgroup multivariable analysis - Risk factors for MetS among young female adults aged 17 to 25 years (n=507), Al Ain, UAE – excluding 48 participants that had HbA1c \geq 6.5

Characteristics	Sample	n (%)	With MetS			
			Crude Odds Ratio (95%CI)	P-value	Adjusted Odds Ratio (95%CI)	P-value
Age (Year)						
17-19	175	6 (3.4)	Reference		Reference	
20-22	278	17 (6.1)	1.83 (0.71, 4.75)	0.211	1.50 (0.53 – 4.28)	0.448
23-25	54	6 (11.1)	3.52 (1.09, 11.41)	0.036	1.89 (0.47 – 7.60)	0.365
Family history of diabetes or hypertension (%)						
No	223	7 (3.1)	Reference		Reference	
Yes	284	22 (7.8)	2.59 (1.09, 6.18)	0.032	1.03 (0.73 – 4.65)	0.19
Body Mass Index (kg/m²)						
Underweight (<18.5)	58	0 (0.0)	0.94 (0.044, 19.88)	0.969	1.54 (0.07 – 35.79)	0.785
Normal-weight (18.5 - <25)	277	2 (0.7)	Reference		Reference	
Overweight (25-29.9)	120	11 (9.2)	11.57 (2.89, 46.26)	0.001	5.62 (1.13 – 27.86)	0.035
Obese (\geq 30.0)	52	16 (30.8)	49.82 (12.61, 196.88)	<0.001	15.35 (2.92 – 80.75)	0.001
Body Fat (%)*						
<35%	296	2 (0.7)	Reference		Reference	
\geq 35%	211	27 (12.8)	21.57 (5.07, 91.78)	<0.001	2.84 (0.57 – 14.06)	0.201
Waist-Hip Ratio**						
<0.8	467	23 (4.9)	Reference		Reference	
\geq 0.8	40	6 (15.0)	3.41 (1.30, 8.93)	0.013	2.12 (0.65 – 6.87)	0.211
Anaemia***						
No	247	18 (7.3)	Reference		Reference	
Yes	260	11 (4.2)	0.56 (0.26, 1.22)	0.143	0.68 (0.28 – 1.69)	0.411
Total Cholesterol (mg/dL)						
< 200	459	22 (4.8)	Reference		Reference	
\geq 200	48	7 (14.6)	3.39 (1.37, 8.41)	0.008	1.38 (0.37 – 5.17)	0.634
Low Density Lipoprotein (mg/dL)						
<130	348	16 (4.6)	Reference		Reference	
\geq 130	157	13 (8.3)	1.87 (0.88, 3.99)	0.104	0.78 (0.27 – 2.26)	0.653
Glycated Haemoglobin (%)						
< 5.6	374	6 (1.6)	Reference		Reference	
5.6 - 6.4	133	23 (17.3)	12.82 (5.09, 32.29)	<0.001	8.11 (3.12 – 21.08)	<0.001

* Physical status: the use and interpretation of anthropometry 1995 [213]

** Waist circumference and waist-hip ratio: Report of a WHO expert consultation 2011 [222]

*** Hemoglobin concentrations for the diagnosis of Anemia according to the WHO 2011 [261]

4.2.4 High Sensitivity C-Reactive Protein and Metabolic Syndrome

Logistic regression analyses showed that the odds of MetS were significantly higher in those with high sensitivity C-reactive protein (Hs-CRP) >3 mg/L (OR = 339.92; 95% CI: 77.6 to 1489.5), when compared to high sensitivity C-reactive protein ≤ 3 mg/L in univariable analyses with the odds remained significant in the adjusted analyses (aOR:217.89; 95% CI: (36.87 to 1287.81) ($P < 0.001$)). Figure 4.5 displays the distribution of Hs-CRP levels after females were classified according to their total number of components of the MetS. The MetS represents an inflammatory state that could be seen by the progressive increase of Hs-CRP levels with the increased number of MetS components according to the harmonized IDF criteria. The graph illustrates the strong linear increase in Hs-CRP levels as the number of MetS components increased. The median Hs-CRP levels for those with 0, 1, 2, ≥ 3 components of the MetS were 0.46, 0.78, 1.405, and 5.14 mg/L, respectively ($P < 0.001$).

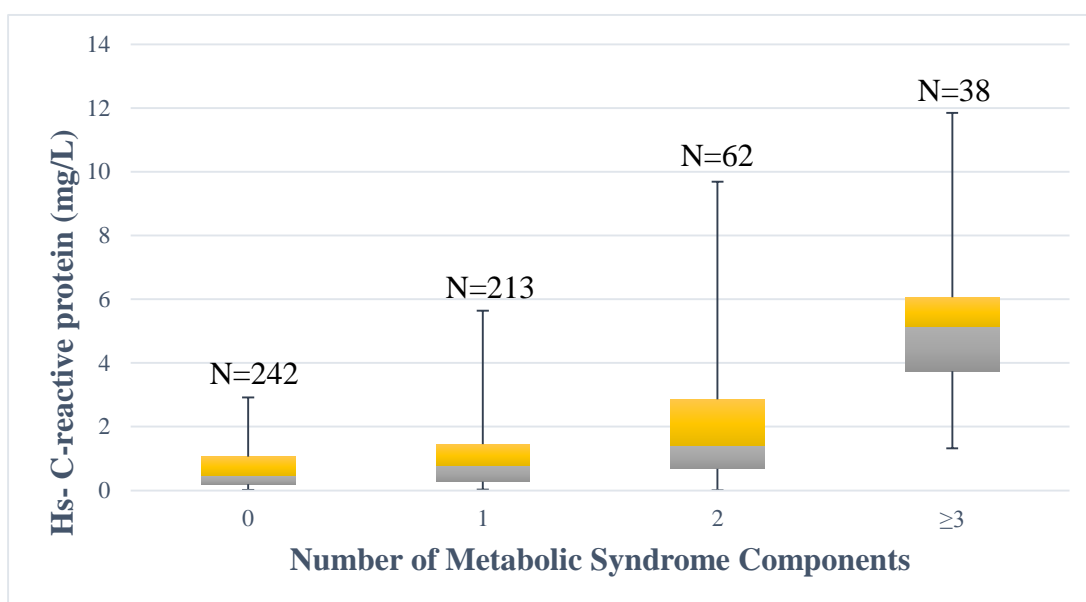


Figure 4.5: Distribution of High Sensitivity C-reactive protein according to number of metabolic syndrome components present among young female adults aged 17 to 25 years (n=555), Al Ain, UAE. Box plots demonstrate minimum, maximum, median, 25th, and 75th percentile values for Hs-CRP

4.2.5 Prevalence of Overweight/Obesity

The distribution of the studied population according to their body mass index (BMI) categories showed that 11.2% were underweight; 55.1% were normal weight; and 33.7% were overweight or obese (23.2% and 10.5%, respectively) as indicated in Figure 4.6.

Table 4.5 demonstrates that 7.6% of the studied population were classified at class I obesity (BMI 30.00 – 34.9 kg/m²); 1.6% met the class II obesity classification (BMI 35.00 – 39.9 kg/m²); and 1.3% had a BMI \geq 40.00 kg/m².

Central obesity was prevalent among 18.2% of the studied population based on the waist circumference (WC) classification. Whereas, only 8.3% of the studied population were found living with central obesity when waist-hip ratio \geq 0.8 was used as a cutoff point. In opposition, measurement of body fat percentage from skinfold thickness at four sites (biceps, triceps, subscapular and suprailiac) via Durnin's regression equation [232] showed that 40.9% of the sample population had a body fat percent $>$ 35%.

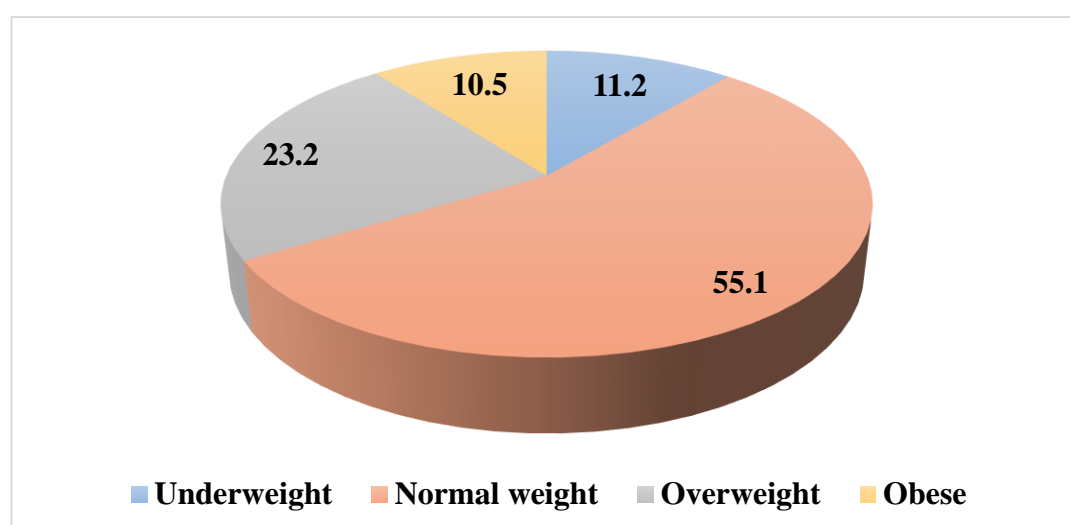


Figure 4.6: Distribution of Emirati young female adults aged 17 to 25 years (n=555) according to body mass index categories

Table 4.5: Distribution of the study population according to different measures of obesity

Variable	N	%
Body Mass Index		
Underweight (<18.50 kg/m ²)	62	11.2
Normal weight (18.50 – 24.99 kg/m ²)	306	55.1
Overweight (25.00 – 29.99 kg/m ²)	129	23.2
Obese class I (30.00 – 34.9 kg/m ²)	42	7.6
Obese class II (35.00 – 39.99 kg/m ²)	9	1.6
Obese class III (\geq 40.00 kg/m ²)	7	1.3
Waist Circumference		
<80 cm	454	81.8
\geq 80 cm (central obesity)	101	18.2
Waist-Hip Ratio		
<0.8	509	91.7
\geq 0.8	46	8.3
Body Fat		
<35%	328	59.1
\geq 35%	227	40.9

4.2.6 Prevalence of Diabetes

According to the standards of medical care in diabetes set by the American Diabetes Association (ADA) in 2016 [255] (previously presented in Table 3.4) diabetes mellitus (DM) could be diagnosed based on the level of fasting plasma glucose (FPG) or glycated hemoglobin (HbA1c). The classification of the study population according to FPG and HbA1c for the diagnosis of diabetes and prediabetes are presented in Table 4.6. The prevalence of diabetes mellitus and prediabetes based on FPG was 0.5% and 9.2%, respectively. However, 8.6% of the studied population were diagnosed with diabetes, and 24% were living with prediabetes according to the percentage of HbA1c. Thus, the crude prevalence of diabetes mellitus and pre-diabetes (shown in Figure 4.7) among the study population was 8.6% and 24.7%, respectively.

Table 4.6: Classification of the study population according to different standards for the diagnosis of diabetes and prediabetes

Variable	N	%
Fasting Plasma Glucose (FPG)		
Normal (≤ 99 mg/dL)	501	90.3
Prediabetes (100-125 mg/dL)	51	9.2
Diabetes (≥ 126 mg/dL)	3	0.5
Glycated Hemoglobin (HbA1C)		
Normal (About 5 %)	374	67.4
Prediabetes (5.7-6.4 %)	133	24.0
Diabetes (≥ 6.5 %)	48	8.6
FPG or HbA1c		
Normal	370	66.7
Prediabetes	137	24.7
Diabetes	48	8.6

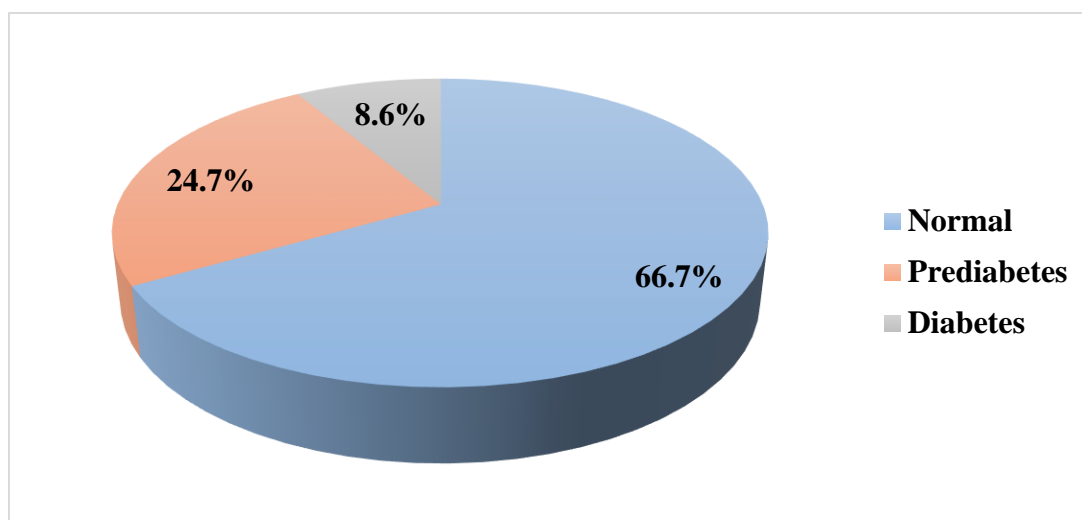


Figure 4.7: Prevalence of abnormal glycemic status based on fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) among Emirati young female adults aged 17 to 25 years (n=555), Al Ain, UAE

Logistic regression was used to determine the association between age; family history; anthropometric measures; and biochemical measures, and the presence of diabetes or prediabetes, as illustrated in Table 4.7. Neither age of the participant, nor having a family history of diabetes or hypertension had a significant association with the risk of DM ($P=0.66$, 0.809 , respectively). Nevertheless, participants living with obesity (n=58) were almost 2.5 times (OR: 2.407; 95% CI: 1.373 to 4.221) more likely to have DM than those with BMI<25.0 (n=368) ($P=0.002$).

Percentage of body fat did not show an association with the risk of DM ($P=0.329$). However, waist circumference of more than 80 cm was significantly associated with increased risk of DM (OR: 2.561; 95% CI: 1.650, 3.975) ($P<0.001$). Hip circumference was also associated with an increased odds of DM (OR: 1.028; 95% CI: 1.011, 1.045). Elevated high sensitivity C-reactive protein ≥ 3 mg/L, conferred a greater likelihood for MD (OR: 12.095; 95% CI: 6.312, 23.176).

Table 4.7: Univariate logistic regression analysis model of the association between abnormal glycemic status based on fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) and selected variables among young Emirati female adults aged 17 to 25 years (n=555), Al Ain, UAE

Variable	N	With abnormal glycemic status			
		n (%)	Odds Ratio (95% CI)	P-value	P-value
Age Year (%)					0.66
17-19	194	62 (32.0)	Reference		
20-22	299	94 (31.4)	0.976 (0.662 - 1.439)	0.903	
23-25	62	29 (46.8)	1.871 (1.044 - 3.352)	0.035	
Parents Diabetic or Hypertensive (%)					0.809
No	250	82 (32.8)	Reference		
Yes	305	103 (33.8)	1.045 (0.732 - 1.490)	0.809	
Blood Pressure (mm Hg)					0.119
Normal	453	147 (32.5)	Reference		
Prehypertension	93	37 (39.8)	1.375 (0.869 - 2.178)	0.174	
Hypertension	9	1 (11.1)	0.260 (0.32 - 2.100)	0.206	
Body Mass Index (kg/m²)					0.013
Underweight (<18.5)	62	16 (25.8)	Reference		
Normal weight (18.5 - <25)	306	92 (30.1)	1.236 (0.665 - 2.296)	0.502	
Overweight (25-29.9)	129	48 (37.2)	1.704 (0.870 - 3.335)	0.120	
Obese (≥30.0)	58	29 (50.0)	2.875 (1.335 - 6.192)	0.007	
Body Fat (%)					0.329
<35	328	104 (31.7)	Reference		
≥35	227	81 (35.7)	1.195 (0.836 - 1.709)	0.329	
Waist Circumference (cm)					<0.001
<80	454	133 (29.3)	Reference		
≥80	101	52 (51.5)	2.561 (1.650 - 3.975)	<0.001	
Hip Circumference (cm)	555	185 (33.3)	1.028 (1.011 - 1.045)	0.001	0.001
Neck Circumference (cm)	555	185 (33.3)	1.022 (0.975 - 1.071)	0.365	0.353
Waist-hip Ratio					0.238
<0.8	509	166 (32.6)	Reference		
≥ 0.8	46	19 (41.3)	1.454 (0.786 - 2.691)	0.233	
Anemia (%)					0.904
No	271	91 (33.6)	Reference		
Yes	284	94 (33.1)	0.979 (0.688 - 1.393)	0.904	
Total Cholesterol (mg/dL)					0.313
< 200	502	164 (32.7)	Reference		
≥200	53	21 (39.6)	1.353 (0.756 - 2.418)	0.309	

Low Density Lipoprotein (mg/dL)					0.078
<130	380	118 (31.1)	Reference		
≥ 130	173	67 (38.7)	1.403 (0.964 - 2.042)	0.077	
High Density Lipoprotein (mg/dL)					<0.001
>45	284	70 (24.6)	Reference		
≤ 45	271	115 (42.4)	2.254 (1.570 - 3.236)	<0.001	
High-sensitivity C-reactive Protein (mg/L)					<0.001
Low (<1)	326	72 (22.1)	Reference		
Intermediate (1-2.9)	167	65 (38.9)	2.248 (1.497 - 3.376)	<0.001	
High (≥ 3)	62	48 (77.4)	12.095 (6.312- 23.176)	<0.001	

Table 4.8: Adjusted analysis model of the association between abnormal glycaemic status based on fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) and selected variables among young Emirati female adults aged 17 to 25 years (n=555), Al Ain, UAE

Variable	Odds Ratio	95% Confidence Interval		P-value
		Lower	Upper	
Body Mass Index (kg/m²)	1.164	0.738	1.835	0.514
Waist circumference (cm)	0.978	0.941	1.016	0.252
Hip circumference (cm)	1.007	0.969	1.046	0.737
Hs-CRP* (mg/L)	1.578	1.360	1.832	<0.001

* High sensitivity C- reactive protein.

4.3 Discussion

In the United Arab Emirates (UAE) rapid socioeconomic growth has resulted in profound lifestyle changes including sedentary behaviors, westernized diets and increased energy intake [167]. The prevalence of MetS among Emirati females has been reported to be higher than that for Emirati males in the adult population (32.9% among men, 45.9% among women) [5]. This research highlights the importance of investigating MetS among young female adults, to facilitate understanding of the prevalence and risk factors of MetS. There is paucity of data on the prevalence of MetS among Emirati females aged 17–25 years. The current study reveals a MetS prevalence of 6.8% among young female Emirati adults aged 17–25 years, and 34.5% among young obese female Emirati adults.

There are few data available about the prevalence of MetS among Emirati females aged 17-25. The current study reveals a MetS prevalence of 6.8% among young female Emirati adults aged 17-25. This percentage is much higher than the 4% prevalence previously reported in a separate study of female Emirati adolescents (12-18 years old) [6] and much lower than that among Emirati female adults (≥ 20 years) 45.9% reported in 2008 [5]. A recent study by Hajat and others in 2012, during the Weqaya screening program in the Emirate of Abu Dhabi [307], found that the overall prevalence of MetS among Emirati adults was 48.7% and 50.3% using the MetS definition of IDF and ATP III, respectively. These findings suggest about 8% increase during the period of 4 years only, when compared with those described in 2008 [5] using the same IDF criteria, where the overall prevalence of MetS was 40.5%. The same prevalence was described in 2010 [308] among overweight and obese adults (18-50 years old) in the UAE. MetS prevalence among the young obese (<12 years old) in

the UAE was stated to be 44% in the same year [309]. In the present study, the prevalence of MetS was 34.5% among obese students.

The results of the current study are in line with findings among college students (18–26 years) in Saudi Arabia [310], where the overall MetS prevalence was 7.8%, and 26.4% in obese students. In the same study, the prevalence of MetS among Saudi female college students was 1.7% compared to 9.9% among males. However, the prevalence of MetS among Saudi female adults was stated to be 34.1% and 36.6% among male adults in 2010[134].

In Kuwait, the prevalence of MetS was even higher among female adolescents (10–19 years) at 9.1% and 14.8% according to ATP III and IDF criteria, respectively [159]. MetS among adult Kuwaitis were similar between men and women at 36.2% and 36.1%, respectively [133]. In Oman, the overall prevalence of MetS among adults (>20 years old) was 23.6%, it was somewhat higher in women 24.4% compared to men 22.8% [131]. These numbers are certainly close to those described in the UAE, which is not surprising, considering the relatively similar rapid increase in obesity and diabetes rates throughout the Gulf region [311, 312]. These trends are as a result of a sedentary and westernised lifestyle [313-316], and could also be partially explained by the “thrifty genes” hypothesis [309], which suggests that the genotype of mankind existed as hunter-gatherers can efficiently store food in the adipose tissue during periods of food abundance, to compensate for periods of food shortage.

Worldwide, the prevalence of MetS among young female adults in the USA (18-21 years) was 4.7% [317], in Brazilian college students it was 1.7% [318], in Chinese female adolescents (14-16 years) it was 2.5% [319], in Spanish female adolescents (10-15 years) it was 3.85% [320], in Tunisian female adolescents (10-19

years) it was 2.4% [321], and 11.7% among Indian female adolescents (10-19 years) [322]. Clearly, the prevalence of MetS could differ between countries depending on the MetS defining criteria used, study method and target population. We have used the IDF and AHA/NHLBI joint statement, as it was an international attempt to harmonize the definition of MetS; central obesity is not an obligatory component of this definition and it is ethnic specific.

The most frequent component of MetS in this study was reduced HDL-C levels, which was also reported in the female Emirati adolescents [6], and female Kuwaiti adolescents [159]. Reduced HDL-C accompanied by elevated triglyceride levels indicates dyslipidemia, which is highly prevalent among the UAE population [323]. Insufficient physical activity and poor dietary habits are associated with low HDL-C levels [324-326]. Elmagd et. al. reported low physical activity (defined as less than 150 minutes/week) in 60% of Emirati college students [327]. The consumption of high caloric diet was also reported in 33.5% of female Emirati adolescents [167]. These findings could explain the high prevalence of low-HDL-C levels observed in our study. Regular checks and screening in this age group could be helpful in identifying participants at an increased risk of developing MetS.

The current study found strong correlations between BMI, body fat, hbA1c, High sensitivity C-reactive protein and the prevalence of MetS in Emirati female students. The relationship between overweight and obesity and MetS has been supported by many other studies [134, 158, 328].

Ridker and his colleagues showed a strong evidence that the MetS represents an inflammatory state that could be seen by the progressive increase of Hs-CRP levels with the increased number of the MetS components according to the ATP III criteria

[67]. Up to date, the only well-standardized marker of inflammation and a predictor of the MetS and future cardiovascular events, is the Hs-CRP [68]. However, the measurement of Hs-CRP is not yet included in the diagnostic criteria of the MetS, although it has been recommended by a large number of investigators [67-71]. Inflammation is usually measured through the measurement of serum Hs-CRP, where a level above 3 mg/L is considered high according to the recommendations of the CDC and the AHA for cardiovascular disease (CVD) risk assessment [63]. However, Hs-CRP is a sensitive marker for acute phase inflammation and has a high within-subject variability [329]. Therefore, a value above 3 mg/L might indicate an increased risk of cardiovascular, but could also be a result of an infection or inflammation, which may vague any projection of coronary risk that might be attributed to the elevated level [330].

The association between HbA1c and the prevalence of MetS has not been previously reported; nevertheless, insulin resistance is a major underlying mechanism accountable for the prevalence of the MetS [331]. Interestingly, the prevalence of diabetes (8.6%) was also high in the study population. Future studies need to explore this finding more closely.

The prevalence of diabetes mellitus and prediabetes based on FPG (0.5% and 9.2%, respectively) was found much lower than that based on the percentage of HbA1c (8.6% and 24%, respectively) However, the crude prevalence of diabetes mellitus and pre-diabetes among Emirati young female adults was 8.6% and 24.7%, respectively.

Our present results are in disagreement with those previously reported in a hospital-based study where the prevalence of diabetes was higher using the FPG criterion (31.6%) compared to the HbA1c criterion (23.5%) [332]. Moreover, in a

retrospective study in Saudi Arabia, the prevalence of impaired glucose tolerance was 54% when using HbA1c as a diagnostic test, and 60.3% when using FPG as a diagnostic test [333]. In contrast, our results are in agreement with a population-based study among Palestinian Arab population that showed a lower prevalence of diabetes diagnoses using the FPG criterion (4.5%) compared to HbA1c criterion (5.3%)[334]. In study among the Korean population, the prevalence of diabetes and prediabetes using FPG only (10.5% and 19.3%, respectively) were lower compared to the prevalence of diabetes and prediabetes using HbA1c as a diagnostic test (12.4% and 38.3%, respectively) [335]. In Canada, using HbA1c only for the diagnoses of overall prediabetes prevalence resulted in a three-fold increase compared to FPG and a six-fold increase among females (FPG 2.22%, HbA1c 13.31%)[336].

Although more participants were diagnosed with diabetes when HbA1c was added as a diagnostic criterion, yet the simultaneous measurement of FPG and HbA1c (FPG and/or HbA1C) is recommended by the American Diabetes Association [255] and has been shown to be a more sensitive and specific screening tool for identifying high-risk individuals with diabetes and IGT at an early stage [337, 338]. Advantages of using HbA1C include: convenience, pre-analytical stability, and minimum day-to-day perturbations during periods of stress and illness. Nevertheless, this test is pricey, and might not be available in certain regions of the developing world [255].

The strengths of this study include a trained researcher who obtained all measurements in the study and each measurement was repeated three times and the average used in the analyses. Anthropometric measures and blood withdrawal were conducted during one 50-minute morning session after assurance of a 12-hour overnight fast. Furthermore, to the best of our knowledge, no other studies exploring

MetS prevalence in college students have been conducted in the UAE. UAEU is the main university in the UAE and it enrolls students from all seven emirates. However, restricting the study to college students makes it not representative of all the Emirati females in this age group.

Moreover, the cross-sectional design is another limitation of this study, as causal inference cannot be drawn. Participants were voluntarily enrolled in the study, which could have caused selection bias (overweight and obese individuals might avoid anthropometric measurements). Additionally, a study conducted by Malik and Razig in 2008 showed that Emirati female adults who had no formal education were 1.27 (95% CI:1.03 to 1.58) times more likely to have MetS than those who had higher education (>12 grade) ($P= 0.028$) according to the IDF definition of MetS [5]. Therefore, limiting participation in the current study to university students only might have introduced underestimation of MetS prevalence among young adults, especially that Malik reported only 25.4% of Emirati female adults (>20) had university degrees [5].

In addition, studying female students only does not allow for examination of gender differences or generalizability of results to all young adults. Therefore, future prospective studies are needed to confirm the prevalence of MetS and its relation to overweight and obesity in Emirati young adults. Additionally, it was challenging to clearly define the “young adult” age group. Some studies reported the MetS prevalence in adolescents and included ages 12–18 years [6] or 10–19 years old [159]. Other studies defined young adults as 18–24 years [317], 17–37 years [339] or college students aged 17–25 years [340]. Having one international definition for the “young adult” age group would be helpful for future data comparisons.

4.4 Conclusion

In summary, we have shown that the prevalence of MetS is high among UAEU female young adults aged 17–25 years (6.8%). One third of the studied population was overweight and obese. The prevalence of diabetes mellitus and prediabetes based on FPG was found much lower than that based on the percentage of HbA1c (0.5% and 9.2% Vs. 8.6% and 24% respectively). However, the crude prevalence of diabetes mellitus and pre-diabetes among Emirati young female adults was 8.6% and 24.7%, respectively.

MetS prevalence was highest among obese participants, as compared with normal-weight and overweight participants. In addition, reduced HDL-C levels followed by central obesity were the most frequent components of MetS. Using logistic regression model MetS was significantly associated with overweight, obesity, waist-hip ratio, glycated hemoglobin (HbA1c) and high sensitivity C-reactive protein ($P < 0.01$).

The high prevalence of MetS highlights the importance of regular screening and intervention programs targeting weight reduction. It is also necessary to design public health policy, clinical practice, and prevention programs for the screening and treatment of Emirati females at high risk for metabolic syndrome. Such strategies should address cultural and community life of Emiratis to ensure relevance and commitment by the community.

Chapter 5: Glycemic Index (GI) Value of Commonly Consumed Foods in the UAE

5.1 Introduction

Diabetes mellitus (DM) is a chronic disease characterized by abnormalities in the metabolism of carbohydrates, fat, and protein along with hyperglycemia. Complications of the disease include macrovascular and microvascular damage in the eyes (retinopathy) [341], kidneys (nephropathy) [342] and nerves (neuropathy) [343]. If left untreated, these complications could progress into blindness, kidney failure, amputation, and even death [205].

During the past three decades, the prevalence of DM in the world has doubled, which presents a great challenge to international public health. According to the recent regional fact sheets of the International Diabetes Federation (IDF), there are 382 million people living with diabetes in the world, with a prevalence of 8.3% [2]. The IDF expects an increase of 205 million patients by 2035. The 9.7% prevalence of diabetes in the Middle East and North Africa (MENA) regions is even higher than the world average [2]. Based on the same fact sheets, United Arab Emirates (UAE) was ranked fifth with regards to the highest prevalence of diabetes mellitus in the region (19%), ranked after other Gulf Cooperation Council (GCC) countries, Saudi Arabia, Kuwait, Bahrain and Qatar which have the prevalence of 23.9%, 23.1%, 21.9% and 19.8% respectively [304].

Type 2 Diabetes Mellitus (T2DM) is the most common type of diabetes. It was thought to occur among adults, but is increasingly reported in children and adolescents. With this type of diabetes, the body is capable of producing insulin but it is either not in a sufficient amount or the body is insulin resistant, leading to the accumulation of glucose in the blood, also known as hyperglycemia [205].

T2DM is an increasing source of concern in the UAE. The prevalence of T2DM was 6% among adults, according to a prevalence survey performed between 1989 and 1990 [344]. A later survey conducted between 1999 and 2000 reported a greater prevalence of about 20% [23]. A recent survey reported the prevalence of diagnosed DM at 10.5%, undiagnosed DM at 6.6%, and pre-diabetes at 20.2% [345]. The latter also reported the prevalence of retinopathy at 54.2%, neuropathy at 34.7%, nephropathy at 40.8%, peripheral vascular disease at 11.1%, and coronary heart disease at 10.5% among patients diagnosed with DM.

The observed increase in incidences of T2DM and pre-diabetes among children, adolescents and younger adults increases the need for understanding the etiology and treatment for the disease. The causes behind the epidemic of T2DM are part of a very complex group of genetic and social systems which, in turn, control behavioral and environmental factors [212]. Moreover, there are several risk factors believed to be mainly contributing to the occurrence of the disease, these include: weight gain and obesity [11]; sedentary lifestyle with low physical activity [12, 13]; ethnicity [14]; family history of diabetes [14]; poor dietary habits [346]; other factors like cigarette smoking and alcohol consumption [255].

The focus on the prevention and treatment of T2DM are lifestyle factors, including diet (for glycemic control), physical activity, and weight reduction [346-348]. The American Diabetes Association (ADA) recommends an effective ongoing support program targeting glycemic control (using carbohydrates counting and selection of low glycemic index foods), weight loss of seven percent of body weight within the first six months of intervention, and increasing physical activity to at least 150 min/week of moderate activity for pre-diabetics [255].

Dietary management and prevention of DM was found to be dependent on the quality and quantity of nutrient intake. Studies suggest that total amount of carbohydrate or fat in the diet does not seem to be associated with T2DM risk but specific forms of carbohydrate or fat were associated with the disease [18, 19]. For example: the ability of dietary carbohydrates to increase blood glucose and insulin levels after ingestion differs, and that is known as the glycemic index (GI) of carbohydrate foods [20]. This concept was developed in 1981 by Jenkins *et. al.* as a tool for predicting blood glucose response to various foods [20]. It is defined as the ratio of the blood glucose response for a tested food as compared to the blood glucose response of a standard (usually a glucose or white bread) [349]. If glucose is used as the reference, then a GI of ≤ 55 is considered low, between 56-69 is intermediate, and ≥ 70 is high [20]. Since the amount of carbohydrate in a food varies according to the serving size, researchers have also introduced the concept of glycemic load (GL), which is the amount of available carbohydrates in a serving size of food item multiplied by its glycemic index [21].

Prospective studies investigating the relations between dietary carbohydrate intake and risk of T2DM using GI and GL supported the protective role that low GI and low GL diets play against the development of T2DM [19, 192, 350]. Since a high GI food would cause a higher increase in the levels of blood glucose after its ingestion, it will, in turn, increase the demand of insulin in the body. Regular consumption of high GI foods would therefore contribute to beta cell distraction, due to the elevated insulin demand and/or continuously raised glucose concentration in the blood [19].

In 2010, a study was conducted in the UAE to determine the factors associated with poor glycemic control among patients with T2DM [22]. The results showed that

poor glycemic control among patients with T2DM was mainly because patients were not following the dietitian's recommended eating plan and/or had a negative attitude towards diabetes [22]. In a different study, researchers were examining differences in the prevalence of DM between different ethnic groups in the UAE [23]. They reported a higher DM prevalence among UAE citizens (25%) compared to expatriates (13–19%, depending on country of origin) [23]. These results advocate the need for better counseling programs focusing on Emirati citizens, which, in turn, requires more information about dietary practices of the Emiratis, food composition tables of locally consumed foods in the UAE, an exchange list of these foods, and glycemic index values of Emirati foods to facilitate the role of dietitians in developing better programs targeting glycemic control in diabetics and pre-diabetics.

This study was designed to test the null hypothesis that Emirati traditional foods are high in their GI and GL values which might be contributing to the high prevalence of DM and CVD. Therefore, it aimed to develop a comprehensive food composition table with GI and GL values of locally consumed foods in the UAE. These tables would serve as a great tool for nutrition therapy planning and dietary management for dietitians in the UAE and other GCC countries.

5.2 Results and Discussion

5.2.1 Proximate Analysis

Proximate analyses including fat, protein, ash, fiber and moisture content were measured according to previously described methods in Section 3.3.5.3. The proximate analyses data were expressed as mean \pm standard derivation on a fresh weight basis of each test food (Table 5.1). There were significant differences in the nutritional composition of the analyzed foods due to different ingredients and preparation methods (see Table 3.8 for main ingredients).

Moisture content ranged from 15.63g/100g in Muhalla bread to 86.66g/100g in Arseyah. Dishes containing meat (all main dishes) like Harees (beef), Thareed (beef), Thareed (chicken), Biryani (chicken), Machbous (fish), Arseyah (chicken), and Marqoqa (chicken) were found to have relatively high moisture content, ranging from 63.26g/100g to 86.66g/100g. Similar results of high moisture values in dishes containing meat have been reported by many other studies investigating food composition of traditional dishes in the Gulf Region [172, 175, 351, 352]. Musaiger *et. al.* (1998) [172], Habib *et. al.* (2011) [175], and Musaiger (2011) [351] reported similar moisture content for Harees: 80.2, 79.9 and 81, respectively. These values are only slightly higher than the 77.7g/100g of moisture in Harees presented in this study. Machbous was found to have 68.3g/100g of moisture, which is comparable to the 68.9g/100g reported by ElObeid *et. al.* (2015)[352], and to the 65.91g/100g described by Habib *et. al.* (2011) [175]. Breads had relatively low moisture content, ranging from 15.6g/100g for Muhalla bread to 35.7g/100g for Chebab bread. Regag bread had a moisture content of 21.9g/100g, which is lower than what ElObeid *et. al.* (2015) reported while investigating Kuwaiti traditional dishes (29.1g/100g) and much higher

than Bahraini and Emirati Regag bread reported in 2011(6.5 and 8.6g/100g, respectively) [175, 351]. The moisture content of Sago was 74.9g/100g. Previous studies reported varying moisture content in Sago, ranging from 39.8g/100g in Kuwaiti Sago [352], to 70.7g/100g in Bahraini Sago [351]. Moisture content of a dish resembles the amount of water available in it. Factors like recipe, cooking time, and cooking method could affect the water content of the same dish, thus explaining the differences found between studies.

Sago and Asida had the lowest protein content (0.80 and 0.99g/100g, respectively) since their main ingredients are sugar and water. Similarly, ElObeid *et. al.* (2015) [352] and Musaiger (2011) [351] found low protein content in Sago (1.9g/100g and 1.0g/100g, respectively) and Asida (1.7 and 3.5g/100g, respectively). However, the protein content was the highest in Chami (15.48g/100g). Chami is the Emirati cottage cheese; therefore, it is expected to have high protein content. Dishes containing meat like Biryani (chicken) and Thareed (beef) were also high in protein (11.55 and 7.04g/100g, respectively). Mugaiger (2011) [351] found 6.8g/100 of protein in Thareed (beef), and 8.1g/100g of protein in Biryani (beef). All six varieties of bread had relatively high protein content, because they either contain milk, egg, or both. The protein content of Regag bread in this study was 10.5g/100g. However, protein content in Bahraini Regag bread was reported to be 12.5g/100g [351], and protein content in Emirati Regag bread was 10.89g/100g [175]. Differences in protein content could result from the type of wheat flour used, as processed wheat flour contains less protein compared to whole grain wheat flour.

Fat content was the lowest in Asida (0.04g/100g), and the highest in Khanfaroosh (30.32g/100g). ElObeid *et. al.* (2015)[352] has also reported very low fat

content in Asida (0.08g/100g), although ghee was one of the ingredients of Asida in both studies.

The preparation of Khanfarooosh (Emirati doughnut) requires deep-frying of the dough in vegetable oil, which accounts for its high fat content. Lower fat content in Bahraini Khanfarooosh (10.1g/100g) was reported by Musaiger (2011)[351]. The difference in fat content could result from differences in cooking methods (deep frying) and recipe (addition of vegetable oil for the dough).

Fiber values varied from 12.20g/100g in Balalet to 0.12g/100g in Chami. Sago, a sweet dish consisting of Sago seeds and ghee, had 0.3g/100g of fiber. Similarly, Bahraini Sago was reported to have 0.5g/100g of fiber [351]. Other dessert dishes like Leqemat, Asida and Khanfarooosh had a fiber content of 1.45, 0.67, and 1.16/100g, respectively. However, fiber content in Leqemat, Asida and Khanfarooosh from Bahrain was 0.5, 2.0, and 0.9/100g, respectively [351]. Differences in fiber content could result from the type of wheat flour used (processed wheat flour or whole grain wheat flour), since whole-wheat flour is a better source of fiber.

Carbohydrate content was calculated by the difference of $\{CHO = 100 - (Protein + fat + ash + water)\}$. Breads were found to have the highest carbohydrate values, ranging from 68.20g in Muhalla bread to 47.08g in Chebab bread. Arabic bread and Regag bread had a carbohydrate content of 63.61 and 65.25g/100g, respectively. However, Musaiger (2011) [351] reported a carbohydrate content of 63.1 and 79.8g/100g in Bahraini Arabic bread and Regag bread, respectively.

Dessert dishes had lower carbohydrate content as compared to breads. Leqemat and Balalet had a carbohydrate content of 45.63 and 40.09g/100g. Slightly lower

values for carbohydrate content of Emirati Leqemat (42.13g/100g) and Balalet (33.76 g/100g) were reported by Habib *et. al.* (2011)[175].

In this study, the carbohydrate content of Harees (beef) was 13.30g/100g. Habib *et. al.* (2011)[175] found similar carbohydrates content in Emirati Harees (beef) (13.71g/100g), whereas Bahraini Harees had somewhat lower carbohydrate content (10.5g/100g) [351].

Khanfaroush provided the highest amount of energy per 100g (462.91 Kcal) due to its high fat content. Whereas, the energy provided by Arseyah was 57.32 Kcal/100g only because of its high water content. Breads provided similar energy values, ranging from 298.26 Kcal/100g in Chebab bread to 382.97Kcal/100g in Khameer bread.

In this study, Regag and Arabic breads provided 307.04, and 302.56 Kcal/100g, respectively. Lower amounts of energy were found in Bahraini Regag and Arabic breads (297 Kcal/100g) [351]. Whereas, Habib *et. al.* (2011)[175] reported much higher energy value for Emirati Regag bread (361.68 Kcal/100g).

Leqemat, a sweet dish similar to doughnut cake, had 416.49 Kcal of energy per 100g. However, a lower energy content was reported in Kuwaiti [173], Emirati [175], and Bahraini [351] Legemat (301, 339.06, and 275 Kcal/100g, respectively). The amount of energy provided by foods differs according to varying content of macronutrients (protein, fat, and carbohydrate).

Table 5.1: Proximate Analysis of United Arab Emirates Traditional Dishes

Test Food	Moisture	Protein	Fat	Ash	Fiber	CHO	Energy (Kcal)
Arabic bread	25.12 ± 0.34	9.45 ± 0.05	1.15 ± 0.04	0.68 ± 0.04	0.13 ± 0.01	63.61 ± 0.38	303 ± 1.14
Regag bread	21.93 ± 0.79	10.49 ± 0.07	0.46 ± 0.01	1.88 ± 0.02	1.23 ± 0.14	65.25 ± 0.73	307 ± 3.25
Chebab bread	35.70 ± 2.15	7.08 ± 0.35	9.07 ± 0.83	1.07 ± 0.07	1.20 ± 0.77	47.08 ± 1.51	298 ± 11.54
Muhalla Bread	15.63 ± 5.91	10.34 ± 0.82	4.24 ± 1.00	1.59 ± 0.27	0.54 ± 0.03	68.20 ± 4.78	352 ± 26.53
Khameer Bread	18.43 ± 6.42	10.45 ± 0.75	12.69 ± 2.13	1.69 ± 0.27	1.81 ± 0.31	56.74 ± 3.42	383 ± 35.07
Gurus	23.89 ± 1.84	8.76 ± 0.16	7.71 ± 0.90	1.07 ± 0.21	4.11 ± 0.73	58.56 ± 2.80	339 ± 5.80
Fendal	62.48 ± 0.20	1.86 ± 0.02	0.57 ± 0.01	0.55 ± 0.06	2.91 ± 0.05	34.54 ± 0.24	151 ± 0.95
Chami	77.25 ± 0.23	15.48 ± 0.28	0.66 ± 0.17	1.17 ± 0.18	0.12 ± 0.10	5.44 ± 0.49	90 ± 0.74
Harees, beef	77.70 ± 1.63	5.55 ± 0.38	2.43 ± 0.84	1.01 ± 0.17	5.56 ± 0.86	13.30 ± 0.92	97 ± 10.83
Thareed, beef	78.40 ± 0.55	7.04 ± 0.23	2.13 ± 0.08	0.31 ± 0.01	1.26 ± 0.19	12.12 ± 0.34	96 ± 2.56
Thareed, chicken	78.33 ± 3.38	5.37 ± 0.58	1.54 ± 0.81	1.01 ± 0.23	1.02 ± 0.36	13.75 ± 2.50	90 ± 16.37
Biryani, chicken	63.26 ± 1.34	11.55 ± 0.86	3.28 ± 0.42	1.26 ± 0.12	0.96 ± 0.45	20.65 ± 1.06	158 ± 3.72
Machbous, fish	68.26 ± 2.24	6.96 ± 1.56	1.98 ± 0.29	1.20 ± 0.12	3.60 ± 0.56	21.60 ± 0.45	132 ± 10.11
Arseyah	86.66 ± 0.34	2.24 ± 0.04	0.88 ± 0.10	0.11 ± 0.01	0.26 ± 0.01	10.11 ± 0.46	57 ± 0.95
Marqoqa	74.18 ± 1.66	5.89 ± 0.89	2.16 ± 1.24	1.20 ± 0.26	0.56 ± 0.13	16.57 ± 0.47	109 ± 12.93
Khabisa	24.91 ± 4.01	5.38 ± 0.87	10.54 ± 1.60	0.32 ± 0.08	2.72 ± 0.41	58.85 ± 4.56	352 ± 16.12
Leqemat	23.26 ± 1.72	7.29 ± 0.28	22.80 ± 2.09	1.02 ± 0.10	1.45 ± 0.34	45.63 ± 1.12	417 ± 16.87
Batheetha	16.40 ± 0.14	5.81 ± 0.04	9.54 ± 0.09	1.14 ± 0.07	6.12 ± 0.44	67.11 ± 0.03	378 ± 0.89
Khanfaroosh	21.57 ± 2.08	6.72 ± 0.20	30.32 ± 1.91	0.60 ± 0.10	1.16 ± 0.28	40.79 ± 0.77	463 ± 18.05
Sago	74.91 ± 2.69	0.80 ± 1.09	0.53 ± 0.17	0.04 ± 0.01	0.30 ± 0.11	23.73 ± 3.56	103 ± 11.56
Asida	77.17 ± 1.30	0.99 ± 0.05	0.04 ± 0.05	0.05 ± 0.01	0.67 ± 0.34	21.75 ± 1.25	91 ± 5.27
Habba Hamra	80.24 ± 1.60	1.23 ± 0.97	1.84 ± 1.31	0.48 ± 0.05	0.25 ± 0.06	16.21 ± 1.22	86 ± 12.70
Balalet	55.67 ± 3.50	2.40 ± 0.26	1.72 ± 0.58	0.12 ± 0.04	12.20 ± 1.31	40.09 ± 3.19	185 ± 15.89
<i>P</i> value*	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Data are expressed as g/100g on a fresh weight basis, (mean ± SD) *P < 0.05.

5.2.2 Minerals Analysis

Major minerals and trace metals (Ca, K, Na, Mg, P, Cu, Fe, Mn, and Zn) were simultaneously determined in locally-consumed Emirati foods by Inductively Coupled Plasma Optical Emission Spectrometer (ICP-OES), as previously described in section 3.3.5.5. The results of major mineral content and trace metal content in the fresh weight basis are presented in Table 5.2 and Table 5.3, respectively.

The calcium content was the highest in Chami (432.971mg/100 g) and the lowest in Sago (18.049mg/100g). Chami is a type of cottage cheese, which explains the high calcium content. Calcium value in Sago was also reported low by ElObeid *et. al.* (2015) [352] (13.91mg/100g) and Musaiger (2011)[351] (18mg/100g). Breads had relatively low calcium values, ranging from 26.345mg/100g in Qurus to 59.395mg/100g in Chebab bread. Chebab and Khameer breads had higher calcium contents when compared to other breads (59.395 and 47.831 mg/100g, respectively), due to the presence of milk powder in their ingredients list. Ali *et. al.*(2013) reported that the mean calcium intakes among Emirati children (6-10 years) and adolescents (11-18 years) were much lower than the estimated average requirement (EAR) values [353]. Consequently, low calcium intake could decrease the level of serum calcium, triggering the secretion of the parathyroid hormone, which partially results in bone resorption leading to a reduction in bone mass and osteoporosis [354]. Several studies showed the association between adequate intakes of calcium and vitamin D, and decreased risk of osteoporosis [354, 355]. Thus, dishes high in calcium like Chami are favorable especially for children and women.

The phosphorus content was high in all main dishes, because they all contained beef, chicken or fish meat. Thareed (beef) had the highest phosphorus content

(374.353mg/100g) among main dishes, followed by Harees (beef) (374.353mg/100g), and Thareed chicken (287.484mg/100g). The phosphorus content was the highest in Chami (761.187mg/100g) as it is a milk product. Moreover, it was very similar among different breads, as it ranged from 120.03mg/100g in Arabic bread to 148.839mg/100g in Khameer bread. This study found 120.03mg/100g of phosphorus in Arabic bread, and 156.958mg/100g in Regag bread. However, Musaiger (2011) [351] reported 100mg/100g of phosphorus in Arabic bread, and 231mg/100g in Regag bread. Conversely, Habib *et. al.* (2011)[175] reported 158.65mg/100g of phosphorus in Regag bread.

Healthy individuals have tightly regulated serum phosphorus levels, however, several studies proposed that high intakes of phosphorus could be associated with an increased risk of cardiovascular disease [356-358]. Moreover, high phosphorus intake with low calcium intake could have negative impact on calcium metabolism, poor bone density and development of osteoporosis [356, 358].

The highest sodium content was found in these main dishes: Arseyah (1569.877mg/100g), Thareed chicken (1413.947mg/100g), thareed (beef) (1320.167mg/100g), and Harees (beef) (1015.980mg/100g). Varied values of sodium were observed in the breads, ranging from 12.910mg/100g in Khameer bread to 355.100mg/100g in Regag bread. ElObeid *et. al.* [352] reported a sodium content of 707.40mg/100g in Regag bread, whereas Habib *et. al.* [175] found 574.21mg/100g of sodium in Regag bread. This variation might be due to different amounts of salt added to the bread dough during the preparation process. The lowest content of sodium was in Batheetha, with only 5.440mg/100g.

Most dishes containing meat and Regag bread presented in this study had very high sodium levels. Consuming a high-sodium diet was found associated with an increased risk of cardiovascular disease (CVD), hypertension, stroke, chronic kidney disease (CKD), heart failure, and stomach cancer [359]. The prevalence of CVD in the Middle East is increasing and becoming a major health problem [360]. According to the 2008 Emirati CVD risk assessment study, 28.4% of Emirati adults had a Framingham risk assessment score (a risk assessment tool for estimating a person's ten-year risk of developing CVD [361]) of more than 20%, and 20.8% of the studied population were found to be hypertensive [362]. Therefore, it is highly important to take serious steps nationally towards reducing the sodium intake in diets in order to lessen the risk of developing CVD, hypertension, and other associated complications.

The potassium content was high in main dishes like Thareed (beef) (670.667 mg/100g), Marqoqa (657.492 mg/100g), and Thareed (chicken) (610.488mg/100g). However, it was the highest in Fendal (1123.920mg/100g), which is expected as it is boiled sweet potato. Khameer bread had the highest content of potassium (215.488mg/100g) among breads, whereas other breads had very similar values, ranging from 100.256mg/100g in Arabic bread to 127.629mg/100g in Muhalla bread. Regag bread had potassium content of 128.672mg/100g. However, Habib *et. al.* (2011)[175] reported a higher potassium value in Regag bread (171.53mg/100g). The lowest potassium content was in Sago (5.816mg/100g); ElObeid *et. al.* (2015) [352] found 18.03mg/100g of potassium in Sago. The use of Sago planted in potassium depleted soil and the use of different cooking methods between countries could be the reason behind such variations.

Reducing sodium intake accompanied by increasing dietary potassium intake (low-sodium/high-potassium diet) could play an essential role in preventing and treating kidney disease, hypertension, stroke, and CVD [363, 364].

The highest magnesium content was found in Fendal (122.697mg/100g) and the lowest was in Sago (13.697mg/100g). Arabic bread, Biryani chicken, Arseyah, Legemat, Asida and Balaleet had similar magnesium concentrations (36.361, 32.286, 37.206, 33.608, 33.475, and 33.733 mg/100g, respectively). The results of this study were not in agreement with ElObeid *et. al.* (2015)[352], who reported the magnesium content of Asida, Thareed (beef), and Sago to be 71.67, 57.97 and 2.42mg/100g respectively. Additionally, Habib *et. al.* (2011) reported lower magnesium content in Regag bread, Qurus, Legemat, and Balalet 51.420, 34.2, 22.93, and 9.42 mg/100g, respectively. Furthermore, Al Nagdy *et. al.* [273], found 44 mg/100g of magnesium in Asida. Musaiger *et. al.* (1998) [172] found only 5.9 mg/100g of magnesium in the Omani Asida. While, Musaiger (2011) [351] reported 32 mg/100g of magnesium in the Bahraini Asida. Such differences could occur due to the use of different recipes in different gulf countries, as well as the use of fortified wheat flour in some countries.

Low serum magnesium level (hypomagnesemia) is associated with metabolic syndrome (MetS) [365, 366], T2DM [367, 368] and hypertension [369]. Accordingly, increasing magnesium intake could prevent high blood pressure [370] and improves the glycemic status of adults with prediabetes and hypomagnesaemia [371]. Moreover, higher serum magnesium levels were found associated with lower risk of MetS [366, 372]. Thus, consuming high magnesium foods like Fendal is recommended for the Emirati population.

The iron content was higher among breads compared to other dishes. It ranged from 9.61mg/100g in Regag bread to 3.11mg/100g in Qurus. ElObeid *et. al.* (2015) [352] found 7.36 mg/100g of iron in Kuwaiti Regag bread. However, Musaiger (2011) [351] found 1.8mg/100g of iron only in Bahraini Regag bread. This difference could be a result of using fortified wheat flour, or due to the use of date paste as an ingredient for the production of Rgag bread in some countries.

Moreover, among main dishes, Margooga and Thareed (beef) had the highest iron content (5.673mg/100g and 5.263mg/100g), followed by Thareed chicken (4.306mg/100g). Low iron content was found in Sago, Khabisa and Arseyah (0.518mg/100g, 0.599 mg/100g, and 0.711 mg/100g, respectively).

Aiming for high iron food sources (like Regag, Chebab, Muhalla, and Khameer bread) as part of the daily diet is recommended to reduce the risk of iron deficiency (anemia). Iron deficiency is considered one of the major public health problems in the Arab Gulf countries. Musaiger (2002) conducted a study in the Arab gulf countries and stated that iron deficiency prevalence among preschoolers ranged from 20% to 67%, and ranged from 12.6% to 50% among school age children. Moreover, the percentage of pregnant women with anemia ranged from 22.7% to 54%. In the UAE, the overall prevalence of anemia (Hb <12g/dL) among female college students attending the University of Sharjah was 26.7% [373]. Moreover, the prevalence of anemia, iron depletion and iron deficiency among Emirati preschoolers were estimated to be 36.1%, 26%, and 9.9%, respectively [374].

The highest zinc content was found in Thareed (beef) and Thareed (chicken) (4.690mg/100g and 4.870mg/100g), while Sago and Khabisa (0.119mg/100g and 0.378mg/100g) had the lowest concentrations of zinc. Breads had very similar zinc

contents, ranging from 1.033mg/100g in Arabic bread to 1.760mg/100g in Regag bread. Lower zinc content was reported by Habib *et. al.* (2011) [175] in Harees, Regag, Qurus, Legemat, and balaleet (0.630, 1.00, 0.77, 0.8, and 0.5mg/100g, respectively). A zinc rich diet is recommended to avoid zinc deficiency and related developmental abnormalities [375].

Lastly, the copper and manganese concentration was very low in all foods. Batheetha had the highest copper content (2.252mg/100g) because its main ingredient is date fruit, and Harees (beef) had the highest manganese value (1.697mg/100g) due to its whole-wheat content.

Table 5.2: Micronutrient (Major Elements) Composition of United Arab Emirates Traditional Dishes

Test Food	Ca	P	Na	K	Mg
Arabic Bread	23.128 ± 0.258	120.030 ± 0.878	163.866 ± 1.190	100.256 ± 0.831	36.361 ± 0.366
Regag Bread	31.526 ± 0.296	156.958 ± 0.030	355.100 ± 1.618	128.672 ± 0.563	54.819 ± 0.121
Chebab Bread	59.395 ± 0.255	143.556 ± 0.609	29.516 ± 0.304	133.194 ± 0.046	48.594 ± 0.483
Muhalla Bread	31.413 ± 0.294	125.495 ± 0.309	258.910 ± 1.532	127.629 ± 0.552	44.236 ± 0.334
Khameer Bread	47.831 ± 0.447	148.839 ± 0.571	12.910 ± 0.147	215.488 ± 0.531	61.460 ± 0.749
Qurus	26.345 ± 0.335	145.118 ± 0.842	343.263 ± 4.430	104.193 ± 0.837	50.187 ± 0.538
Fendal	92.019 ± 0.153	250.605 ± 0.093	185.567 ± 1.276	1123.920 ± 0.065	122.697 ± 0.265
Chami	432.971 ± 3.000	761.187 ± 3.291	661.612 ± 8.737	363.357 ± 3.126	44.450 ± 3.263
Harees, beef	307.293 ± 4.636	322.488 ± 4.038	1015.980 ± 1.365	229.026 ± 4.631	67.878 ± 4.700
Thareed, beef	164.897 ± 0.714	374.353 ± 0.616	1320.167 ± 3.758	670.667 ± 0.770	91.784 ± 0.462
Thareed, chicken	73.908 ± 0.246	287.484 ± 0.958	1413.947 ± 5.294	610.488 ± 0.468	76.364 ± 0.219
Biryani, chicken	87.003 ± 0.758	241.033 ± 0.313	711.897 ± 7.661	241.940 ± 0.047	32.286 ± 0.137
Machbous, fish	94.765 ± 0.352	251.137 ± 0.330	818.807 ± 1.688	338.951 ± 0.527	45.076 ± 0.666
Arseyah	123.126 ± 0.422	186.523 ± 0.649	1569.877 ± 10.597	154.863 ± 0.741	37.206 ± 0.355
Marqoqa	67.646 ± 0.391	218.739 ± 0.924	1513.897 ± 9.729	657.492 ± 0.790	62.527 ± 0.274
Khabisa	24.154 ± 0.266	56.100 ± 0.993	143.485 ± 2.912	65.303 ± 0.118	17.403 ± 0.196
Legemat	37.515 ± 0.055	124.039 ± 0.937	26.431 ± 0.204	113.173 ± 0.768	33.608 ± 0.421
Batheetha	63.539 ± 0.346	116.993 ± 0.826	5.440 ± 0.035	416.591 ± 0.625	65.605 ± 0.190
Khanfaroosh	24.135 ± 0.482	103.445 ± 0.274	55.498 ± 1.397	61.774 ± 0.971	24.332 ± 0.003
Sago	18.049 ± 0.102	2.998 ± 0.112	141.127 ± 1.332	5.816 ± 0.097	13.697 ± 0.061
Asida	37.980 ± 0.155	105.896 ± 0.474	161.374 ± 1.285	99.773 ± 0.459	33.475 ± 0.474
Habba Hamra	235.230 ± 1.256	159.128 ± 1.071	100.352 ± 0.693	196.556 ± 1.186	66.875 ± 1.610
Balaleet	29.176 ± 0.070	116.748 ± 0.248	120.472 ± 0.665	76.901 ± 0.622	33.733 ± 0.129

Data are expressed as mg/100 g on a fresh weight basis, (mean ± SD)

Table 5.3: Micronutrient (Trace Elements) Composition of United Arab Emirates Traditional Dishes

Test Food	Fe	Zn	Cu	Mn
Arabic Bread	4.815 ± 0.072	1.033 ± 0.049	0.206 ± 0.009	0.718 ± 0.003
Regag bread	9.613 ± 0.041	1.760 ± 0.024	0.302 ± 0.001	1.581 ± 0.003
Chebab Bread	8.479 ± 0.036	1.462 ± 0.041	0.241 ± 0.005	1.526 ± 0.003
Muhalla Bread	8.238 ± 0.060	1.343 ± 0.063	0.245 ± 0.010	1.221 ± 0.003
Khameer	8.212 ± 0.074	1.423 ± 0.028	0.306 ± 0.005	1.335 ± 0.003
Qurus	3.111 ± 0.026	1.401 ± 0.055	0.244 ± 0.002	1.133 ± 0.003
Fendal	2.399 ± 0.010	0.765 ± 0.037	0.462 ± 0.008	0.632 ± 0.003
Chammi	0.798 ± 0.008	2.184 ± 0.037	0.093 ± 0.009	0.024 ± 0.003
Harees, beef	2.142 ± 0.027	1.841 ± 0.019	0.279 ± 0.005	1.697 ± 0.003
Thareed, beef	5.263 ± 0.021	4.690 ± 0.040	0.311 ± 0.004	1.558 ± 0.003
Thareed, chicken	4.306 ± 0.023	4.870 ± 0.067	0.320 ± 0.008	1.195 ± 0.003
Biryani, chicken	1.253 ± 0.011	1.920 ± 0.043	0.215 ± 0.006	0.447 ± 0.003
Machbous, fish	1.742 ± 0.018	1.338 ± 0.038	0.211 ± 0.002	0.789 ± 0.003
Arseyah	0.711 ± 0.009	1.777 ± 0.062	0.189 ± 0.009	0.489 ± 0.003
Marqooqa	5.673 ± 0.021	2.077 ± 0.038	0.246 ± 0.004	0.666 ± 0.003
Khabisa	0.599 ± 0.006	0.378 ± 0.039	0.127 ± 0.004	0.501 ± 0.003
Legemat	3.019 ± 0.024	0.965 ± 0.024	0.161 ± 0.006	0.721 ± 0.003
Batheetha	6.311 ± 0.083	1.090 ± 0.033	2.252 ± 0.029	1.029 ± 0.003
Khanfarooosh	3.138 ± 0.052	0.996 ± 0.013	0.170 ± 0.005	0.706 ± 0.003
Sago	0.518 ± 0.002	0.119 ± 0.014	0.021 ± 0.009	0.095 ± 0.003
Asida	3.544 ± 0.043	1.004 ± 0.011	0.180 ± 0.003	0.940 ± 0.003
Habba Hamra	4.781 ± 0.027	1.434 ± 0.038	0.079 ± 0.003	0.307 ± 0.003
Balaleet	2.651 ± 0.011	1.134 ± 0.020	0.297 ± 0.004	0.744 ± 0.003

Data are expressed as mg/100 g on a fresh weight basis, (mean ± SD).

5.2.3 Vitamins Analysis

Seven water-soluble and all fat-soluble vitamins (Vitamin C, Thiamin, Riboflavin, Niacin, Vitamin B-6, Folate, Vitamin B-12, Vitamin A, Vitamin E (alpha-tocopherol), Vitamin D (D2 + D3), and Vitamin K (phylloquinone)) were simultaneously found in foods using the High-Performance Liquid Chromatography (HPLC) method, as described earlier in section 3.3.5.8. Data on the analysis of water-soluble and fat-soluble vitamins per 100g of fresh weight is presented in Table 5.4, and Table 5.5 respectively.

Vitamin C (total ascorbic acid) content was the highest in Biryani chicken (631.01mg/100g) and Arseyah (624.86mg/100g). However, no vitamin C was found in Arabic bread, Regag bread, Gurus and Asida. Musaiger (2011) [351] reported similar results for Arabic bread (0 mg/100g), and traces of vitamin C in Asida (0.6mg/100g). Breads usually do not contain vitamin C, yet Chebab bread, Khameer bread and Muhallah bread were found to have 247.82mg/100g, 245.71mg/100g, and 362.51mg/100g of vitamin C respectively, due to their milk content.

The concentration of thiamin in breads ranged from 1.40mg/100g in Regag bread to 7.83mg/100g in Gurus. This study reported 3.94 mg/100g of thiamin in Arabic bread, whereas lower thiamin content was found in Bahraini Arabic bread (0.1mg/100g) [351]. These differences could result from the use of fortified wheat flour in some recipes. Moreover, thiamin was found to be the highest in Sago (13.98mg/100g), and no thiamin was found in Fendal, Thareed (beef), Thareed (chicken) and Marqooqa.

As expected, dishes containing meat had relatively higher riboflavin content: Thareed (beef) (183.14mg/100g), Biryani chicken (76.51mg/100g), Margooga (64.20mg/100g), and Harees (beef) (56.89mg/100g). Whereas, Arabic bread, Regag bread, Muhallah bread, Khameer bread, Gurus, Chami, Khabisa, Batheetha, Khanfaroosh, Sago, Asida, and Balaleet did not contain riboflavin.

High niacin content was found in Thareed chicken (13.73mg/100g), Fendal (11.89 mg/100g) and Thareed (beef) (9.86mg/100g). However, no niacin was present in Khameer bread, Gurus, Chami, Biryani chickin, Machbous fish, Khabisa, Batheetha, Khanfaroosh, and Sago. The results of this study were not in agreement with Musaiger (2011) [351], who reported the niacin content of Asida, Batheetha, Harees, Khabisah, Khanfaroosh, Marqoqa, and Sago to be 1.2, 6.5, 1.9, 1.2, 1.0, 0.3 and 0.3 mg/100g respectively. The use of different ingredients and cooking methods could account for these differences between studies.

The highest concentration of vitamin B-6 was found in Batheetha (8.49mg/100g), followed by Thareed (beef) (5.70mg/100g) and Biryani chicken (4.06mg/100g). Vitamin B-6 was not present in Regag bread, Khameer bread, Khanfaroosh, Habba Hamra and Sago.

Habba Hamra, a red seed added in milk drink had the highest folic acid content (188.07mg/100g), followed by Thareed (beef) (13.59mg/100g) and Thareed Chicken (9.70mg/100g). Folate content was low in all breads, ranging from zero in Arabic and Regag bread, to 0.97mg/100g in Qurus. The results of this study were not in agreement with Musaiger (2011) [351], who reported much higher folic acid content in Thareed (26.7mg/100g). This could be due to the national flour fortification program adopted by the Kingdom of Bahrain in collaboration with the World Health Organization

(WHO) in 2001 to control and prevent anemia by fortifying flour with iron and folic acid [376].

All foods included in this study did not contain vitamin B-12 or vitamin A except for Habba Hamra, which had 0.64mg/100g of vitamin B-12, due to its milk content. Likewise, in Bahrain, B-12 was not present in Sago, Kabisah, Marqoqa, or batheetha [351]. Vitamin E concentration was the highest in Fendal (1.79mg/100g). However, Arabic bread (0.03mg/100g), Sago (0.05mg/100g) and Chami (0.08mg/100g) had the lowest vitamin E content. On the other hand, Bahraini Sago did not have vitamin E (0mg/100g), and Bahraini Asida, Marqoqa, and khanfarooash had slightly higher vitamin E content as compared to values reported in this study (0.7, 0.7, and 1.6 mg/100g vs. 0.31, 0.51, and 0.27 mg/100g, respectively) [351].

Some traditional foods did not contain vitamin D, such as Thareed (beef), Thareed (chicken), Gurus, Arseya, Marqoqa, Khabisa, Sago, and Asida. Similarly, Musaiger (2011) [351] reported 0 mg/100g of vitamin D in Sago and Marqoqa. Vitamin K was also low in all tested foods. It ranged from 0.01mg/100g in Margooga and Habba Hamra, to 0.11mg/100g in Thareed (beef) and Biryani (chicken).

The duration, temperature of cooking, food recipe composition, and storage conditions are all factors that substantially influence the stability and status of vitamins in foods [377]. The major micronutrient deficiencies reported in the UAE are iron deficiency anemia [373, 374] and vitamin D deficiency [378, 379]. Programs aiming to prevent and control these deficiencies could include: nutrition education; fortification of drinks with vitamin D [380]; flour fortification with iron and folic acid [376]; vitamin D and iron supplementation [381, 382]; and sensible sun exposure.

Table 5.4: Water-Soluble Vitamin Composition of United Arab Emirates Traditional Dishes

Test Food	Vitamin C	Thiamin	Riboflavin	Niacin	Vitamin B-6	Folate	Vitamin B-12
Arabic bread	0.00	3.94 ± 0.10	0.00	0.31 ± 0.06	0.73 ± 0.05	0.00	0.00
Regag bread	0.00	1.40 ± 0.16	0.00	0.11 ± 0.05	0.00	0.00	0.00
Chebab bread	247.82 ± 0.06	3.46 ± 0.03	28.21 ± 0.68	0.35 ± 0.06	0.46 ± 0.28	0.63 ± 0.10	0.00
Muhalla Bread	245.71 ± 0.53	2.04 ± 0.24	0.00	0.31 ± 0.01	2.27 ± 0.03	0.49 ± 0.20	0.00
Khameer Bread	362.51 ± 6.22	1.50 ± 0.02	0.00	0.00	0.00	0.67 ± 6.02	0.00
Gurus	0.00	7.83 ± 0.10	0.00	0.00	0.98 ± 0.03	0.97 ± 0.06	0.00
Fendal	351.28 ± 7.95	0.00	121.53 ± 0.34	11.89 ± 1.62	1.55 ± 0.16	5.28 ± 7.05	0.00
Chami	121.77 ± 1.68	0.33 ± 0.02	0.00	0.00	1.01 ± 0.72	0.25 ± 1.05	0.00
Harees, beef	591.50 ± 17.54	2.12 ± 0.42	56.89 ± 3.54	0.38 ± 0.04	2.63 ± 0.06	0.25 ± 17.03	0.00
Thareed, beef	453.91 ± 9.52	0.00	183.14 ± 4.55	9.86 ± 0.53	5.70 ± 0.44	13.59 ± 9.45	0.00
Thareed, chicken	636.92 ± 1.84	0.00	81.77 ± 6.60	13.73 ± 1.48	2.67 ± 0.47	9.70 ± 1.56	0.00
Biryani, chicken	631.01 ± 26.81	2.07 ± 0.56	76.51 ± 1.98	0.00	4.06 ± 0.17	2.14 ± 26.29	0.00
Machbous, fish	583.00 ± 0.18	2.02 ± 0.11	30.83 ± 1.11	0.00	1.92 ± 1.53	0.13 ± 0.01	0.00
Arseyah	624.86 ± 11.49	2.56 ± 0.31	36.90 ± 1.15	3.12 ± 1.15	2.60 ± 0.00	1.07 ± 11.08	0.00
Marqoqa	581.81 ± 26.16	0.00	64.20 ± 1.09	4.23 ± 1.89	1.11 ± 0.19	0.81 ± 26.09	0.00
Khabisa	339.24 ± 1.95	3.99 ± 0.08	0.00	0.00	1.28 ± 0.17	2.08 ± 1.12	0.00
Leqemat	322.43 ± 2.91	1.80 ± 0.14	15.65 ± 0.40	0.71 ± 0.08	0.74 ± 0.23	0.15 ± 2.02	0.00
Batheetha	244.09 ± 6.49	4.30 ± 0.26	0.00	0.00	8.49 ± 2.17	0.58 ± 6.00	0.00
Khanfaroosh	200.69 ± 8.33	1.17 ± 0.22	0.00	0.00	0.00	2.41 ± 8.07	0.00
Sago	133.34 ± 6.27	13.98 ± 1.21	0.00	0.00	0.00	0.13 ± 6.00	0.00
Asida	0.00	5.10 ± 0.27	0.00	0.11 ± 0.04	0.54 ± 0.11	1.38 ± 0.00	0.00
Habba Hamra	252.23 ± 21.24	8.52 ± 0.92	28.69 ± 1.41	0.01 ± 0.01	0.00	188.07 ± 21.84	0.64 ± 0.05
Balalet	289.65 ± 3.67	1.22 ± 0.07	0.00	1.17 ± 0.15	1.79 ± 0.37	0.82 ± 3.04	0.00

Data are expressed as mg/100 g on a fresh weight basis, (mean ± SD)

Table 5.5: Fat-Soluble Vitamin Composition of United Arab Emirates Traditional Dishes

Food	Vitamin A	Vitamin E	Vitamin D	Vitamin K
Arabic bread	0.00	0.03 ± 0.00	0.04 ± 0.00	0.04 ± 0.00
Regag bread	0.00	0.17 ± 0.00	0.04 ± 0.00	0.02 ± 0.00
Chebab bread	0.00	0.16 ± 0.00	0.03 ± 0.00	0.03 ± 0.00
Muhalla Bread	0.00	0.26 ± 0.00	0.03 ± 0.00	0.03 ± 0.00
Khameer Bread	0.00	0.67 ± 0.00	0.01 ± 0.00	0.08 ± 0.00
Gurus	0.00	0.41 ± 0.00	0.00	0.08 ± 0.00
Fendal	0.00	1.79 ± 0.00	0.02 ± 0.00	0.04 ± 0.00
Chami	0.00	0.08 ± 0.00	0.01 ± 0.00	0.02 ± 0.00
Harees, beef	0.00	0.09 ± 0.00	0.07 ± 0.01	0.04 ± 0.00
Thareed, beef	0.00	0.79 ± 0.00	0.00	0.11 ± 0.00
Thareed, chicken	0.00	0.77 ± 0.00	0.00	0.02 ± 0.00
Biryani, chicken	0.00	0.54 ± 0.12	0.04 ± 0.00	0.11 ± 0.12
Machbous, fish	0.00	0.44 ± 0.00	0.02 ± 0.00	0.01 ± 0.00
Arseyah	0.00	0.06 ± 0.00	0.00	0.02 ± 0.00
Marqoqa	0.00	0.51 ± 0.00	0.00	0.01 ± 0.00
Khabisa	0.00	0.20 ± 0.00	0.00	0.04 ± 0.00
Leqemat	0.00	0.11 ± 0.00	0.06 ± 0.00	0.04 ± 0.00
Batheetha	0.00	0.50 ± 0.00	0.02 ± 0.00	0.04 ± 0.00
Khanfaroosh	0.00	0.27 ± 0.00	0.02 ± 0.00	0.05 ± 0.00
Sago	0.00	0.05 ± 0.00	0.00	0.00
Asida	0.00	0.31 ± 0.00	0.00	0.06 ± 0.00
Habba Hamra	0.00	0.13 ± 0.00	0.01 ± 0.00	0.01 ± 0.00
Balalet	0.00	0.11 ± 0.00	0.10 ± 0.03	0.06 ± 0.00

Data are expressed as mg/100 g on a fresh weight basis, (mean ± SD).

5.2.4 Lipids Analysis

Lipid analysis, including fatty acids and cholesterol, were found in Emirati foods using the Gas Chromatograph (GC) and High-Performance Liquid Chromatography (HPLC) respectively, as previously described in section 3.3.5.7. Data on the analysis of fatty acids (saturated, monounsaturated, polyunsaturated, and trans fatty acids) and cholesterol is presented in Table 5.6.

Khanfarooosh and Legemat had the highest content of saturated fatty acids (13.520g/100g and 9.586g/100g, respectively). This could be a result of the deep-frying preparation method used in these sweet dishes. Among breads, Chebab bread had the highest saturated fatty acids content (6.178g/100g) followed by Khameer bread (5.633g/100g), and Gurus (4.373g/100g). Excessive dietary fat consumption, especially saturated fat, has been linked to an increase in many health risks such as obesity, coronary heart disease (CHD), and certain types of cancer [383]. The 2015 USDA's dietary guidelines recommends limiting intake of saturated fats to less than 10% of energy intake, to acquire optimal cardiovascular health [384]. Moreover, the WHO dietary guidelines for the Eastern Mediterranean Region (2012) recommends reducing saturated fats and trans fats to <10% and < 1% of total energy intake, respectively, and replacing both types of fat with unsaturated fats [385].

Monounsaturated fatty acid content ranged from 0g/100g in Asida and Regag bread to 4.202g/100g in Legemat. Whereas, polyunsaturated fatty acids content ranged from 0.018g/100g in Asida to 15.99g/100g in Khanfarooosh. The USDA and the WHO emphasize on replacing saturated fatty acids - usually found in red meat, processed meats, and fried foods - with healthier monounsaturated and polyunsaturated fatty acid-rich foods, including fatty fish (salmon, tuna, and sardines) and nuts. These

should be combined with healthy sources of carbohydrates (fiber-rich whole-grain sources), and adequate consumption of fruits and vegetables [384, 385].

Most foods studied did not contain trans fatty acids such as Regag bread, Chebab bread, Muhallah bread, Khameer bread, Harees (beef), and Thareed (beef). However, Khabisa contained the highest trans fatty acids content (1.731g/100g). Observational studies and controlled trials provide evidence that trans fatty acid intake from partially hydrogenated oils adversely affects several cardiovascular risk factors and contributes significantly to increased risk of CHD events [386].

Cholesterol content ranged from 1.10mg/100g in Regag bread and Legemat, to 27.25mg/100g in Thareed chicken. Among breads, Khameer had the highest cholesterol content (9.70mg/100g), followed by Chebab bread (6.25mg/100g) and Muhallah bread (4.60mg/100g). These breads contain eggs, and according to Schärer and Schulthess [387], egg yolk has the highest content of cholesterol detected in different kinds of food.

Table 5.6: Lipid profile of United Arab Emirates Traditional Dishes

Test Food	Fatty acids, total saturated (g)	Fatty acids, total monounsaturated (g)	Fatty acids, polyunsaturated (g)	Trans Fatty acids (g)	Cholesterol (mg)
Arabic bread	0.574 ± 0.130	0.027 ± 0.013	0.536 ± 0.143	0.013 ± 0.000	1.40 ± 0.14
Regag bread	0.116 ± 0.014	0.000 ± 0.000	0.316 ± 0.021	0.00	1.10 ± 0.00
Chebab bread	6.178 ± 0.085	0.199 ± 0.029	2.693 ± 0.114	0.00	6.25 ± 0.07
Muhalla Bread	2.741 ± 0.264	0.147 ± 0.074	1.352 ± 0.189	0.00	4.60 ± 0.00
Khameer Bread	5.633 ± 0.187	0.401 ± 0.152	6.656 ± 0.035	0.00	9.70 ± 0.00
Gurus	4.373 ± 0.352	0.095 ± 0.049	3.197 ± 0.238	0.046 ± 0.065	1.70 ± 0.14
Fendal	0.322 ± 0.013	0.010 ± 0.009	0.222 ± 0.009	0.016 ± 0.004	1.60 ± 0.00
Chami	0.395 ± 0.013	0.010 ± 0.002	0.225 ± 0.027	0.030 ± 0.042	6.40 ± 0.00
Harees, beef	0.856 ± 0.011	0.066 ± 0.002	1.415 ± 0.019	0.00	11.60 ± 0.00
Thareed, beef	0.826 ± 0.022	0.052 ± 0.012	1.169 ± 0.011	0.00	15.75 ± 0.07
Thareed, chicken	0.656 ± 0.005	0.043 ± 0.001	0.744 ± 0.006	0.008 ± 0.011	27.25 ± 0.07
Biryani, chicken	1.797 ± 0.326	0.097 ± 0.029	1.365 ± 0.268	0.021 ± 0.029	26.25 ± 0.07
Machbous, fish	0.861 ± 0.003	0.062 ± 0.003	1.042 ± 0.016	0.015 ± 0.022	14.15 ± 0.07
Arseyah	0.234 ± 0.007	0.119 ± 0.016	0.365 ± 0.013	0.129 ± 0.008	14.05 ± 0.07
Marqoqa	0.840 ± 0.008	0.054 ± 0.001	1.170 ± 0.006	0.00	22.30 ± 0.14
Khabisa	3.226 ± 0.178	1.640 ± 0.049	3.795 ± 0.033	1.731 ± 0.111	1.15 ± 0.07
Leqemat	9.586 ± 0.439	4.202 ± 0.131	7.989 ± 0.154	0.00	1.10 ± 0.00
Batheetha	3.758 ± 0.022	0.093 ± 0.005	5.052 ± 0.030	0.157 ± 0.047	6.55 ± 0.49
Khanfarooth	13.520 ± 0.021	0.178 ± 0.007	15.990 ± 0.095	0.725 ± 0.021	3.50 ± 0.28
Sago	0.338 ± 0.036	0.005 ± 0.003	0.147 ± 0.018	0.041 ± 0.057	2.95 ± 0.07
Asida	0.020 ± 0.000	0.000 ± 0.000	0.018 ± 0.001	0.00	1.30 ± 0.00
Habba Hamra	0.538 ± 0.029	0.170 ± 0.018	1.127 ± 0.054	0.005 ± 0.007	5.85 ± 0.07
Balalet	0.739 ± 0.008	0.00	0.913 ± 0.008	0.019 ± 0.001	1.90 ± 0.00

Data are expressed (Unit/100g) on a fresh weight basis, (mean ± SD).

5.2.5 Sugars Analysis

Various types of sugars including monosaccharides (glucose, and fructose), disaccharides (sucrose, maltose, and lactose), and trisaccharide (raffinose) were found in foods using the High-Performance Liquid Chromatography (HPLC) method, as previously described in section 3.3.5.6. Data on the analysis of sugar is presented in Table 5.7.

Khameer and Muhalla bread had the highest content of fructose, glucose and total sugars among breads; this might be due to sugar added during preparation or the use of date paste. However, fructose and glucose were not present in Chami and Marqoqa. Recent human studies suggest that high dietary fructose levels might elevate serum triglyceride levels and cause other metabolic diseases, dyslipidemia, non-alcoholic fatty liver, obesity and weight gain [388-390].

The highest content of sucrose was found in Sago (3.046g/100g) and Gurus (3.005g/100g), which could be explained by the high amount of sugar in the recipe.

Most foods did not contain maltose and raffinose sugars, such as Arabic bread, Harees (beef), Marqoqa, Leqemat, Sago, and Balalet. However, Gurus contained the highest maltose content (0.373g/100g) and Habba Hamra had the highest content of raffinose (0.261g/100g).

Lactose content was the highest in Fendal (1.091g/100g), followed by Chami (0.899g/100g). However, Gurus, Thareed (Chicken), Biryani (Chicken), Machbous (Fish), Marqoqa, and Batheetha did not contain lactose.

Total sugars ranged from 0.009g/100g in Marqoqa to 5.018g/100g in Batheetha. Dessert dishes like Batheetha, Sago, and Habba Hamra had higher amounts of total sugars compared to other foods.

It is important to monitor the total amount of added sugar in the diet. According to the 2015 World Health Organization's (WHO) guidelines on sugar intake [391], adults and children are recommended to reduce their daily intake of added sugars to less than 10% of their total energy intake. Welsh et al. (2011) [392] suggested that an additional reduction to below 5% of the total energy intake might provide added health benefits.

Table 5.7: Sugar Composition of United Arab Emirates Traditional Dishes

Test Food	Fructose	Glucose	Sucrose	Maltose	Lactose	Raffinose	Total, Sugars
Arabic bread	0.387 ± 0.005	0.258 ± 0.008	0.000	0.000	0.257 ± 0.014	0.000	0.902 ± 0.027
Regag bread	0.009 ± 0.001	0.017 ± 0.001	0.039 ± 0.007	0.030 ± 0.001	0.162 ± 0.012	0.000	0.257 ± 0.018
Chebab bread	1.263 ± 0.002	0.769 ± 0.004	0.000	0.021 ± 0.008	0.450 ± 0.019	0.000	2.503 ± 0.005
Muhalla Bread	1.336 ± 0.001	1.214 ± 0.004	0.009 ± 0.002	0.014 ± 0.005	0.302 ± 0.012	0.022 ± 0.002	2.897 ± 0.007
Khameer Bread	1.685 ± 0.001	1.333 ± 0.006	0.000	0.010 ± 0.010	0.264 ± 0.010	0.000	3.291 ± 1.006
Gurus	0.045 ± 0.002	0.040 ± 0.000	3.005 ± 0.003	0.373 ± 0.009	0.000	0.050 ± 0.070	3.512 ± 0.068
Fendal	0.042 ± 0.001	0.085 ± 0.000	0.139 ± 0.003	0.080 ± 0.008	1.091 ± 0.007	0.126 ± 0.009	1.562 ± 0.009
Chami	0.000	0.000	0.000	0.010 ± 0.010	0.899 ± 0.059	0.000	0.909 ± 0.069
Harees, beef	0.012 ± 0.002	0.047 ± 0.001	0.10 ± 0.001	0.000	0.297 ± 0.019	0.000	0.366 ± 0.022
Thareed, beef	0.017 ± 0.001	0.000	0.012 ± 0.001	0.015 ± 0.001	0.059 ± 0.005	0.022 ± 0.014	0.124 ± 0.019
Thareed, chicken	0.011 ± 0.002	0.012 ± 0.001	0.024 ± 0.001	0.018 ± 0.003	0.000	0.000	0.064 ± 0.000
Biryani, chicken	0.015 ± 0.006	0.007 ± 0.001	0.024 ± 0.002	0.004 ± 0.004	0.000	0.000	0.050 ± 0.001
Machbous, fish	0.017 ± 0.002	0.012 ± 0.001	0.028 ± 0.005	0.005 ± 0.002	0.000	0.000	0.062 ± 0.006
Arseyah	0.000	0.005 ± 0.002	0.011 ± 0.001	0.006 ± 0.007	0.187 ± 0.018	0.000	0.208 ± 0.029
Marqoqa	0.000	0.000	0.009 ± 0.000	0.000	0.000	0.000	0.009 ± 0.000
Khabisa	0.094 ± 0.002	1.285 ± 0.005	0.086 ± 0.000	0.001 ± 0.000	0.053 ± 0.000	0.000	1.518 ± 0.002
Leqemat	1.240 ± 0.006	1.168 ± 0.004	0.053 ± 0.002	0.000	0.396 ± 0.005	0.000	2.856 ± 0.000
Batheetha	1.309 ± 0.015	1.181 ± 0.000	2.468 ± 0.002	0.000	0.000	0.060 ± 1.001	5.018 ± 0.016
Khanfaroosh	0.912 ± 0.007	0.771 ± 0.006	0.000	0.019 ± 0.001	0.299 ± 0.005	0.000	2.001 ± 0.010
Sago	0.294 ± 0.001	1.576 ± 0.004	3.046 ± 0.015	0.000	0.095 ± 0.006	0.000	5.010 ± 0.015
Asida	0.227 ± 0.002	0.847 ± 0.004	2.621 ± 0.020	0.040 ± 0.000	0.063 ± 0.011	0.045 ± 0.064	3.842 ± 0.027
Habba Hamra	0.561 ± 0.001	1.192 ± 0.007	1.808 ± 0.026	0.018 ± 0.001	0.397 ± 0.006	0.261 ± 0.005	4.237 ± 0.046
Balalet	0.260 ± 0.004	1.688 ± 0.018	0.276 ± 0.001	0.000	0.154 ± 0.006	0.000	2.377 ± 0.017

Data are expressed as g/100g on a fresh weight basis, (mean ± SD).

5.2.6 Characteristics of Study Population

One hundred and twelve normal weight, healthy female students and staff participants were recruited from United Arab Emirates University (UAEU) to voluntarily participate in this study, as described earlier in section 3.3.1. The physical characteristics of the population of this study are presented as mean \pm standard deviation, in Table 5.8. The mean age of the study population was 22.8 ± 5.01 years old, the mean weight was 58.26 ± 8.25 kg, and the mean body mass index (BMI) was 22.86 ± 3.57 kg/m². Other physical characteristics such as waist circumference, fat mass and fat-free mass are also reported in Table 5.8.

Table 5.8: Physical Characteristics of the Study Population

Characteristics	Mean \pm SD, (n=112)
Age (years)	22.8 ± 5.01
Height (m)	1.60 ± 0.06
Weight (kg)	58.26 ± 8.25
Body Mass Index (kg/m²)	22.86 ± 3.57
Waist Circumference (cm)	74.12 ± 14.31
Fat Mass (%)	29.00 ± 5.60
Fat Mass (kg)	17.57 ± 6.27
Fat-Free Mass (%)	65.54 ± 13.47
Fat-Free Mass (kg)	44.27 ± 12.11
Fasting Plasma Glucose (mg/dL)	88.32 ± 7.82

5.2.7 Glycemic Response

Twenty-three traditional foods commonly consumed in the United Arab Emirates (UAE) were chosen to investigate their glycemic index and glycemic load, according to the previously described methodology in section 3.3.6. The mean incremental area under the glycemic response curves of the standard food and each test food are presented in Figures A to W of Appendix 11.

The mean incremental areas under the glycemic response curves (IAUCs) for low glycemic index traditional meals (Chebab bread, Khameer bread, Harees (beef), Biryani (chicken), Leqemat, Khanfaroosh, and Habba Hamra) are presented in Figure 5.1. Biryani (chicken) showed the highest increase in glycemic response compared with other low GI foods, whereas, Harees and Khanfashoosh produced a slower and lower raise in the glycemic response over 120 minutes. Differences in glucose response between the meals were calculated using the Kruskal-Wallis test. The incremental increase in blood glucose at 15 min was significantly different between Chebab bread, Khanfaroosh, and Habba Hamra ($P = 0.009$, median = 0.90, 0.20, and 0.80 respectively). At 30 min the incremental increase in blood glucose was significantly different between Chebab bread, Khameer bread, Harees (beef), Biryani (chicken), Leqemat, Khanfaroosh, and Habba Hamra ($P = 0.008$, median = 1.60, 0.90, 1.10, 2.00, 1.10, 0.50, and 2.10 respectively). At 45 min the significant differences in incremental blood glucose were between Chebab bread, Harees (beef), Biryani (chicken), Leqemat, Khanfaroosh, and Habba Hamra ($P = 0.006$, median = 1.10, 1.40, 2.40, 1.10, 1.20, and 1.50 respectively). At 60 min the significant differences in incremental blood glucose were between Harees (beef), Biryani (chicken), Leqemat, Khanfaroosh, and Habba Hamra ($P = 0.02$, median = 1.00, 1.50, 1.30, 0.90 and 0.50

respectively). At 90 min the significant differences in incremental blood glucose were between Chebab bread, Leqemat, and Habba Hamra ($P = 0.015$, median = 1.10, 1.10, and 0.40 respectively).

Several factors may alter the GI of food (Section 2.5.3), including the presence of macronutrients such as fat and protein, the type of starch, processing, and the addition of acids, sugars, gelling fibers, or amylase inhibitors [196].

The high protein content in Biryani (11.55g/100g) along with added vegetables (onion, garlic and pepper) could explain its low GI [393]. Likewise, the addition of vegetable mixed curry showed considerable reduction of GI and GL of traditional Sri Lankan breakfast foods [394]. The high amount of dietary fibers present in Harees (5.56g/100g) may cause its low GI, because the presence of dietary fibers in test foods may delay its glycemic response, as it contributes to slower nutrient absorption and a delayed transit time in the small intestines [193, 395].

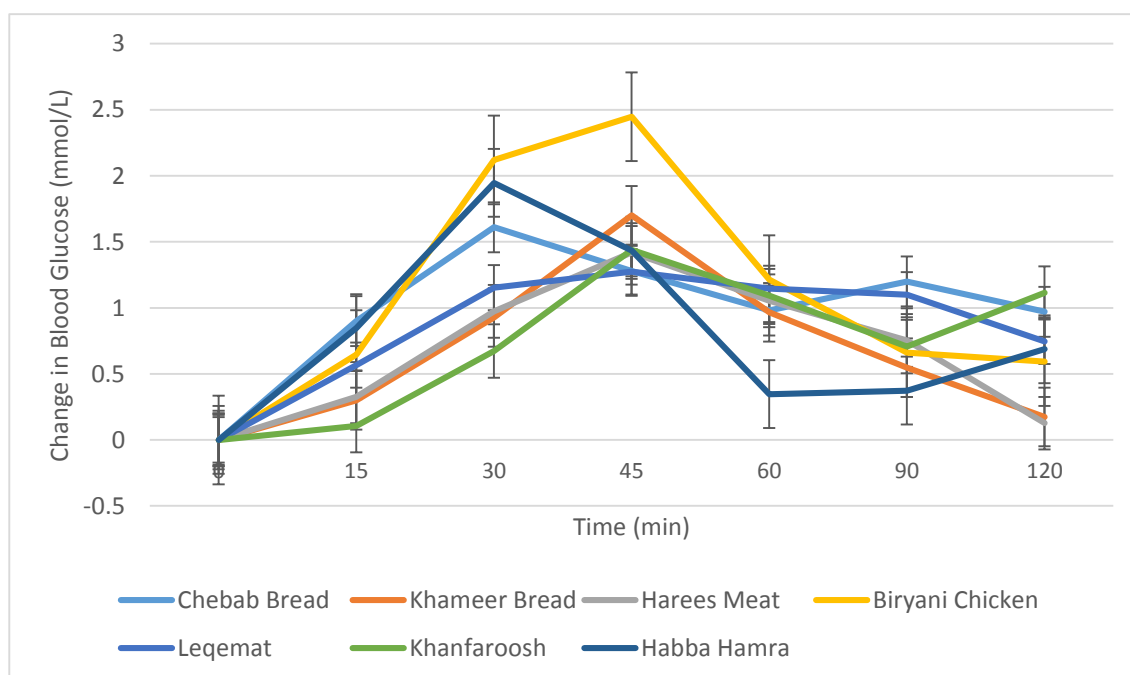


Figure 5.1: The incremental area under the blood glucose curves (IAUC) for low glycemic index traditional meals. Standard errors of the mean values are represented by vertical bars

The IAUCs value for low GI test foods is presented in Table 5.9 as mean \pm standard error. The table illustrates that the IAUC for all seven low GI test foods are significantly different from the IAUCs of the reference food ($P \leq 0.002$). Among low GI test foods, Harees and Khanfarosh had the smallest IAUC (88.08 and 93.39, respectively). However, Biryani (chicken) had the highest IAUC (138.13 ± 10.64).

Table 5.9: Incremental Area Under the Curve (IAUC) for low GI test foods

Test Foods	IAUC \pm SE	P-Value
Reference Food	267.96 \pm 32.86	0.002
Chebab Bread	134.83 \pm 18.08	
Reference Food	215.56 \pm 16.19	<0.001
Khameer Bread	93.51 \pm 5.65	
Reference Food	215.61 \pm 14.55	<0.001
Harees	88.08 \pm 4.67	
Reference Food	285.57 \pm 26.68	<0.001
Biryani, chicken	138.13 \pm 10.64	
Reference Food	285.67 \pm 26.68	<0.001
Leqemat	117.38 \pm 8.85	
Reference Food	215.56 \pm 16.19	<0.001
Khanfarosh	93.39 \pm 7.34	
Reference Food	215.56 \pm 16.19	<0.001
Habba Hamra	97.61 \pm 5.94	

The mean IAUCs of medium glycemic index traditional meals (Arabic bread, Chami, Machbous (Fish), Khabisah, Batheetha, and Balalet) are shown in Figure 5.2. Differences in glucose response between the meals were calculated using the Kruskal-Wallis test. The incremental increase in blood glucose at 30 min was significantly different between Arabic bread, Chami, Machbous (Fish), Khabisah, Batheetha, and Balalet ($P = 0.029$, median = 1.00, 0.16, 1.50, 1.60, 1.75, and 1.70 respectively). At 45 min the incremental increase in blood glucose was significantly different between Arabic bread, Chami, Machbous (Fish), Khabisah, Batheetha, and Balalet ($P = 0.039$, median = 1.40, 0.16, 1.75, 2.10, 1.60, and 1.80 respectively). At 60 min the significant

differences in incremental blood glucose were between Arabic bread, Chami, Machbous (Fish), Khabisah, Batheetha, and Balalet ($P = 0.034$, median = 1.70, 0.13, 1.00, 1.90, 1.10, and 1.60 respectively). At 90 min the significant differences in incremental blood glucose were between Arabic bread, Chami, Machbous (Fish), Khabisah, Batheetha, and Balalet ($P = 0.016$, median = 1.40, 0.19, 0.45, 1.20, 0.70, and 0.80 respectively). At 120 min the significant differences in incremental blood glucose were between Arabic bread, Chami, Machbous (Fish), Khabisah, Batheetha, and Balalet ($P = 0.006$, median = 1.00, 0.80, 0.20, 0.90, 0.20, and 0.10 respectively). Chami created a slow and low rise in the glycemic response over 120 minutes, due to its very low carbohydrate content (5.44g/100g), which was expected as it is a dairy product [187].

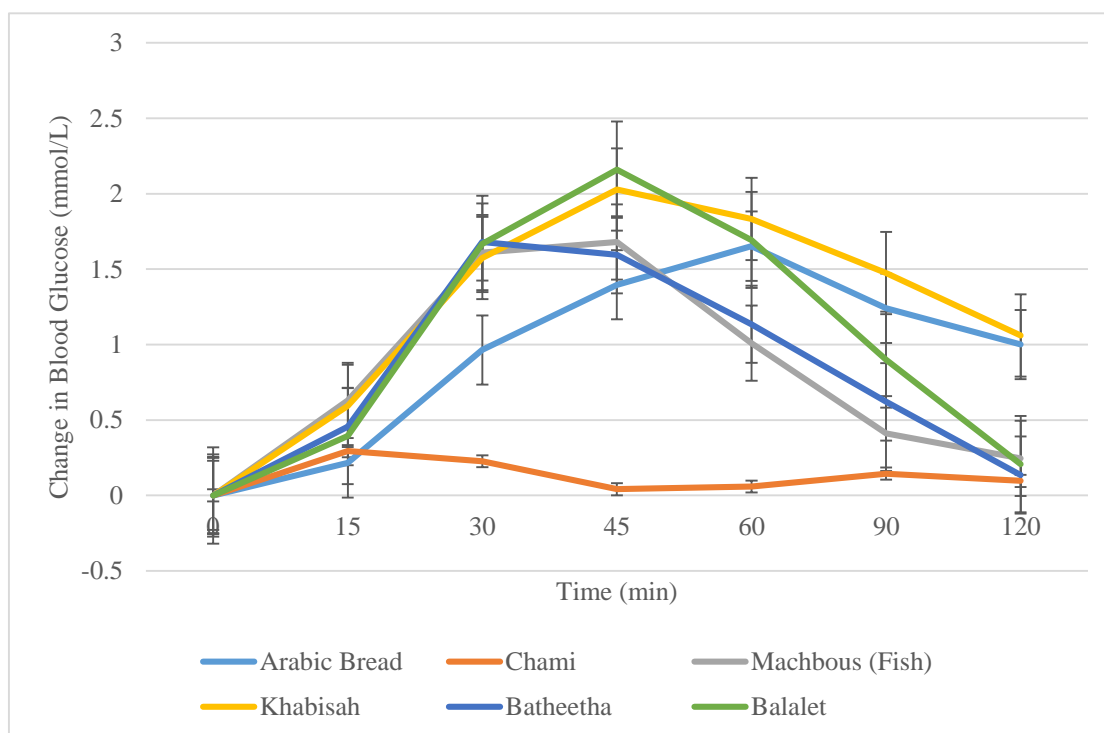


Figure 5.2: The incremental area under the blood glucose curves (IAUC) for medium glycemic index traditional meals. Standard errors of the mean values are represented by vertical bars

Table 5.10 shows the IAUC for medium GI foods. The IAUC for all medium GI test foods showed a significant difference compared to the IAUC of the reference food ($P < 0.001$), except for Chami ($P=0.739$). Khabisah had the highest IAUC value (IAUC \pm SE: 164.23 ± 13.58), while Chami reported the lowest IAUC value (IAUC \pm SE: 76.23 ± 8.36) among medium GI foods.

Table 5.10: Incremental Area Under the Curve (IAUC) for medium GI test foods

Test Foods	IAUC \pm SE	P-Value
Reference Food	224.77 ± 11.61	<0.001
Arabic Bread	145.33 ± 11.14	
Reference Food *	77.9313 ± 5.49	0.739
Chami	76.23 ± 8.36	
Reference Food	186.37 ± 10.41	<0.001
Machbous, fish	103.47 ± 7.99	
Reference Food	250.01 ± 16.87	<0.001
Khabisa	164.23 ± 13.58	
Reference Food	186.3650 ± 10.41	<0.001
Batheetha	107.93 ± 7.76	
Reference Food	215.61 ± 14.55	<0.001
Balalet	132.82 ± 13.34	

*(25g glucose)

The mean IAUCs of high glycemic index traditional meals (Regag bread, Muhalla bread, Qurus, Fendal, Thareed (beef), Thareed (chicken), Arseyah, Marqoqa, Sago, and Asida) are presented in Figure 5.3. Muhalla bread, Asida and Sago produced the highest increase in glycemic response, compared with other high GI foods. Whereas, Thareed (beef) showed the highest peak (at 45 minutes) among high GI foods. Regag bread showed a slow and low rise in glycemic response during the first 15 minutes, and then a sharp increase between 15 to 60 minutes. Differences in glucose response between the meals were calculated using the Kruskal-Wallis test. The incremental increase in blood glucose at 15 min was significantly different between Regag bread, Fendal, Thareed (beef), Sago, and Asida ($P = 0.025$, median = 0.20, 0.65,

0.90, 0.90, and 0.90 respectively). At 30 min the incremental increase in blood glucose was significantly different between Regag bread, Qurus, Thareed (beef), Sago, and Asida ($P = 0.006$, median = 1.10, 1.20, 2.30, 2.60, and 2.70 respectively). At 90 min the significant differences in incremental blood glucose were between Regag bread, Muhalla bread, Fendal, and Thareed (beef) ($P = 0.01$, median = 1.50, 1.30, 0.20, and 1.30 respectively).

At 120 min the significant differences in incremental blood glucose were between Regag bread, Muhalla bread, Qurus, Fendal, Thareed (beef), Arseyah, and Asida ($P = 0.04$, median = 1.10, 0.90, 1.00, 0.00, 0.90, 1.10, and 0.10 respectively). Sago had the highest incremental blood glucose response at 15 and 30 min compared to other high GI foods. The high moisture content (humidity) of Sago (74.91g/100g), along with high cooking temperatures and prolonged cooking durations (time) could have increased starch gelatinization and digestibility degree, thus, resulting in high glycemic responses for this traditional desserts [396, 397].

The IAUC's values for high GI test foods are presented in Table 5.11 as mean \pm standard error. The table demonstrates that the IAUC for all high GI test foods are significantly different from the IAUCs of the reference food ($P \leq 0.001$), except for Marqoqa, Sago, and Asida ($P = 0.074$, $P = 0.654$, and $P = 0.745$, respectively). Among high GI test foods, Fendal had the smallest IAUC (IAUC \pm SE: 123.95 \pm 12.02), and Muhalla bread showed the highest IAUC (IAUC \pm SE: 198.78 \pm 12.39).

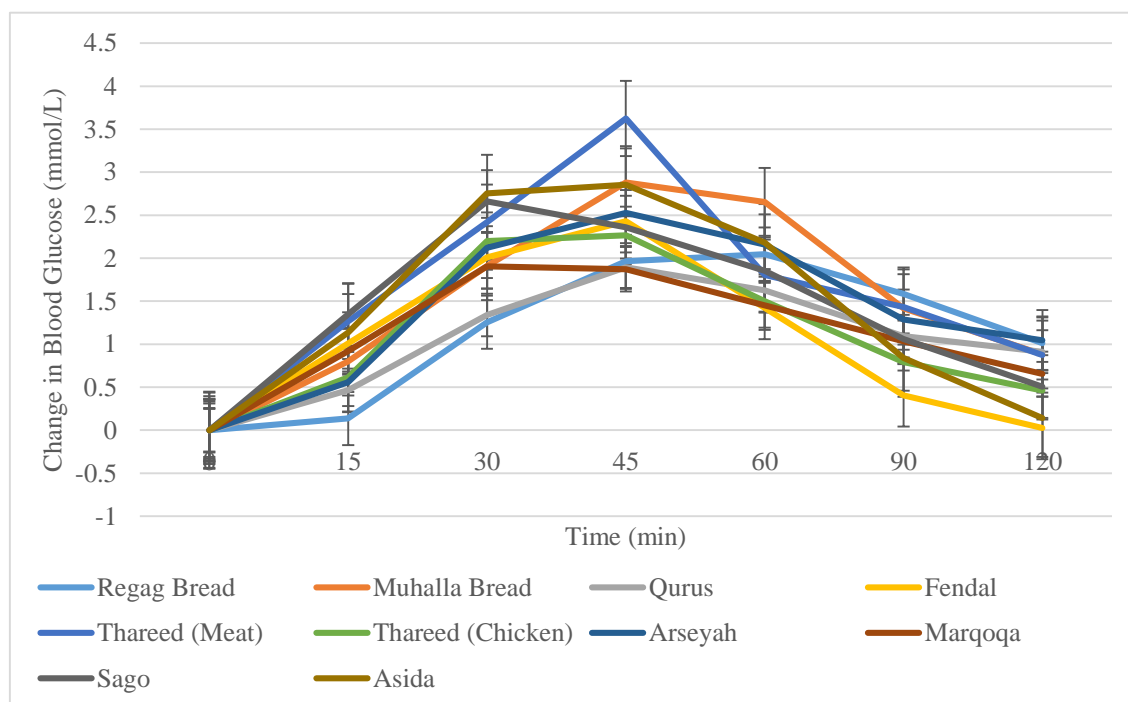


Figure 5.3: The incremental area under the blood glucose curves (IAUC) for the high glycemic index traditional meals. Standard errors of the mean values are represented by vertical bars

Table 5.11: Incremental Area Under the Curve (IAUC) for high GI test foods

Test Foods	IAUC \pm SE	P-Value
Reference Food	224.77 \pm 11.61	0.001
Regag Bread	162.98 \pm 14.88	
Reference Food	261.9 \pm 20.57	<0.001
Muhalla Bread	198.78 \pm 12.39	
Reference Food	202.58 \pm 8.92	0.001
Qurus	143.65 \pm 14.5	
Reference Food	186.37 \pm 10.41	0.001
Fendal	123.95 \pm 12.02	
Reference Food	250.01 \pm 16.87	<0.001
Thareed, beef	186 \pm 55.45	
Reference Food	202.8 \pm 8.92	<0.001
Thareed, chicken	142.41 \pm 8.36	
Reference Food	250.01 \pm 16.87	<0.001
Arseyah	176.5 \pm 10.77	
Reference Food	202.58 \pm 8.92	0.074
Marqoqa	162.65 \pm 19.17	
Reference Food	202.58 \pm 8.92	0.654
Sago	194.17 \pm 14.95	
Reference Food	202.58 \pm 8.92	0.745
Asida	197.38 \pm 15.02	

5.2.8 Glycemic Index and Glycemic Load Classifications

The GI and GL values for all tested foods are presented in Table 5.12. GI values are given as means with their standard errors. GI calculation was performed according to the previously described procedure in Section 3.3.6.1. For practicality, GI and GL values are usually grouped into categories of low, medium or high. If the GI of test food was ≤ 55 , then it is labeled as low GI food, between 56 and 69 (inclusive) is medium GI food, and ≥ 70 is high GI food [398].

The four groups of food included in this study (breads, entrée, main, and dessert) produced a wide range of GI values, with seven foods producing low GI (Chebab bread, Khameer bread, Harees (beef), Biryani (chicken), Leqemat, Khanfaroosh, and Habba Hamra), six foods were classified as medium GI (Arabic bread, Chami, Machbous (fish), Khabisah, Batheetha, and Balalet), and ten foods had high GI (Regag bread, Muhalla bread, Qurus, Fendal, Thareed (beef), Thareed (chicken), Arseyah, Marqoqa, Sago, and Asida). Several factors may alter the GI of food (Section 2.5.3), including the presence of macronutrients such as fat and protein, the type of starch, processing, and the addition of acids, sugars, gelling fibers, or amylase inhibitors [196]. Other factors may include the degree of chewing, the concentration of amylase in the gut, the presence of other food components in the gut, the amount of the insulin response and the rate of gastric emptying [399].

Rice is the main staple and energy source for almost half the world's population. Hence, it has significant nutrition and health implications. Many studies on rice and rice products led to the conclusion that rice should generally be classified as high GI food [349, 400-402]. However, many factors could affect the GI of rice and

rice products, including: rice variety and starch content (Amylose and Amylopectin), cooking, processing, cooling, soaking, fiber content, and particle size [402].

Additionally, rice is barely ever consumed on its own, it is often accompanied with other foods such as pulses, legumes, vegetables, seafood, nuts, and meats, which could alter the high GI of rice. In this study, Biryani, Machbous, and Arseyah - mixed rice dishes (rice with chicken or fish) - were found to have low (52), medium (60) and high (72) GI values, respectively. The high protein content in Biryani (11.55g/100g) along with added vegetables (onion, garlic and pepper) could explain its low GI. In Sri Lanka, parboiled Mottai Karupan red rice showed a mean GI value of 47 when it was consumed with Amaranthus leaf curry, and 56 for parboiled rice with soya meat gravy [393]. Moreover, the addition of vegetable mixed curry showed considerable reduction of GI and GL of traditional Sri Lankan breakfast foods [394]. Many studies have also showed a reduction in the GI of rice when consumed with legumes and pulses such as lentils [403-405]. Furthermore, the addition of acidic condiments (vinegar or pickles), emulsifiers, dairy products (milk, cheese, and yogurt), vegetables, pulses, and viscous fibers seems to decrease the GI of rice [402].

Harees is a traditional dish with a porridge-like consistency; it is prepared from crushed wheat with meat (beef or chicken) [175]. The GI value of Harees was the lowest among the traditional dishes studied (42). The presence of dietary fibers in test foods may delay its glycemic response, as it contributes to slower nutrient absorption and a delayed transit time in the small intestines [193, 395]. Hence, the high amount of dietary fibers presents in Harees (5.56g/100g) may cause its low GI.

The GI values of potatoes reported in the literature ranged from low (23) to high (111). Similarly, the GI values for sweet potatoes ranged from 44 (low) to 78

(high) [186]. In the current study, Fendal (boiled sweet potato) had a GI of 74 (high). Jenkins *et. al.* (1981) found that sweet potato from Canada had a GI of 48 [20] while sweet potato (*Ipomoea Batatas*) in Australia had a GI of 44 only [406]. However, in New Zealand, Perry *et. al.* (2000) reported a GI of 77 for Kumara (sweet potato) [407]. Whereas, the GI for sweet potato that has been peeled, cubed, boiled (in salted water for 15 minutes) was 59 [408]. Furthermore, three varieties of boiled Australian potatoes (Sebago, Desiree, and Pontiac) showed varying high GI values (87, 101, and 88, respectively) [409].

According to the International Tables of Glycemic Index and Glycemic Load Values (2008), boiled potato has a mean GI value of 78, whereas, the mean GI for boiled sweet potato is 63 [187]. Thus, the consumption of sweet potatoes seems to be a better choice for individuals wishing to reduce their dietary GI. However, studies have shown that the GI value of potatoes could differ depending on the variety, maturity, cooking method (baking, steaming, roasting, frying, or boiling), cutting method (cubing, peeling, mashing, or slicing), cooling process, and storage conditions (period and temperature) [398, 409-411].

To reduce the GI value of potatoes, researchers advise to precook potatoes and consume them cold (potato salad, for example) or reheated [410, 412]. Moreover, consuming potatoes with other ingredients such as acetic acid (vinegar) [413], vinaigrette dressing (vinegar and olive oil added to potato salad) [414], or topping baked potatoes with cheddar cheese [415] may lower the GI of potatoes but could limit the form in which they can be consumed. Additionally, aiming to develop low GI potato cultivars is also a valid proposition [398].

Bread is a staple food that is prepared usually by baking a dough of flour (wheat, rye, rice, oat, or barley) and water. In this study, six kinds of wheat bread and three main dishes containing wheat bread were tested. Two of the breads were classified as high GI - Regag bread (mean GI = 76) and Muhalla bread (mean GI = 77) - and all three main dishes containing bread were also high in GI - beef Thareed (mean GI = 74), chicken thareed (mean GI = 72), and Marqoqa (mean GI = 85). Likewise, the International Tables of Glycemic Index and Glycemic Load Values (2008) reported a mean GI value of 75 (high) for White wheat bread and 70 (high) for unleavened wheat bread [187].

Chebab bread and Khameer bread showed low GI values, which might be due to their high protein (7.08g/100 and 10.45g/100g, respectively) and fat content (9.07g/100g, and 12.69g/100g, respectively). Studies suggest that adding fat and protein to foods containing carbohydrates could possibly reduce their glycemic response and decrease their overall GI [416, 417]. It has been proposed that protein stimulates greater gastric inhibitory peptide (GIP) and higher insulin responses, which, in turn, lowers the postprandial peak of glucose and reduces the glycemic response of high GI foods [418]. High fat content was shown to delay the rate of gastric emptying, thus, reducing the rate of glucose digestion and absorption [415]. High fat content in Legemat and Khanfarooch (22.8g/100g, and 30.32g/100g, respectively) might be the reason behind their low GI values (44 and 45, respectively). However, food choice should not solely depend on the GI value of the food, since high fat content - especially saturated fats as in the case of Khanfarooch and Legemat (13.520g/100g and 9.586g/100g, respectively) - defeats the purpose of choosing low GI foods.

White Arabic wheat bread, also referred to as “Lebanese bread” or “Pita bread”, was also tested in the current study and showed a mean GI value of 67 (Medium). Similarly, Ali *et. al.* (2012) evaluated the GI of eight different types of traditional Omani wheat breads and reported a GI value of 63 (Medium) for white Lebanese wheat bread [419]. Moreover, Wolever *et. al.* (1994) found that white Pita bread has a GI of 57 (medium) when tested on subjects with type one or type 2 diabetes [408].

Khameer, Chebab and Arabic bread are considered leavened breads (fermented by yeast), which could explain their lower GI values compared to other breads. The effect of sourdough fermentation of leavened baked breads on the glycemic index has been reported by many researchers [420-423]. Several hypotheses were proposed to explain the reduced effect that sourdough fermentation has on the GI of bread including the synthesis of lactic acid which in turn lowers the rate of starch digestion [420], the synthesis of acetic and propionic acids, causing a reduction in the gastric emptying rate [421], or the synthesis/release of amino acids and peptides, resulting in better regulation of glucose metabolism [422].

Batheetha is a date paste made out of date fruit, wheat flour, ghee, sugar, cardamom and cinnamon. Various studies have reported the low GI value of date fruit [424, 425]. In 2002, Miller *et. al.* determined the GI of three different varieties of dates - Khalas = 35.5, Barhi = 49.7, and Bo ma'an= 30.5. In another study, Alkaabi *et. al.* (2011) reported the mean GIs of Fara'd, Lulu, Bo ma'an, Dabbas and Khalas dates tested in thirteen healthy individuals to be 54.0, 53.5, 46.3, 49.1, and 55.1, respectively [425]. However, in the current study, Batheetha is classified as a medium GI food (59), possibly due to the sugar added during the preparation of Batheetha, as the sugar

analysis showed a content of 5.02g total sugar in every 100g. The sugar analysis also revealed that 2.468g of sugar are in the form of sucrose and 1.181g were found to be glucose. Additionally, based on the International Tables of Glycemic Index and Glycemic Load Values (2008), the mean GIs for sucrose and glucose are 65 (medium) and 103 (high) respectively [187].

The low GI (47) of Habba Hamra (milk with red seed) was expected, since the literature indicated low GI for full-fat milk, ranging from 11 to 40 [186] and the mean GI for full-fat milk reported in the International Tables of Glycemic Index and Glycemic Load Values (2008) was 39 [187]. Conversely, other dessert dishes tested in the current study, like Sago and Asida, showed high GI values (mean GI = 99 for both dishes). The high moisture content (humidity) of Sago (74.91g/100g) and Asida (77.17g/100g), along with high cooking temperatures and prolonged cooking durations (time) could have increased starch gelatinization and digestibility degree, thus, resulting in high glycemic responses for these two traditional desserts [396, 397].

The GL of a standard serving size of each test food was calculated using the equation described in Section 3.3.6.2. A $GL \leq 10$ is considered low, between 11 and 19 is medium, and ≥ 20 is high. The results of the study demonstrated that the majority of test foods were classified as high GL, which is expected as most of the test foods had high GI, and they remained high when calculating their GL value.

Table 5.12: Glycemic Index (GI) and Glycemic Load (GL) Values of United Arab Emirates Traditional Dishes

Test Food	Available Carbohydrate (g/100g)*	Experimental portion (g)	GI		Standard serving size (g)	Carbohydrate (g/ serving)	GL (per serving)	Subjects (n)	GI classification	GL classification
			Mean	SE						
Arabic bread	63.47 ± 0.32	78.8	67	5	90	57.1	38.3	25	Medium	High
Regag bread	44.37 ± 0.49	112.7	76	7	21	9.3	7.1	25	High	Low
Chebab bread	45.88 ± 1.17	109.0	54	8	77	35.3	19.2	15	Low	Medium
Muhalla bread	67.66 ± 4.79	73.9	77	2	47	31.8	24.5	15	High	High
Khameer bread	54.93 ± 3.47	91.0	47	3	76	41.7	19.5	15	Low	Medium
Gurus	54.45 ± 2.60	91.8	72	8	91	49.5	35.5	15	High	High
Fendal	31.64 ± 0.24	158.0	74	7	150	47.5	35.3	20	High	High
Chami	5.31 ± 0.58	470.8	60	9	170	9.0	5.4	16	Medium	Low
Harees	7.74 ± 1.15	323.0	42	2	212	16.4	6.9	15	Low	Low
Thareed (beef)	10.87 ± 0.24	460.0	74	3	245	26.6	19.7	15	High	Medium
Thareed (chicken)	12.73 ± 2.65	392.8	72	4	393	50.0	36.0	15	High	High
Biryani (chicken)	19.69 ± 2.05	253.9	52	4	261	51.4	27.0	15	Low	High
Machbous (fish)	18.00 ± 0.82	277.8	60	3	250	45.0	26.8	20	Medium	High
Arseyah	9.85 ± 0.37	507.6	72	4	261	25.7	18.5	15	High	Medium
Marqoqa	16.00 ± 0.52	312.5	85	9	313	50.1	42.4	15	High	High
Khabisa	56.13 ± 4.65	89.1	67	4	86	48.3	32.1	15	Medium	High
Leqemat	44.19 ± 1.35	113.1	44	4	90	39.8	17.6	15	Low	Medium
Batheetha	38.24 ± 0.97	130.8	59	4	100	38.2	22.7	20	Medium	High
Kanfaroosh	39.62 ± 0.92	126.2	45	3	100	39.6	18.0	15	Low	Medium
Saqa	23.43 ± 3.54	213.4	99	8	214	50.1	49.8	15	High	High
Asida	21.09 ± 1.14	237.1	99	5	237	50.0	49.6	15	High	High
Habba Hamra	15.96 ± 1.20	313.3	47	3	98	15.6	7.4	15	Low	Low
Balalet	27.89 ± 2.19	179.3	63	5	144	40.2	25.4	15	Medium	High

*Date are expressed as Mean ± SD.

5.2.9 Exchange List

The food exchange list helps in monitoring food portion sizes and energy intake, because it translates scientific nutrition knowledge into a practical tool. Foods from the same category can be used interchangeably without changing estimated amounts of carbohydrates, fat, protein, and total energy obtainable in a meal [286]. The rounding-off method described by Wheeler *et. al.*[285] was used to fit food items into exchanges as previously described in Section 3.3.5.4.

The exchange list for traditional dishes commonly consumed in the UAE was established based on the proximate analysis data presented in Table 5.1. The exchanges per serving of test foods are shown in Table 5.13. Serving sizes of test foods are presented in standard measures (weight in grams) and equivalent cooking methods.

All test foods contained one exchange of starch, except Chami and Habba Hamra. Habba Hamra, a red seed added to milk drink contained one exchange of low fat milk. Whereas, Chami, a type of cottage cheese, was considered an exchange of very lean meat, as it belongs to the low-fat meat substitute food group.

Khanfarosh, a fried dessert dish, contained the highest number of fat exchanges (2 exchanges). All main dishes contained 1 exchange of starch and 1 exchange of lean meat, which is expected as they are all composed of grain products (wheat or rice) with a type of meat (chicken, beef, or fish).

Table 5.13: Meal Planning Exchange List of Twenty-Three United Arab Emirates Traditional Dishes

Test Food	Description	Food Group	Serving Size	Portion Size (g)	Macronutrient (Serving)			Kcal (serving)	Exchanges per serving
					CHO	Protein	Fat		
Arabic bread	Baked bread	Bread	1 oz.	30	19.1	2.8	0.3	90.7	1 Starch
Regag bread	Thin crispy crepe	Bread	1 oz.	30	13.7	3.1	0.1	68.5	1 Starch
Chebab bread	Emirati pancake	Bread	1 oz.	30	14.1	2.1	2.7	89.5	1 Starch
Muhalla Bread	Emirati crepe	Bread	1 oz.	30	20.5	3.1	1.3	105.7	1 Starch
Khameer Bread	Baked bread	Bread	1 oz.	30	17.0	3.1	3.8	114.9	1 Starch, 1/2 Fat
Gurus	Fried bread	Bread	1 oz.	30	17.6	2.6	2.3	101.6	1 Starch
Fendal	Boiled sweet potato	Starchy vegetable	1 small potato	60	20.7	1.1	0.3	90.4	1 Starch
Chami	Cottage cheese	Low-fat meat substitute's	1 oz.	30	1.6	4.6	0.2	26.9	1 Very Lean Meat
Harees (beef)	Crushed wheat with meat	Starch/Meat	1/2 cup composite dish	75	10.0	4.2	1.8	73.0	1 Starch, 1 Lean Meat
Thareed (beef)	Bread with meat stew	Starch/Meat	1/2 cup composite dish	75	9.1	5.3	1.6	71.9	1 Starch, 1 Lean Meat
Thareed (chicken)	Bread with chicken stew	Starch/Meat	1/2 cup composite dish	75	10.3	4.0	1.2	67.8	1 Starch, 1 Lean Meat
Biryani (chicken)	Rice with chicken	Starch/Meat	1/2 cup composite dish	75	15.5	8.7	2.5	118.8	1 Starch, 1 Lean Meat
Machbous (fish)	Rice with fish	Starch/Meat	1/2 cup composite dish	75	16.2	5.2	1.5	99.0	1 Starch, 1 Lean Meat
Arseyah	Rice with chicken	Starch/Meat	1/2 cup composite dish	180	18.2	4.0	1.6	103.2	1 Starch, 1 Lean Meat

Margoga	Bread with chicken stew	Starch/Meat	1/2 cup composite dish	75	12.4	4.4	1.6	82.0	1 Starch, 1 Lean Meat
Khabisa	Cardamom Pudding	Starchy food Prepared with fat	1/2 cup Pudding	25	14.7	1.3	2.6	87.9	1 Starch
Leqemat	Doughnut cake	Starchy food Prepared with fat	1 oz.	30	13.7	2.2	6.8	125.1	1 Starch, 1 Fat
Batheetha	Date paste	Starchy food Prepared with fat	1 oz.	30	13.3	1.7	2.9	86.0	1 Starch, 1/2 Fat
Khanfaroosh	Doughnut cake	Starchy food Prepared with fat	1 oz.	30	12.2	2.0	9.1	138.9	1 Starch, 2 Fat
Sago	Sago seed with sugar	Starch	2 oz.	60	14.2	0.5	0.3	61.7	1 Starch
Asida	Flour with Sugar	Starch	2 oz.	60	13.1	0.6	0.0	54.8	1 Starch
Habba Hamra	Red seed Milk drink	Evaporated Milk	1/2 cup	126	20.4	1.6	2.3	108.8	1 ½ Low Fat Milk
Balalet	Sweet vermicelli	Starch	1 oz.	30	12.0	0.7	0.5	55.7	1 Starch

5.3 Conclusion

In conclusion, this study provides a comprehensive food composition table including proximate data, minerals, vitamins, lipids, and sugars contents, along with GI and GL values of twenty-three locally consumed foods in the UAE including breads, entrées, mains and desserts. Determining the nutritional composition and the glycemic response of Emirati traditional foods is needed to assess the dietary intake of the population and identifying their effects on health promotion and disease prevention. Additionally, these tables will serve as a great tool of nutrition therapy planning and dietary management for dietitians in the UAE and other GCC countries. Furthermore, the availability of this data should be useful for assembling national and international food composition tables. Moreover, the knowledge of the GI and GL values of traditional Emirati foods helps in developing better dietary guidelines for individuals living with diabetes and/or obesity, and could be utilized by further research studies interested in the application of GL and GI.

Most dishes containing meat presented in this study had very high sodium levels. Therefore, it is highly important to take serious national steps towards reducing the sodium intake in diets in order to lessen the risk of developing CVD, hypertension, and other associated complications. Aiming for high iron food sources (like Regag, Chebab, Muhalla, and Khameer bread) as part of the daily diet is recommended to reduce the risk of iron deficiency (anemia).

The findings of this study advocate attention to the nutritive value and health aspects of traditional desserts when establishing dietary guidelines for UAE. Traditional desserts should be consumed in moderation due to their high content of

saturated fat (Khanfarooosh and Legemat), cholesterol (batheetha), and sugar (Sago, Asida, and Batheetha), in addition to their medium to high glycemic response.

Using whole-grain wheat flour instead of refined wheat flour for the preparation of breads is recommended in order to reduce their high GI values. The presence of dietary fibers in foods could also delay its glycemic response, as it contributes to slower nutrient absorption and delayed transit time in the small intestines [193, 395]. Furthermore, the addition of acidic condiments (vinegar or pickles), emulsifiers, dairy products (milk, cheese, and yogurt), vegetables, pulses, or viscous fiber to rice is recommended, since they appear to decrease its high GI value [402]. It is also recommended that foods containing carbohydrate should be consumed with protein-rich foods (rice and yogurt, bread and cheese, and rice with pulses) to reduce their glycemic response [416, 417]. Controlling the portion size of foods consumed can contribute to lowering their GL values, thus, reducing the risk of non-communicable diseases [107, 108, 209].

It is also advised to precook potatoes and consume them cold (potato salad, for example) or reheated [410, 412]. Moreover, consuming potatoes with other ingredients such as acetic acid (vinegar) [413], vinaigrette dressing (vinegar and olive oil added to potato salad) [414], or topping baked potatoes with cheddar cheese [415] are all good ways to lower the GI of potatoes.

Dietitians are advised to consider the macronutrient content of foods, such as fiber, total fat, saturated fat and trans fats along with the GI value when making dietary recommendations for patients with MetS.

The calculated exchange lists serve as a user-friendly guide, which enable consumers to exchange foods from the same group without changing energy or macronutrient contents. In addition, exchange lists assist dietitians in establishing culturally appropriate meal plans for Emiratis - mainly individuals living with diabetes and/or obesity.

Chapter 6: Summary and Recommendations

6.1 Overall Summary

Non-communicable diseases (NCDs) are the leading cause of deaths worldwide, and diabetes mellitus (DM) is the fourth major cause of NCD deaths [1]. United Arab Emirates (UAE) was ranked as having the fifth highest prevalence of DM in the Middle East and North Africa (MENA) region [304].

The transition in socioeconomic status accompanied with the adoption of a western lifestyle and diet for Emirati citizens, have led to a rise in the prevalence of overweight and obesity, particularly among females [167]. The existence of pre-diabetes, dyslipidemia, elevated blood pressure, and obesity is known as metabolic syndrome (MetS) [33]. There is paucity of data available about the prevalence of MetS and its relation with overweight and obesity among young female adults (17-25 years) in the UAE; therefore, determination of the prevalence of MetS in Emirati females aged 17–25 years and its relation to overweight and obesity in Al Ain, UAE has been part of our study.

The American Diabetes Association (ADA) recommends the use of low GI diets for DM management and prevention [255]. Additionally, the Food and Agriculture Organization (FAO) of the United Nations and the World Health Organization (WHO) have also validated the use of GI for the classification of carbohydrate-containing foods [188]. Likewise, they recommended the use of the GI classification system along with food composition tables to guide better food choices. Designing public health policy, clinical practice, and prevention programs for the screening and treatment of Emirati at high risk for metabolic syndrome is highly

important. Unfortunately, very little information is available about the food composition and glycemic index of Emirati traditional foods.

The current study provides a comprehensive food composition table with GI and GL values of locally consumed foods in the UAE, which could serve as a tool for nutrition therapy planning. In addition, the produced data will facilitate the role of dietitians in the dietary management of disease in the UAE and other GCC countries. Moreover, the current dataset will add on the existing international table of GI and GL values.

The following is summary of some main findings of both studies performed in this dissertation:

1. One third of the studied population were overweight and obese.
2. The prevalence of diabetes mellitus and prediabetes based on FPG was found much lower than that based on the percentage of HbA1c (0.5% and 9.2% Vs. 8.6% and 24% respectively). However, the crude prevalence of diabetes mellitus and pre-diabetes among Emirati young female adults was 8.6% and 24.7%, respectively.
3. Screening for pre-diabetes using IFG was independent than screening using HbA1c.
4. The overall prevalence of MetS was 6.8% (95% CI: 5% to 9%).
5. The most frequent component of MetS was reduced HDL-C levels, followed by central obesity and impaired fasting glucose.
6. MetS prevalence was highest among obese participants, as compared with normal-weight and overweight participants.

7. Using logistic regression model MetS was significantly associated with overweight, obesity, waist-hip ratio, glycated hemoglobin (HbA1c) and high sensitivity C-reactive protein ($P < 0.01$).
8. Most dishes containing meat and Regag bread presented in this study had very high sodium levels.
9. Traditional desserts had high content of saturated fat (Khanfaroosh and Legemat), cholesterol (batheetha), and sugar (Sago, Asida, and Batheetha), in addition to their medium to high glycemic response.
10. The exchange list developed in this study could be used by food and nutrition professionals to plan culturally sensitive meal plans.

6.2 Recommendations

1. Future research needs to include both male and female genders to investigate gender differences and generalize the results to all young adults
2. Additional prospective studies with bigger sample size and including all segments of the society are needed to confirm the prevalence of MetS and its most predictive risk factors among Emirati young adults.
3. Developing screening programs for early detection of metabolic syndrome among young Emirati adults, so that lifestyle interventions and treatment may prevent the development of diabetes and/or cardiovascular disease.
4. Developing population-based strategies that address cultural and community life of Emiratis to ensure relevance and commitment by the community.
5. Developing food-based dietary guidelines specific for the Emirati population, to be used for improving health policies and nutrition education programs.
6. Advising the Emirati population to consume traditional desserts in moderation due to their high content of saturated fat, cholesterol and sugar, in addition to their medium to high glycemic response.
7. Conducting a national flour fortification program to control and prevent anemia by fortifying flour with iron and folic acid.
8. Future intervention studies investigating the beneficial effects of low GI diets on the prevalence of the metabolic syndrome in the UAE.
9. Running awareness campaigns about the effect of the GI on certain non-communicable diseases common in the UAE including diabetes and obesity.
10. Encouraging dietitians to use the exchange list of Emirati foods when establishing culturally appropriate meal plans particularly for individuals living with diabetes and/or obesity.

References

1. WHO: Global status report on noncommunicable diseases 2014. Geneva: World Health Organization; 2014.
2. International Diabetes F: IDF Diabetes Atlas 2013, 6.
3. Saadi H, Al-Kaabi J, Benbarka M, Khalili A, Almahmeed W, Nagelkerke N, Abdel-Wareth L, Al Essa A, Yasin J, Al-Dabbagh B et al: Prevalence of Undiagnosed Diabetes and Quality of Care in Diabetic Patients Followed at Primary and Tertiary Clinics in Abu Dhabi, United Arab Emirates. The review of diabetic studies : RDS 2010, 7(4):293-302.
4. Alberti K, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, Fruchart J-C, James WPT, Loria CM, Smith SC: Harmonizing the Metabolic Syndrome A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation* 2009, 120(16):1640-1645.
5. Malik M, Razig SA: The prevalence of the metabolic syndrome among the multiethnic population of the United Arab Emirates: a report of a national survey. *Metabolic syndrome and related disorders* 2008, 6(3):177-186.
6. Mehairi AE, Khouri AA, Naqbi MM, Muhairi SJ, Maskari FA, Nagelkerke N, Shah SM: Metabolic syndrome among Emirati adolescents: a school-based study. *PloS one* 2013, 8(2):e56159.
7. Babusik P, Duris I: Comparison of obesity and its relationship to some metabolic risk factors of atherosclerosis in Arabs and South Asians in Kuwait. *Medical principles and practice : international journal of the Kuwait University, Health Science Centre* 2010, 19(4):275-280.
8. Sirdah MM, Al Laham NA, Abu Ghali AS: Prevalence of metabolic syndrome and associated socioeconomic and demographic factors among Palestinian adults (20-65 years) at the Gaza Strip. *Diabetes & metabolic syndrome* 2011, 5(2):93-97.
9. Yang G, Yuan S, Fu H, Wan G, Zhu L, Bu X, Zhang J, Du X, Li Y, Ji Y et al: Neck Circumference Positively Related With Central Obesity, Overweight, and Metabolic Syndrome in Chinese Subjects With Type 2 Diabetes: Beijing

- Community Diabetes Study 4. In: *Diabetes Care*. Volume 33, edn.; 2010: 2465-2467.
10. Gibson RS: *Principles of Nutritional Assessment*. In., edn.: Oxford University Press; 2005: 233-402.
 11. Colditz GA, Willett WC, Rotnitzky A, Manson JE: Weight gain as a risk factor for clinical diabetes mellitus in women. *Annals of internal medicine* 1995, 122(7):481-486.
 12. Hu FB, Sigal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Willett WC, Speizer FE, Manson JE: Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. *Jama* 1999, 282(15):1433-1439.
 13. Manson JE, Stampfer MJ, Colditz GA, Willett WC, Rosner B, Hennekens CH, Speizer FE, Rimm EB, Krolewski AS: Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *The Lancet* 1991, 338(8770):774-778.
 14. Carulli L, Rondinella S, Lombardini S, Canedi I, Loria P, Carulli N: Review article: diabetes, genetics and ethnicity. *Alimentary pharmacology & therapeutics* 2005, 22(s2):16-19.
 15. Schwingshackl L, Missbach B, König J, Hoffmann G: Adherence to a Mediterranean diet and risk of diabetes: a systematic review and meta-analysis. *Public health nutrition* 2015, 18(07):1292-1299.
 16. ADA: Standards of medical care in diabetes—2015. *Diabetes Care* 2015, 38:S1-S94.
 17. Anderson JW: *Nutrition Management of Diabetes*. *Modern Nutrition in Health and Disease*, Lea and Fieberger, Philadelphia 1988:1201-1229.
 18. Salmeron J, Hu FB, Manson JE, Stampfer MJ, Colditz GA, Rimm EB, Willett WC: Dietary fat intake and risk of type 2 diabetes in women. *The American journal of clinical nutrition* 2001, 73(6):1019-1026.
 19. Willett W, Manson J, Liu S: Glycemic index, glycemic load, and risk of type 2 diabetes. *The American journal of clinical nutrition* 2002, 76(1):274S-280S.
 20. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL, Goff DV: Glycemic index of foods: a physiological basis for carbohydrate exchange. *The American journal of clinical nutrition* 1981, 34(3):362-366.

21. Salmerón J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes care* 1997, 20(4):545-550.
22. Khattab M, Khader YS, Al-Khawaldeh A, Ajlouni K: Factors associated with poor glycemic control among patients with type 2 diabetes. *Journal of diabetes and its complications* 2010, 24(2):84-89.
23. Malik M, Bakir A, Saab BA, Roglic G, King H: Glucose intolerance and associated factors in the multi-ethnic population of the United Arab Emirates: results of a national survey. *Diabetes research and clinical practice* 2005, 69(2):188-195.
24. Reaven GM: Role of insulin resistance in human disease. *Diabetes* 1988, 37(12):1595-1607.
25. Alberti KGMM, Zimmet PZ: Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO Consultation. *Diabetic Medicine* 1998, 15(7):539-553.
26. Crepaldi G, Nosadini R: Diabetic cardiopathy: is it a real entity? *Diabetes/metabolism reviews* 1988, 4(3):273-288.
27. Kaplan NM: The deadly quartet: upper-body obesity, glucose intolerance, hypertriglyceridemia, and hypertension. *Archives of internal medicine* 1989, 149(7):1514-1520.
28. DeFronzo RA, Ferrannini E: Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes care* 1991, 14(3):173-194.
29. Shafrir E: *Animal models of syndrome X*. 1993.
30. Sarafidis PA, Nilsson PM: The metabolic syndrome: a glance at its history. *Journal of hypertension* 2006, 24(4):621-626.
31. Blaha MJ, Tota-Maharaj R: *Metabolic Syndrome : From Risk Factors to Management*. Torino, ITA: SEEd Srl; 2012.
32. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *Jama* 2001, 285(19):2486-2497.

33. Alberti K, Zimmet P, Shaw J: Metabolic syndrome—a new world-wide definition. A consensus statement from the international diabetes federation. *Diabetic Medicine* 2006, 23(5):469-480.
34. Kahn R, Buse J, Ferrannini E, Stern M: The metabolic syndrome: time for a critical appraisal Joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes care* 2005, 28(9):2289-2304.
35. Balkau B, Charles M-A: Comment on the provisional report from the WHO consultation. *Diabetic medicine* 1999, 16(5):442-443.
36. Einhorn M, FACP, FACE, Daniel: American College of Endocrinology position statement on the insulin resistance syndrome*. *Endocrine Practice* 2003, 9(Supplement 2):5-21.
37. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith SC: Diagnosis and management of the metabolic syndrome an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation* 2005, 112(17):2735-2752.
38. Grundy SM: Atlas of atherosclerosis and metabolic syndrome. DE: Springer Verlag; 2011.
39. Grundy SM: Atlas of atherosclerosis and metabolic syndrome, vol. 5th. DE: Springer Verlag; 2011.
40. Pollex RL, Hegele RA: Genetic determinants of the metabolic syndrome. *Nature clinical practice Cardiovascular medicine* 2006, 3(9):482-489.
41. Roche HM, Phillips C, Gibney MJ: The metabolic syndrome: the crossroads of diet and genetics. *Proceedings of the Nutrition Society* 2005, 64(03):371-377.
42. Frayling TM, Timpson NJ, Weedon MN, Zeggini E, Freathy RM, Lindgren CM, Perry JR, Elliott KS, Lango H, Rayner NW: A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. *Science* 2007, 316(5826):889-894.
43. Attaoua R, El Mkaem SA, Radian S, Fica S, Hanzu F, Albu A, Gheorghiu M, Coculescu M, Grigorescu F: FTO gene associates to metabolic syndrome in women with polycystic ovary syndrome. *Biochemical and biophysical research communications* 2008, 373(2):230-234.
44. Cameron AJ, Boyko EJ, Sicree RA, Zimmet PZ, Söderberg S, Alberti K, Tuomilehto J, Chitson P, Shaw JE: Central obesity as a precursor to the

- metabolic syndrome in the AusDiab study and Mauritius. *Obesity* 2008, 16(12):2707-2716.
45. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K: Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. *Journal of Clinical Investigation* 2006, 116(7):1784-1792.
 46. Saleem U, Khaleghi M, Morgenthaler NG, Bergmann A, Struck J, Mosley TH, Kullo IJ: Plasma Carboxy-Terminal Provasopressin (Copeptin): A Novel Marker of Insulin Resistance and Metabolic Syndrome. *The Journal of clinical endocrinology and metabolism* 2009, 94(7):2558-2564.
 47. de Luca C, Olefsky JM: Inflammation and Insulin Resistance. *FEBS letters* 2008, 582(1):97-105.
 48. IKEDA Y, SUEHIRO T, NAKAMURA T, KUMON Y, HASHIMOTO K: Clinical significance of the insulin resistance index as assessed by homeostasis model assessment. *Endocrine journal* 2001, 48(1):81-86.
 49. Lemieux I, Pascot A, Couillard C, Lamarche B, Tchernof A, Alméras N, Bergeron J, Gaudet D, Tremblay G, Prud'homme D: Hypertriglyceridemic Waist A Marker of the Atherogenic Metabolic Triad (Hyperinsulinemia; Hyperapolipoprotein B; Small, Dense LDL) in Men? *Circulation* 2000, 102(2):179-184.
 50. Rasouli N, Molavi B, Elbein SC, Kern PA: Ectopic fat accumulation and metabolic syndrome. *Diabetes, Obesity and Metabolism* 2007, 9(1):1-10.
 51. Jensen M: Health consequences of fat distribution. *Hormone research in paediatrics* 1997, 48(Suppl. 5):88-92.
 52. Han TS, Lean MEJ: Metabolic syndrome. *Medicine* 2011, 39(1):24-31.
 53. Lind L: Circulating markers of inflammation and atherosclerosis. *Atherosclerosis* 2003, 169(2):203-214.
 54. Pouliot M-C, Després J-P, Lemieux S, Moorjani S, Bouchard C, Tremblay A, Nadeau A, Lupien PJ: Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *The American journal of cardiology* 1994, 73(7):460-468.
 55. Roberts CK, Sindhu KK: Oxidative stress and metabolic syndrome. *Life sciences* 2009, 84(21):705-712.

56. Rahmouni K, Correia ML, Haynes WG, Mark AL: Obesity-associated hypertension new insights into mechanisms. *Hypertension* 2005, 45(1):9-14.
57. Hall JE: Pathophysiology of obesity hypertension. *Current hypertension reports* 2000, 2(2):139-147.
58. Sowers JR: Insulin resistance and hypertension. *American Journal of Physiology-Heart and Circulatory Physiology* 2004, 286(5):H1597-H1602.
59. Grundy SM: Hypertriglyceridemia, atherogenic dyslipidemia, and the metabolic syndrome. *The American journal of cardiology* 1998, 81(4):18B-25B.
60. Perk J, De Backer G, Gohlke H, Graham I, Reiner Ž, Verschuren M, Albus C, Benlian P, Boysen G, Cifkova R: European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). *European heart journal* 2012, 33(13):1635-1701.
61. Krauss RM, Siri PW: Metabolic abnormalities: triglyceride and low-density lipoprotein. *Endocrinology and metabolism clinics of North America* 2004, 33(2):405-415.
62. Grundy SM, Vega GL, Otvos JD, Rainwater DL, Cohen JC: Hepatic lipase activity influences high density lipoprotein subclass distribution in normotriglyceridemic men: genetic and pharmacological evidence. *Journal of lipid research* 1999, 40(2):229-234.
63. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO, Criqui M, Fadl YY, Fortmann SP, Hong Y, Myers GL: Markers of inflammation and cardiovascular disease application to clinical and public health practice: a statement for healthcare professionals from the centers for disease control and prevention and the American Heart Association. *Circulation* 2003, 107(3):499-511.
64. Nishida M, Moriyama T, Ishii K, Takashima S, Yoshizaki K, Sugita Y, Yamauchi-Takahara K: Effects of IL-6, adiponectin, CRP and metabolic syndrome on subclinical atherosclerosis. *Clinica Chimica Acta* 2007, 384(1):99-104.
65. Pischon T, Hu FB, Rexrode KM, Girman CJ, Manson JE, Rimm EB: Inflammation, the metabolic syndrome, and risk of coronary heart disease in women and men. *Atherosclerosis* 2008, 197(1):392-399.

66. Maury E, Brichard S: Adipokine dysregulation, adipose tissue inflammation and metabolic syndrome. *Molecular and cellular endocrinology* 2010, 314(1):1-16.
67. Ridker PM, Buring JE, Cook NR, Rifai N: C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events an 8-year follow-up of 14 719 initially healthy American women. *Circulation* 2003, 107(3):391-397.
68. Devaraj S, Singh U, Jialal I: Human C-reactive protein and the metabolic syndrome. *Current opinion in lipidology* 2009, 20(3):182.
69. Fröhlich M, Imhof A, Berg G, Hutchinson WL, Pepys MB, Boeing H, Muche R, Brenner H, Koenig W: Association between C-reactive protein and features of the metabolic syndrome: a population-based study. *Diabetes care* 2000, 23(12):1835-1839.
70. Sattar N, Gaw A, Scherbakova O, Ford I, O'Reilly DSJ, Haffner SM, Isles C, Macfarlane PW, Packard CJ, Cobbe SM: Metabolic syndrome with and without C-reactive protein as a predictor of coronary heart disease and diabetes in the West of Scotland Coronary Prevention Study. *Circulation* 2003, 108(4):414-419.
71. Ridker PM, Wilson PWF, Grundy SM: Should C-reactive protein be added to metabolic syndrome and to assessment of global cardiovascular risk? *Circulation* 2004, 109(23):2818-2825.
72. Ninomiya JK, L'Italien G, Criqui MH, Whyte JL, Gamst A, Chen RS: Association of the metabolic syndrome with history of myocardial infarction and stroke in the Third National Health and Nutrition Examination Survey. *Circulation* 2004, 109(1):42-46.
73. Olijhoek JK, van der Graaf Y, Banga J-D, Algra A, Rabelink TJ, Visseren FL: The metabolic syndrome is associated with advanced vascular damage in patients with coronary heart disease, stroke, peripheral arterial disease or abdominal aortic aneurysm. *European heart journal* 2004, 25(4):342-348.
74. Isomaa B, Almgren P, Tuomi T, Forsén B, Lahti K, Nissén M, Taskinen M-R, Groop L: Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes care* 2001, 24(4):683-689.
75. Lakka H-M, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, Salonen JT: The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *Jama* 2002, 288(21):2709-2716.

76. Mottillo S, Filion KB, Genest J, Joseph L, Pilote L, Poirier P, Rinfret S, Schiffrin EL, Eisenberg MJ: The metabolic syndrome and cardiovascular risk: a systematic review and meta-analysis. *Journal of the American College of Cardiology* 2010, 56(14):1113-1132.
77. Ogbera AO: Prevalence and gender distribution of the metabolic syndrome. *Diabetology & metabolic syndrome* 2010, 2(1):4.
78. Ruggenti P, Fassi A, Ilieva AP, Bruno S, Iliev IP, Brusegan V, Rubis N, Gherardi G, Arnoldi F, Ganeva M: Preventing microalbuminuria in type 2 diabetes. *New England Journal of Medicine* 2004, 351(19):1941-1951.
79. Relimpio F, Martinez-Brocca M, Leal-Cerro A, Losada F, Mangas M, Pumar A, Astorga R: Variability in the presence of the metabolic syndrome in type 2 diabetic patients attending a diabetes clinic: influences of age and gender. *Diabetes research and clinical practice* 2004, 65(2):135-142.
80. Alexander CM, Landsman PB, Teutsch SM, Haffner SM: NCEP-defined metabolic syndrome, diabetes, and prevalence of coronary heart disease among NHANES III participants age 50 years and older. *Diabetes* 2003, 52(5):1210-1214.
81. Ford ES, Li C, Sattar N: Metabolic Syndrome and Incident Diabetes Current state of the evidence. *Diabetes care* 2008, 31(9):1898-1904.
82. Lorenzo C, Williams K, Stern MP, Haffner SM: The metabolic syndrome as predictor of type 2 diabetes the San Antonio heart study. *Diabetes care* 2003, 26(11):3153-3159.
83. Riddle MC, Ambrosius WT, Brillon DJ, Buse JB, Byington RP, Cohen RM, Goff DC, Malozowski S, Margolis KL, Probstfield JL: Epidemiologic relationships between A1C and all-cause mortality during a median 3.4-year follow-up of glycemic treatment in the ACCORD trial. *Diabetes care* 2010, 33(5):983-990.
84. Dilley J, Ganesan A, Deepa R, Deepa M, Sharada G, Williams OD, Mohan V: Association of A1C with cardiovascular disease and metabolic syndrome in Asian Indians with normal glucose tolerance. *Diabetes care* 2007, 30(6):1527-1532.
85. Sung K, Rhee E: Glycated haemoglobin as a predictor for metabolic syndrome in non-diabetic Korean adults. *Diabetic medicine* 2007, 24(8):848-854.

86. Ford ES: Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome a summary of the evidence. *Diabetes care* 2005, 28(7):1769-1778.
87. Galassi A, Reynolds K, He J: Metabolic syndrome and risk of cardiovascular disease: a meta-analysis. *The American journal of medicine* 2006, 119(10):812-819.
88. Gami AS, Witt BJ, Howard DE, Erwin PJ, Gami LA, Somers VK, Montori VM: Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. *Journal of the American College of Cardiology* 2007, 49(4):403-414.
89. Li W, Ma D, Liu M, Liu H, Feng S, Hao Z, Wu B, Zhang S: Association between metabolic syndrome and risk of stroke: a meta-analysis of cohort studies. *Cerebrovascular Diseases* 2008, 25(6):539-547.
90. Hui WS, Liu Z, Ho SC: Metabolic syndrome and all-cause mortality: a meta-analysis of prospective cohort studies. *European journal of epidemiology* 2010, 25(6):375-384.
91. Wild SH, Byrne CD: The Epidemiology of the Metabolic Syndrome and its Association with Diabetes, Cardiovascular Disease and Other Conditions. *The Metabolic Syndrome* 2011:1.
92. Ogawa H, Nakayama M, Morimoto T, Uemura S, Kanauchi M, Doi N, Jinnouchi H, Sugiyama S, Saito Y, Investigators JPPoAWAfDT: Low-dose aspirin for primary prevention of atherosclerotic events in patients with type 2 diabetes: a randomized controlled trial. *Jama* 2008, 300(18):2134-2141.
93. Blaha MJ, Bansal S, Rouf R, Golden SH, Blumenthal RS, DeFilippis AP: A practical "ABCDE" approach to the metabolic syndrome. In: *Mayo Clinic Proceedings: 2008: Elsevier; 2008: 932-943.*
94. Tota-Maharaj R, Defilippis AP, Blumenthal RS, Blaha MJ: A practical approach to the metabolic syndrome: review of current concepts and management. *Current opinion in cardiology* 2010, 25(5):502-512.
95. Wannamethee SG, Shaper AG, Lennon L, Morris RW: Metabolic syndrome vs Framingham Risk Score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus. *Archives of internal medicine* 2005, 165(22):2644-2650.
96. Conroy R, Pyörälä K, Fitzgerald Ae, Sans S, Menotti A, De Backer G, De Bacquer Dr, Ducimetiere P, Jousilahti P, Keil U: Estimation of ten-year risk of

- fatal cardiovascular disease in Europe: the SCORE project. *European heart journal* 2003, 24(11):987-1003.
97. Group DS: Prediction of the risk of cardiovascular mortality using a score that includes glucose as a risk factor. The DECODE Study. *Diabetologia* 2004, 47(12):2118-2128.
 98. Krause T, Lovibond K, Caulfield M, McCormack T, Williams B: Management of hypertension: summary of NICE guidance. *Bmj* 2011, 343.
 99. Group AS: Effects of intensive blood-pressure control in type 2 diabetes mellitus. *The New England journal of medicine* 2010, 362(17):1575.
 100. Barclay AW, Petocz P, McMillan-Price J, Flood VM, Prvan T, Mitchell P, Brand-Miller JC: Glycemic index, glycemic load, and chronic disease risk—a meta-analysis of observational studies. *The American journal of clinical nutrition* 2008, 87(3):627-637.
 101. Katzmarzyk PT, Leon AS, Wilmore JH, Skinner JS, Rao D, Rankinen T, Bouchard C: Targeting the metabolic syndrome with exercise: evidence from the HERITAGE Family Study. *Medicine and science in sports and exercise* 2003, 35(10):1703-1709.
 102. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obesity research* 1998, 6 Suppl 2:51s-209s.
 103. Case C, Jones P, Nelson K, O'Brian Smith E, Ballantyne C: Impact of weight loss on the metabolic syndrome. *Diabetes, Obesity and Metabolism* 2002, 4(6):407-414.
 104. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S: A randomized trial of a low-carbohydrate diet for obesity. *New England Journal of Medicine* 2003, 348(21):2082-2090.
 105. Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ: Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *Jama* 2005, 293(1):43-53.
 106. Kastorini C-M, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB: The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534,906 individuals. *Journal of the American College of Cardiology* 2011, 57(11):1299-1313.

107. Ebbeling CB, Leidig MM, Feldman HA, Lovesky MM, Ludwig DS: Effects of a low-glycemic load vs low-fat diet in obese young adults: a randomized trial. *Jama* 2007, 297(19):2092-2102.
108. Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, Hennekens CH, Manson JE: A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *The American journal of clinical nutrition* 2000, 71(6):1455-1461.
109. Sofi F, Cesari F, Abbate R, Gensini GF, Casini A: Adherence to Mediterranean diet and health status: meta-analysis. *Bmj* 2008, 337.
110. Cappuccio FP, Taggart FM, Kandala N-B, Currie A: Meta-analysis of short sleep duration and obesity in children and adults. *Sleep* 2008, 31(5):619.
111. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA: Quantity and Quality of Sleep and Incidence of Type 2 Diabetes A systematic review and meta-analysis. *Diabetes care* 2010, 33(2):414-420.
112. Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA: Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *European heart journal* 2011, 32(12):1484-1492.
113. Lindström J, Ilanne-Parikka P, Peltonen M, Aunola S, Eriksson JG, Hemiö K, Hämäläinen H, Härkönen P, Keinänen-Kiukaanniemi S, Laakso M: Sustained reduction in the incidence of type 2 diabetes by lifestyle intervention: follow-up of the Finnish Diabetes Prevention Study. *The Lancet* 2006, 368(9548):1673-1679.
114. Ratner RE, Prevention Program Research Group D: An update on the diabetes prevention program. *Endocrine Practice* 2006, 12(Supplement 1):20-24.
115. van de Laar FA, Lucassen PL, Akkermans RP, van de Lisdonk EH, Rutten GE, van Weel C: α -Glucosidase Inhibitors for Patients With Type 2 Diabetes Results from a Cochrane systematic review and meta-analysis. *Diabetes care* 2005, 28(1):154-163.
116. Loke YK, Singh S, Furberg CD: Long-term use of thiazolidinediones and fractures in type 2 diabetes: a meta-analysis. *Canadian Medical Association Journal* 2009, 180(1):32-39.
117. Wu T, Gao X, Chen M, Van Dam R: Long-term effectiveness of diet-plus-exercise interventions vs. diet-only interventions for weight loss: a meta-analysis. *Obesity Reviews* 2009, 10(3):313-323.

118. Vaughan C, Schoo A, Janus ED, Philpot B, Davis-Lameloise N, Lo SK, Laatikainen T, Vartiainen E, Dunbar JA: The association of levels of physical activity with metabolic syndrome in rural Australian adults. *BMC public health* 2009, 9(1):273.
119. Dunstan D, Salmon J, Owen N, Armstrong T, Zimmet P, Welborn T, Cameron A, Dwyer T, Jolley D, Shaw J: Associations of TV viewing and physical activity with the metabolic syndrome in Australian adults. *Diabetologia* 2005, 48(11):2254-2261.
120. Chan CB, Ryan DA, Tudor-Locke C: Health benefits of a pedometer-based physical activity intervention in sedentary workers. *Preventive medicine* 2004, 39(6):1215-1222.
121. Kalter-Leibovici O, Younis-Zeidan N, Atamna A, Lubin F, Alpert G, Chetrit A, Novikov I, Daoud N, Freedman LS: Lifestyle intervention in obese Arab women: a randomized controlled trial. *Archives of internal medicine* 2010, 170(11):970-976.
122. Jurca R, Lamonte MJ, Church TS, Earnest CP, Fitzgerald SJ, Barlow CE, Jordan AN, Kampert JB, Blair SN: Associations of muscle strength and fitness with metabolic syndrome in men. *Medicine and science in sports and exercise* 2004, 36(8):1301-1307.
123. Méndez-Hernández P, Flores Y, Siani C, Lamure M, Dosamantes-Carrasco LD, Halley-Castillo E, Huitrón G, Talavera JO, Gallegos-Carrillo K, Salmerón J: Physical activity and risk of metabolic syndrome in an urban Mexican cohort. *BMC public health* 2009, 9(1):276.
124. Leon AS, Franklin BA, Costa F, Balady GJ, Berra KA, Stewart KJ, Thompson PD, Williams MA, Lauer MS: Cardiac rehabilitation and secondary prevention of coronary heart disease an american heart association scientific statement from the council on clinical cardiology (subcommittee on exercise, cardiac rehabilitation, and prevention) and the council on nutrition, physical activity, and metabolism (subcommittee on physical activity), in collaboration with the american association of cardiovascular and pulmonary rehabilitation. *Circulation* 2005, 111(3):369-376.
125. Stiles S, Cummings S, Klein S, McCullough PA, Fielding CR: Bariatric Surgery and Cardiovascular Risk Factors. *Circulation* 2011, 123:1683-1701.
126. Gloy VL, Briel M, Bhatt DL, Kashyap SR, Schauer PR, Mingrone G, Bucher HC, Nordmann AJ: Bariatric surgery versus non-surgical treatment for obesity: a systematic review and meta-analysis of randomised controlled trials. *Bmj* 2013, 347:f5934.

127. Mingrone G, Panunzi S, De Gaetano A, Guidone C, Iaconelli A, Leccesi L, Nanni G, Pomp A, Castagneto M, Ghirlanda G: Bariatric surgery versus conventional medical therapy for type 2 diabetes. *New England Journal of Medicine* 2012, 366(17):1577-1585.
128. Schauer PR, Kashyap SR, Wolski K, Brethauer SA, Kirwan JP, Pothier CE, Thomas S, Abood B, Nissen SE, Bhatt DL: Bariatric surgery versus intensive medical therapy in obese patients with diabetes. *New England Journal of Medicine* 2012, 366(17):1567-1576.
129. Gracia-Solanas JA, Elia M, Aguilera V, Ramirez J, Martínez J, Bielsa M, Martinez M: Metabolic syndrome after bariatric surgery. Results depending on the technique performed. *Obesity surgery* 2011, 21(2):179-185.
130. Kelli H, Kassas I, Lattouf O: Cardio Metabolic Syndrome: A Global Epidemic. *J Diabetes Metab* 2015, 6(513):2.
131. El-Aty MA, Mabry R, Morsi M, Al-Lawati J, Al-Riyami A, El-Sayed M: Metabolic Syndrome and Its Components: Secondary analysis of the World Health Survey, Oman. *Sultan Qaboos University medical journal* 2014, 14(4):e460.
132. Bener A, Zirie M, Musallam M, Khader YS, Al-Hamaq AO: Prevalence of metabolic syndrome according to Adult Treatment Panel III and International Diabetes Federation criteria: a population-based study. *Metabolic syndrome and related disorders* 2009, 7(3):221-230.
133. Al Rashdan I, Al Neseif Y: Prevalence of overweight, obesity, and metabolic syndrome among adult Kuwaitis: results from community-based national survey. *Angiology* 2010, 61(1):42-48.
134. Al-Daghri NM, Al-Attas OS, Alokail MS, Alkharfy KM, Sabico SL, Chrousos GP: Decreasing prevalence of the full metabolic syndrome but a persistently high prevalence of dyslipidemia among adult Arabs. *PloS one* 2010, 5(8):e12159.
135. Henry CJK, Lightowler HJ, Al-Hourani HM: Physical activity and levels of inactivity in adolescent females ages 11–16 years in the United Arab Emirates. *American Journal of Human Biology* 2004, 16(3):346-353.
136. Gu D, Reynolds K, Wu X, Chen J, Duan X, Reynolds RF, Whelton PK, He J, Group IC: Prevalence of the metabolic syndrome and overweight among adults in China. *The Lancet* 2005, 365(9468):1398-1405.

137. Lim S, Shin H, Song JH, Kwak SH, Kang SM, Yoon JW, Choi SH, Cho SI, Park KS, Lee HK: Increasing Prevalence of Metabolic Syndrome in Korea The Korean National Health and Nutrition Examination Survey for 1998–2007. *Diabetes care* 2011, 34(6):1323-1328.
138. Kozan O, Oguz A, Abaci A, Erol C, Ongen Z, Temizhan A, Celik S: Prevalence of the metabolic syndrome among Turkish adults. *European journal of clinical nutrition* 2007, 61(4):548-553.
139. Khader Y, Bateiha A, El-Khateeb M, Al-Shaikh A, Ajlouni K: High prevalence of the metabolic syndrome among Northern Jordanians. *Journal of Diabetes and its Complications* 2007, 21(4):214-219.
140. Sibai A-M, Obeid O, Batal M, Adra N, El Khoury D, Hwalla N: Prevalence and correlates of metabolic syndrome in an adult Lebanese population. *CVD Prevention and Control* 2008, 3(2):83-90.
141. Harzallah F, Alberti H, Ben Khalifa F: The metabolic syndrome in an Arab population: a first look at the new International Diabetes Federation criteria. *Diabetic medicine* 2006, 23(4):441-444.
142. Azizi F, Salehi P, Etemadi A, Zahedi-Asl S: Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes research and clinical practice* 2003, 61(1):29-37.
143. Santos A, Lopes C, Barros H: Prevalence of metabolic syndrome in the city of Porto. *Revista portuguesa de cardiologia: orgao oficial da Sociedade Portuguesa de Cardiologia= Portuguese journal of cardiology: an official journal of the Portuguese Society of Cardiology* 2004, 23(1):45-52.
144. Guallar-Castillón P, Pérez RF, López García E, León-Muñoz LM, Aguilera MT, Graciani A, Gutiérrez-Fisac JL, Banegas JR, Rodríguez-Artalejo F: Magnitude and Management of Metabolic Syndrome in Spain in 2008-2010: The ENRICA Study. *Revista Española de Cardiología (English Edition)* 2014, 67(5):367-373.
145. Alvarez LE, Ribas BL, Serra ML: [Prevalence of the metabolic syndrome in the population of Canary Islands, Spain]. *Medicina clinica* 2003, 120(5):172-174.
146. Miccoli R, Bianchi C, Odoguardi L, Penno G, Caricato F, Giovannitti MG, Pucci L, Del Prato S: Prevalence of the metabolic syndrome among Italian adults according to ATP III definition. *Nutrition, Metabolism and Cardiovascular Diseases* 2005, 15(4):250-254.

147. Arai H, Yamamoto A, Matsuzawa Y, Saito Y, Yamada N, Oikawa S, Mabuchi H, Teramoto T, Sasaki J, Nakaya N: Prevalence of metabolic syndrome in the general Japanese population in 2000. *Journal of atherosclerosis and thrombosis* 2006, 13(4):202-208.
148. Gupta R, Deedwania PC, Gupta A, Rastogi S, Panwar RB, Kothari K: Prevalence of metabolic syndrome in an Indian urban population. *International journal of cardiology* 2004, 97(2):257-261.
149. Xi B, He D, Hu Y, Zhou D: Prevalence of metabolic syndrome and its influencing factors among the Chinese adults: The China Health and Nutrition Survey in 2009. *Preventive medicine* 2013, 57(6):867-871.
150. Sidorenkov O, Nilssen O, Brenn T, Martiushov S, Arkhipovsky VL, Grjibovski AM: Prevalence of the metabolic syndrome and its components in Northwest Russia: the Arkhangelsk study. *BMC public health* 2010, 10(1):23.
151. Velásquez-Meléndez G, Gazzinelli A, Côrrea-Oliveira R, Pimenta AM, Kac G: Prevalence of metabolic syndrome in a rural area of Brazil. *São Paulo medical journal* 2007, 125(3):155-162.
152. Ervin RB: Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States. *National health statistics reports* 2009, 13:1-8.
153. Riediger ND, Clara I: Prevalence of metabolic syndrome in the Canadian adult population. *Canadian Medical Association Journal* 2011, 183(15):E1127-E1134.
154. Cameron AJ, Magliano DJ, Zimmet PZ, Welborn T, Shaw JE: The Metabolic Syndrome in Australia: Prevalence using four definitions. *Diabetes Research and Clinical Practice* 2007, 77(3):471-478.
155. Gentles D, Metcalf P, Dyal L, Sundborn G, Schaaf D, Black P, Scragg R, Jackson R: Metabolic syndrome prevalence in a multicultural population in Auckland, New Zealand. *NZ Med J* 2007, 120(1248):U2399.
156. Daniels SR, Arnett DK, Eckel RH, Gidding SS, Hayman LL, Kumanyika S, Robinson TN, Scott BJ, Jeor SS, Williams CL: Overweight in children and adolescents pathophysiology, consequences, prevention, and treatment. *Circulation* 2005, 111(15):1999-2012.
157. Rodríguez-Morán M, Salazar-Vázquez B, Violante R, Guerrero-Romero F: Metabolic syndrome among children and adolescents aged 10–18 years. *Diabetes Care* 2004, 27(10):2516-2517.

158. Al-Daghri NM: Extremely high prevalence of metabolic syndrome manifestations among Arab youth: a call for early intervention. *European journal of clinical investigation* 2010, 40(12):1063-1066.
159. Al-Isa A, Akanji AO, Thalib L: Prevalence of the metabolic syndrome among female Kuwaiti adolescents using two different criteria. *British journal of nutrition* 2010, 103(01):77-81.
160. Badran M, Laher I: Obesity in Arabic-speaking countries. *Journal of Obesity* 2011, 2011.
161. Muttappallymyalil J, Mathew E, Sreedharan J, Al Sharbatii S, Shaikh RB, Basha SA: Self reported physical activity among University Students in Ajman, UAE. *Pak J Med Sci* 2010, 26(4):782-786.
162. Musaiger AO: Risk factors for cardiovascular disease in the United Arab Emirates. *International journal of food sciences and nutrition* 1998:S65.
163. Belal AM: Nutrition-related chronic diseases Epidemic in UAE: can we stand to STOP it? *Sudanese Journal of Public Health* 2009, 4(4):383-392.
164. Berger G, Peerson A: Giving young Emirati women a voice: Participatory action research on physical activity. *Health & Place* 2009, 15(1):117-124.
165. Musaiger AO, Abuirmeileh NM: Food consumption patterns of adults in the United Arab Emirates. *The journal of the Royal Society for the Promotion of Health* 1998, 118(3):146-150.
166. Bin Zaal A, Musaiger A, D'Souza R: Dietary habits associated with obesity among adolescents in Dubai, United Arab Emirates. *Nutricion hospitalaria* 2009, 24(4):437-444.
167. Ng SW, Zaghoul S, Ali H, Harrison G, Yeatts K, El Sadig M, Popkin BM: Nutrition transition in the United Arab Emirates. *European journal of clinical nutrition* 2011, 65(12):1328-1337.
168. Al Matroushi M, Fikry MU-G: United Arab Emirates global school-based student health survey 2005. *Centers for Disease Control, World Health Organization* 2005.
169. Al Dhaheri A, Al Ma'awali A, Laleye L, Washi S: Nutritional knowledge of Emirati traditional foods and body image perceptions among UAE University students. *Emirates Journal of Food and Agriculture* 2014, 26(10):853.
170. Al-Jebrin A, Sawaya WN, Salji JP, Ayaz M, Khalil JK: Chemical and nutritional quality of some Saudi Arabian dishes based on cereals and legumes

1. Proximate composition, Amino Acid contents and nutritive value. *Ecology of food and nutrition* 1985, 17(2):157-164.
171. Musaiger AO, Al-Saad JA, Al-Hooti DS, Khunji ZA: Chemical composition of fermented dairy products consumed in Bahrain. *Food chemistry* 1998, 61(1):49-52.
172. Musaiger AO, Ahmed MA, Rao MV: Chemical composition of some traditional dishes of Oman. *Food chemistry* 1998, 61(1):17-22.
173. Dashti BH, Al-Awadi F, Khalafawi MS, Al-Zenki S, Sawaya W: Nutrient contents of some traditional Kuwaiti dishes: proximate composition and phytate content. *Food Chem* 2001, 74(2):169-175.
174. Buttriss JL, Benelam B: Nutrition and health claims: the role of food composition data. *European journal of clinical nutrition* 2010, 64:S8-S13.
175. Habib HM, Ali HI, Ibrahim WH, Afifi HS: Nutritional value of 10 traditional dishes of the United Arab Emirates. *Ecology of food and nutrition* 2011, 50(6):526-538.
176. Trowell H: The development of the concept of dietary fiber in human nutrition. *The American journal of clinical nutrition* 1978, 31(10):S3-S11.
177. Trowell H: Dietary fibre, ischaemic heart disease and diabetes mellitus. *Proceedings of the Nutrition Society* 1973, 32(03):151-157.
178. Trowell H: Dietary-fiber hypothesis of the etiology of diabetes mellitus. *Diabetes* 1975, 24(8):762-765.
179. Brand-Miller JC, Holt SH, Pawlak DB, McMillan J: Glycemic index and obesity. *The American journal of clinical nutrition* 2002, 76(1):281S-285S.
180. Van Dam R, Visscher A, Feskens E, Verhoef P, Kromhout D: Dietary glycemic index in relation to metabolic risk factors and incidence of coronary heart disease: the Zutphen Elderly Study. *European journal of clinical nutrition* 2000, 54(9):726-731.
181. Rahelić D, Jenkins A, Božikov V, Pavić E, Jurić K, Fairgrieve C, Romić D, Kokić S, Vuksan V: Glycemic index in diabetes. *Collegium antropologicum* 2011, 35(4):1363-1368.
182. Ludwig DS: The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *Jama* 2002, 287(18):2414-2423.

183. Raatz SK, Torkelson CJ, Redmon JB, Reck KP, Kwong CA, Swanson JE, Liu C, Thomas W, Bantle JP: Reduced glycemic index and glycemic load diets do not increase the effects of energy restriction on weight loss and insulin sensitivity in obese men and women. *The Journal of nutrition* 2005, 135(10):2387-2391.
184. Alfenas RCG, Mattes RD: Influence of glycemic index/load on glycemic response, appetite, and food intake in healthy humans. *Diabetes Care* 2005, 28(9):2123-2129.
185. Foster-Powell K, Miler JB: International tables of glycemic index. *The American journal of clinical nutrition* 1995, 62(2):871S-890S.
186. Foster-Powell K, Holt SH, Brand-Miller JC: International table of glycemic index and glycemic load values: 2002. *The American journal of clinical nutrition* 2002, 76:5-56.
187. Atkinson FS, Foster-Powell K, Brand-Miller JC: International tables of glycemic index and glycemic load values: 2008. *Diabetes care* 2008, 31(12):2281-2283.
188. FAO/WHO: Carbohydrates in human nutrition. In: Report of a Joint FAO/WHO Expert Consultation FAO Food and Nutrition Paper no 66. Rome: FAO; 1998.
189. Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, Jenkins AL, Axelsen M: Glycemic index: overview of implications in health and disease. *The American journal of clinical nutrition* 2002, 76(1):266S-273S.
190. Buyken AE, Toeller M, Heitkamp G, Karamanos B, Rottiers R, Muggeo M, Fuller JH: Glycemic index in the diet of European outpatients with type 1 diabetes: relations to glycosylated hemoglobin and serum lipids. *The American journal of clinical nutrition* 2001, 73(3):574-581.
191. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of non—insulin-dependent diabetes mellitus in women. *Jama* 1997, 277(6):472-477.
192. Brand-Miller J, Foster-Powell K, Burani J: *The New Glucose Revolution Low GI Guide to Diabetes: The Only Authoritative Guide to Managing Diabetes Using the Glycemic Index*: Da Capo Press; 2005.
193. Wolever TM, Jenkins DJ, Jenkins AL, Josse RG: The glycemic index: methodology and clinical implications. *The American journal of clinical nutrition* 1991, 54(5):846-854.

194. Brouns F, Bjorck I, Frayn KN, Gibbs AL, Lang V, Slama G, Wolever TMS: Glycaemic index methodology. *Nutrition research reviews* 2005, 18(01):145-171.
195. Henry CJ, Lightowler HJ, Strik CM, Renton H, Hails S: Glycaemic index and glycaemic load values of commercially available products in the UK. *The British journal of nutrition* 2005, 94(6):922-930.
196. Arvidsson-Lenner R, Asp N-G, Axelsen M, Bryngelsson S, Haapa E, Anette J, Karlström B, Raben A, Sohlström A, Thorsdottir I: Glycaemic index. Relevance for health, dietary recommendations and food labelling. *Food & Nutrition Research* 2004, 48(2):84-94.
197. Fryar CD, Carroll MD, Ogden CL: Prevalence of Overweight, Obesity, and Extreme Obesity Among Adults: United States, 1960–1962 Through 2011–2012. In.; 2015.
198. WHO: Obesity: preventing and managing the global epidemic. In: WHO Technical Report Series 894. Edited by Consultation W. Geneva: World Health Organization; 2000.
199. Rytting KR, Flaten H, Rossner S: Long-term effects of a very low calorie diet (Nutrilett) in obesity treatment. A prospective, randomized, comparison between VLCD and a hypocaloric diet+behavior modification and their combination. *International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity* 1997, 21(7):574-579.
200. Hopkins M, Finlayson G, Duarte C, Whybrow S, Ritz P, Horgan G, Blundell J, Stubbs R: Modelling the associations between fat-free mass, resting metabolic rate and energy intake in the context of total energy balance. *International journal of obesity* 2015.
201. Karl J, Schaefer E, Fuss P, Das SK, Saltzman E, Roberts S: Dietary glycemic index does not influence adaptation of resting energy expenditure during weight loss (371.7). *The FASEB Journal* 2014, 28(1 Supplement):371.377.
202. Kong AP, Choi KC, Chan RS, Lok K, Ozaki R, Li AM, Ho CS, Chan MH, Sea M, Henry CJ: A randomized controlled trial to investigate the impact of a low glycemic index (GI) diet on body mass index in obese adolescents. *BMC public health* 2014, 14(1):180.
203. Juanola-Falgarona M, Salas-Salvadó J, Ibarrola-Jurado N, Rabassa-Soler A, Díaz-López A, Guasch-Ferré M, Hernández-Alonso P, Balanza R, Bulló M: Effect of the glycemic index of the diet on weight loss, modulation of satiety,

inflammation, and other metabolic risk factors: a randomized controlled trial. *The American journal of clinical nutrition* 2014, 100(1):27-35.

204. Aller E, Larsen TM, Claus H, Lindroos AK, Kafatos A, Pfeiffer A, Martinez J, Handjieva-Darlenska T, Kunesova M, Stender S: Weight loss maintenance in overweight subjects on ad libitum diets with high or low protein content and glycemic index: the DIOGENES trial 12-month results. *International journal of obesity* 2014.
205. Wolfsdorf JI, Sperling MA: *Diabetes Mellitus*. In., edn. Berlin, Heidelberg: Springer Berlin Heidelberg; 2012: 3759-3789.
206. Inzucchi SE, Bergenstal RM, Buse JB, Diamant M, Ferrannini E, Nauck M, Peters AL, Tsapas A, Wender R, Matthews DR: Management of hyperglycemia in type 2 diabetes: a patient-centered approach position statement of the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetes care* 2012, 35(6):1364-1379.
207. Estruch R, Ros E, Salas-Salvadó J, Covas M-I, Corella D, Arós F, Gómez-Gracia E, Ruiz-Gutiérrez V, Fiol M, Lapetra J: Primary prevention of cardiovascular disease with a Mediterranean diet. *New England Journal of Medicine* 2013, 368(14):1279-1290.
208. Gögebakan Ö, Kohl A, Osterhoff MA, van Baak MA, Jebb SA, Papadaki A, Martinez JA, Handjieva-Darlenska T, Hlavaty P, Weickert MO: Effects of Weight Loss and Long-Term Weight Maintenance With Diets Varying in Protein and Glycemic Index on Cardiovascular Risk Factors The Diet, Obesity, and Genes (DiOGenes) Study: A Randomized, Controlled Trial. *Circulation* 2011, 124(25):2829-2838.
209. Mirrahimi A, Chiavaroli L, Srichaikul K, Augustin LS, Sievenpiper JL, Kendall CW, Jenkins DJ: The role of glycemic index and glycemic load in cardiovascular disease and its risk factors: a review of the recent literature. *Current atherosclerosis reports* 2014, 16(1):1-10.
210. de Vries PG: Stratified random sampling. In: *Sampling Theory for Forest Inventory*. edn.: Springer; 1986: 31-55.
211. Brožek J: *Body Measurements and Human Nutrition*. In., edn.: Wayne University Press; 1956: 78-81.
212. Chen L, Magliano DJ, Zimmet PZ: The worldwide epidemiology of type 2 diabetes mellitus, present and future perspectives. *Nature reviews Endocrinology* 2012, 8(4):228-236.

213. WHO: Physical Status: The Use and Interpretation of Anthropometry. In: WHO Technical Report Series. Geneva: World Health Organization; 1995: 22-28.
214. NIH: Bioelectrical impedance analysis in body composition measurement: National Institutes of Health Technology Assessment Conference Statement. *The American journal of clinical nutrition* 1996, 64(3 Suppl):524s-532s.
215. Chinn S, Rona RJ: International definitions of overweight and obesity for children: a lasting solution? *Annals of human biology* 2002, 29(3):306-313.
216. Després J-P, Nadeau A, Tremblay A, Ferland M, Moorjani S, Lupien PJ, Thériault G, Pinault S, Bouchard C: Role of deep abdominal fat in the association between regional adipose tissue distribution and glucose tolerance in obese women. *Diabetes* 1989, 38(3):304-309.
217. Van der Kooy K, Seidell JC: Techniques for the measurement of visceral fat: a practical guide. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity* 1993, 17(4):187-196.
218. Lohman TG: Skinfolts and body density and their relation to body fatness: a review. *Human Biology* 1981:181-225.
219. Jones PR, Hunt MJ, Brown TP, Norgan NG: Waist-hip circumference ratio and its relation to age and overweight in British men. *Human nutrition Clinical nutrition* 1986, 40(3):239-247.
220. Suk S-H, Sacco RL, Boden-Albala B, Cheun JF, Pittman JG, Elkind MS, Paik MC: Abdominal obesity and risk of ischemic stroke the Northern manhattan stroke study. *Stroke* 2003, 34(7):1586-1592.
221. Canoy D, Boekholdt SM, Wareham N, Luben R, Welch A, Bingham S, Buchan I, Day N, Khaw K-T: Body Fat Distribution and Risk of Coronary Heart Disease in Men and Women in the European Prospective Investigation Into Cancer and Nutrition in Norfolk Cohort A Population-Based Prospective Study. *Circulation* 2007, 116(25):2933-2943.
222. WHO: Waist circumference and waist-hip ratio: Report of a WHO expert consultation, Geneva, 8-11 December 2008: World Health Organization; 2011.
223. Zhou JY, Ge H, Zhu MF, Wang LJ, Chen L, Tan YZ, Chen YM, Zhu HL: Neck circumference as an independent predictive contributor to cardio-metabolic syndrome. *Cardiovascular diabetology* 2013, 12:76.

224. Ben-Noun L, Sohar E, Laor A: Neck circumference as a simple screening measure for identifying overweight and obese patients. *Obesity research* 2001, 9(8):470-477.
225. Fernández MAL, Delchevalerie P, Van Herp M: Accuracy of MUAC in the detection of severe wasting with the new WHO growth standards. *Pediatrics* 2010, 126(1):e195-e201.
226. Jeyakumar A, Ghugre P, Gadhawe S: Mid-Upper-Arm Circumference (MUAC) as a Simple Measure to Assess the Nutritional Status of Adolescent Girls as Compared With BMI. *ICAN: Infant, Child, & Adolescent Nutrition* 2013, 5(1):22-25.
227. James WP, Mascie-Taylor GC, Norgan NG, Bistrrian BR, Shetty PS, Ferro-Luzzi A: The value of arm circumference measurements in assessing chronic energy deficiency in Third World adults. *European journal of clinical nutrition* 1994, 48(12):883-894.
228. Borkan GA, Hults DE, Cardarelli J, Burrows BA: Comparison of ultrasound and skinfold measurements in assessment of subcutaneous and total fatness. *American journal of physical anthropology* 1982, 58(3):307-313.
229. Martin AD, Ross WD, Drinkwater DT, Clarys JP: Prediction of body fat by skinfold caliper: assumptions and cadaver evidence. *International journal of obesity* 1984, 9:31-39.
230. Lohman TG, Roche AF, Martorell R: *Anthropometric standardization reference manual*. 1988.
231. Durnin J, De Bruin H, Feunekes GIJ: Skinfold thicknesses: is there a need to be very precise in their location? *British Journal of Nutrition* 1997, 77(01):3-7.
232. Durnin J, Womersley J: Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition* 1974, 32(01):77-97.
233. Vaught JB: Blood collection, shipment, processing, and storage. *Cancer Epidemiology Biomarkers & Prevention* 2006, 15(9):1582-1584.
234. WHO: *WHO guidelines on drawing blood: best practices in phlebotomy*. 2010.
235. DiMagno EP, Corle D, O'Brien JF, Masnyk IJ, Go VL, Aamodt R: Effect of long-term freezer storage, thawing, and refreezing on selected constituents of serum. In: 1989 1989: Elsevier; 1989: 1226-1234.

236. Bowling JL, Katayev A: An Evaluation of the Roche Cobas c 111. *Lab Medicine* 2010, 41(7):398-402.
237. Després J-P, Lamarche B, Mauriège P, Cantin B, Dagenais GR, Moorjani S, Lupien P-J: Hyperinsulinemia as an independent risk factor for ischemic heart disease. *New England Journal of Medicine* 1996, 334(15):952-958.
238. Halperin RO, Sesso HD, Ma J, Buring JE, Stampfer MJ, Gaziano JM: Dyslipidemia and the risk of incident hypertension in men. *Hypertension* 2006, 47(1):45-50.
239. Smith Jr SC: Multiple risk factors for cardiovascular disease and diabetes mellitus. *The American journal of medicine* 2007, 120(3):S3-S11.
240. NIH: Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents: Summary Report. *Pediatrics* 2011, 128(Supplement 5):S213-S256.
241. Ginsberg HN, Zhang Y-L, Hernandez-Ono A: Regulation of plasma triglycerides in insulin resistance and diabetes. *Archives of medical research* 2005, 36(3):232-240.
242. Kekki M, Nikkilä EA: Plasma triglyceride metabolism in the adult nephrotic syndrome. *European journal of clinical investigation* 1971, 1(5):345-351.
243. Siedel J, Schmuck R, Staepels J, Town MH: Long term stable, liquid ready-to-use monoreagent for the enzymatic assay of serum or plasma triglycerides (GPO-PAP method). *AACC Meeting Abstract* 34. *Clin Chem* 1993, 39:1127.
244. Tietz NW: *Clinical guide to laboratory tests*: WB Saunders Co; 1995.
245. Brown MS, Goldstein JL: Lipoprotein metabolism in the macrophage: implications for cholesterol deposition in atherosclerosis. *Annual review of biochemistry* 1983, 52(1):223-261.
246. Allain GC, Poon LS, Chan CSG, Richmond W, Fu PC: Quantitative determination of cholesterol using enzymatic colorimetric method. *Clin Chem* 1974, 20:470-475.
247. NIH: *Recommendations for Improving Cholesterol Measurement: A Report From the Laboratory Standardization Panel of the National Cholesterol Education Program*. In.: US Department of Health and Human Services, Public Health Service, National Institutes of Health; 1990.
248. Itabe H: Oxidative modification of LDL: its pathological role in atherosclerosis. *Clinical reviews in allergy & immunology* 2009, 37(1):4-11.

249. Bachorik PS, Ross JW: National Cholesterol Education Program recommendations for measurement of low-density lipoprotein cholesterol: executive summary. The National Cholesterol Education Program Working Group on Lipoprotein Measurement. *Clinical chemistry* 1995, 41(10):1414-1420.
250. Cooper GR, Myers GL, Smith SJ, Sampson EJ: Standardization of lipid, lipoprotein, and apolipoprotein measurements. *Clinical chemistry* 1987, 34(8B):B95-105.
251. Barter P, Gotto AM, LaRosa JC, Maroni J, Szarek M, Grundy SM, Kastelein JJP, Bittner V, Fruchart J-C: HDL cholesterol, very low levels of LDL cholesterol, and cardiovascular events. *New England Journal of Medicine* 2007, 357(13):1301-1310.
252. Sugiuchi H, Uji Y, Okabe H, Irie T, Uekama K, Kayahara N, Miyauchi K: Direct measurement of high-density lipoprotein cholesterol in serum with polyethylene glycol-modified enzymes and sulfated alpha-cyclodextrin. *Clinical Chemistry* 1995, 41(5):717-723.
253. Kimberly MM, Leary ET, Cole TG, Waymack PP: Selection, validation, standardization, and performance of a designated comparison method for HDL-cholesterol for use in the cholesterol reference method laboratory network. *Clinical chemistry* 1999, 45(10):1803-1812.
254. Tietz: clinical guide to laboratory tests, 4th edn. Philadelphia: Saunders/Elsevier St. Louis; 2006.
255. ADA: Standards of Medical Care in Diabetes—2016 Abridged for Primary Care Providers. *Clinical Diabetes* 2016, 34(1):3-21.
256. Folsom AR, Aleksic N, Catellier D, Juneja HS, Wu KK: C-reactive protein and incident coronary heart disease in the Atherosclerosis Risk In Communities (ARIC) study. *American heart journal* 2002, 144(2):233-238.
257. Price CP, Trull AK, Berry D, Gorman EG: Development and validation of a particle-enhanced turbidimetric immunoassay for C-reactive protein. *Journal of immunological methods* 1987, 99(2):205-211.
258. Ridker PM: Clinical application of C-reactive protein for cardiovascular disease detection and prevention. *Circulation* 2003, 107(3):363-369.
259. Von Schenck H, Falkensson M, Lundberg B: Evaluation of "HemoCue," a new device for determining hemoglobin. *Clinical chemistry* 1986, 32(3):526-529.

260. Burger S, Pierre-Louis J: A procedure to estimate the accuracy and reliability of HemoCue measurements of survey workers. Washington: ILSI 2003.
261. WHO: Haemoglobin Concentrations for the Diagnosis of Anaemia and Assessment of Severity. Vitamin and Mineral Nutrition Information System (WHO/NMH/NHD/MNM/11.1). In. Geneva: World Health Organization; 2011.
262. Little RR, Sacks DB: HbA1c: how do we measure it and what does it mean? *Current Opinion in Endocrinology, Diabetes and Obesity* 2009, 16(2):113-118.
263. WHO: Use of glycated haemoglobin (HbA1c) in diagnosis of diabetes mellitus: abbreviated report of a WHO consultation. 2011.
264. Evaluation of HemoCue HbA1c 501 system [[http://www.hemocue.com/~media/hemocue-images/hemocuedotcom-images/product-images/hba1c/nsm174int-130826-posterhemocue-hba1c-501-\(2\).pdf?la=en](http://www.hemocue.com/~media/hemocue-images/hemocuedotcom-images/product-images/hba1c/nsm174int-130826-posterhemocue-hba1c-501-(2).pdf?la=en)]
265. Bartges JW, Willis AM, Polzin DJ: Hypertension and renal disease. *The Veterinary clinics of North America Small animal practice* 1996, 26(6):1331-1345.
266. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, Jones DW, Kurtz T, Sheps SG, Roccella EJ: Recommendations for blood pressure measurement in humans and experimental animals part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension* 2005, 45(1):142-161.
267. El Assaad MA, Topouchian JA, Darne BM, Asmar RG: Validation of the Omron HEM-907 device for blood pressure measurement. *American Journal of Hypertension* 2002, 15(S3):87A-87A.
268. White WB, Anwar YA: Evaluation of the overall efficacy of the Omron office digital blood pressure HEM-907 monitor in adults. *Blood pressure monitoring* 2001, 6(2):107-110.
269. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, Jones DW, Materson BJ, Oparil S, Wright Jr JT: The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. *Jama* 2003, 289(19):2560-2571.
270. FAO/WHO: Carbohydrates in Human Nutrition: Report of a Joint FAO/WHO Expert Consultation; 1998.

271. Stork ADM, Kemperman H, Erkelens DW, Veneman TF: Comparison of the accuracy of the HemoCue glucose analyzer with the Yellow Springs Instrument glucose oxidase analyzer, particularly in hypoglycemia. *European journal of endocrinology* 2005, 153(2):275-281.
272. Sawaya WN, Al-Jebrin A, Salji JP, Ayaz M, Khalil JK: Cholesterol contents and fatty acid composition of selected Saudi Arabian dishes. *Nutrition reports international (USA)* 1985.
273. Al Nagdy SA, Abd-El Ghani SA, Abdel-Rahman MO: Chemical assessment of some traditional Qatari dishes. *Food chemistry* 1994, 49(3):261-264.
274. Dashti BH, Al-Awadi F, Khalafawi MS, Al-Zenki S, Sawaya W: Nutrient contents of some traditional Kuwaiti dishes: proximate composition, and phytate content. *Food chemistry* 2001, 74(2):169-175.
275. Anderson JW, Lin W-J, Ward K: Composition of foods commonly used in diets for persons with diabetes. *Diabetes care* 1978, 1(5):293-302.
276. AOAC, Horwitz W: *Official Methods of Analysis of the AOAC International*. Washington, DC: Association of Official Analytical Chemists; 2003.
277. Bakircioglu D, Kurtulus YB, Ucar G: Determination of some traces metal levels in cheese samples packaged in plastic and tin containers by ICP-OES after dry, wet and microwave digestion. *Food and Chemical Toxicology* 2011, 49(1):202-207.
278. Shreve B, Thiex N, Wolf M: *National Forage Testing Association Reference Method: Dry matter by oven drying for 3 hours at 105 C*. NFTA Reference Methods. National Forage Testing Association, Omaha, NE. In.; 2006.
279. Thiex N, Novotny L, Crawford A: Determination of ash in animal feed: AOAC official method 942.05 revisited. *Journal of AOAC International* 2012, 95(5):1392-1397.
280. Liang S, Yang G, Ma Y: Chemical characteristics and fatty acid profile of foxtail millet bran oil. *Journal of the American Oil Chemists' Society* 2010, 87(1):63-67.
281. Sporring S, Bøwadt S, Svensmark B, Björklund E: Comprehensive comparison of classic Soxhlet extraction with Soxtec extraction, ultrasonication extraction, supercritical fluid extraction, microwave assisted extraction and accelerated solvent extraction for the determination of polychlorinated biphenyls in soil. *Journal of Chromatography A* 2005, 1090(1):1-9.

282. Loranger J, Meyer ST, Shipley B, Kattge J, Loranger H, Roscher C, Weisser WW: Predicting invertebrate herbivory from plant traits: evidence from 51 grassland species in experimental monocultures. *Ecology* 2012, 93(12):2674-2682.
283. ANKOM: Neutral Detergent Fiber in Feeds - Filter Bag Technique (for A2000 and A2000I), NDF Method 13. In. Edited by Technology A; 2013.
284. ANKOM: Analytical Methods of Dietary Fiber. In.; 2015.
285. Wheeler ML, Franz M, Barrier P, Holler H, Cronmiller N, Delahanty LM: Macronutrient and energy database for the 1995 exchange lists for meal planning: a rationale for clinical practice decisions. *Journal of the American Dietetic Association* 1996, 96(11):1167-1171.
286. Bawadi HA, Al-Shwaiyat NM, Tayyem RF, Mekary R, Tuuri G: Developing a food exchange list for Middle Eastern appetisers and desserts commonly consumed in Jordan. *Nutrition & Dietetics* 2009, 66(1):20-26.
287. Link DD, Walter PJ, Kingston HM: Wastewater standards and extraction chemistry in validation of microwave-assisted EPA method 3015A. *Environmental science & technology* 1999, 33(14):2469-2473.
288. Heckman M: Collaborative study of a copper in feeds by atomic absorption spectrophotometry. *Journal-Association of Official Analytical Chemists* 1971, 54(3):666.
289. Swami K, Judd CD, Orsini J, Yang KX, Husain L: Microwave assisted digestion of atmospheric aerosol samples followed by inductively coupled plasma mass spectrometry determination of trace elements. *Fresenius' journal of analytical chemistry* 2001, 369(1):63-70.
290. Yuan J-P, Chen F: Simultaneous separation and determination of sugars, ascorbic acid and furanic compounds by HPLC—dual detection. *Food Chemistry* 1999, 64(3):423-427.
291. Smith js, villalobos mc, kottemann cm: Quantitative determination of sugars in various food products. *Journal of Food Science* 1986, 51(5):1373-1375.
292. Sigma-Aldrich: Comparison of 37 Component FAME Standard on Four Capillary GC Columns. In: Supelco. 2015.
293. Berger S, Raman G, Vishwanathan R, Jacques PF, Johnson EJ: Dietary cholesterol and cardiovascular disease: a systematic review and meta-analysis. *The American journal of clinical nutrition* 2015:ajcn100305.

294. Indyk H: Simultaneous liquid chromatographic determination of cholesterol, phytosterols and tocopherols in foods. *Analyst* 1990, 115(12):1525-1530.
295. Essaka DC: Reversed-Phase HPLC Determination of Cholesterol in Food Items. 2007.
296. Rizzolo A, Polesello S: Chromatographic determination of vitamins in foods. *Journal of Chromatography A* 1992, 624(1):103-152.
297. Ekinci R, Kadakal C: Determination of seven water-soluble vitamins in tarhana, a traditional Turkish cereal food, by high-performance liquid chromatography. *ACTA chromatographica* 2005, 15:289.
298. Moreno P, Salvado V: Determination of eight water-and fat-soluble vitamins in multi-vitamin pharmaceutical formulations by high-performance liquid chromatography. *Journal of chromatography A* 2000, 870(1):207-215.
299. Srdjenovic B, Djordjevic-Milic V, Grujic N, Injac R, Lepojevic Z: Simultaneous HPLC determination of caffeine, theobromine, and theophylline in food, drinks, and herbal products. *Journal of chromatographic science* 2008, 46(2):144-149.
300. Erickson J: Determination of the concentration of caffeine, theobromine and gallic acid in commercial teasamples. *Concord. Coll. J of Anal Chem* 2011, 2:31-35.
301. Wolever TM: Carbohydrate and the regulation of blood glucose and metabolism. *Nutr Rev* 2003, 61(5 Pt 2):S40-S48.
302. ADFCA: A Photographic Atlas of Food Portions for the Emirate of Abu Dhabi. In. Abu Dhabi United Arab Emirates: Abu Dhabi Food Control Authority; 2014.
303. Al Dhaheri A, Al Ma'awali A, Laleye L, Washi S, Jarrar A, Al Meqbaali F, Mohamad M, Masuadi E: The effect of nutritional composition on the glycemic index and glycemic load values of selected Emirati foods. *BMC Nutrition* 2015, 1(1):4.
304. IDF: International Diabetes Federation: Diabetes Atlas, 6th edn; 2013.
305. WHO: United Arab Emirates: WHO statistical profile. In. Edited by Organization WH; 2015.
306. Ali HI, Baynouna LM, Bernsen RM: Barriers and facilitators of weight management: perspectives of Arab women at risk for type 2 diabetes. *Health & Social Care in the Community* 2010, 18(2):219-228.

307. Hajat C, Shather Z: Prevalence of metabolic syndrome and prediction of diabetes using IDF versus ATPIII criteria in a Middle East population. *Diabetes research and clinical practice* 2012, 98(3):481.
308. Al-Sarraj T, Saadi H, Volek JS, Fernandez ML: Metabolic syndrome prevalence, dietary intake, and cardiovascular risk profile among overweight and obese adults 18-50 years old from the United Arab Emirates. *Metabolic syndrome and related disorders* 2010, 8(1):39-46.
309. Eapen V, Mabrouk A, Yousef S: Metabolic syndrome among the young obese in the United Arab Emirates. *Journal of tropical pediatrics* 2010, 56(5):325-328.
310. Abolfotouh MA, Al-Alwan IA, Al-Rowaily MA: Prevalence of metabolic abnormalities and association with obesity among Saudi college students. *International journal of hypertension* 2012, 2012.
311. ALNohair S: Obesity in gulf countries. *International journal of health sciences* 2014, 8(1):79.
312. Alharbi NS, Almutari R, Jones S, Al-Daghri N, Khunti K, de Lusignan S: Trends in the prevalence of type 2 diabetes mellitus and obesity in the Arabian Gulf States: Systematic review and meta-analysis. *Diabetes research and clinical practice* 2014, 106(2):e30-e33.
313. Musaiger AO, Al-Roomi K, Bader Z: Social, dietary and lifestyle factors associated with obesity among Bahraini adolescents. *Appetite* 2014, 73:197-204.
314. Selmi S, Daghsh G, Mathis S, Nahas K, Wilbur K: A program for obese youth at-risk for diabetes in Qatar. *Avicenna* 2015.
315. Musaiger AO, Al-Kandari FI, Al-Mannai M, Al-Faraj AM, Bouriki FA, Shehab FS, Al-Dabous LA, Al-Qalaf WB: Perceived barriers to weight maintenance among university students in Kuwait: the role of gender and obesity. *Environmental health and preventive medicine* 2014, 19(3):207-214.
316. Al-Kaabi J, Al-Maskari F, Afandi B, Parkar H, Nagelkerke N: Physical activity and reported barriers to activity among type 2 diabetic patients in the United Arab Emirates. *The review of diabetic studies: RDS* 2009, 6(4):271.
317. Fernandes J, Lofgren IE: Prevalence of metabolic syndrome and individual criteria in college students. *Journal of American College Health* 2011, 59(4):313-321.

318. F de Freitas Jr RW, M de Araújo MF, P Marinho NB, A de Vasconcelos HC, S Lima AC, R Pereira DC, Almeida PC, Zanetti ML, C Damasceno MM: Prevalence of the metabolic syndrome and its individual components in brazilian college students. *Journal of clinical nursing* 2013, 22(9-10):1291-1298.
319. Yi-Qun X, Cheng-Ye J: Prevalence of the metabolic syndrome in secondary school adolescents in Beijing, China. *Acta Paediatrica* 2008, 97(3):348-353.
320. González-Jiménez E, Montero-Alonso MA, Schmidt-RioValle J, García-García CJ, Padez C: Metabolic syndrome in Spanish adolescents and its association with birth weight, breastfeeding duration, maternal smoking, and maternal obesity: a cross-sectional study. *European journal of nutrition* 2014, 54(4):589-597.
321. Aounallah-Skhiri H, El Ati J, Traissac P, Gartner A, Ben Gharbia H, Hsairi M, Ben Rayana C, Delpeuch F: Prevalence of metabolic syndrome and associated behavioural factors in Tunisian adolescents. *Obesity Facts* 2015, 8(Suppl. 1).
322. Bhalavi V, Deshmukh PR, Goswami K, Garg N: Prevalence and correlates of metabolic syndrome in the adolescents of Rural Wardha. *Indian journal of community medicine: official publication of Indian Association of Preventive & Social Medicine* 2015, 40(1):43.
323. Smitha F, Meenakshi J, Padma R, Multani S: Survey and evaluation of various epidemiological factors in a multiethnic Diabetic Population in Ras Al-Khaimah, UAE. *Indian Journal of Pharmacy Practice* 2011, 4(1).
324. Hamer M, Stamatakis E, Steptoe A: Effects of substituting sedentary time with physical activity on metabolic risk. 2014.
325. Ahmed HM, Blaha MJ, Nasir K, Rivera JJ, Blumenthal RS: Effects of physical activity on cardiovascular disease. *The American journal of cardiology* 2012, 109(2):288-295.
326. Kant A, Whitley M, Graubard B: Away from home meals: associations with biomarkers of chronic disease and dietary intake in American adults, NHANES 2005–2010. *International journal of obesity* 2015, 39(5):820-827.
327. Elmagd MA, Mossa AH, Sami MM, El-Marsafawy TS, Al Jadaan O, Eldin MS: The Impact of Physical Activity on the Academic Performance among Medical and Health Sciences Students: A Cross Sectional Study from RAKMHSU-Ras Alkhaimah-UAE. 2015.

328. Mabry RM, Reeves MM, Eakin EG, Owen N: Gender differences in prevalence of the metabolic syndrome in Gulf Cooperation Council Countries: a systematic review. *Diabetic medicine : a journal of the British Diabetic Association* 2010, 27(5):593-597.
329. Macy EM, Hayes TE, Tracy RP: Variability in the measurement of C-reactive protein in healthy subjects: implications for reference intervals and epidemiological applications. *Clinical chemistry* 1997, 43(1):52-58.
330. den Engelsen C, Koekkoek PS, Gorter KJ, van den Donk M, Salomé PL, Rutten GE: High-sensitivity C-reactive protein to detect metabolic syndrome in a centrally obese population: a cross-sectional analysis. *Cardiovascular diabetology* 2012, 11(1):1.
331. Guo S: Insulin signaling, resistance, and metabolic syndrome: insights from mouse models into disease mechanisms. *Journal of Endocrinology* 2014, 220(2):T1-T23.
332. Kim C-H, Kim H-K, Bae S-J, Park J-Y, Lee K-U: Discordance between fasting glucose-based and hemoglobin A1c-based diagnosis of diabetes mellitus in Koreans. *Diabetes research and clinical practice* 2011, 91(1):e8-e10.
333. Alqahtani N, Khan WAG, Alhumaidi MH, Ahmed YAAR: Use of glycated hemoglobin in the diagnosis of diabetes mellitus and pre-diabetes and role of fasting plasma glucose, oral glucose tolerance test. *International journal of preventive medicine* 2013, 4(9).
334. Kharroubi AT, Darwish HM, Al-Halaweh AIA, Khammash UM: Evaluation of glycated hemoglobin (HbA1c) for diagnosing type 2 diabetes and prediabetes among Palestinian Arab population. *PloS one* 2014, 9(2):e88123.
335. Jeon JY, Ko S-H, Kwon H-S, Kim NH, Kim JH, Kim CS, Song K-H, Won JC, Lim S, Choi SH: Prevalence of diabetes and prediabetes according to fasting plasma glucose and HbA1c. *Diabetes & metabolism journal* 2013, 37(5):349-357.
336. Rosella LC, Lebenbaum M, Fitzpatrick T, Zuk A, Booth GL: Prevalence of prediabetes and undiagnosed diabetes in Canada (2007–2011) according to fasting plasma glucose and HbA1c screening criteria. *Diabetes care* 2015, 38(7):1299-1305.
337. Hu Y, Liu W, Chen Y, Zhang M, Wang L, Zhou H, Wu P, Teng X, Dong Y, wen Zhou J: Combined use of fasting plasma glucose and glycated hemoglobin A1c in the screening of diabetes and impaired glucose tolerance. *Acta diabetologica* 2010, 47(3):231-236.

338. McCane D, Hanson RL, Charles M-A, Jacobsson LT, Pettitt DD, Bennett PH, Knowler WC: Comparison of tests for glycated haemoglobin and fasting and two hour plasma glucose concentrations as diagnostic methods for diabetes. *Bmj* 1994, 308(6940):1323-1328.
339. Jain J, Rao T, Desmukh P, Banait S: Prevalence and Correlates of Metabolic Syndrome in Young Population: A Cross Sectional Study. *J Diabetes Metab* 2015, 6(503):2.
340. Kim D-i, Kim JY, Lee MK, Lee H-D, Lee J-W, Jeon JY: The relationship between fitness, BMI and risk factors of metabolic syndrome among university students in Korea. *The Korean Journal of Obesity* 2012, 21(2):99-107.
341. Kifley A, Wang JJ, Cugati S, Wong TY, Mitchell P: Retinal vascular caliber, diabetes, and retinopathy. *American journal of ophthalmology* 2007, 143(6):1024-1026.
342. Caramori ML, Mauer M: Diabetes and nephropathy. *Current opinion in nephrology and hypertension* 2003, 12(3):273-282.
343. Ahamed MM, Banji O: A review on diabetic neuropathy and nephropathy. *International Journal of Pharmaceutical Sciences and Research* 2012, 3(2):300.
344. El Mugamer IT, Ali ZAS, Hossain MM, Pugh RN: Diabetes, obesity and hypertension in urban and rural people of bedouin origin in the United Arab Emirates. *The Journal of tropical medicine and hygiene* 1995, 98(6):407-415.
345. Saadi H, Carruthers SG, Nagelkerke N, Al-Maskari F, Afandi B, Reed R, Lukic M, Nicholls MG, Kazam E, Algawi K: Prevalence of diabetes mellitus and its complications in a population-based sample in Al Ain, United Arab Emirates. *Diabetes research and clinical practice* 2007, 78(3):369-377.
346. Wennehorst K, Mildenstein K, Saliger B, Tigges C, Diehl H, Keil T, Englert H: A Comprehensive Lifestyle Intervention to Prevent Type 2 Diabetes and Cardiovascular Diseases: the German CHIP Trial. *Prevention Science* 2016:1-12.
347. Hensrud D: Dietary treatment and long-term weight loss and maintenance in type 2 diabetes. *Obesity research* 2001, 9(Suppl 4):348S-353S.
348. Ramachandran A, Snehalatha C, Mary S, Mukesh B, Bhaskar A, Vijay V: The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia* 2006, 49(2):289-297.

349. Jenkins DA, Jenkins A, Wolever TS, Josse R, Wong G: The glycaemic response to carbohydrate foods. *The Lancet* 1984, 324(8399):388-391.
350. Markovic TP, Muirhead R, Overs S, Ross GP, Louie JCY, Kizirian N, Denyer G, Petocz P, Hyett J, Brand-Miller JC: Randomized controlled trial investigating the effects of a low-glycemic index diet on pregnancy outcomes in women at high risk of gestational diabetes mellitus: the GI Baby 3 Study. *Diabetes care* 2016, 39(1):31-38.
351. Musaiger A: Food composition tables for Kingdom of Bahrain. Food composition tables for Kingdom of Bahrain 2011.
352. ElObeid T, Phoboo S, Magdad Z: proximate and mineral composition of indigenous qatari dishes: comparative study with similar middle eastern dishes. *Journal of food chemistry and nutrition* 2015, 3(1):27-34.
353. Ali HI, Ng SW, Zaghloul S, Harrison GG, Qazaq HS, El Sadig M, Yeatts K: High proportion of 6 to 18-year-old children and adolescents in the United Arab Emirates are not meeting dietary recommendations. *Nutrition research* 2013, 33(6):447-456.
354. Al-Rahawi DA: Intakes of Calcium and Vitamin D and the Relationship to Bone Health: Incidence and Prevalence of Osteoporosis: University of Cincinnati; 2008.
355. Sadiya A, Ahmed SM, Skaria S, Abusnana S: Vitamin D status and its relationship with metabolic markers in persons with obesity and type 2 diabetes in the UAE: a cross-sectional study. *Journal of diabetes research* 2014, 2014.
356. Trautvetter U, Jahreis G, Kiehntopf M, Gleit M: Consequences of a high phosphorus intake on mineral metabolism and bone remodeling in dependence of calcium intake in healthy subjects—a randomized placebo-controlled human intervention study. *Nutrition journal* 2016, 15(1):1.
357. Chang AR, Lazo M, Appel LJ, Gutiérrez OM, Grams ME: High dietary phosphorus intake is associated with all-cause mortality: results from NHANES III. *The American journal of clinical nutrition* 2014, 99(2):320-327.
358. Dhingra R, Sullivan LM, Fox CS, Wang TJ, D'Agostino RB, Gaziano JM, Vasan RS: Relations of serum phosphorus and calcium levels to the incidence of cardiovascular disease in the community. *Archives of internal medicine* 2007, 167(9):879-885.
359. Go A, Bauman M, Coleman King S, Fonarow G, Lawrence W, Williams K, Sanchez E: AHA/ACC/CDC science advisory: an effective approach to high

- blood pressure control a science advisory from the American Heart Association, the American College of Cardiology, and the Centers for Disease Control and Prevention. *J Am Coll Cardiol* 2013, 11:19.
360. Motlagh B, O'Donnell M, Yusuf S: Prevalence of cardiovascular risk factors in the Middle East: a systematic review. *European Journal of Cardiovascular Prevention & Rehabilitation* 2009, 16(3):268-280.
 361. Lloyd-Jones DM, Wilson PW, Larson MG, Beiser A, Leip EP, D'Agostino RB, Levy D: Framingham risk score and prediction of lifetime risk for coronary heart disease. *The American journal of cardiology* 2004, 94(1):20-24.
 362. Baynouna LM, Revel AD, Nagelkerke NJ, Jaber TM, Omar AO, Ahmed NM, Nazirudeen MK, Al Sayed MF, Nour FA, Abdouni S: Associations of cardiovascular risk factors in Al Ain, United Arab Emirates. *Cardiovascular diabetology* 2009, 8:21.
 363. Aaron KJ, Sanders PW: Role of dietary salt and potassium intake in cardiovascular health and disease: a review of the evidence. In: *Mayo Clinic Proceedings: 2013: Elsevier; 2013: 987-995.*
 364. Castro H, Raij L: Potassium in hypertension and cardiovascular disease. In: *Seminars in nephrology: 2013: Elsevier; 2013: 277-289.*
 365. Guerrero-Romero F, Rodriguez-Moran M: Low serum magnesium levels and metabolic syndrome. *Acta diabetologica* 2002, 39(4):209-213.
 366. He K, Song Y, Belin RJ, Chen Y: Magnesium intake and the metabolic syndrome: epidemiologic evidence to date. *Journal of the cardiometabolic syndrome* 2006, 1(5):351-355.
 367. Simmons D, Joshi S, Shaw J: Hypomagnesaemia is associated with diabetes: Not pre-diabetes, obesity or the metabolic syndrome. *Diabetes research and clinical practice* 2010, 87(2):261-266.
 368. Song Y, Manson JE, Buring JE, Liu S: Dietary magnesium intake in relation to plasma insulin levels and risk of type 2 diabetes in women. *Diabetes care* 2004, 27(1):59-65.
 369. Joffres M, Reed D, Yano K: Relationship of magnesium intake and other dietary factors to blood pressure: the Honolulu heart study. *The American journal of clinical nutrition* 1987, 45(2):469-475.
 370. Annapurna D, Sujatha P, Sudha J: A study of oral magnesium supplementation in patients with essential hypertension. *Hypertension* 2015, 4(10):02.

371. Guerrero-Romero F, Simental-Mendía L, Hernández-Ronquillo G, Rodríguez-Morán M: Oral magnesium supplementation improves glycaemic status in subjects with prediabetes and hypomagnesaemia: A double-blind placebo-controlled randomized trial. *Diabetes & metabolism* 2015, 41(3):202-207.
372. Song Y, Ridker PM, Manson JE, Cook NR, Buring JE, Liu S: Magnesium intake, C-reactive protein, and the prevalence of metabolic syndrome in middle-aged and older US women. *Diabetes care* 2005, 28(6):1438-1444.
373. Sultan AH: Anemia among female college students attending the University of Sharjah, UAE: prevalence and classification. *The Journal of the Egyptian Public Health Association* 2006, 82(3-4):261-271.
374. Miller CJ, Dunn EV, Abdouni SF, Shaheen HM, Ullah MS: Factors associated with iron depletion and iron deficiency anemia among Arabic preschool children of the United Arab Emirates. *Saudi medical journal* 2004, 25(7):843-847.
375. Hambidge M: Human zinc deficiency. *The Journal of nutrition* 2000, 130(5):1344S-1349S.
376. Al-Dallal ZS, Hussain KM: Impact of the National Flour Fortification Program on the Prevalence of Iron Deficiency and Anemia among Women at Reproductive Age in the Kingdom of Bahrain.
377. Reddy MB, Love M: The impact of food processing on the nutritional quality of vitamins and minerals. In: *Impact of processing on food safety*. edn.: Springer; 1999: 99-106.
378. Muhairi SJ, Mehairi AE, Khouri AA, Naqbi MM, Maskari FA, Al Kaabi J, Al Dhaheri AS, Nagelkerke N, Shah SM: Vitamin D deficiency among healthy adolescents in Al Ain, United Arab Emirates. *BMC public health* 2013, 13(1):1.
379. Al Anouti F, Thomas J, Abdel-Wareth L, Rajah J, Grant WB, Haq A: Vitamin D deficiency and sun avoidance among university students at Abu Dhabi, United Arab Emirates. *Dermato-endocrinology* 2011, 3(4):235-239.
380. Calvo MS, Whiting SJ: Survey of current vitamin D food fortification practices in the United States and Canada. *The Journal of steroid biochemistry and molecular biology* 2013, 136:211-213.
381. Lips P, Gielen E, van Schoor NM: Vitamin D supplements with or without calcium to prevent fractures. *BoneKEy reports* 2014, 3.

382. Thompson J, Biggs B-A, Pasricha S-R: Effects of daily iron supplementation in 2-to 5-year-old children: systematic review and meta-analysis. *Pediatrics* 2013, 131(4):739-753.
383. Hruby A, Hu FB: Saturated fat and heart disease: The latest evidence. *Lipid Technology* 2016, 28(1):7-12.
384. McGuire S: Scientific Report of the 2015 Dietary Guidelines Advisory Committee. Washington, DC: US Departments of Agriculture and Health and Human Services, 2015. *Advances in Nutrition: An International Review Journal* 2016, 7(1):202-204.
385. WHO: Promoting a healthy diet for the WHO Eastern Mediterranean Region: user-friendly guide; 2012.
386. Mozaffarian D, Aro A, Willett W: Health effects of trans-fatty acids: experimental and observational evidence. *European journal of clinical nutrition* 2009, 63:S5-S21.
387. Schärer M, Schulthess G: [Egg intake and cardiovascular risk]. *Therapeutische Umschau Revue thérapeutique* 2005, 62(9):611-613.
388. Stanhope KL, Havel PJ: Fructose consumption: recent results and their potential implications. *Annals of the New York Academy of Sciences* 2010, 1190:15-24.
389. Faeh D, Minehira K, Schwarz J-M, Periasamy R, Park S, Tappy L: Effect of fructose overfeeding and fish oil administration on hepatic de novo lipogenesis and insulin sensitivity in healthy men. *Diabetes* 2005, 54(7):1907-1913.
390. Bray GA, Nielsen SJ, Popkin BM: Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *The American journal of clinical nutrition* 2004, 79(4):537-543.
391. Organization WH: Sugars Intake for Adults and Children. Guideline. World Health Organization: Geneva, Switzerland 2015.
392. Welsh JA, Sharma AJ, Grellinger L, Vos MB: Consumption of added sugars is decreasing in the United States. *The American journal of clinical nutrition* 2011, 94(3):726-734.
393. Pirasath S, Thayaananthan K, Balakumar S, Arasaratnam V: Effect of dietary curries on the glycaemic index. *Ceylon Medical Journal* 2010, 55(4).
394. Perera M, Sivakanesan R, Abeysekara D, Sarananda K: Effect of Vegetable Mixed Curry on Glycaemic Index and Glycaemic Load of Soy Flour

- Incorporated Traditional Sri Lankan Breakfast Foods in Healthy Adults. *Pakistan Journal of Nutrition* 2014, 13(11):616.
395. Trinidad TP, Valdez DH, Loyola AS, Mallillin AC, Askali FC, Castillo JC, Masa DB: Glycaemic index of different coconut (*Cocos nucifera*)-flour products in normal and diabetic subjects. *British Journal of Nutrition* 2003, 90(03):551-556.
 396. Juansang J, Puttanlek C, Rungsardthong V, Pucha-arnon S, Uttapap D: Effect of gelatinisation on slowly digestible starch and resistant starch of heat-moisture treated and chemically modified canna starches. *Food Chem* 2012, 131(2):500-507.
 397. Lehmann U, Robin F: Slowly digestible starch - its structure and health implications: a review. *Trends Food Sci Technol* 2007, 18(7):346-355.
 398. Ek KL, Brand-Miller J, Copeland L: Glycemic effect of potatoes. *Food Chem* 2012, 133:1230-1240.
 399. Englyst HN, Veenstra J, Hudson GJ: Measurement of rapidly available glucose (RAG) in plant foods: a potential in vitro predictor of the glycaemic response. *British Journal of Nutrition* 1996, 75(03):327-337.
 400. Frei M, Siddhuraju P, Becker K: Studies on the in vitro starch digestibility and the glycemic index of six different indigenous rice cultivars from the Philippines. *Food Chemistry* 2003, 83(3):395-402.
 401. Miller JB, Pang E, Bramall L: Rice: a high or low glycemic index food? *The American journal of clinical nutrition* 1992, 56(6):1034-1036.
 402. Kaur B, Ranawana V, Henry J: The Glycemic Index of Rice and Rice Products: A Review, and Table of GI Values. *Critical reviews in food science and nutrition* 2016, 56(2):215-236.
 403. Chew I, Brand JC, Thorburn AW, Truswell A: Application of glycemic index to mixed meals. *The American journal of clinical nutrition* 1988, 47(1):53-56.
 404. Araya H, Pak N, Vera G, Alviña M: Digestion rate of legume carbohydrates and glycemic index of legume-based meals. *International journal of food sciences and nutrition* 2003, 54(2):119-126.
 405. Widanagamage RD, Ekanayake S, Welihinda J: Carbohydrate-rich foods: glycaemic indices and the effect of constituent macronutrients. *International journal of food sciences and nutrition* 2009, 60(sup4):215-223.

406. Thorburn AW, Brand JC: Digestion and absorption of carbohydrate in Australian Aboriginal, Pacific Island and Western Foods. 1987.
407. Perry T, Mann J, Mehalski K, Gayya C, Wilson J, Thompson C: Glycaemic index of New Zealand foods. *The New Zealand medical journal* 2000, 113(1108):140.
408. Wolever TM, Katzman-Relle L, Jenkins AL, Vuksan V, Josse RG, Jenkins DJ: Glycaemic index of 102 complex carbohydrate foods in patients with diabetes. *Nutrition research* 1994, 14(5):651-669.
409. Soh N, Brand-Miller J: The glycaemic index of potatoes: the effect of variety, cooking method and maturity. *European journal of clinical nutrition* 1999, 53(4):249-254.
410. Fernandes G, Velangi A, Wolever TM: Glycemic index of potatoes commonly consumed in North America. *Journal of the American Dietetic Association* 2005, 105(4):557-562.
411. Bahado-Singh PS, Riley CK, Wheatley AO, Lowe HI: Relationship between processing method and the glycemic indices of ten sweet potato (*Ipomoea batatas*) cultivars commonly consumed in Jamaica. *J Nutr Metab* 2011, 584832:6.
412. Tahvonen R, Hietanen R, Sihvonen J, Salminen E: Influence of different processing methods on the glycemic index of potato (Nicola). *Journal of Food Composition and Analysis* 2006, 19(4):372-378.
413. Liljeberg H, Björck I: Delayed gastric emptying rate may explain improved glycaemia in healthy subjects to a starchy meal with added vinegar. *European journal of clinical nutrition* 1998, 52(5):368-371.
414. Leeman M, Östman E, Björck I: Vinegar dressing and cold storage of potatoes lowers postprandial glycaemic and insulinaemic responses in healthy subjects. *European journal of clinical nutrition* 2005, 59(11):1266-1271.
415. Henry CJ, Lightowler HJ, Kendall FL, Storey M: The impact of the addition of toppings/fillings on the glycaemic response to commonly consumed carbohydrate foods. *European journal of clinical nutrition* 2006, 60(6):763-769.
416. Henry C, Lightowler H, Kendall F, Storey M: The impact of the addition of toppings/fillings on the glycaemic response to commonly consumed carbohydrate foods. *European journal of clinical nutrition* 2006, 60(6):763-769.

417. Flint A, Moller BK, Raben A, Pedersen D, Tetens I, Holst JJ, Astrup A: The use of glycaemic index tables to predict glycaemic index of composite breakfast meals. *The British journal of nutrition* 2004, 91(6):979-989.
418. Hatonen KA, Virtamo J, Eriksson JG, Sinkko HK, Sundvall JE, Valsta LM: Protein and fat modify the glycaemic and insulinaemic responses to a mashed potato-based meal. *The British journal of nutrition* 2011, 106(2):248-253.
419. Ali A, Al-Nassri HAS, Al-Rasasi B, Akhtar MS, Al-Belushi BS: Glycemic index and chemical composition of traditional Omani breads. *International Journal of Food Properties* 2010, 13(1):198-208.
420. HELENA GL: Sourdough Fermentation or Addition of Organic Acids or Corresponding Salts to Bread Improves Nutritional Properties of Starch in Healthy Humans¹. 1995.
421. De Angelis M, Rizzello CG, Alfonsi G, Arnault P, Cappelle S, Di Cagno R, Gobbetti M: Use of sourdough lactobacilli and oat fibre to decrease the glycaemic index of white wheat bread. *British Journal of Nutrition* 2007, 98(06):1196-1205.
422. Novotni D, Ćurić D, Bituh M, Colić Barić I, Škevin D, Čukelj N: Glycemic index and phenolics of partially-baked frozen bread with sourdough. *International journal of food sciences and nutrition* 2011, 62(1):26-33.
423. Gobbetti M, Rizzello CG, Di Cagno R, De Angelis M: How the sourdough may affect the functional features of leavened baked goods. *Food microbiology* 2014, 37:30-40.
424. Miller CJ, Dunn EV, Hashim IB: Glycemic index of 3 varieties of dates. *Saudi medical journal* 2002, 23(5):536-538.
425. Alkaabi JM, Al-Dabbagh B, Ahmad S, Saadi HF, Gariballa S, Ghazali M: Glycemic indices of five varieties of dates in healthy and diabetic subjects. *Nutrition journal* 2011, 10(1):1.

List of Publications

Conference Abstract: entitled by “The relationship between anthropometric measurements and diagnosis of pre-diabetes mellitus among United Arab Emirates University female students” Presented in the 10th international symposium on body composition, Portugal. (June, 2014) ISBN 978 972 735 200 5 (Appendix 12).

Conference Abstract: entitled by “The relationship between anthropometric measurements and diagnosis of pre-diabetes mellitus and pre-hypertension among United Arab Emirates University female students”. Presented in the 12th Asian Congress of Nutrition, Yokohama, Japan. (May 2015) (Appendix 13).

Research Article: Al Dhaheri, Ayesha S., Asila K. Al Ma’awali, Louis C. Laleye, Sidiga A. Washi, Amjad H. Jarrar, Fatima T. Al Meqbaali, Maysm N. Mohamad, and Emad M. Masuadi. "The effect of nutritional composition on the glycemic index and glycemic load values of selected Emirati foods." BMC Nutrition 1, no. 1 (2015): 1. (<http://www.biomedcentral.com/2055-0928/1/4>) (Appendix 14).

Research Article: Al Dhaheri, Ayesha S., Maysm N. Mohamad, Amjad H. Jarrar, Eric O. Ohuma, Leila Cheikh Ismail, Fatima T. Al Meqbaali, Usama Souka, and Syed M. Shah. "A Cross-Sectional Study of the Prevalence of Metabolic Syndrome among Young Female Emirati Adults." PloS one 11, no. 7 (2016): e0159378. (<http://dx.doi.org/10.1371/journal.pone.0159378>) (Appendix 15).

Research Article: entitled by “Glycaemic index and glycaemic load values of commonly consumed foods in the United Arab Emirates” [Submitted]

Research Article: entitled by “Developing an exchange list for commonly consumed foods in the United Arab Emirates” [Under Review]

Appendices

Appendix 1: Information Sheet

Anthropometric measures and their association with metabolic syndrome risk factors in Emirati young female adults

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully.

What is the purpose of the study?

The objective of this research is to identify the anthropometric measures of obesity that best identify metabolic syndrome in UAE early adulthood female students of the United Arab Emirates University. The results obtained might show that simple measurements are useful for predicting metabolic syndrome risk.

Why have I been chosen to take part?

The subjects chosen to take part in the study are females, aged 17-25 years. We will evaluate the screening performance of eight anthropometric measurements, Body mass index (BMI), waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR), waist to height ratio (WHtR), neck circumference (NC), Mid-upper-arm circumference (MUAC) and skin-fold thickness at four sites, in identifying metabolic syndrome risk factors.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time, without giving a reason, and to withdraw any unprocessed data previously supplied. If you are involved in a dependent relationship (i.e. teacher/student) with the Project Principle Investigator (PI), your involvement in the study will not affect your grades.

What will happen to me if I do take part?

The time you will spend participating in this study will be no longer than 40 min. You will be asked to visit the Nutrition and Health clinic, UAE University only once. Prior to your test session you will be asked to fast overnight (12-14hr), do not come during your menstrual cycle, and we would like you to come wearing appropriate clothing to facilitate skin-fold measurements. When you come for your appointment the researchers will take your anthropometric (e.g. height, weight, waist and hip circumference) and body composition measurements, fasting venous blood (by well-trained nurse) and blood pressure. You will also be asked to fill a one-page questionnaire. All measurements will be taken by trained personnel.

Are there any risks involved?

All measurements taken in the study are simple, easy and painless. Trained personnel will collect the blood samples in a designated clean and private area. The Nutrition and Health clinic allows privacy where only females will be involved in taking the measurements and counselling the participants.

What are the possible benefits of taking part?

You will receive your own “health check” profile, including anthropometric and body composition measurements. You will also be provided with a participating certificate and complementary gift to show appreciation for your valued participation.

What will happen to the results of the study?

Confidentiality of information provided can only be protected within the limitations of the law. All information collected will be kept strictly confidential. All samples and records will be coded and will only be available to the researchers involved in the study; your name will never appear in any published work. The Project PI and Project Co-PI will carry out data analysis and dissemination of results. All data from the study will be owned by the Department of Nutrition and Health and will be stored at the department for a minimum of 5 years.

Who can I contact if I have any questions?

If you have any questions regarding this study, you can contact the Project PI:

Ms. Maysm Nezar Mohamad

Department of Nutrition and Health, College of Food and Agriculture.

United Arab Emirates University, Maqam Campus, P. O. Box 17555

Mob: 00971 55 236 8727

Email: maysmnezar88@uaeu.ac.ae

Appendix 2: Consent Form



Consent form

Anthropometric measures and their association with metabolic syndrome risk factors in Emirati young female adults.

Contacts:

Ms. Maysm Nezar Moaham d, PhD student
 Department of Nutrition and Health
 United Arab Emirates University
 Maqam Campus
 Tel: 055 236 8727
 Email: *maysmnezar88@uaeu.ac.ae*

This consent form establishes that you have read and understood what taking part in this research study will involve. Please initial all boxes that apply.

1. I confirm that I have read and understand the information sheet for the above research project.
2. I confirm that I have had the opportunity to ask questions and have received satisfactory answers to all my questions.
3. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving reason, or to withdraw any unprocessed data previously supplied.
4. I understand that any information that I give will only be used anonymously and I will not be identified when my views are presented to other participants or in any publications and reports.
5. I agree to take part in the above study
6. I agree to the research team having the following personal details for the purpose of contacting me directly to arrange further research interview.

Participant's Details

Name (Block capitals): _____
 Date: _____
 Email: _____
 Mobile: _____
 Signature: _____

Researcher:

Name (Block capitals): _____
 Date: _____
 Signature: _____

Appendix 3: Ethical Approvals

UAEU

جامعة الإمارات العربية المتحدة
United Arab Emirates University

No: DVCRGS/ 370/2014
29/06/2014

To: Dr. Maysm Nezar Mohamad
CFA

Subject: Anthropometric measures and their association with metabolic syndrome risk factors in Emirati young female adult

Dear Dr. Nezar,

Please be advised that the UAEU Scientific Research Ethics Committee, in its meeting No. 45 on June 25, 2014, reviewed the ethical principles involved in your submission.

The decision reached is:

Approved as is

On behalf of the Committee, I wish you every success with your study.

Sincerely,



Prof. Reyadh Al Mehaideb
Deputy Vice Chancellor for Research and Graduate Studies

Deputy Vice Chancellor for
Research and Graduate Studies

PO BOX 15551, Al Ain, UAE
T +971 3 713 5900 F +971 3 713 4910
vprgs.office@uaeu.ac.ae www.uaeu.ac.ae

نائب مدير الجامعة
للدراستات العليا والبحث العلمي

ص.ب. 15551، العين، الإمارات العربية المتحدة
ت +971 3 713 5900 ف +971 3 713 4910
vprgs.office@uaeu.ac.ae www.uaeu.ac.ae

19 November 2014

DR. Ayesha Al Dhaheeri
Assistant Professor & Chair,
Department of Food & Health
College of Food & Agriculture
Al Ain, UAE.

Dear Dr. Ayesha,

Re: Al Ain Medical District Human Research Ethics Committee - Protocol No. 14/48 Anthropometric measures and their association with metabolic syndrome risk factors in Emirati young female adults.

Thank you very much for submitting your application to the Ethics Committee.

Your submitted documents were reviewed by the committee and I am pleased to provide you ethical approval of your project.

May I reiterate, should there be any ethical concern arising from the study in due course the Committee should be informed.

Annual reports plus a terminal report are necessary and the Committee would appreciate receiving copies of abstracts and publications should they arise.

I wish to take this opportunity to wish you success with this important study.

This Ethics Committee is an approved organization of Federal Wide Assurance (FWA) and compliant with ICH/GCP standards.

With kind regards,

Yours sincerely,




Dr. Fawaz Torab
Chair, Al Ain Medical District Human Research Ethics Committee

Appendix 4: Data Sheet



Data sheet



Anthropometric measures and their association with metabolic syndrome risk factors in Emirati young female adults



Make sure that you are using calibrated devices

Participants' Code: _____

Age: _____

Tanita Data	T1	T2	T3	Mean
Height (cm)				
Weight (kg)				
BMI				
Fat %				
Fat Mass (kg)				
FFM (kg)				
Measurements using Tape				
Waist circumference (cm)				
Hip circumference (cm)				
Waist to hip ratio (calculated)				
Neck circumference (cm)				
Mid- upper-arm circumference (cm)				
Skin-fold thicknesses				
Biceps (mm)				
Triceps (mm)				
Subscapular (mm)				
Suprailiac (mm)				
Total fat mass (%)				
Blood Pressure				
Systolic Blood pressure (mmHg)				
Diastolic Blood pressure (mmHg)				
Pulse (bpm)				
Blood analysis using Hemocue				
Glycated Haemoglobin (%)				
Haemoglobin (g/dL)				
Fasting Blood Glucose				
Blood analysis using Cobas C111				
Total Cholesterol (mg/dL)				
Fasting Blood Glucose (mg/dL)				
High Density Lipoprotein (mg/dL)				
Low Density Lipoprotein (mg/dL)				
Triglycerides (mg/dL)				
C-Reactive Protein(mg/L)				
C-Reactive Protein High Sensitive(mg/L)				

Appendix 5: Health Questionnaire

 UAEU College of Food and Agriculture	Health questionnaire	 جامعة الإمارات العربية المتحدة United Arab Emirates University
Subject Code: -----		College: -----
1- From which Emirate you are? <ul style="list-style-type: none"> <input type="radio"/> Abu Dhabi <input type="radio"/> Dubai <input type="radio"/> Sharjah <input type="radio"/> Ajman <input type="radio"/> Umm al qaiwain <input type="radio"/> Ras Al khaimah <input type="radio"/> Fujairah 		
2- How you classify your weight? <ul style="list-style-type: none"> <input type="radio"/> Underweight <input type="radio"/> Normal <input type="radio"/> Overweight <input type="radio"/> Obese 		
3- Did you notice any change in your body weight during the last 6 months? <ul style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No 		
4- How was your appetite during the last two weeks? <ul style="list-style-type: none"> <input type="radio"/> Very poor <input type="radio"/> Poor <input type="radio"/> Average <input type="radio"/> Good <input type="radio"/> Very good 		
5- Do you take any dietary supplements (Vitamins, Minerals, Natural supplements, etc)? <ul style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <ul style="list-style-type: none"> <input type="radio"/> Please specify ----- 		
6- Do you take any medication? <ul style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <ul style="list-style-type: none"> <input type="radio"/> Please specify ----- 		
7- Do you practice any physical activity at least 30 min/day (such as walking, swimming, etc)? <ul style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No 		

Health Questionnaire, Page2

8- Do you suffer from any of the following health problems?

- Diabetes
- Blood pressure
- Renal disease
- Obesity
- Liver disease
- Cardiovascular diseases
- Anemia
- Dental problems
- Food allergy
- None
- Others?
 - Please specify -----

9- Does anyone from your first relatives (father, mother, brother, sister, etc) suffer from any of the previous health problems?

- Yes
- No
 - If the answer was yes, please specify the health problem and who has it -----

10- Are you a smoker?

- Yes
- No

11- How much time do you sleep during the night?

- Less than 4 hours
- 4 – 6 hours
- 6 – 8 hours
- More than 8 hours

12- Do you take a nap during the day?

- Yes
- No
 - If the answer was yes specify the duration?
 - 30 minutes or less
 - 30 – 60 minutes
 - 1 – 1:30 hour
 - More

Appendix 6: Health Screening Questionnaire

Subject Code:	
---------------	--

GLYCAEMIC INDEX VALUE OF COMMONLY CONSUMED FOODS IN UAE GENERAL SCREENING QUESTIONNAIRE

D.O.B.	
Weight (kg)	
Height (m)	
Gender (M/F)	
Would you be prepared to be contacted again in 3-5 years?	

Please tick yes / no in answer to the following questions:	No	Yes	If yes, please give details, where appropriate:
1. Do you suffer from any heart / blood related conditions?			
2. Do you suffer from any kidney-related conditions?			
3. Do you suffer from any gastro-intestinal conditions?			
4. Do you suffer from any metabolic conditions, e.g. diabetes?			
5. Are you on any medication?			
6. Are you on any special diet or suffer from any food allergies?			
7. Are you currently trying to lose weight by means of dietary restriction and / or exercise?			
8. Has your weight fluctuated within the last 3 months by more than 3 kg?			
9. Do you suffer from any other medical condition not covered here?			
10. Did you ever make yourself sick after having eaten in order to lose weight or not to gain weight?			
11. Do you smoke?			
12. Do you regularly eat breakfast – please give details			

Health Screening Questionnaire, Page 2

Please answer the following questions by circling the number above the appropriate response

12.	How often are you dieting in a conscious effort to control your weight?			
	1 Rarely	2 Sometimes	3 Usually	4 Always
13.	How likely are you to consciously eat less than you want?			
	1 Unlikely	2 Slightly unlikely	3 Moderately likely	4 Very likely
14.	On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never "giving in"), what number would you give yourself?			
	0 = Eat whatever you want whenever want it 1 = Usually eat whatever you want, when ever you want it 2 = Often eat whatever you want, when ever you want it 3 = Often limit food intake but often 'give in' 4 = Usually limit food intake rarely 'give in' 5 = Constantly limit food intake never 'giving in'			

Appendix 7: Information Sheet

Glycaemic Index (GI) value of commonly consumed foods in UAE

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully.

What is the purpose of the study?

The glycaemic index (GI) of foods has potential implications for the prevention and treatment of major chronic diseases. The GI concept was developed to predict post-prandial increases in blood glucose concentration after the consumption of food. Type 2 diabetes has been seen in various populations whose lifestyle has changed from traditional patterns to a modern 'westernized' model. Accordingly, dietary control could have a positive impact on the number of people affected by diabetes. The consumption of some traditional foods in the UAE (e.g. Camel milk, Cow milk, "Reqaq bread" and "Arabic bread"....etc) raises the question, should the UAE population and especially the diabetic patients eat these foods?

Why have I been chosen to take part?

The subjects chosen to take part in the study are aged 18-50 years. At the beginning of the study, your fasting plasma glucose level will be tested (this will involve taking a finger-prick blood sample after you have fasted overnight). If you are identified as being at risk of developing impaired glucose tolerance or diabetes you will be excluded from taking part in this study.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time, without giving a reason, and to withdraw any unprocessed data previously supplied. If you are involved in a dependent relationship (i.e. teacher/student) with the Project Principle Investigator (PI), your involvement in the study will not affect your grades.

What will happen to me if I do take part?

It is anticipated that your time spent taking part in the study will be approximately 2 hours per test session (total study duration = 6 sessions). Each test session will be conducted in the morning at the Nutrition and Health laboratory, UAE University. Prior to each test session, you will be asked to fast overnight before anthropometric (e.g. height, weight, waist and hip circumference) and body composition measurements are taken. During each test session, you will be asked to eat a portion of food (milk or bread). Finger-prick blood samples will be taken at 0 minutes (fasting) and at 15, 30, 45, 60, 90 and 120 minutes after the start of the meal, and your blood glucose measured. For each test session, the blood glucose measurements will require a maximum of seven finger-pricks, however you will not experience any negative consequences. You will be asked to rate your feelings of hunger/satiety at various intervals during the test session. All measurements will be taken by trained personnel. Tests will be undertaken with at least 1 day between sessions.

Are there any risks involved?

During each test session, the blood glucose measurements will require a maximum of seven finger-pricks, however you will not experience any negative consequences. Trained personnel will take finger-prick blood samples in a designated clean area. During the course of the study,

if you are identified as being at risk of developing diabetes, you will be provided with immediate information and given details to take to your physician. It must be stressed that the diagnosis of diabetes is never made on the basis of a single abnormal blood glucose value.

What are the possible benefits of taking part?

You will receive your own “health check” profile, including anthropometric and body composition measurements

What will happen to the results of the study?

Confidentiality of information provided can only be protected within the limitations of the law. All information collected will be kept strictly confidential. All samples and records will be coded and will only be available to the researchers involved in the study; your name will never appear in any published work. The Project PI and Project Co-PI will carry out data analysis and dissemination of results. All data from the study will be owned by the Department of Nutrition and Health and will be stored at the department for a minimum of 5 years.

Who can I contact if I have any questions?

If you have any questions regarding this study, you can contact either the Project PI or Project Co-PI:

Dr. Ayesha Al Dhaheri (Project PI)
Department of Nutrition and Health
College of Food and Agriculture
UAE University
Maqam Campus
P. O. Box 17555
Tel: 03 7134539
Email: ayesha_aldhaheri@uaeu.ac.ae

Ms. Maysm Mohamad (Project Co-PI)
Department of Nutrition and Health
College of Food and Agriculture
UAE University
Maqam Campus
P. O. Box 17555
Tel: 055 236 8727
Email: maysmnezar88@uaeu.ac.ae

Appendix 8: Data Sheet

Test Food Name:.....

Subject Date _____

Age	(Yrs)	
Height	(m)	
Weight	(kg)	
BMI	(kg/m ²)	
Waist circumference	(cm)	
FM	(%)	
FM	(kg)	
FFM	(%)	
FFM	(kg)	

Blood glucose

0 minutes	(mmol/l)	
15 minutes	(mmol/l)	
30 minutes	(mmol/l)	
45 minutes	(mmol/l)	
60 minutes	(mmol/l)	
90 minutes	(mmol/l)	
120 minutes	(mmol/l)	

0 mins



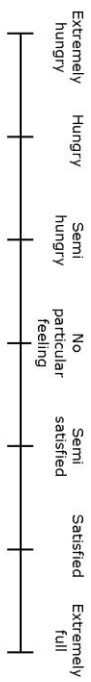
15 mins



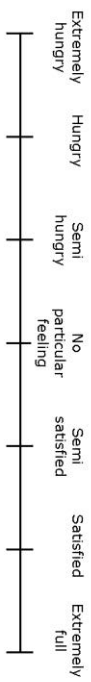
30 mins



45 mins



60 mins



90 mins



120 mins



Appendix 9: Ethical Approval

UNITED ARAB EMIRATES UNIVERSITY



جامعة الإمارات العربية المتحدة

RESEARCH AFFAIRS

شؤون البحث العلمي

516/09

March 10, 2009

To: Dr. Ayesha Salem Al Dhaheri
Faculty of Food and Agriculture

Subject: Glycaemic Index (GI) value of commonly consumed foods in UAE

Dear Dr. Al Dhaheri,

Please be advised that the UAEU Scientific Research Ethics Committee, in its meeting No. 14th dated February 16, 2009, reviewed the ethical principles involved in your submission.

The Decision reached is:

- Approved as is
 Not Approved
 Approved with the following comments

On behalf of the committee, I wish you every success with your study.

With my best regards.

Sincerely,

Abdel-Mohsen Onsy Mohamed
Professor of Civil & Environmental Engineering and
Director of Research



Appendix 10: Consent Form



Consent form



Glycaemic Index (GI) value of commonly consumed foods in UAE

Contacts:

Dr Ayesha Al Dhaheri , Assistant Professor
Mr. Amjad Jarrar, Instructor

Department of Nutrition and Health
United Arab Emirates University
Maqam Campus
P.O.Box: 17555, Al Ain
Tel: 03 7134539/03 7134804
Email: ayesha_aldhaheri@uaeu.ac.ae / amjadj@uaeu.ac.ae

Please initial the appropriate box

	Yes	No
1. I confirm that I have read and understand the information sheet for the above research project.	<input type="checkbox"/>	<input type="checkbox"/>
2. I confirm that I have had the opportunity to ask questions and have received satisfactory answers to all my questions.	<input type="checkbox"/>	<input type="checkbox"/>
3. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving reason, or to withdraw any unprocessed data previously supplied.	<input type="checkbox"/>	<input type="checkbox"/>
4. I understand that confidentiality of information provided can only be protected within the limits of the law.	<input type="checkbox"/>	<input type="checkbox"/>
5. I agree to take part in the above study	<input type="checkbox"/>	<input type="checkbox"/>

Name of Participant ----- Date -----
(block capitals)

Signature -----

Name of Researcher ----- Date -----
(block capitals)

Signature -----

Appendix 11: Graphs of the IAUC for all Test Foods

The mean incremental area under the glycemc response curves of the standard food and each test food are presented in Figures A to W.

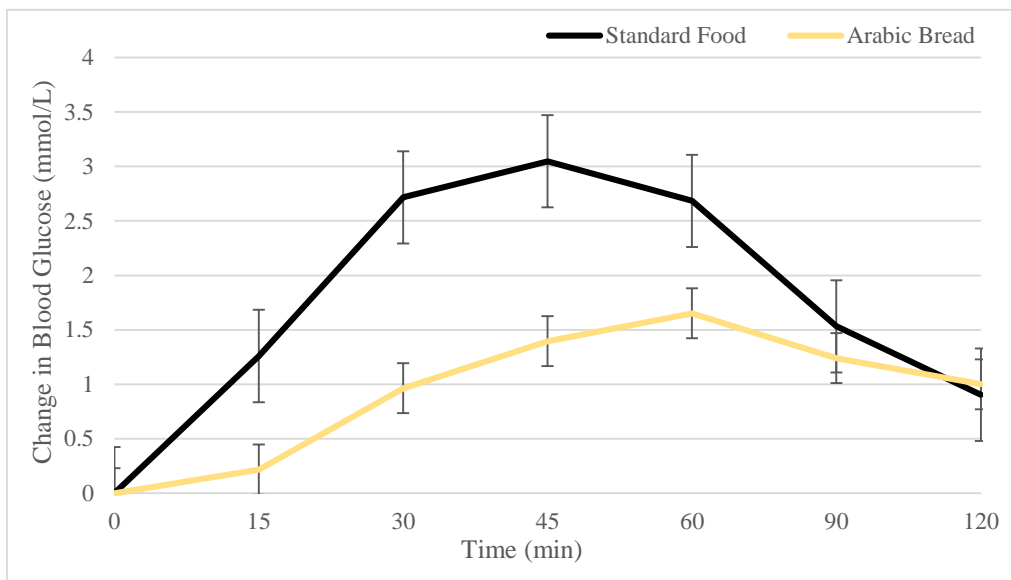


Figure A: Incremental area under the blood glucose curves (IAUC) for the standard food and Arabic bread. Standard errors of the mean are represented by vertical bars.

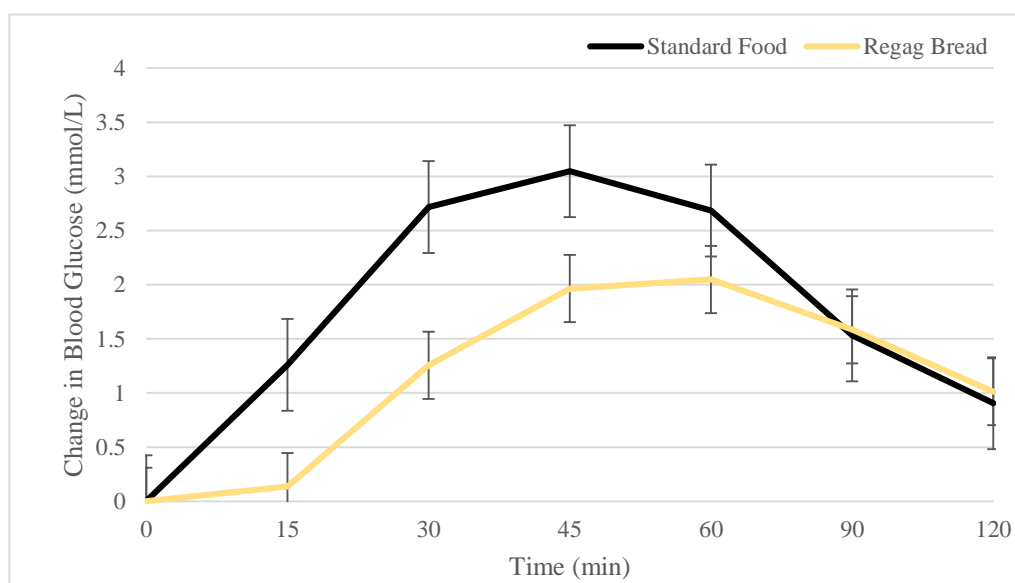


Figure B: Incremental area under the blood glucose curves (IAUC) for the standard food and Regag bread. Standard errors of the mean are represented by vertical bars.

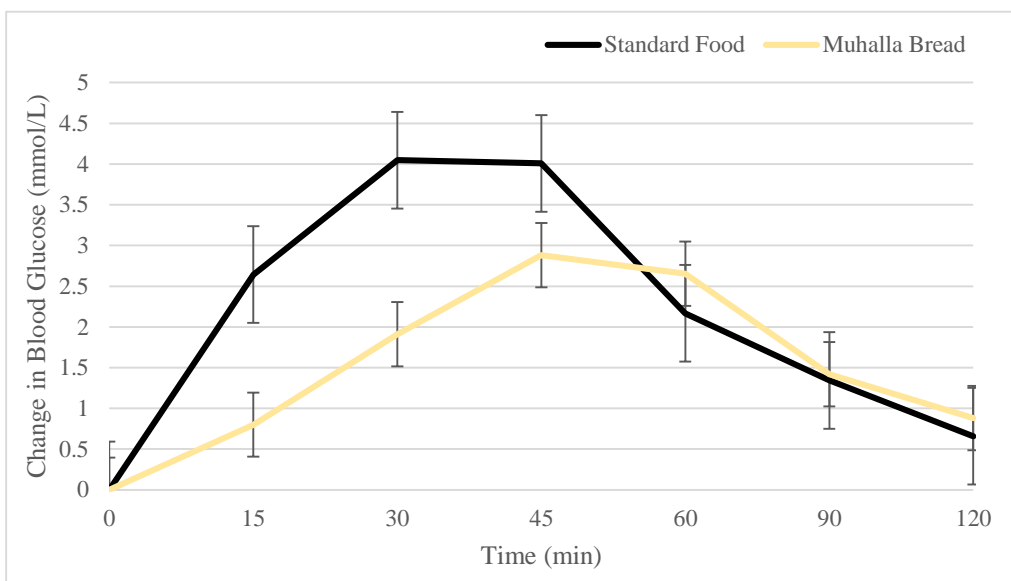


Figure C: Incremental area under the blood glucose curves (IAUC) for the standard food and Muhalla bread. Standard errors of the mean are represented by vertical bars.

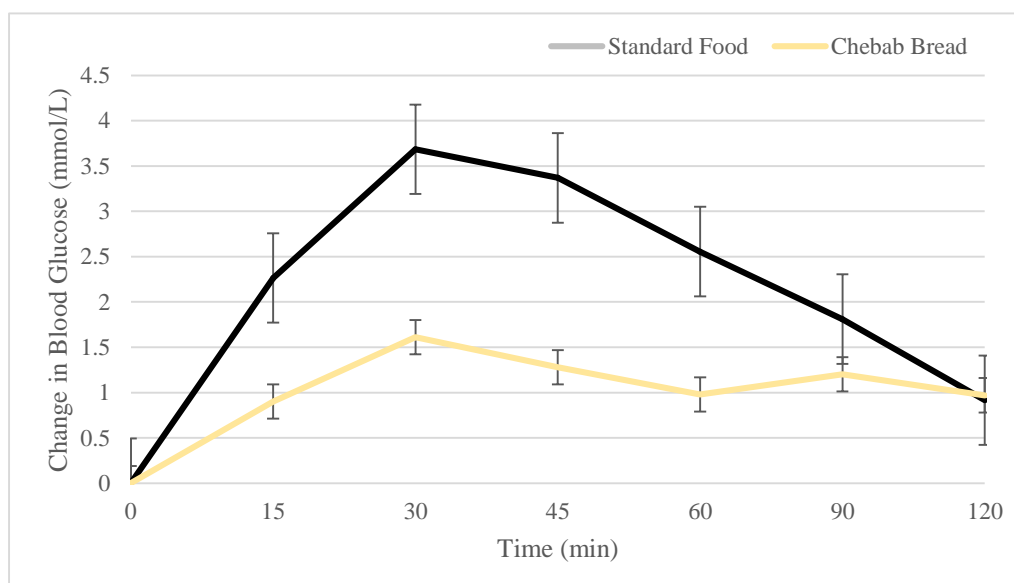


Figure D Incremental area under the blood glucose curves (IAUC) for the standard food and Chebab bread. Standard errors of the mean are represented by vertical bars.

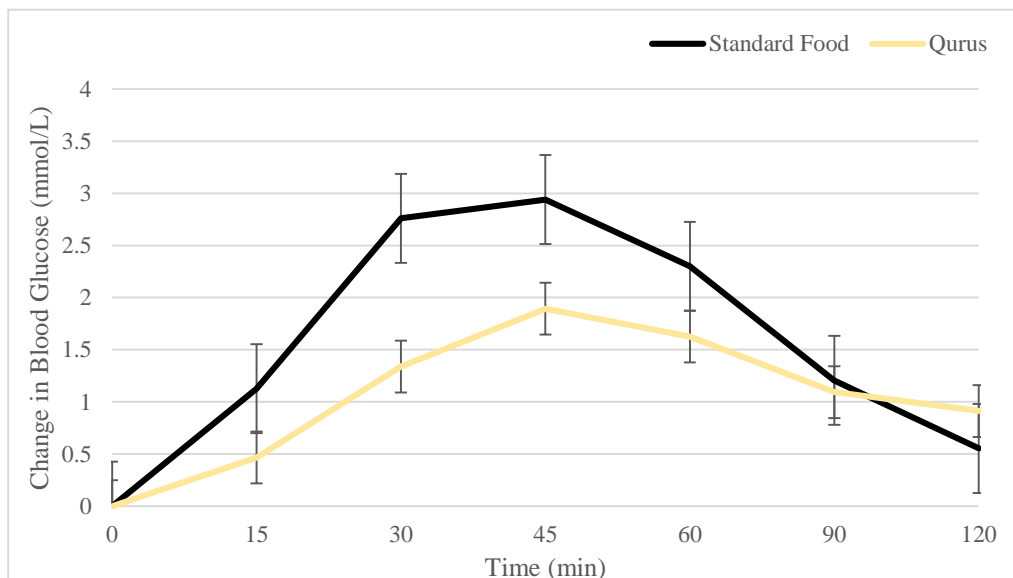


Figure E: Incremental area under the blood glucose curves (IAUC) for the standard food and Qurus. Standard errors of the mean are represented by vertical bars.

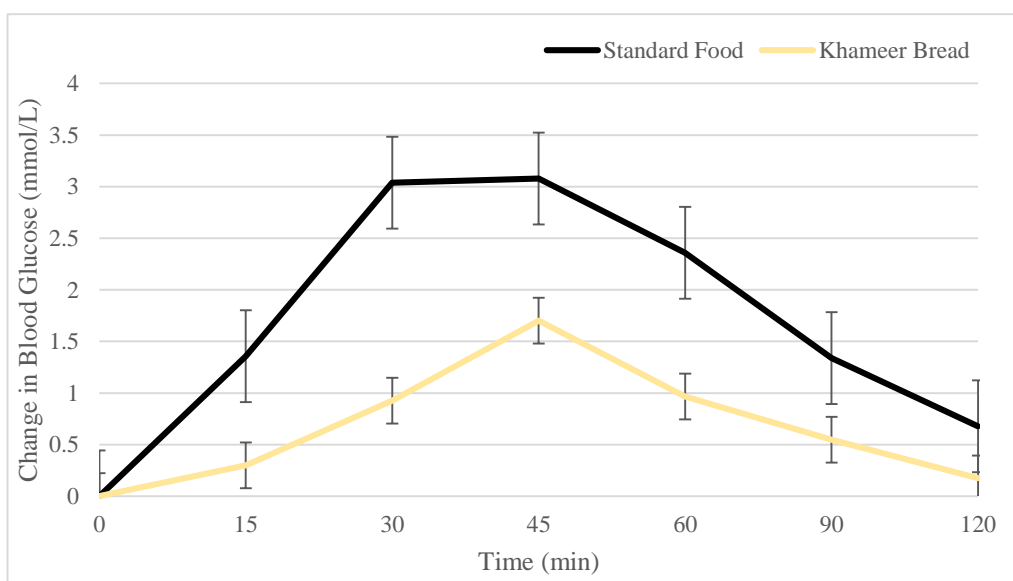


Figure F: Incremental area under the blood glucose curves (IAUC) for the standard food and Khameer bread. Standard errors of the mean are represented by vertical bars.

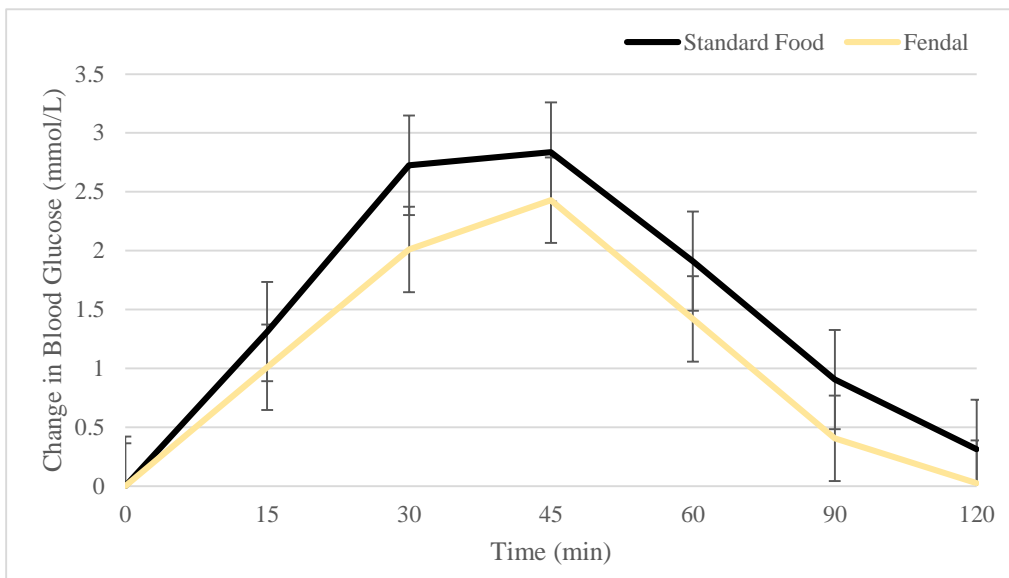


Figure G: Incremental area under the blood glucose curves (IAUC) for the standard food and Fendal. Standard errors of the mean are represented by vertical bars.

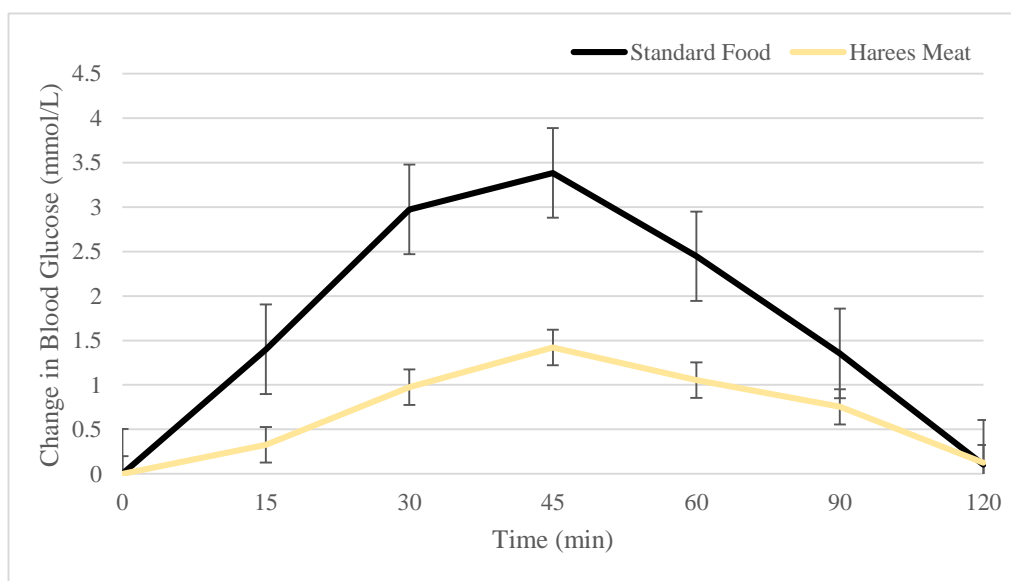


Figure H: Incremental area under the blood glucose curves (IAUC) for the standard food and Harees. Standard errors of the mean are represented by vertical bars.

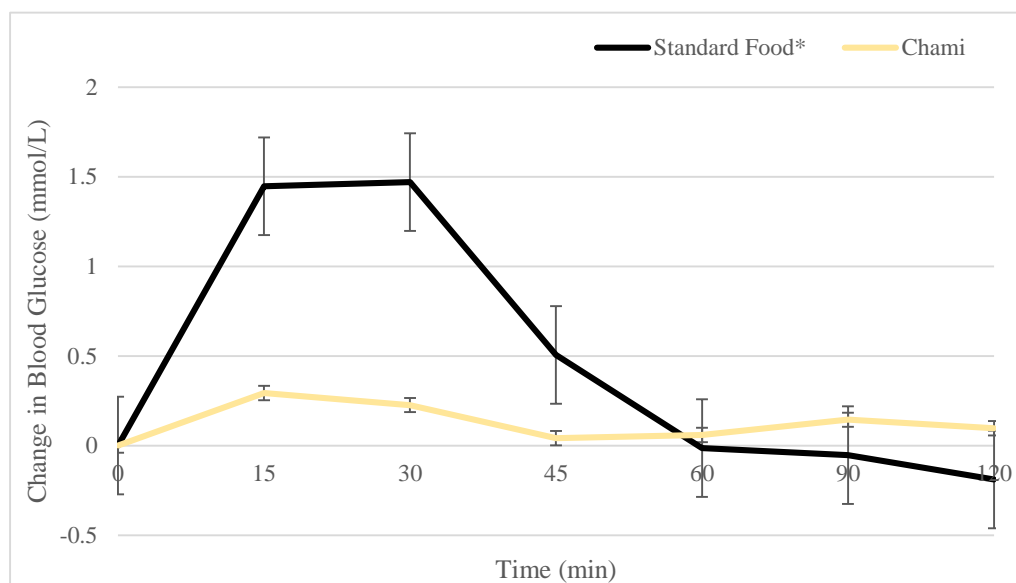


Figure I: Incremental area under the blood glucose curves (IAUC) for the standard food and Chammi. Standard errors of the mean are represented by vertical bars.

* 25g of Glucose.

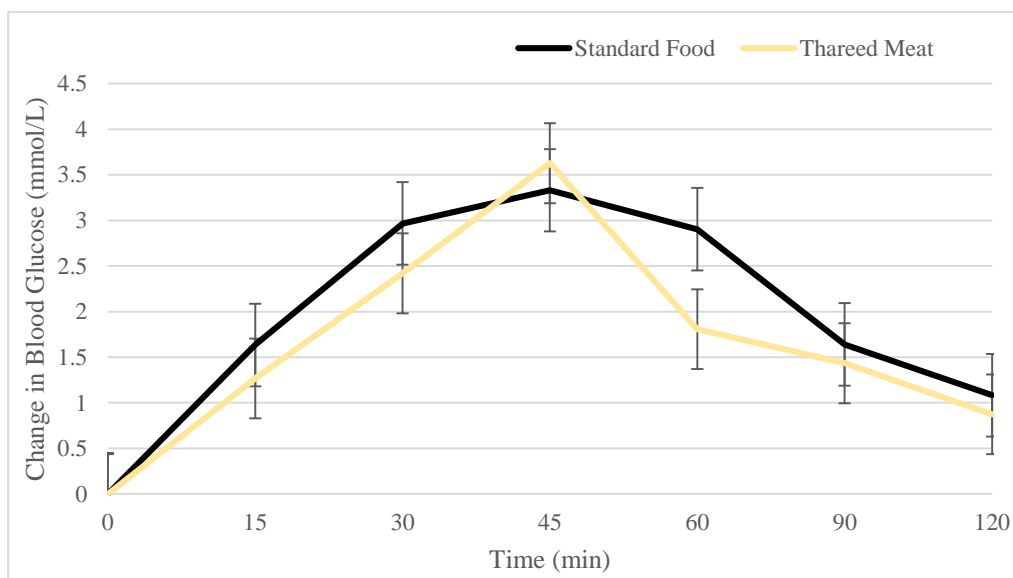


Figure J: Incremental area under the blood glucose curves (IAUC) for the standard food and Thareed beef. Standard errors of the mean are represented by vertical bars.

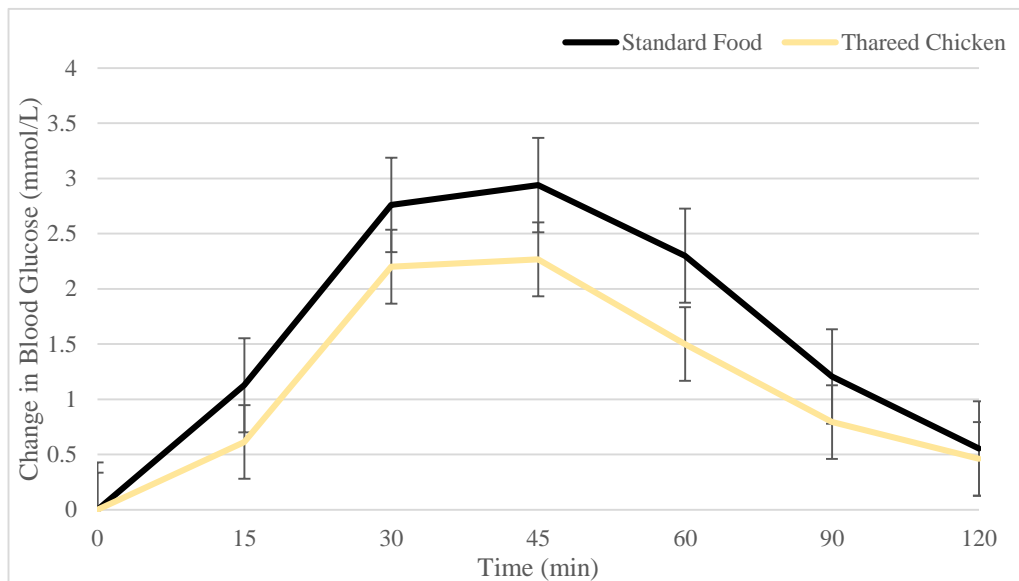


Figure K: Incremental area under the blood glucose curves (IAUC) for the standard food and Thareed chicken. Standard errors of the mean are represented by vertical bars.

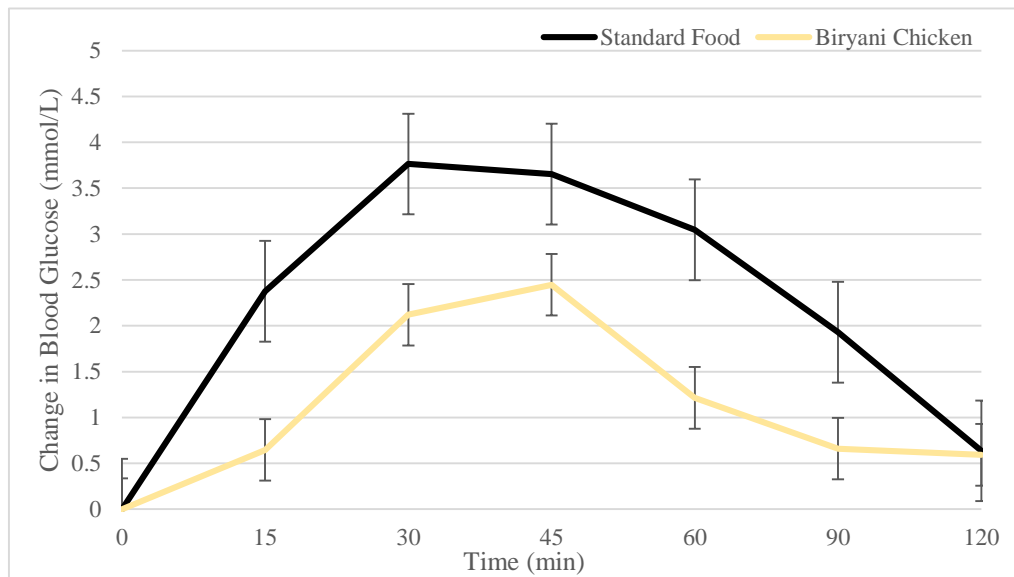


Figure L: Incremental area under the blood glucose curves (IAUC) for the standard food and Biryani chicken. Standard errors of the mean are represented by vertical bars.

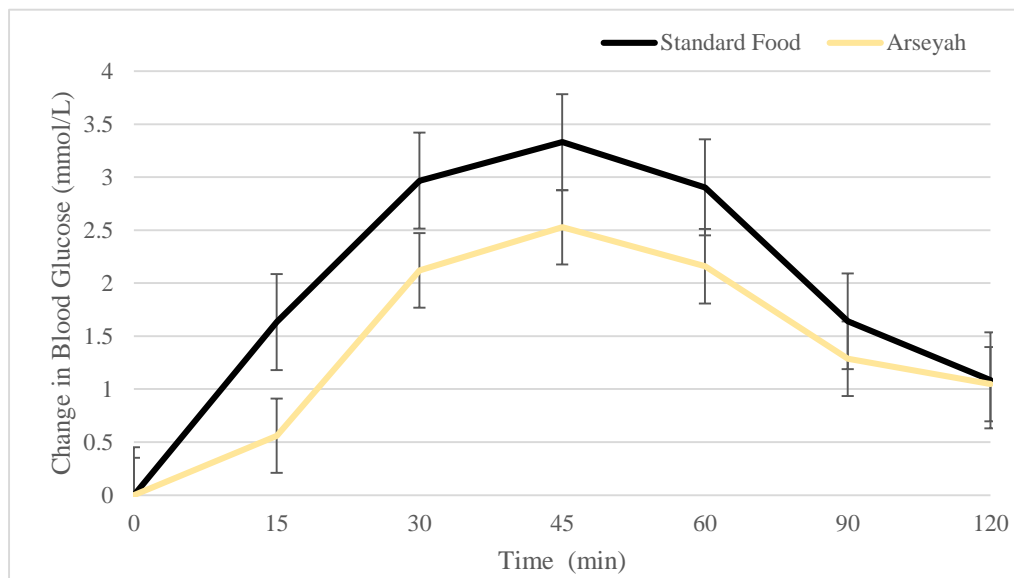


Figure M: Incremental area under the blood glucose curves (IAUC) for the standard food and Arseyah. Standard errors of the mean are represented by vertical bars.

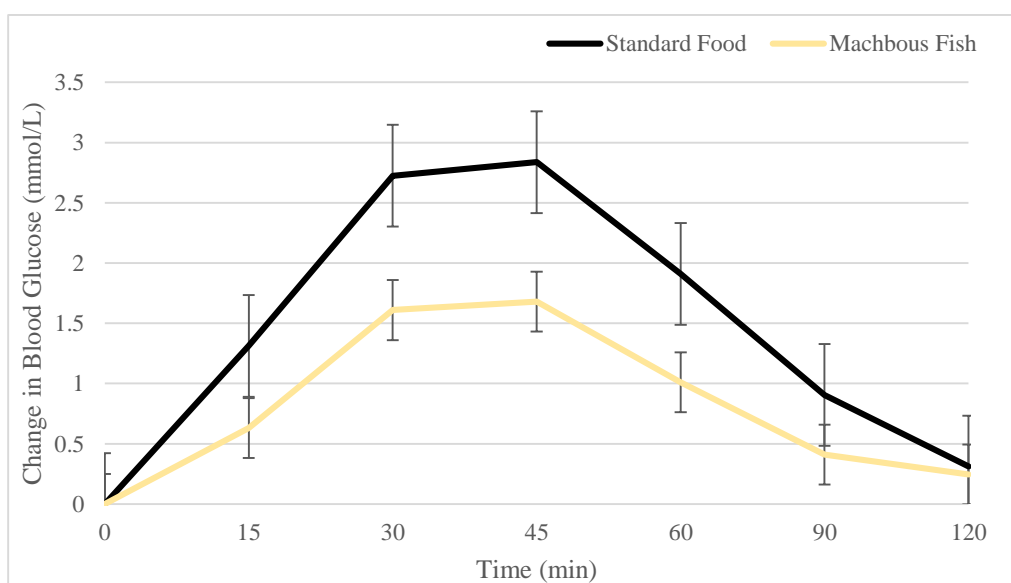


Figure N: Incremental area under the blood glucose curves (IAUC) for the standard food and Machbous fish. Standard errors of the mean are represented by vertical bars.

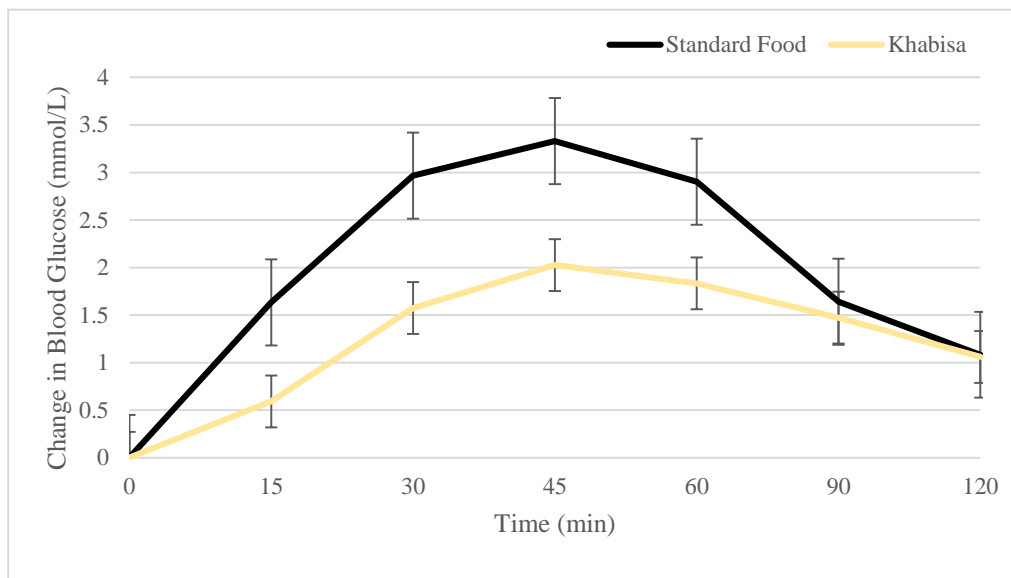


Figure O: Incremental area under the blood glucose curves (IAUC) for the standard food and Khabisa. Standard errors of the mean are represented by vertical bars.

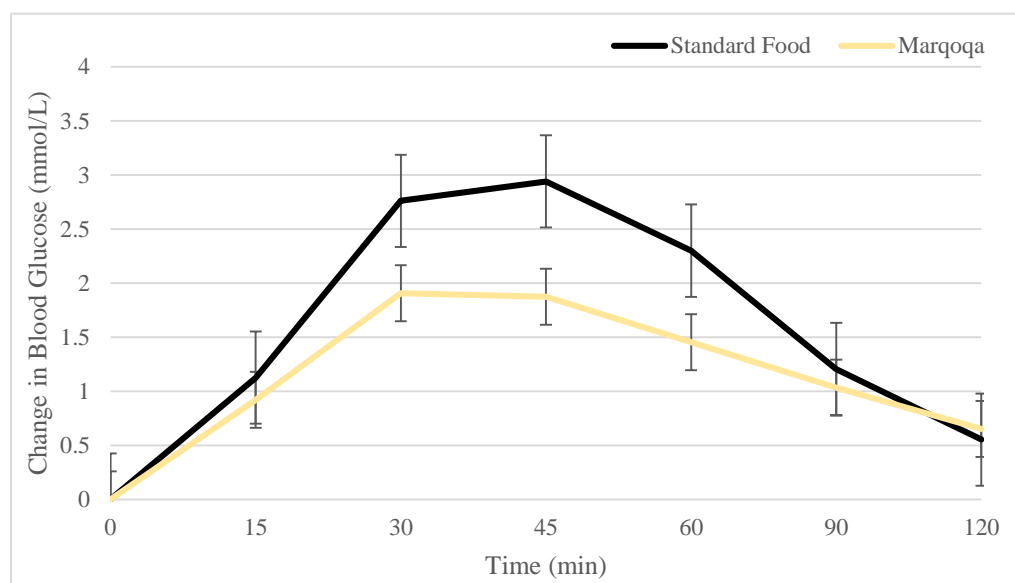


Figure P: Incremental area under the blood glucose curves (IAUC) for the standard food and Marqoqa. Standard errors of the mean are represented by vertical bars.

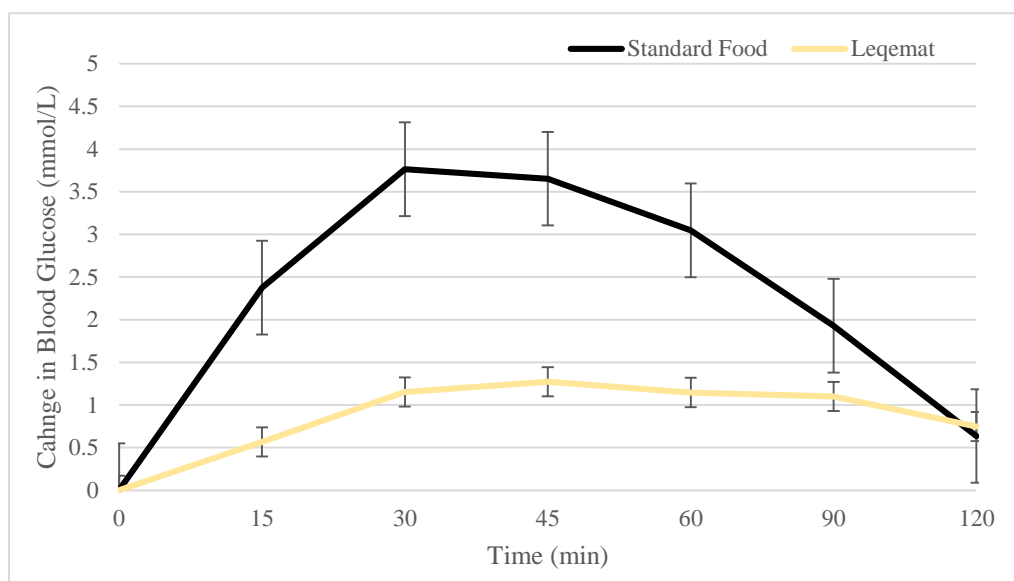


Figure Q: Incremental area under the blood glucose curves (IAUC) for the standard food and Leqemat. Standard errors of the mean are represented by vertical bars.

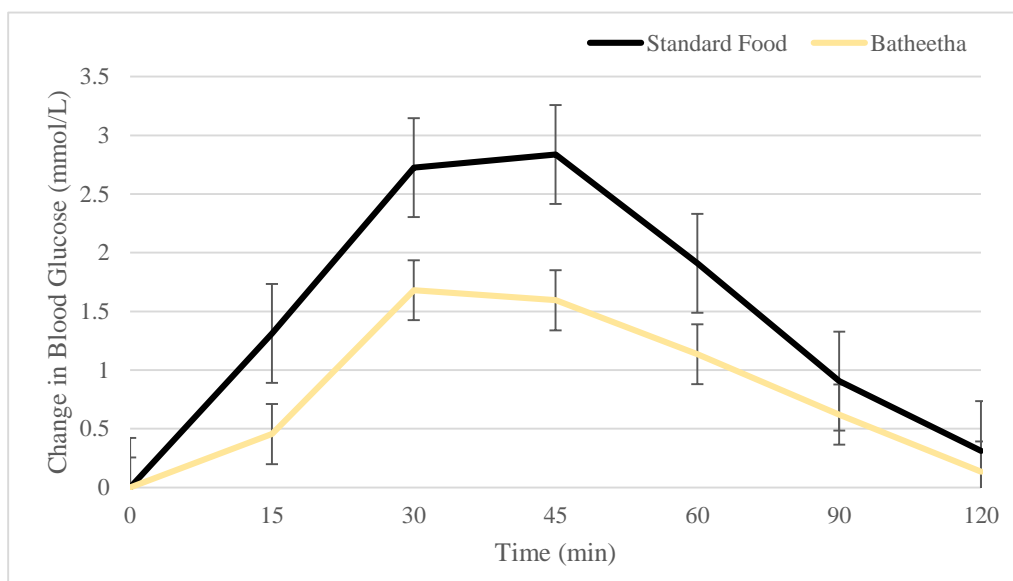


Figure R: Incremental area under the blood glucose curves (IAUC) for the standard food and Batheetha. Standard errors of the mean are represented by vertical bars.

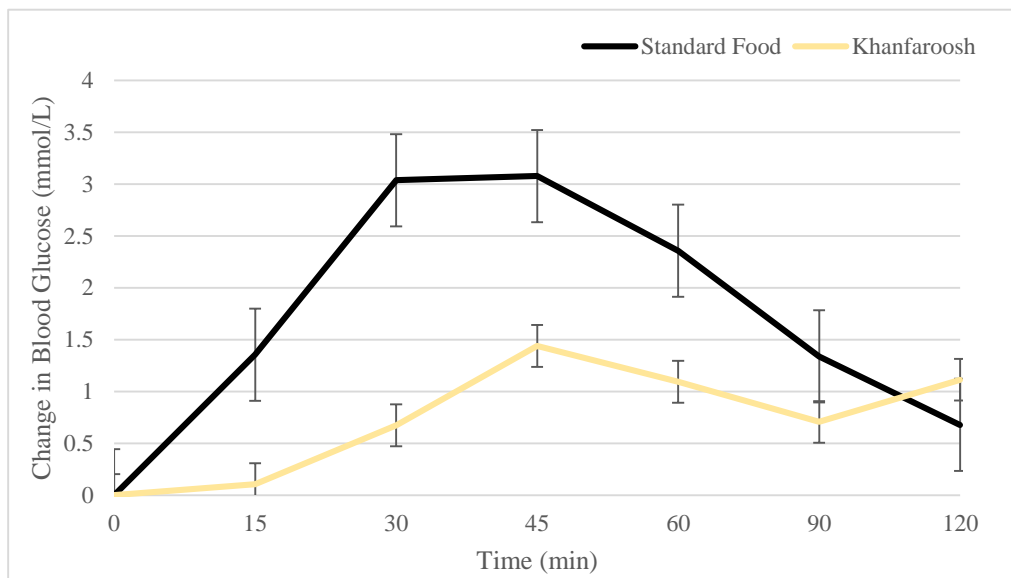


Figure S: Incremental area under the blood glucose curves (IAUC) for the standard food and Khanfaroosh. Standard errors of the mean are represented by vertical bars.

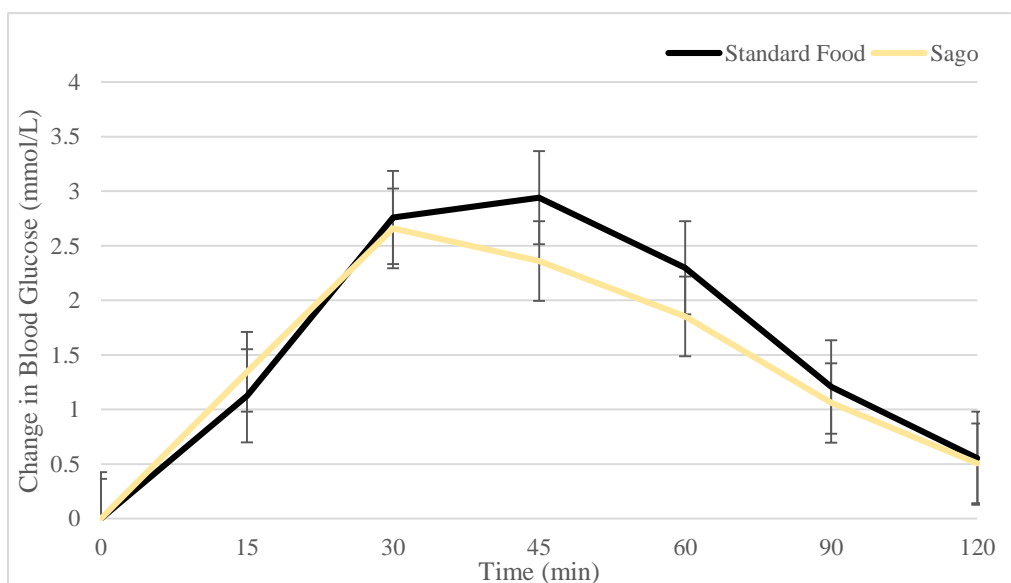


Figure T: Incremental area under the blood glucose curves (IAUC) for the standard food and Sago. Standard errors of the mean are represented by vertical bars.

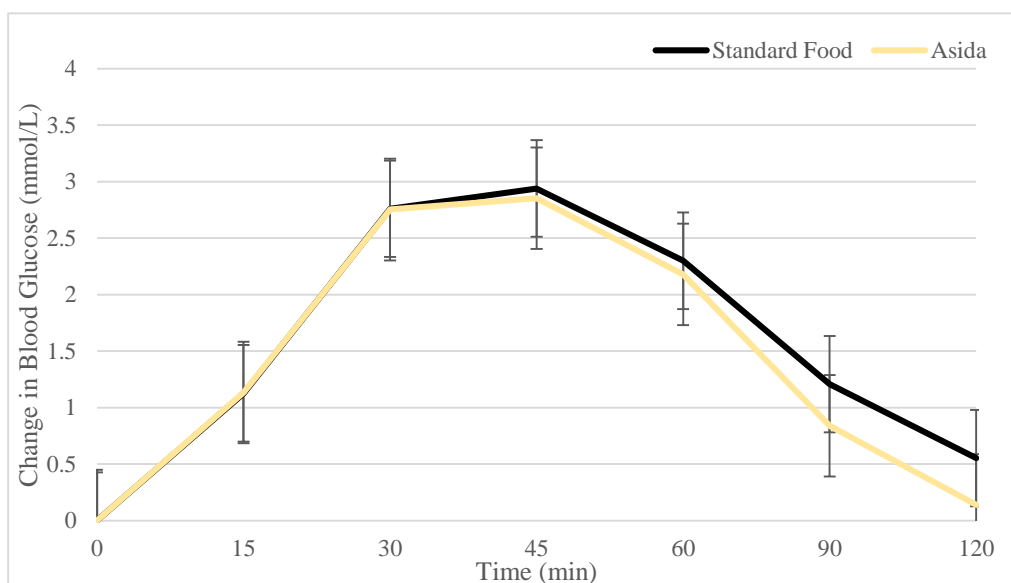


Figure U: Incremental area under the blood glucose curves (IAUC) for the standard food and Asida. Standard errors of the mean are represented by vertical bars.

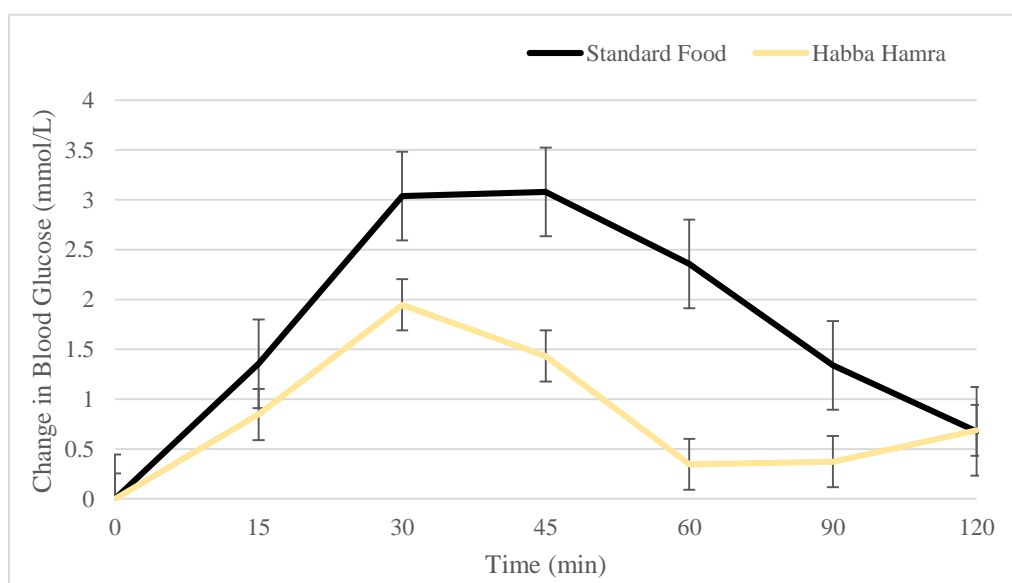


Figure V: Incremental area under the blood glucose curves (IAUC) for the standard food and Habba Hamra. Standard errors of the mean are represented by vertical bars.

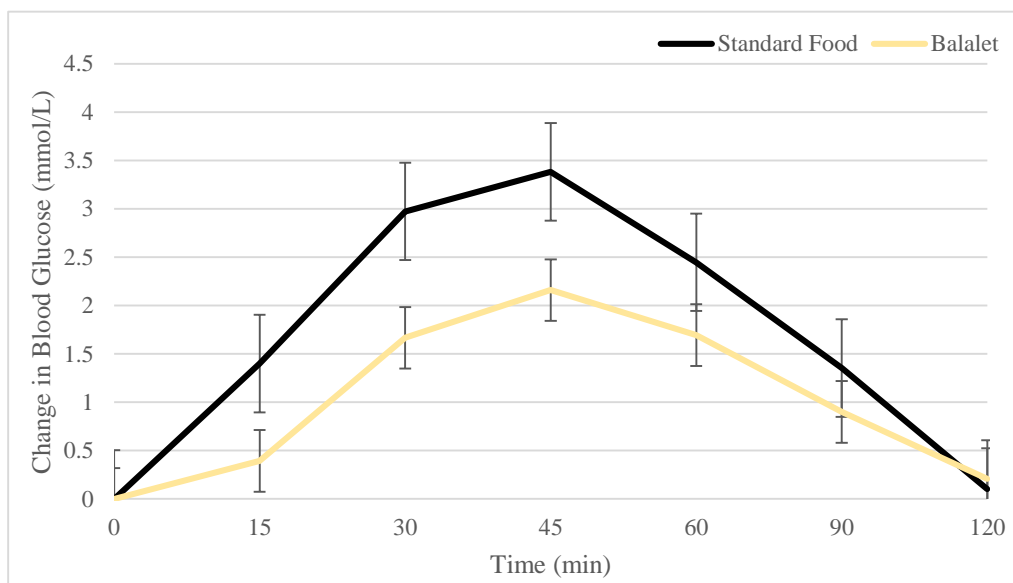


Figure W: Incremental area under the blood glucose curves (IAUC) for the standard food and Balalet. Standard errors of the mean are represented by vertical bars.

Appendix 12: Abstract for the 10th International Symposium on Body Composition.



FACULDADE DE MOTRICIDADE HUMANA

Erratum

Due to an error on the part of the Organization, this paper has not been included in the Book of Abstract. This paper is an integral part of the Congress Program. We regret this mistake.

THE RELATIONSHIP BETWEEN ANTHROPOMETRIC MEASUREMENTS AND DIAGNOSIS OF PRE-DIABETES MELLITUS AMONG UNITED ARAB EMIRATES UNIVERSITY FEMALE STUDENTS

Ayesha S. Al Dhaheri, Maysm N. Mohamad, Amjad H. Jarrar

Nutrition and Health Department, College of Food and Agriculture, United Arab Emirates University, Al Ain, United Arab Emirates.

BACKGROUND/OBJECTIVES: Diabetes mellitus (DM) is one of the most common non-communicable diseases (NCDs) globally. Population-based diabetes studies consistently show that a substantial proportion of those found to have diabetes had not been previously diagnosed; this could lead to increased morbidity and mortality. Identification of anthropometric variables and their clustering as important markers of diabetes risk have not been investigated in UAE early adulthood age group. The objective of this study was to assess anthropometric measures of obesity and their association with pre-diabetes. In addition, to identify the anthropometric measures that best identify pre-diabetes mellitus in early adulthood female students of the United Arab Emirates University (UAEU).

Design: A cross-sectional population-based study.

SUBJECTS/METHODS: A total of 300 female students aged 17-25 years were recruited from United Arab Emirates University (UAEU), and only 182 were considered for the study. Body mass index [BMI], waist circumference [WC], hip circumference [HC], waist to hip ratio [WHR], skin-fold thickness at four sites [Biceps, Triceps, Subscapular, Suprailiac], and percentage of body fat [PBF] were measured using standard equipment and procedures. Fasting blood glucose [FBG] and glycated Hemoglobin [HbA1c] were measured.

PRELIMINARY RESULTS: The prevalence of overweight and obesity amongst UAEU female students defined by BMI, waist circumference and WHR was 33.5%, 10.9% and 4.9% respectively. Furthermore, the prevalence of pre-diabetes diagnosed by fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) was 5.5% and 34.4% respectively. BMI, WC and HC showed a strong positive correlation with PBF r 0.83, r 0.81 and r 0.77 respectively ($P < 0.01$). HC showed the highest association with FBG ($P = 0.01$). However, there was no significant relationship between HbA1c and any of the anthropometric measures.

CONCLUSIONS: Using glycated hemoglobin (HbA1c) for diagnosis of pre-diabetes showed an alarming percentage. Moreover, general obesity measures had stronger correlation with PBF among UAEU female students.

Keywords: Anthropometric measurements; Fasting Blood Glucose (FBG); Glycated Hemoglobin (HbA1c); Pre-diabetes mellitus.

Appendix 13: Abstract for the 12th Asian Congress of Nutrition.

8/29/2016

Details | ACN2015 Abstract Database



ACN2015 Abstract Database

[Home](#) » [Search Result](#) » [Details](#)

Details

Program Category	Poster/Oral Presentation
Program Date/Time	2015/05/16 (Sat) 11:50-12:20
Room	Exhibition Hall
Program No.	PS-02-a-256
Session	Nutritional Epidemiology 3
Title	The relationship between anthropometric measurements and diagnosis of pre-diabetes mellitus and pre-hypertension among United Arab Emirates University female students
Author	Ayesha Salem Al Dhaheeri, Maysm Nezar Mohamad, Amjad Hasan Jarrar
Affiliation	United Arab Emirates University, United Arab Emirates
Abstract	<p>BACKGROUND: Non-communicable diseases (NCDs) such as diabetes mellitus (DM) and hypertension contribute to the increasing morbidity and mortality rates worldwide. Identification of anthropometric variables and their clustering as important markers of diabetes and hypertension risk have not been investigated in UAE "early adulthood" age group. Furthermore, the objective of this study was to assess anthropometric measures of obesity and their association with pre-diabetes and pre-hypertension. In addition, to identify the anthropometric measures that best identify pre-diabetes mellitus and pre-hypertension in early adulthood female students of the United Arab Emirates.</p> <p>METHODS: A total of 555 female students aged 17-25 years were recruited from United Arab Emirates University. Body mass index [BMI], waist circumference [WC], hip circumference [HC], waist to hip ratio [WHR], neck circumference [NC], skin-fold thickness at four sites [Biceps, Triceps, Subscapular, Suprailiac], and percentage of body fat [PBF] were measured using standard equipments. Blood pressure was measured using Omron HEM-907 automated blood pressure monitor. Fasting blood glucose [FBG] and glycated Hemoglobin [HbA1c] were determined by HemoCue instruments in a cross-sectional population-based epidemiological study.</p> <p>PRIMARY RESULTS: The prevalence of obesity amongst Emirati young female adults defined by BMI, waist circumference and WHR was 10.5, 17.1 and 7.7% respectively and of overweight defined by BMI was 23.1%. Furthermore, the prevalence of pre-diabetes diagnosed by FBG and HbA1c was 5.9% and 32.1% respectively. BMI, WC and HC showed a strong positive correlation with PBF r 0.79, r 0.80 and r 0.75 respectively ($P < 0.01$). NC showed the highest association with pre-hypertension and FBG ($P < 0.01$). However, there was no significant relationship between HbA1c and any of the anthropometric measures.</p> <p>CONCLUSIONS: Using HbA1c for diagnosis of pre-diabetes showed an alarming percentage. Neck circumference might be expanding the ability for the identification of pre-hypertension and pre-diabetes among young female students of the UAE.</p>

https://member.jsnfs.or.jp/acn2015/index.php?t_search_seq=2641

1/2

Appendix 14: Research Paper 1

Al Dhaheri et al. *BMC Nutrition* 2015, 1:4
<http://www.biomedcentral.com/bmcnutr/content/1/1/4>



RESEARCH ARTICLE

Open Access

The effect of nutritional composition on the glycemic index and glycemic load values of selected Emirati foods

Ayesha S Al Dhaheri^{1*}, Asila K Al Ma'awali¹, Louis C Laleye², Sidiga A Washi¹, Amjad H Jarrar¹, Fatima T Al Meqbaali¹, Maysm N Mohamad¹ and Emad M Masuadi³

Abstract

Background: Population-based studies have shown an association between health, food composition, and diets; therefore, data on the composition of traditional foods for meal planning, nutritional assessment, and clinical nutrition research to build up a relevant database are needed.

Methods: The objective of this study was to assess the effect of the nutritional composition of five commonly consumed traditional Emirati foods, *Threed chicken*, *Marqoqa*, *Gurus*, *Assidah*, and *Saqa*, on the glycemic index (GI) and glycemic load (GL) values. Fifteen healthy subjects aged between 18 and 25 years old participated in this study.

Results: The proximate analysis showed high amounts of protein in *Gurus* and *Threed chicken* and high-fiber content in *Gurus*. The carbohydrate percentages for the foods tested were as follows 54.4% in *Gurus*, 23.4% in *Saqa*, 21.1% in *Assidah*, 13.3% in *Marqoqa*, and 12.3% in *Threed chicken*. The corresponding GI values were high: 71.7, 99.4, 99.2, 84.6, and 71.9, respectively. The GL values of the foods tested were also considered high, varying from 35.85 to 49.7. The incremental increase in blood glucose was monitored and calculated for each food and when compared with the standard food (glucose) showed significant differences ($P < 0.001$) for all foods except *Saqa* and *Assidah* at 30 min, with similar responses at 45 min. At 120 min, no significant differences in blood glucose levels were observed ($P > 0.05$). The types of carbohydrate, different ingredients of foods, and cooking method used all contributed to the GI value.

Conclusions: The GI value of traditional foods can be modified through altering the ingredients, cooking method, or the portion size served. This data will help to inform decisions on the diet and health of consumers in the UAE.

Keywords: Glycemic index, Glycemic load, Traditional foods, Emirati foods, University students, United Arab Emirates (UAE)

Background

The incidence of diabetes is dramatically increasing worldwide, reflecting current lifestyle trends, characterized by calorific abundance in foods and low physical activity. The incidence of type 2 diabetes mellitus (T2DM) not only influences the individual's health but also causes an economic loss to society, with increased health-care costs. Obesity is a well-known major independent risk

factor for developing T2DM [1] and is strongly correlated with insulin-sensitivity reduction, especially in people with excess abdominal fat distribution and physical inactivity [2].

In general, insulin resistance increases with increased body fat mass (FM), and this often exists in patients long before their diagnosis with T2DM. It has been estimated that more than 135 million people globally have T2DM, particularly in the United States, with more than 20 million diabetic patients [3]. The United Arab Emirates (UAE) has been ranked by the International Diabetes Federation (IDF) as having the 15th highest prevalence

* Correspondence: ayasha_aldhaheri@uaeu.ac.ae

¹Nutrition and Health Department, College of Food and Agriculture, United Arab Emirates University, Al Ain, United Arab Emirates
 Full list of author information is available at the end of the article



© 2015 Al Dhaheri et al.; licensee BioMed Central. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly credited. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated.

rate of T2DM in the world [4]. Owing to the severity of this disease, interest in the underlying mechanism has become increasingly important [5]. However, the high prevalence of diabetes in the UAE could be related to the individual's lifestyle choices, with the main reason being their dietary habits. Positive lifestyle modification and preventive measures are needed to decrease the rapid growth of this problem. Several alternative therapies have been identified such as surgical, physical, and dietary therapy and low-carbohydrate, high-fiber, low-calorie, and low-glycemic index (GI) diets.

The GI concept has clinically important benefits for preventing, managing, and treating a number of chronic diseases such as diabetes, cardiovascular disease (CVD), and some forms of cancer and obesity [6]. Foods that are classified as low GI provide a better response to postprandial glucose, causing a slight increase in circulating levels of insulin and gastrointestinal hormones. Therefore, satiety is increased and voluntary food intake is reduced [7]. However, increased insulin secretion, caused by foods with high GI, leads to postprandial hyperinsulinemia along with an increase in both hunger and voluntary food intake [7]. This suggests that a low-GI diet may provide some level of prevention against developing diabetes and obesity and for managing existing CVD.

Several international GI tables have been published, generally Australian, British, or Canadian in origin [8-10]. Currently, no published GI table is available for Emirati foods. Given this lack of information, it has always been a challenging task for dietitians in UAE to design meal plans and to improve advice for preventing and managing obesity and other chronic diseases. Therefore, the main objective of the present study is to provide this data on the nutritional composition, GI, and GL values of five selected traditional Emirati foods commonly consumed in UAE and to assess the effect of the nutritional composition on the GI and GL values of these foods.

Methods

Ethical approval for the study protocol was obtained from the Scientific Research Ethics Committee at UAE University (UAEU, Reference No: 516/09), and all subjects gave written informed consent to participate. The subjects were given full details of the study protocol with the opportunity to ask questions.

All subjects were recruited from UAEU for voluntary participation in the study. A number of different methods were used for recruitment: email circulation, posters displayed in different UAEU buildings, and word of mouth. Fifteen healthy female subjects aged between 18 and 25 years old were recruited. The subjects were excluded if they had a fasting blood glucose value >7.0 mmol/L. They were also asked to complete a health questionnaire before the study. The subjects were asked not to undertake vigorous activities on the day before the test, to avoid caffeine-containing drinks, and not to smoke for 24 h before the test; instructions concerning meals on the previous day were not provided, because the fat and carbohydrate content of the evening meal before GI testing does not influence blood glucose response [11].

Anthropometric measurements

Measurements of body size and body composition were all carried out in the Nutrition and Health Department laboratory at UAEU. All anthropometric measurements were taken after a 12-h fast (fasting stage) with the subjects wearing light clothes and no shoes. The measurements were made of height (cm) using a stadiometer (Seca Ltd., Birmingham, UK) [12], waist circumference (WC; cm) using a measuring tape and body weight (kg), FM, and fat-free mass (FFM) using a Segmental Body Composition Analyzer (TBF-410 MA; Tanita Corp., Tokyo, Japan) [13]. BMI was calculated as the weight in kilograms divided by the square of the height in meters: $BMI = \text{Weight (kg)} / \text{Height}^2 \text{ (m}^2\text{)}$, using cut-off values for normal weight, overweight, and the various levels of obesity in adults from WHO [14].

Test foods

The present study is part of an ongoing research project funded by the Emirates Foundation to assess the GI of 20 traditional foods commonly consumed in UAE. The five test foods were selected as highly reproducible and the most acceptable to all subjects. The selected foods were obtained from three popular restaurants in Al Ain that specialized in Emirati foods and could prepare the foods from standardized recipes. The selected test foods were sweet dishes: *Gurus*, *Saqo*, and *Assidah*, and main dishes: *Threed chicken* and *Marqoqa* (Table 1).

Table 1 Main ingredients used in the preparation of five traditional foods commonly consumed in UAE

Local name (description)	Ingredients
<i>Gurus</i> (fried bread)	Wheat flour, vegetable oil, egg, sugar, salt, and water
<i>Assidah</i> (flour with ghee)	Wheat flour, sugar, ghee, salt, and water
<i>Saqo</i> (saqo seeds with ghee)	<i>Saqo</i> seeds, sugar, ghee, saffron, and water
<i>Marqoqa</i> (bread with chicken stew)	Wheat flour, chicken, potatoes, onions, tomatoes, carrots, tomato paste, vegetable oil, garlic, spices, and water
<i>Threed chicken</i> (bread with chicken stew)	Wheat flour, chicken, potatoes, onions, tomatoes, zucchini, tomato paste, vegetable oil, spices, and water

Chemical analyses

Proximate analysis for the test foods was carried out in the Nutrition and Health Department laboratory at UAEU. The proximate analysis was done for each test food using the standard method of the Association of Official Analytical Chemists [15]. Each test food was analyzed three times on separate occasions at the beginning, middle, and end of the month; this was done to ensure that the restaurants were consistent in using the food recipe. The test foods were also separately homogenized and prepared in triplicate, and average results were determined for the proximate analyses of moisture, protein, fat, fiber, and ash content using the following methods.

The moisture content was determined using the forced air draft oven method by drying 1 g of sample at 105°C for 16 h in an air oven [15]; the ash content was determined by adding 1 g of sample to a crucible and ashing in a muffle furnace maintained at 500°C for 4 h [15]; the total protein was determined by the Kjeldahl method (2300 Kjeltac Analyzer Unit, Foss A/S, Hillerød, Denmark) and was calculated using the general factor 6.25 [15]; the fat content was determined by extraction with light petroleum ether and then the solvent was removed by distillation using a Soxhlet extraction equipment. The residue was dried at 103°C and the fat content was determined gravimetrically [15]; the fiber content was determined using sequential extraction of food samples with sulfuric acid and sodium hydroxide. The insoluble residue collected by filtration was dried, weighed, and ashed [15]. After these analyses, the carbohydrate content was estimated by subtraction of the mean percentage values of moisture, ash, protein, lipids, and dietary fiber from 100 [16]. The energy content was calculated by multiplying the amounts of protein, fat, and carbohydrates by factors of 4, 9, and 4, respectively [17].

Procedures for determining GI

The GI value of the test foods was determined by feeding them to the 15 healthy subjects. The study of the subjects started in the morning after a 12-h overnight fast. A fasting blood sample was taken at 0 min; then immediately after this, the subjects consumed a standard or test food within 15 min in a comfortable place. All the test and standard foods were served with 200 mL water. Further blood samples were taken at 15, 30, 45, 60, 90, and 120 min after starting to eat.

The standard food provided was 50 g glucose powder (glucose dextrose monohydrate) dissolved in 200 mL water. This was consumed by the subject on two separate occasions and the other test foods were consumed only once in a random order, with a gap of at least a day between measurements to minimize any carry-over effects.

Blood was obtained from a finger prick using the One-touch® UltraSoft Lancing Device (One-Touch® Ultra², LifeScan, Livingstone, UK). The third finger on the left hand was used for all finger-prick blood samples. Before the finger prick, the subjects were encouraged to warm their hands to increase blood flow. The fingers were not squeezed to extract blood from the fingertip as squeezing may dilute the blood with plasma. A 0.6 µL blood sample was used to measure the blood glucose using an automatic analyzer (One-Touch® Ultra², LifeScan). The blood glucose meters were calibrated daily using control solutions from the manufacturer.

Calculation of GI and GL

The incremental area under the blood glucose response curve (IAUC), ignoring the area beneath the baseline, was calculated geometrically [18]. The IAUC for each test meal eaten by each subject was expressed as a percentage of the mean IAUC for the standard food eaten by the same subject as follows: $GI = (IAUC \text{ for the test food containing } 50 \text{ g of available carbohydrate} / IAUC \text{ of a standard food with an equal carbohydrates portion}) \times 100$. The GI of each tested food was taken as the mean value from the whole group of subjects [18-20]. The glycemic load (GL) was calculated according to the formula [18,20]: $GL = (GI \text{ of test food} \times \text{amount of carbohydrate in a serving of test food (g)}) / 100$.

The power of analysis

A sample size of 15 was considered sufficient with 90% power and an alpha of 0.05 using a paired *t*-test statistic considering an effect size of 1.4 for glucose response over time and 1.6 for glucose response between the foods.

Statistical analysis

Statistical analysis was performed using the statistical package SPSS 20.0 (Statistical Package for the Social Sciences, IBM, Cary, NC, USA). The Kruskal-Wallis test was used to compare the medians of measurements of nutrients for the types of food because the measurements did not satisfy the normality assumption of ANOVA. The paired *t*-test was used to compare the mean of the IAUC of the standard food with each of the test foods. Statistical significance was set at $P < 0.05$.

Results

Physical characteristics

The physical characteristics of the study subjects were as follows: mean weight 54.2 ± 4.7 kg, BMI 21.2 ± 2.1 kg/m², and WC 64.8 ± 4.7 cm (Table 2). The subjects for the present study were selected according to specific criteria. The reason for setting these inclusion criteria was to assess the GI value of selected commonly consumed

Table 2 Physical characteristics of the study population (mean \pm SD, n = 15)

Characteristics	Mean \pm SD
Age (years)	23.3 \pm 2.1
Height (m)	1.60 \pm 0.04
Weight (kg)	54.2 \pm 4.7
BMI (kg/m ²)	21.2 \pm 2.1
WC (cm)	64.8 \pm 4.7
FM (%)	23.0 \pm 4.0
FM (kg)	12.5 \pm 2.7
FFM (%)	0.8 \pm 0.1
FFM (kg)	41.6 \pm 3.6

BMI body mass index, WC waist circumference, FM fat mass, FFM fat-free mass.

traditional foods by subjects with a normal BMI with respect to their age, weight, and height. Additionally, other parameters, WC, FM, and FFM, were also taken into consideration when selecting subjects within the normal range.

Chemical analyses of test foods

The first stage involved in the calculation of the GI value was the proximate composition of the selected foods. Data on the proximate analysis per 100 g of each test food are given in Table 3. There were considerable variations in the nutritional composition of the analyzed foods, owing to the different ingredients and preparation methods. The moisture content ranged from 23.8 g for test food 1 (*Gurus*) to 77.1 g for test food 2 (*Assidah*). The protein content was lowest in test food 3 (*Saqo*) at 0.80 g and highest in test food 1 (*Gurus*) at 8.7 g.

The preparation of test food 1 (*Gurus*) involved frying bread in oil, thus giving it a high-fat profile (7.7 g) as illustrated in Table 3. Fiber analysis showed that test food 1 (*Gurus*) had the highest fiber level at 4.1 g, whereas test food 3 (*Saqo*) had the lowest level at 0.30 g.

The preparation methods for the test foods were based on different ingredients, which can be related to the

carbohydrate content and energy value of each food. For example, test food 5 (*Threed chicken*) had a lower carbohydrate content (12.2 g) than test food 1 (*Gurus*; 54.4 g). This large difference of 42.2 g was because of the ingredients of test food 1 (*Gurus*), which comprised mainly wheat flour, vegetable oil, salt, and water, after which frying in oil is required for the bread preparation. Thus, it had the highest energy value (322.2 kcal) of the test foods. Test foods 4 (*Marqoqa*) and 1 (*Gurus*) had the highest ash contents of 1.2 and 1.0 g, respectively.

Portion sizes, GI, and GL classification of test foods

The GI test is based on 50 g in each test food of available carbohydrate, defined as the total carbohydrate minus the dietary fiber. Therefore, the portion size of each test food, shown in Table 4, could vary according to the quantity of carbohydrate available in that food. The standard food (glucose) was tested using an equivalent amount of carbohydrate (50 g). The portion sizes of the test foods ranged from 91.8 g for test food 1 (*Gurus*) to 406.8 g for test food 5 (*Threed chicken*).

Table 4 also shows the GI and GL values and classification of the five test foods. These results showed that the GI values for the five test foods ranged from 71.7 to 99.4, which classified them all as high-GI foods. Test foods 1 (*Gurus*) and 5 (*Threed chicken*) had the lowest GI value (71.7 and 71.9, respectively), whereas test foods 2 (*Assidah*) and 3 (*Saqo*) had the highest GI value (99.2 and 99.4, respectively). The results also showed that the GL values for the five test foods ranged from 35.8 to 49.7, falling into the high-GL category. Test foods 3 (*Saqo*) and 2 (*Assidah*) had the highest GL values of 49.7 and 49.6, respectively, which corresponded with their high-GI values. Similar patterns were observed for test foods 1 (*Gurus*) and 5 (*Threed chicken*), which had lower GI values and also lower GL values of 35.8 and 35.9, respectively.

Glycemic response of food

The mean incremental areas under the glycemic response curves for the standard and test foods are shown in Figure 1.

Table 3 Proximate analysis (three analyses/food) of UAE five traditional foods (100 g); (mean \pm SD)

	Test food 1	Test food 2	Test food 3	Test food 4	Test food 5	P value*
	<i>Gurus</i>	<i>Assidah</i>	<i>Saqo</i>	<i>Marqoqa</i>	<i>Threed chicken</i>	
Protein (g)	8.76 \pm 0.16	0.99 \pm 0.05	0.80 \pm 1.09	5.89 \pm 0.89	7.81 \pm 1.76	0.012
Fat (g)	7.71 \pm 0.90	0.04 \pm 0.05	0.53 \pm 0.17	2.16 \pm 1.24	2.27 \pm 1.28	0.014
Fiber (g)	4.11 \pm 0.73	0.67 \pm 0.34	0.30 \pm 0.11	3.24 \pm 4.30	0.51 \pm 0.31	0.034
Moisture (g)	23.89 \pm 1.84	77.17 \pm 1.30	74.91 \pm 2.69	74.18 \pm 1.66	76.48 \pm 3.46	0.017
Ash (g)	1.07 \pm 0.21	0.05 \pm 0.01	0.04 \pm 0.01	1.20 \pm 0.26	0.64 \pm 0.04	0.014
Carbohydrate (g)	54.45 \pm 2.60	21.08 \pm 1.14	23.43 \pm 3.54	13.32 \pm 4.48	12.29 \pm 1.1	0.014
Energy (kcal)	322.26 \pm 6.55	88.65 \pm 4.74	101.66 \pm 11.54	96.31 \pm 15.50	100.82 \pm 14.93	0.025

Data expressed as 100 g on a fresh weight basis.

*P < 0.05.

Table 4 GI and GL value and classification of five foods commonly consumed in the United Arab Emirates

Test foods	Available CHO (g)	Portion size (g)	GI value	Classification	GL value	Classification
Test food 1 (<i>Gurus</i>)	50	91.8	71.7	High	35.85	High
Test food 2 (<i>Assidah</i>)	50	237.1	99.2	High	49.6	High
Test food 3 (<i>Saqa</i>)	50	213.4	99.4	High	49.7	High
Test food 4 (<i>Marqoqa</i>)	50	375.3	84.6	High	42.3	High
Test food 5 (<i>Threed chicken</i>)	50	406.8	71.9	High	35.95	High

The differences in glucose response between the test foods were analyzed using a *t*-test. The incremental increase in blood glucose at 15 min was significantly different between test food 1 (*Gurus*) and the standard food ($P = 0.017$; mean = 0.66 and 1.12, respectively); at 30 min, it was significantly different between test foods 1 (*Gurus*) and 4 (*Marqoqa*) ($P = 0.001$ and 0.027 ; mean = 1.42 and 0.85, respectively). At 45 min, the significant differences were between test foods 1 (*Gurus*), 4 (*Marqoqa*), and 5 (*Threed chicken*) ($P < 0.001$, 0.01 , and 0.01 ; mean = 1.04, 1.06, and 0.67, respectively). Test foods 4 (*Marqoqa*) and 5 (*Threed chicken*) showed a significant difference in incremental blood glucose at 60 min ($P = 0.02$ and 0.01 ; mean = 0.84 and 0.80, respectively); at 90 min, test food 5 showed a significant difference compared with the standard food ($P = 0.036$; mean = 0.41 and 1.20, respectively). At 120 min, there were no significant differences in the incremental blood glucose levels between any test foods ($P > 0.05$).

IAUC for the standard and test foods (mean \pm SD)

Table 5 shows the IAUC for the five test foods. Significant differences were found in the IAUC between the

standard and test food 1 (*Guru*) and test food 5 (*Threed chicken*) ($P = 0.001$ and <0.001 , respectively).

Discussion

Although the glycemic response is easy to measure, it is complicated to identify the mechanism of the glycemic response of food in the body. In fact, GI does not just measure the carbohydrate absorption in the small intestine directly but also indicates the effect of other factors in the foods tested that can influence the rate of carbohydrate absorption in the small intestine [9,21]. Adding fat and protein to carbohydrate-containing foods has the potential to reduce the glycemic response and lower the overall GI [22,23]. The mechanisms by which these nutrients affect blood glucose concentration have been proposed in many studies: high levels of protein produces greater gastric inhibitory peptide (GIP) and insulin responses resulting in a lower postprandial glucose peak and a reduced glycemic response from high-GI foods [24], while higher levels of fat content has the potential to delay gastric emptying, thereby slowing digestion and the absorption of glucose [23]. Fat may also affect the interaction of plasma glucose, insulin, and GIP [25]. This

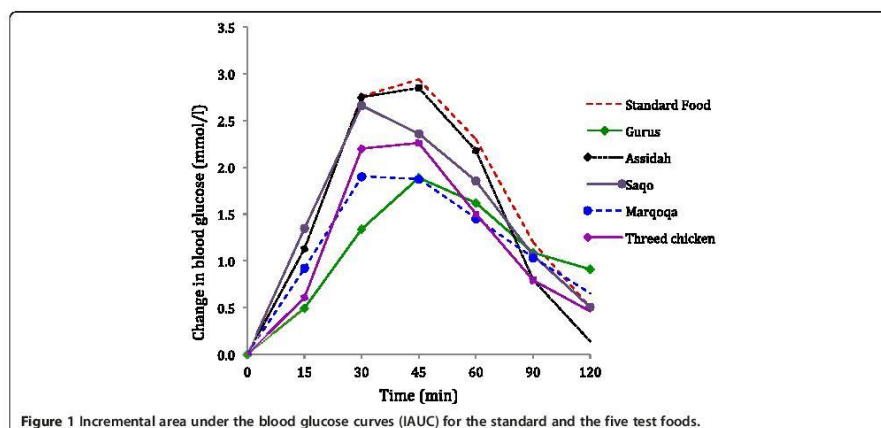


Figure 1 Incremental area under the blood glucose curves (IAUC) for the standard and the five test foods.

Table 5 Incremental area under the blood glucose response curve (IAUC) for test foods

Test foods	IAUC ± SD	P value
Standard food	202.57 ± 34.56	
Test food 1 (<i>Gurus</i>)	143.65 ± 56.15	0.001
Test food 2 (<i>Assidah</i>)	197.38 ± 58.15	0.745
Test food 3 (<i>Saqa</i>)	194.17 ± 57.91	0.654
Test food 4 (<i>Marqoqa</i>)	162.64 ± 74.22	0.074
Test food 5 (<i>Threed chicken</i>)	142.41 ± 32.37	0.001

could explain why test foods 5 (*Threed chicken*) and 1 (*Gurus*) with the high-protein content was observed to have a reduction of glycemic response in the IAUC compared with the standard food as well as having a GI value of 71.9 and 71.7, which is close to the cut-off point (70) for the high-GI category.

In the present study, for *Threed chicken*, the blood glucose peak response from the IAUC was at 45 min which was significantly different from that for the standard food ($P = 0.01$). Owen and Wolever [25] studied the consumption of 50 g available carbohydrate from white bread with 0, 5, 10, 20, or 40 g fat of non-hydrogenated-fat margarine in healthy subjects. Their results showed that there was no significant IAUC for blood glucose reduction when white bread was consumed with 5, 10, or 20 g of fat, but a significant reduction in the IAUC for blood glucose (30%) was observed when 40 g of fat was consumed with the white bread [26]. In contrast, a number of studies in foods commonly consumed in the UK [19] and in China [26] have found that the amount of protein or fat does not affect the glycemic value of foods. The present study found that there was a reduction in the IAUC for *Threed chicken* compared with the standard food where the amounts of protein may have affected the postprandial glycemic responses of that food [27]. It is assumed that the decrease in the postprandial glycemic response from the IAUC of *Gurus* was owed to its high-fat and fiber content (7.7 and 4.1 g), which has an effect on the GI value of the food. A study by Livesey and Tagami [28] found that increasing the viscous soluble fiber consumption has a great effect on lowering the glycemic response but limits its palatability. The viscous fiber blend significantly reduced the glycemic index of food by 74% in healthy participants and by 63% in participants with diabetes [27]. Similarly, Jenkins et al. [29] showed a reduction in the IAUC of commonly consumed meals in healthy subjects when 5 g of novel viscous polysaccharide (NVP) was added.

All traditional foods tested contained white flour in their ingredients and different moisture contents and were prepared using different cooking time, which can

all be related to explain the differences in the degree of starch gelatinization and consequently the GI values. Heat, moisture, and cooking method have been shown to be factors that can affect the GI of foods [30] and the GI of starchy food can be altered by the level of gelatinization [31].

Since all the test foods contained flour, we found that the effects of cooking method played a role in increasing the moisture content and therefore the GI value of foods. This was observed particularly in the preparation of *Saqa*, *Assidah*, and *Marqoqa*, which had the highest GI values (99.4, 99.2, and 84.6, respectively) compared with the other foods. The cooking process for *Saqa* involved mixing starchy seeds with sugar and fat and then boiling them slowly in water to form a viscous slurry, thus resulting in the maximum hydrolysis of the starch present in the *Saqa* seeds. These methods are the reasons behind the high glycemic response (IAUC) to *Saqa*. However, the preparation method for *Marqoqa* included a long cooking time (about 3 h) for all the ingredients with water at a high temperature, which was then poured over the white bread. High temperature and increased cooking time in a large quantity of water were associated with increased starch gelatinization and degree of digestibility, as well as increased blood glucose levels [32]. Conversely, although *Gurus* had a high-fiber content, its GI value was still high (71.7). The increased GI value of *Gurus* could be because of the cooking method, which involved frying the ingredients with vegetable oil. Bahado-Singh et al. [33], found that fried sweet potato had an intermediate to moderately high GI value (63 ± 2 to 77 ± 4), which was close to the GI value of *Gurus* found in the present study.

Different nutritional and physiological factors might have an effect on the blood glycemic response and the GI value of traditional foods. Included among these factors are the digestibility rate of the starch, the interactions of starch absorption with the amount of fiber, fat and protein present, and the cooking methods. In *Threed chicken*, the high-protein content led to a lower postprandial glucose peak and a decrease in glycemic response compared with other high-GI foods. Similar findings of a glycemic response reduction were observed in *Gurus*, with its high-fiber, fat, and protein content. The preparation method for *Gurus*, given its high-fat and protein content, led to delayed gastric emptying, thereby slowing down the rate of glucose digestion and absorption, while the traditional cooking procedures for *Assidah*, *Saqa*, and *Marqoqa* were associated with increases in the degree of starch gelatinization and consequently an increased GI value. The GI value was affected to varying degrees by the different preparation methods and ingredients of the five test foods.

Conclusions

In the assessment of the nutrient composition and GI value of traditional foods, the present study can conclude that all the selected test foods, commonly consumed in UAE culture, had high GI values. These findings emphasize that the dietary habits and the consumption of traditional foods need to be assessed in connection with other factors with the evidence of the increasing prevalence of obesity in the UAE.

A limitation of this study was the effect of the different cooking methods used by the restaurants, which may have affected the GI values of these foods.

Since the traditional foods tested are frequently consumed by the Emirati, the authors of this study recommend the consumption of smaller portion sizes along with low-GI foods to overcome the high-GI level.

Recommendation

To completely address the objectives of this study, additional research should be performed using other traditional Emirati foods and obese and diabetic individuals as subjects to examine how this links to the increased prevalence of diabetes and obesity in the population. Studies on the chemical analysis and GI of other traditional foods are strongly recommended to be used as preliminary references for setting up a GI and GL database for traditional Emirati foods. The evaluation of an acceptable portion size for a low-GL diet is also needed. Moreover, preliminary studies to evaluate a low-GI diet using commonly consumed foods and the blood glucose and insulin responses among healthy subjects or diabetic patients are essential.

Abbreviations

BMI: Body mass index; CVD: Cardiovascular disease; DM: Diabetes mellitus; FM: Fat mass; FFM: Fat-free mass; GI: Glycemic index; GIP: Gastric inhibitory peptide; GL: Glycemic load; IAUC: Incremental area under the blood glucose response curve; IDF: International Diabetes Federation; NVP: Novel viscous polysaccharide; UAE: United Arab Emirates; UAEU: United Arab Emirates University; WC: Waist circumference; WHO: World Health Organization.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

ASAD, LCL, and SAW conceived, designed, and supervised the experiments. ASAD and LCL wrote the paper. AKAM, FTAM, and MNM performed the experiments. ASAD, AKAM, and EMM analyzed the data. ASAD contributed reagents/materials/analysis tools. ASAD, LCL, AHJ, and AKAM contributed to the drafting of the manuscript. All authors read and approved the final manuscript.

Acknowledgements

The authors would like to thank the Emirates Foundation for funding this project. We would also like to acknowledge the university students for their participation as subjects and the laboratory staff who performed the chemical analyses.

Author details

¹Nutrition and Health Department, College of Food and Agriculture, United Arab Emirates University, Al Ain, United Arab Emirates. ²Department of Biotechnology, Faculty of Technology, Maharakham University, Maharakham 44150, Thailand. ³Department of Statistics, College of Business, United Arab Emirates University, Al Ain, United Arab Emirates.

Received: 30 June 2014 Accepted: 20 November 2014
 Published: 21 January 2015

References

- Steinberger J, Daniels SR: Obesity, insulin resistance, diabetes, and cardiovascular risk in children: an American Heart Association scientific statement from the Atherosclerosis, Hypertension, and Obesity in the Young Committee (Council on Cardiovascular Disease in the Young) and the Diabetes Committee (Council on Nutrition, Physical Activity, and Metabolism). *Circulation* 2003, **107**(10):1448-1453.
- Hensrud D: Dietary treatment and long-term weight loss and maintenance in type 2 diabetes. *Obes Res* 2001, **9**(Suppl 4):348S-353S.
- Li SC, Liu YH, Liu JF, Chang WH, Chen CM, Chen CY: Almond consumption improved glycemic control and lipid profiles in patients with type 2 diabetes mellitus. *Metabolism* 2011, **60**(4):474-479.
- International Diabetes Federation: *IDF Diabetes Atlas*. 6th edition. Brussels: International Diabetes Federation; 2013. [<http://www.idf.org/diabetesatlas>]. Accessed 19 February 2014.
- Hummasti S, Hotamisliqilil GS: Endoplasmic reticulum stress and inflammation in obesity and diabetes. *Circ Res* 2010, **107**(5):579-591.
- Martin CL, Murphy SP, Au LMD: Compiling glycemic index and glycemic load values for addition to a food composition database. *J Food Compos Anal* 2008, **21**(6):469-473.
- Aller EEJG, Abete I, Astrup A, Martinez JA, Baak MA: Starches, sugars and obesity. *Nutrients* 2011, **3**(3):341-369.
- Foster-Powell K, Holt SH, Brand-Miller JC: International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr* 2002, **76**:5-56.
- Aston LM, Gambell JM, Lee DM, Bryant SP, Jebb SA: Determination of the glycemic index of various staple carbohydrate-rich foods in the UK diet. *Eur J Clin Nutr* 2008, **62**(2):279-285.
- Foster-Powell K, Miller JB: International tables of glycemic index. *Am J Clin Nutr* 1995, **62**(2):871S-890S.
- Brown RC, Ning B, Williams S, Venn B, Green TJ: The macronutrient composition of the evening meal before glycemic index testing has no effect on glycemic response. *FASEB J* 2009, **23**(Meeting Abstract Supplement):544A.
- Gibson RS: *Principles of Nutritional Assessment*. 2nd edition. New York: Oxford University Press; 2005.
- Chang C-H, Chen C-Y, Huang K-C, Wu C-H, Hsiung CA, Hsu C-C, Chen C-Y: Comparison of three BIA muscle indices for sarcopenia screening in old adults. *Eur Geriatr Med* 2013, **4**(3):145-149.
- World Health Organization (WHO): *BMI Classification*. Geneva: World Health Organization; 2006. [http://apps.who.int/bmi/index.jsp?introPage=intro_3.html]. Accessed 20 February 2014.
- AOAC: *Official Methods of Analyses* 2003. Washington, DC: Association of Official Analytical Chemists; 2003.
- Bouaziz MA, Besbes S, Blecker C, Attia H: Chemical composition and some functional properties of soluble fibro-protein extracts from Tunisian date palm seeds. *Afr J Biotechnol* 2013, **12**(10):1121-1131.
- Dashti BH, Al-Awadi F, Khabafawi MS, Al-Zenk S, Sawaya W: Nutrient contents of some traditional Kuwaiti dishes: proximate composition and phytate content. *Food Chem* 2001, **74**(2):169-175.
- Food and Agriculture Organization/World Health Organization: *Carbohydrates in Human Nutrition 1998, Report of a Joint FAO/WHO Expert Consultation*. Rome: FAO; 1998.
- Henry CJ, Lightowler HJ, Strik CM, Renton H, Hails S: Glycaemic index and glycaemic load values of commercially available products in the UK. *Br J Nutr* 2005, **94**(6):922-930.
- Kris Y, Lok RC, Chan D, Li L, Leung G, Woo J, Lightowler HJ, Henry CJ: Glycaemic index and glycaemic load values of a selection of popular foods consumed in Hong Kong. *Br J Nutr* 2010, **103**:556-560.
- Wolever TM: Carbohydrate and the regulation of blood glucose and metabolism. *Nutr Rev* 2003, **61**(5 Pt 2):S40-S48.
- Flint A, Moller BK, Raben A, Pedersen D, Tetens I, Holst JJ, Astrup A: The use of glycaemic index tables to predict glycaemic index of composite breakfast meals. *Br J Nutr* 2004, **91**(6):979-989.
- Henry CJ, Lightowler HJ, Kendall FL, Storey M: The impact of the addition of toppings/fillings on the glycaemic response to commonly consumed carbohydrate foods. *Eur J Clin Nutr* 2006, **60**(6):763-769.
- Hätönen KA, Virtamo J, Eriksson JG, Sinkko HK, Sundvall JE, Valsta LM: Protein and fat modify the glycaemic and insulinemic responses to a mashed potato-based meal. *Br J Nutr* 2011, **106**(2):248-253.

25. Owen B, Wolever TM: **Effect of fat on glycaemic responses in normal subjects: a dose-response study.** *Nutr Res* 2003, **23**(10):1341-1347.
26. Chen YJ, Sun FH, Wong SH, Huang YJ: **Glycemic index and glycemic load of selected Chinese traditional foods.** *World J Gastroenterol* 2010, **16**(12):1512-1517.
27. Jenkins AL, Jenkins DJ, Wolever TM, Rogovik AL, Jovanovski E, Bozilov V, Rahelić D, Vuksan V: **Comparable postprandial glucose reductions with viscous fiber blend-enriched biscuits in healthy subjects and patients with diabetes mellitus: acute randomized controlled clinical trial.** *Croat Med J* 2008, **49**(6):772-782.
28. Livesey G, Tagami H: **Interventions to lower the glycemic response to carbohydrate foods with a low-viscosity fiber (resistant maltodextrin): meta-analysis of randomized controlled trials.** *Am J Clin Nutr* 2009, **89**(1):114-125.
29. Jenkins AL, Kadinik V, Lyon M, Wolever TM: **Effect of adding the novel fiber, PGX®, to commonly consumed foods on glycemic response, glycemic index and GRIP: a simple and effective strategy for reducing post prandial blood glucose levels—a randomized, controlled trial.** *Nutr J* 2010, **9**:58.
30. Ek KL, Brand-Miller J, Copeland L: **Glycemic effect of potatoes.** *Food Chem* 2012, **133**:1230-1240.
31. Juansang J, Puttanlek C, Rungsardthong V, Pancha-arnon S, Uttapap D: **Effect of gelatinisation on slowly digestible starch and resistant starch of heat-moisture treated and chemically modified canna starches.** *Food Chem* 2012, **131**(2):500-507.
32. Lehmann U, Robin F: **Slowly digestible starch - its structure and health implications: a review.** *Trends Food Sci Technol* 2007, **18**(7):346-355.
33. Bahado-Singh PS, Riley CK, Wheatley AQ, Lowe HJ: **Relationship between processing method and the glycemic indices of ten sweet potato (*Ipomoea batatas*) cultivars commonly consumed in Jamaica.** *J Nutr Metab* 2011, **58**:48326.

doi:10.1186/2055-0928-1-4

Cite this article as: Al Dhaheeri *et al.*: The effect of nutritional composition on the glycemic index and glycemic load values of selected Emirati foods. *BMC Nutrition* 2015 **1**:4.

Submit your next manuscript to BioMed Central and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at
www.biomedcentral.com/submit



Appendix 15: Research Paper 2



RESEARCH ARTICLE

A Cross-Sectional Study of the Prevalence of Metabolic Syndrome among Young Female Emirati Adults

Ayesha S. Al Dhaheri^{1*}, Maysm N. Mohamad¹, Amjad H. Jarrar¹, Eric O. Ohuma^{2,3}, Leila Cheikh Ismail⁴, Fatima T. Al Meqbaali¹, Usama Souka¹, Syed M. Shah⁵

1 Nutrition and Health Department, College of Food and Agriculture, United Arab Emirates University, Al Ain, UAE, **2** Centre for Statistics in Medicine, Nuffield Department of Orthopaedics, Rheumatology & Musculoskeletal Sciences, University of Oxford, Oxford, United Kingdom, **3** Centre for Tropical Medicine and Global Health, Nuffield Department of Medicine, University of Oxford, Oxford, United Kingdom, **4** Nuffield Department of Obstetrics & Gynaecology and Oxford Maternal & Perinatal Health Institute, Green Templeton College, University of Oxford, Oxford, United Kingdom, **5** Institute of Public Health, College of Medicine and Health Sciences, United Arab Emirates University, Al Ain, UAE

* ayesha_aldhaheri@uaeu.ac.ae



OPEN ACCESS

Citation: Al Dhaheri AS, Mohamad MN, Jarrar AH, Ohuma EO, Ismail LC, Al Meqbaali FT, et al. (2016) A Cross-Sectional Study of the Prevalence of Metabolic Syndrome among Young Female Emirati Adults. *PLoS ONE* 11(7): e0159378. doi:10.1371/journal.pone.0159378

Editor: Rasheed Ahmad, Dasman Diabetes Institute, KUWAIT

Received: February 27, 2016

Accepted: July 2, 2016

Published: July 14, 2016

Copyright: © 2016 Al Dhaheri et al. This is an open access article distributed under the terms of the [Creative Commons Attribution License](https://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Data Availability Statement: The United Arab Emirates University ethical restrictions prohibit the authors from making the dataset publicly available. However, the data can be provided upon request to all interested researchers by contacting the lead author Ayesha Salem Al Dhaheri, email: Ayesha_aldhaheri@uaeu.ac.ae.

Funding: This study was supported by a research start-up grant from the United Arab Emirates University (CFA-31F038) and was given to ASA. The funding agent had no role with the study design, data collection and analysis, decision to publish, or

Abstract

Introduction

Metabolic syndrome (MetS) is a growing problem in the United Arab Emirates (UAE). Moreover, the prevalence of overweight and obesity is rapidly increasing in the UAE especially among young females. However, few studies have evaluated the prevalence of MetS among young female adults in the UAE. This study determined the prevalence of MetS in Emirati females aged 17–25 years and its relation to overweight and obesity.

Methods

In total, 555 Emirati female college students were enrolled in a cross-sectional study, conducted during 2013–2014 at United Arab Emirates University in Al Ain, UAE. Anthropometric measurements, blood pressure and biochemical measurements were collected. MetS was defined according to the harmonised International Diabetes Federation criteria.

Results

Of the 555 participants enrolled, 23.1% were overweight and 10.4% were classified as obese. The overall prevalence of MetS was 6.8%. MetS prevalence was highest among obese participants (34.5%), as compared with normal-weight (1.7%) and overweight (10.1%) participants. MetS was significantly associated with overweight (adjusted odds ratio [aOR] = 3.8, 95% confidence interval [CI]; 1.15–12.52) and obesity (aOR = 11.2, 95% CI; 3.1–40.9), as compared with normal-weight. Waist-hip ratio ≥ 0.8 (aOR = 3.04, 95% CI; 1.10–8.44) was significantly associated with MetS, as compared with waist-hip ratio <0.8 . The odds of MetS were 22 fold higher in participants with glycated haemoglobin (HbA1c) $\geq 6.5\%$ (aOR = 22.5, 95% CI; 6.37–79.42) compared to HbA1c $<6.5\%$. This difference was 9

preparation of the manuscript. <http://www.uaeu.ac.ae/en/>

Competing Interests: The authors have declared that no competing interests exist.

fold higher when HbA1c between 5.6%–6.4% was compared to HbA1c <5.6% (aOR = 8.9, 95% CI; 3.4–23.5).

Conclusion

The prevalence of MetS among obese Emirati female students was significantly higher than overweight and normal weight students. The high prevalence of MetS highlights the importance of regular screening and intervention programmes targeting weight reduction.

Introduction

Non-communicable diseases (NCDs) are the leading cause of deaths worldwide, and diabetes mellitus (DM) is the fourth major cause of NCD deaths [1].

A diagnosis of metabolic syndrome (MetS) is based on the existence of pre-diabetes combined with dyslipidaemia (elevated levels of total or low-density lipoprotein [LDL] cholesterol, or low high-density lipoprotein [HDL] cholesterol levels), elevated blood pressure and obesity [2]. Based on the International Diabetes Federation (IDF) definition for MetS; a study conducted in Emirati adults (>20 years old) by Malik and Razig in 2008 reported the total MetS prevalence was 40.5% and was higher among women (45.9%) than men (32.9%) [3]. The study by Mehairi et al. in 2013 showed an increase in the prevalence of MetS with higher body mass index (BMI) values [4].

The lifestyle of the Emirati population has changed considerably over the past 40 years due to the rapid improvement in socioeconomic status. This transition has led to less physical activity and altered eating habits. These changes, in addition to the adoption of a western lifestyle and diet, have led to the rise in the prevalence of overweight and obesity in the UAE, particularly among females [5]. There is a paucity of data available about the prevalence of MetS and its relation with overweight and obesity among young female adults in the UAE. Moreover, the population structure of UAE is mainly young and has therefore been greatly affected by the rapid socioeconomic changes.

This study aimed to determine the prevalence of MetS in Emirati females aged 17–25 years as this age range has not been studied previously, and its relation to overweight and obesity in Al Ain, UAE.

Design and Methods

Study population

A cross-sectional population-based study was conducted during the academic year 2013/2014 at United Arab Emirates University (UAEU) in Al Ain, UAE. The university currently enrolls around 14,000 students each year.

The study population included students from all nine colleges of the university. Participants were asked to read the information sheet carefully and were given the chance to ask any question related to the study before providing written informed consent to participate. Each participant was assigned a personal identification number to maintain anonymity and data confidentiality. Ethical approval was obtained from the United Arab Emirates University Scientific Research Ethics Committee (Reference number DVCRGS/370/2014).

A stratified random sampling approach was used to select eligible participants [6]. All female students were divided into strata by college (nine strata), and then a random subsample

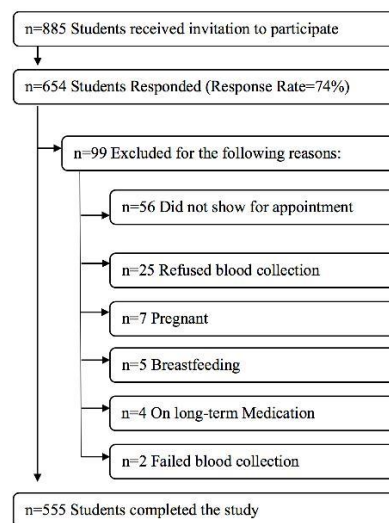


Fig 1. Study participants enrolment process

doi:10.1371/journal.pone.0159378.g001

proportional to size that consisted of 10% of the students from each college were selected. All eligible students were then contacted via e-mail to request their participation in the study. Of the total number of females registered in the university during the 2013/2014 academic year ($n = 8846$), a subsample of 885 students received a request to participate in the study. The enrolment process of the study participants is shown in Fig 1.

Questionnaire

Each participant completed a self-reported questionnaire on demographic data, supplements and medication use, tobacco use, diet, physical activity, sleeping patterns, perceptions about obesity, personal history of NCDs and family history (first-degree relatives) of NCDs. Participants completed the questionnaire under the supervision of the research team to respond to any clarification needed on any aspects of the questionnaire or the study as a whole.

Anthropometric measurements and physical examination

Height was measured using a portable stadiometer (Seca Stadiometer, Seca Ltd, Birmingham, UK), in the standing position, without shoes and recorded to the nearest millimetre [7]. Body weight (Kg) and body composition were measured using the Tanita Segmental Body Composition Analyser (Tanita BC-418, Tanita Corp., Tokyo, Japan) [8]. The World Health Organization classification of the BMI ($\text{weight} / \text{height}^2$; kg/m^2) was used to classify underweight, normal-weight, overweight and obesity in the studied population [9]. Waist circumference (WC) was measured in centimetres (cm) using a plastic tape, at the midway between the inferior margin of the ribs and the superior border of the iliac crest or at umbilicus level for obese

participants [10]. Hip circumference (HC) was measured at the level of maximum posterior extension of the buttocks [11]. Waist-hip ratio (WHR) was calculated by dividing WC by HC. Total body fat was measured from skin-fold thickness at four sites (biceps, triceps, subscapular and suprailliac) using the equation described by Durnin and Womersley in 1974 [12]. Cut-off points for body fat percentage, WHR, and anaemia were based on World Health Organization recommended values [13–15].

The anthropometric measurements were carried out by a trained anthropometrist to reduce inter-observer variations. All measurements were completed during a single 50-minute session (to eliminate missing data), with the participants reporting to the clinic having fasted for 12–14 hours prior to testing, although drinking water was allowed in moderation. Measurements were taken in the morning between 7:00–10:00 a.m. to minimize inter-day fluctuations. Participants were asked not to visit the clinic during their menstrual cycle. Participants were encouraged to rest for 15 minutes before any measures were performed to enable them to relax before performing any of the tests. Each measurement was taken three times and averaged to improve accuracy. All measuring devices were calibrated on a daily basis.

Blood pressure (BP) was measured by a registered nurse using a validated and calibrated digital automated sphygmomanometer (Omron Hem-907, Omron Healthcare, Kyoto, Japan), after the participant had rested for at least 15 minutes [16]. Two consecutive measurements were obtained 5-minutes apart and the average of the two readings recorded [17].

Laboratory measurements

A registered nurse collected a 5-ml venous blood sample from each participant after 12 hours of fasting via a vacuum system (vacuette 0.64 × 19mm, Greiner Bio-One, Kremsmünster, Austria), into a serum separator tube with clot activator (Vacutest Kima srl, Arzergrande, Italy). Blood samples were centrifuged (2,500 rpm, 15 minutes) and the serum was properly separated, identified and stored at –80°C until the time of analyses.

Samples were thawed on ice for 30 minutes with proper handling during thawing and storage [18]. The total cholesterol, triglyceride (TG), LDL-cholesterol, HDL-cholesterol (HDL-C), high-sensitivity C-reactive protein and fasting blood glucose concentrations in human serum analyses were performed using the Cobas C111 automated biochemical analyser (Roche Diagnostics, Indianapolis, IN, USA) [19]. The HemoCue Hb 201+ portable photometer system (HemoCue AB, Ängelholm, Sweden) was used for the assessment of haemoglobin concentration and the HemoCue HbA1c 501 system was used for assessing glycated haemoglobin (HbA1c) percentage in whole blood. MetS was defined according to the harmonised definition established in 2009 by the IDF and the American Heart Association/the National Heart, Lung, and Blood Institute (AHA/NHLBI) as the presence of any three of the following five factors: elevated WC (≥ 80 cm in women); hypertriglyceridaemia (TG ≥ 150 mg/dL or drug treatment for elevated TG); reduced HDL-C (< 50 mg/dL in women or drug treatment for reduced HDL-C); elevated BP (systolic BP ≥ 130 mmHg and/or diastolic BP ≥ 85 mmHg or use of anti-hypertensive drugs); and elevated fasting blood glucose ≥ 100 mg/dL or use of hypoglycaemic medication [20].

Sample size calculation

Sample size was calculated using the Minitab software (version 16, Minitab Inc, PA, State College, USA) and was based on an expected prevalence of 4%. At 80% power and 5% significance level, a sample size of 555 would achieve a 1.58% margin of error for the survey of the female student population.

Statistical analyses

Data analyses were carried out using Stata version 13 (Stata Corp, College Station, TX, USA). Descriptive statistics were computed and summarised; continuous variables were summarised using means and standard deviations (SD) and categorical variables using proportions. The Student's *t*-test was used for continuous variables to compare mean differences between participants with and without MetS. Univariable and multivariable logistic regression analysis was used to study the association between anthropometric and chemical measures and the presence or absence of MetS as the outcome variable. To account for perfect prediction of MetS by BMI categories and the small sample size across BMI class, we applied the Firth logistic regression to obtain reasonable and robust estimates. All statistical significance was assessed at the 5% significance level.

Results

Of the 885 students invited to participate, the response rate was 74% ($n = 654$). The overall prevalence of MetS was 6.8% (95% CI: 5% to 9%). The demographic and clinical characteristics of the study population by MetS status are presented as mean \pm SD, in Table 1. The mean age of the study population was 20.4 ± 1.7 years. The average age of participants with MetS was not significantly different from those without MetS (20.9 vs. 20.4 years, $P = 0.057$). However, participants with MetS had a significantly higher weight, height, HC, BMI, body fat percentage, serum LDL (mg/dL) and HbA1c level ($P < 0.01$).

No MetS defining components were found in 242 (43.6%) participants. At least one MetS component was found in 213 participants (38.4%); two MetS components were present in 62 participants (11.2%); three MetS components were found in 27 participants (4.9%); four components of MetS were present in 10 participants (1.8%); and all five MetS components were found in only one participant (0.2%). The most frequent component of MetS was reduced HDL-C levels (48.8%), followed by central obesity (18.2%) and impaired fasting glucose (9.7%) (Fig 2).

A Chi-square test of association between MetS and BMI categories among young female adults showed a statistically significant association ($P < 0.001$), and was particularly high among obese participants (34.5%) compared to 10.1% overweight, and 1.7% normal-weight. None of the five MetS components were observed in 69% of the normal-weight participants whereas all obese participants had at least one MetS component. Obese participants were more likely to have three or more MetS components (52.6%) than overweight (34.2%) and normal-

Table 1. Demographic and clinical characteristics by metabolic syndrome status.

	With Metabolic Syndrome (N = 38)	Without Metabolic Syndrome (N = 517)	Student's <i>t</i> -test <i>P</i> -value
	Mean \pm SD	Mean \pm SD	
Age (Year)	20.9 \pm 1.7	20.4 \pm 1.7	0.057
Weight (Kg)	82.1 \pm 17.1	58.9 \pm 12.2	< 0.001
Height (cm)	161.3 \pm 5.2	158.9 \pm 5.8	0.013
Hip Circumference (cm)	115.42 \pm 12.35	98.67 \pm 9.71	<0.001
Body Mass Index (Kg/m ²)	31.5 \pm 6.3	23.2 \pm 4.6	<0.001
Body Fat (%)	40.3 \pm 3.9	32.9 \pm 5.3	<0.001
Serum Low Density Lipoprotein (mg/dL)	102.5 \pm 30.9	91.0 \pm 24.7	0.006
Serum Total Cholesterol (mg/dL)	165.7 \pm 37.9	155.6 \pm 31.9	0.063
Glycated Haemoglobin (%)	6.3 \pm 1.1	5.5 \pm 0.8	<0.001
Haemoglobin (g/dL)	12.00 \pm 1.6	11.8 \pm 1.5	0.482

doi:10.1371/journal.pone.0159378.t001

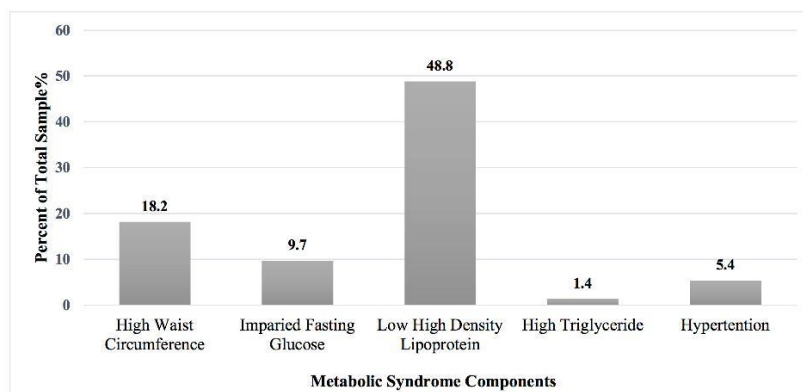


Fig 2. Prevalence of metabolic syndrome components among UAEU young female adults 17–25 years (N = 555), Al Ain, UAE.

doi:10.1371/journal.pone.0159378.g002

weight (13.2%) participants. Furthermore, none of the underweight participants had three or more MetS components (Fig 3).

Table 2 shows the univariable and multivariable logistic regression results for the odds of MetS by potential risk factors. In the univariable analyses, older participants (23–25 years) were three times more likely to have MetS (odds ratio [OR]: 2.96; 95% CI: 1.03 to 8.52) than younger participants (17–19 years) but this effect was not significant in the multivariable

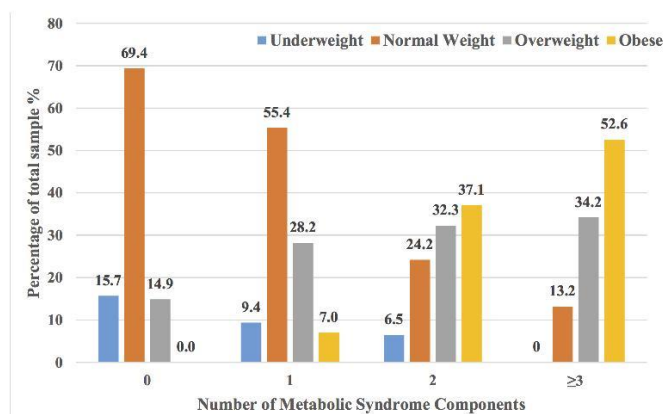


Fig 3. Percentage of participants per number of metabolic syndrome components and BMI category among young female adults aged 17–25 years (n = 555), Al Ain, UAE.

doi:10.1371/journal.pone.0159378.g003

Table 2. Risk factors for MetS among young female adults aged 17–25 years (n = 555), Al Ain, UAE.

Characteristics	Sample	n (%)	With MetS			
			Crude Odds Ratio (95%CI)	P-value	Adjusted Odds Ratio (95%CI)	P-value
Age (Year)						
17–19	194	8 (4.1)	Reference		Reference	
20–22	299	23 (7.7)	1.94 (0.85, 4.42)	0.116	1.89 (0.72–4.94)	0.19
23–25	62	7 (11.3)	2.96 (1.03, 8.52)	0.044	1.14 (0.30–4.30)	0.85
Family history of diabetes or hypertension (%)						
No	250	9 (3.6)	Reference		Reference	
Yes	305	29 (9.5)	2.81 (1.31, 6.06)	0.008	1.85 (0.73–4.65)	0.19
Body Mass Index (Kg/m²)						
Underweight (<18.5)	62	0 (0.0)	0.44 (0.02, 8.03)	0.58	0.85 (0.04–17.26)	0.92
Normal-weight (18.5–<25)	306	5 (1.6)	Reference		Reference	
Overweight (25–29.9)	129	13 (10.1)	6.35 (2.30, 17.51)	<0.001	3.80 (1.15–12.52)	0.028
Obese (≥30.0)	58	20 (34.5)	29.19 (10.75, 79.28)	<0.001	11.19 (3.06–40.86)	<0.001
Body Fat (%)						
<35%	328	4 (1.2)	Reference		Reference	
≥35%	227	34 (14.9)	14.27 (4.99, 40.83)	<0.001	3.12 (0.91–10.68)	0.07
Waist-Hip Ratio						
<0.8	509	28 (5.5)	Reference		Reference	
≥0.8	46	10 (21.7)	4.77 (2.15, 10.59)	<0.001	3.04 (1.10–8.44)	0.033
Anaemia						
No	271	22 (8.1)	Reference		Reference	
Yes	284	16 (5.6)	0.67 (0.35, 1.32)	0.249	1.04 (0.46–2.35)	0.92
Total Cholesterol (mg/dL)						
<200	502	29 (5.8)	Reference		Reference	
≥200	53	9 (16.9)	3.34 (1.48, 7.49)	0.004	1.71 (0.53–5.55)	0.37
Low Density Lipoprotein (mg/dL)						
<130	380	21 (5.5)	Reference		Reference	
≥130	173	17 (9.8)	1.86 (0.96, 3.63)	0.067	0.92 (0.37–2.32)	0.86
Glycated Haemoglobin (%)						
<5.6	374	6 (1.6)	Reference		Reference	
5.6–6.4	133	23 (17.3)	12.82 (5.09, 32.29)	<0.001	8.92 (3.39–23.48)	<0.001
≥6.5	48	9 (18.8)	14.15 (4.78, 41.86)	<0.001	22.49 (6.37–79.42)	<0.001

doi:10.1371/journal.pone.0159378.t002

analyses (odds ratio [OR]: 1.14; 95% CI: 0.30 to 4.30). Participants who reported having a family history of diabetes or hypertension (n = 305) had a 2.8 times elevated risk of MetS (OR: 2.81; 95% CI: 1.31 to 6.06) compared with participants without a family history of diabetes or hypertension in the univariable analyses but not in the adjusted analyses (OR: 1.85; 95% CI: 0.73 to 4.65). Participants who were overweight or obese were, respectively, 6.4 (95% CI: 2.3 to 17.5) and 29 (95% CI: 29.2 to 79.3) times more likely to have MetS than those of normal-weight in the univariable analysis. These findings remained significant even after adjusting for other potential confounders (i.e. OR = 3.8; 95% CI: 1.15–12.52 for overweight; and OR = 11.2; 95% CI: 3.06–40.86 for obese participants). Participants with percentage body fat ≥35% (n = 227) showed a significantly higher risk for the development of MetS in the univariable analyses (OR: 14.27; 95% CI: 4.99 to 40.83; *P* < 0.001), but the difference was not significant after adjusting for other factors (OR = 3.12; 95% CI: 0.91–10.68). A WHR of more than 0.8 was significantly associated with at least three times increased risk of MetS (*P* < 0.001) in the adjusted analyses when compared with those with a WHR < 0.8 (aOR = 3.04; 95% CI: 1.10–

8.44). Elevated HbA1c ($\geq 6.5\%$) showed a high significant association with the presence of MetS (OR: 14.15; 95% CI: 4.78 to 41.86; $P < 0.001$) in univariable analyses and remained significant in the adjusted analyses (adjusted OR [aOR]: 22.49, 95% CI: 6.37 to 79.42; $P < 0.001$). Total cholesterol ≥ 200 mg/dL and LDL ≥ 130 mg/dL conferred a greater likelihood for MetS: OR: 3.34 (95% CI: 1.48 to 7.49) and OR: 1.86 (95% CI: 0.96 to 3.63), respectively, in the univariable analyses but not in the adjusted analyses.

A subgroup analysis was conducted for the study population excluding all females with HbA1c $> = 6.5$ resulting in a total sample size of 507 participants. Results of the subgroup analysis remain largely unchanged based on the magnitude of the effect sizes, direction of significance and overall conclusions. However, in the multivariate analysis, only waist hip-ratio was no longer significant in the subgroup analysis (OR = 2.12; 95% CI: 0.65–6.87; $P = 0.211$)

Discussion

In the United Arab Emirates (UAE) rapid socioeconomic growth has resulted in profound lifestyle changes including sedentary behaviours, westernized diets and increased energy intake [5]. The prevalence of MetS among Emirati females has been reported to be higher than that for Emirati males in the adult population (32.9% among men, 45.9% among women) [3]. This research highlights the importance of investigating MetS among young female adults, to facilitate understanding of the prevalence and risk factors of MetS. There is paucity of data on the prevalence of MetS among Emirati females aged 17–25 years. The current study reveals a MetS prevalence of 6.8% among young female Emirati adults aged 17–25 years, and 34.5% among young obese female Emirati adults.

The results of the current study are in line with findings among college students (18–26 years) in Saudi Arabia [21], where the overall MetS prevalence was 7.8%, and 26.4% in obese students. In Kuwait, the prevalence of MetS was even higher among female adolescents (10–19 years) at 9.1% and 14.8% according to ATP III and IDF criteria, respectively [22]. These numbers are certainly close to those described in the UAE, which is not surprising, considering the relatively similar rapid increase in obesity and diabetes rates throughout the Gulf region [23, 24]. These trends are likely to be a result of the sedentary and westernised lifestyle [25–28], and could also be partially explained by the “thrifty genes” hypothesis [29], which suggests that the genotype of mankind existed as hunter-gatherers can efficiently store food in the adipose tissue during periods of food abundance, to compensate for periods of food shortage.

Worldwide, the prevalence of MetS among young female adults in the USA (18–21 years) was 4.7% [30], in Brazilian college students it was 1.7% [31], in Chinese female adolescents (14–16 years) it was 2.5% [32], in Spanish female adolescents (10–15 years) it was 3.85% [33], in Tunisian female adolescents (10–19 years) it was 2.4% [34], and 11.7% among Indian female adolescents (10–19 years) [35]. Clearly, the prevalence of MetS could differ between countries depending on the MetS defining criteria used, study method and target population. We have used the IDF and AHA/NHLBI joint statement, as it was an international attempt to harmonise the definition of MetS; central obesity is not an obligatory component of this definition and it is ethnic specific.

The most frequent component of MetS in this study was reduced HDL-C levels, which was also reported in female Emirati adolescents [4], and female Kuwaiti adolescents [22]. Reduced HDL-C accompanied by elevated triglyceride levels indicates dyslipidaemia, which is highly prevalent among the UAE population [36]. Insufficient physical activity and poor dietary habits are associated with low HDL-C levels [37–39]. Elmagd et al. [40] reported low physical activity (defined as less than 150 minutes/week) in 60% of Emirati college students. The consumption of high caloric diet was also reported in 33.5% of female Emirati adolescents [5].

These findings could explain the high prevalence of low-HDL-C levels observed in our study. Regular checks and screening in this age group could be helpful in identifying participants at an increased risk of developing MetS.

The current study found strong correlations between BMI, body fat, HbA1c and the prevalence of MetS in Emirati female students. The relationship between overweight and obesity and MetS has been supported by many other studies [41–43]. The association between HbA1c and the prevalence of MetS has not been previously reported; nevertheless, insulin resistance is a major underlying mechanism accountable for the prevalence of the MetS [44]. Interestingly, the prevalence of diabetes (8.5%) was also high in the study population. Future studies need to explore this finding more closely.

The strengths of this study include a trained researcher who obtained all measurements in the study and each measurement was repeated three times and the average used in the analyses. Anthropometric measures and blood withdrawal were conducted during one 50-minute morning session after assurance of a 12-hour overnight fast. Furthermore, to the best of our knowledge, no other studies exploring MetS prevalence in college students have been conducted in the UAE. UAEU is the main university in the UAE and it enrolls students from all seven emirates. However, restricting the study to college students makes it not representative of all the Emirati females in this age group. Moreover, the cross-sectional design is another limitation of this study, as causal inference cannot be drawn. Participants were voluntarily enrolled in the study, which could have caused selection bias (overweight and obese individuals might avoid anthropometric measurements). In addition, studying female students only does not allow for examination of gender differences or generalisability of results to all young adults. Therefore, future prospective studies are needed to confirm the prevalence of MetS and its relation to overweight and obesity in Emirati young adults. Additionally, it was challenging to clearly define the “young adult” age group. Some studies reported the MetS prevalence in adolescents and included ages 12–18 years [4] or 10–19 years old [22]. Other studies defined young adults as 18–24 years [30], 17–37 years [45] or college students aged 17–25 years [46]. Having one international definition for the “young adult” age group would be helpful for future data comparisons.

Conclusions

In summary, we have shown that the prevalence of MetS is high among UAEU female young adults aged 17–25 years (6.8%). Identification and possible intervention programmes may be useful for this age group in order to improve their future health. In addition, reduced HDL-C levels followed by central obesity were the most frequent components of MetS. BMI, body fat percentage and HbA1c were significantly associated with MetS.

Acknowledgments

This study was supported by a research start-up grant from the United Arab Emirates University (CFA-31F038). The authors wish to thank Ms. Rahla Daneshi, Mr. Noura Alneyadi, Ms. Sara Alyousefi, Ms. Abeer Alwahedi, Ms. Moza Alkaabi, Ms. Haneen Ateya and Mr. Asma Numan for their help in recruiting study participants. We thank all UAEU female students who participated in this study.

Author Contributions

Conceived and designed the experiments: ASA. Performed the experiments: ASA MNM AHJ FTA US. Analyzed the data: ASA MNM SMS EO FTA US. Contributed reagents/materials/analysis tools: ASA. Wrote the paper: ASA MNM. Contributed to drafting of the manuscript:

SMS EO LCI. Contributed to critical revision of the manuscript: EO LCI. Read and approved the final version of the manuscript: ASA MNM AHJ EO LCI FTA US SMS.

References

1. WHO. Global status report on noncommunicable diseases 2014. Geneva: World Health Organization; 2014. 298 p.
2. Alberti K, Zimmet P, Shaw J. Metabolic syndrome—a new world-wide definition. A consensus statement from the international diabetes federation. *Diabetic Medicine*. 2006; 23(5):469–80. PMID: [16681555](#)
3. Malik M, Razig SA. The prevalence of the metabolic syndrome among the multiethnic population of the United Arab Emirates: a report of a national survey. *Metabolic syndrome and related disorders*. 2008; 6(3):177–86. Epub 2008/08/14. doi: [10.1089/met.2008.0006](#) PMID: [18699721](#)
4. Mehairi AE, Khouri AA, Naqbi MM, Muhairi SJ, Maskari FA, Nagelkerke N, et al. Metabolic syndrome among Emirati adolescents: a school-based study. *PLoS One*. 2013; 8(2):e56159. doi: [10.1371/journal.pone.0056159](#) PMID: [23418529](#); PubMed Central PMCID: [PMC3572014](#).
5. Ng SW, Zaghoul S, Ali H, Harrison G, Yeatts K, El Sadig M, et al. Nutrition transition in the United Arab Emirates. *European journal of clinical nutrition*. 2011; 65(12):1328–37. doi: [10.1038/ejcn.2011.135](#) PMID: [21772317](#)
6. de Vries PG. Stratified random sampling. *Sampling Theory for Forest Inventory*: Springer; 1986. p. 31–55.
7. Gibson RS. *Principles of Nutritional Assessment*. Oxford University Press; 2005. p. 233–402.
8. Subhedar R, Subhedar V, Dave P, Mishra P, Kaur A. Bioelectrical impedance technology for evaluating human body composition parameters: “an advanced diagnostic technology for body composition and physical fitness analysis”. *Int J Physiother Res*. 2014; 2(6):824–30.
9. WHO. Obesity: preventing and managing the global epidemic. Geneva: World Health Organization, 2000.
10. Van der Kooy K, Seidell JC. Techniques for the measurement of visceral fat: a practical guide. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*. 1993; 17(4):187–96.
11. Lohman TG. Skinfolds and body density and their relation to body fatness: a review. *Human Biology*. 1981; 53(2):181–225. PMID: [7239496](#)
12. Durin J, Womersley J. Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition*. 1974; 32(01):77–97.
13. WHO. Physical Status: The Use and Interpretation of Anthropometry. Geneva: World Health Organization, 1995 Contract No.: 854.
14. WHO. Waist circumference and waist-hip ratio: Report of a WHO expert consultation, Geneva, 8–11 December 2008: World Health Organization; 2011.
15. WHO. Haemoglobin Concentrations for the Diagnosis of Anaemia and Assessment of Severity. Vitamin and Mineral Nutrition Information System (WHO/NMH/NHD/MNM/11.1). Geneva: World Health Organization; 2011.
16. El Assaad MA, Topouchian JA, Darme BM, Asmar RG. Validation of the Omron HEM-907 device for blood pressure measurement. *American Journal of Hypertension*. 2002; 15(S3):87A–A.
17. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, et al. Recommendations for blood pressure measurement in humans and experimental animals part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension*. 2005; 45(1):142–61. PMID: [15611362](#)
18. Dimagno, E. P., CORLE, D., O'BRIEN, J. F., MASNYK, I. J., GO, V. L., & AAMODT, R. (1989, October). Effect of long-term freezer storage, thawing, and refreezing on selected constituents of serum. In *Mayo Clinic Proceedings* (Vol. 64, No. 10, pp. 1226–1234). Elsevier.
19. Bowling JL, Katayev A. An Evaluation of the Roche Cobas c 111. *Lab Medicine*. 2010; 41(7):398–402.
20. Alberti K, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the Metabolic Syndrome: A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*. 2009; 120(16):1640–5. doi: [10.1161/CIRCULATIONAHA.109.192644](#) PMID: [19805654](#)

21. Abolfotouh MA, Al-Alwan IA, Al-Rowaily MA. Prevalence of metabolic abnormalities and association with obesity among Saudi college students. *International journal of hypertension*. 2012;2012.
22. Al-Isa A, Akanji AO, Thalib L. Prevalence of the metabolic syndrome among female Kuwaiti adolescents using two different criteria. *British journal of nutrition*. 2010; 103(01):77–81.
23. ALNohair S. Obesity in gulf countries. *International journal of health sciences*. 2014; 8(1):79. PMID: [24899882](#)
24. Alharbi NS, Almutari R, Jones S, Al-Daghri N, Khunti K, de Lusignan S. Trends in the prevalence of type 2 diabetes mellitus and obesity in the Arabian Gulf States: Systematic review and meta-analysis. *Diabetes research and clinical practice*. 2014; 106(2):e30–e3. doi: [10.1016/j.diabres.2014.08.019](#) PMID: [25241351](#)
25. Musaiger AO, Al-Roomi K, Bader Z. Social, dietary and lifestyle factors associated with obesity among Bahraini adolescents. *Appetite*. 2014; 73:197–204. doi: [10.1016/j.appet.2013.11.002](#) PMID: [24231426](#)
26. Selmi S, Daghash G, Mathis S, Nahas K, Wilbur K. A program for obese youth at-risk for diabetes in Qatar. *Avicenna*. 2015; 1:1–13.
27. Musaiger AO, Al-Kandari FI, Al-Mannai M, Al-Faraj AM, Bouriki FA, Shehab FS, et al. Perceived barriers to weight maintenance among university students in Kuwait: the role of gender and obesity. *Environmental health and preventive medicine*. 2014; 19(3):207–14. doi: [10.1007/s12199-013-0377-z](#) PMID: [24402439](#)
28. Al-Kaabi J, Al-Maskari F, Afandi B, Parkar H, Nagelkerke N. Physical activity and reported barriers to activity among type 2 diabetic patients in the United Arab Emirates. The review of diabetic studies: RDS. 2009; 6(4):271. doi: [10.1900/RDS.2009.6.271](#) PMID: [20043039](#)
29. Eapen V, Mabrouk A, Yousef S. Metabolic syndrome among the young obese in the United Arab Emirates. *Journal of tropical pediatrics*. 2010; 56(5):325–8. Epub 2009/12/25. doi: [10.1093/tropj/tpj128](#) PMID: [20032259](#)
30. Fernandes J, Lofgren IE. Prevalence of metabolic syndrome and individual criteria in college students. *Journal of American College Health*. 2011; 59(4):313–21. doi: [10.1080/07448481.2010.508084](#) PMID: [21308592](#)
31. F de Freitas RW Jr, M de Araújo MF, P Marinho NB, A de Vasconcelos HC, S Lima AC, R Pereira DC, et al. Prevalence of the metabolic syndrome and its individual components in Brazilian college students. *Journal of clinical nursing*. 2013; 22(9–10):1291–8. doi: [10.1111/jocn.12015](#) PMID: [23574292](#)
32. Yi-Qun X, Cheng-Ye J. Prevalence of the metabolic syndrome in secondary school adolescents in Beijing, China. *Acta Paediatrica*. 2008; 97(3):348–53. doi: [10.1111/j.1651-2227.2008.00665.x](#) PMID: [18298784](#)
33. González-Jiménez E, Montero-Alonso MA, Schmidt-RíoValle J, García-García CJ, Padez C. Metabolic syndrome in Spanish adolescents and its association with birth weight, breastfeeding duration, maternal smoking, and maternal obesity: a cross-sectional study. *European journal of nutrition*. 2014; 54(4):589–97. doi: [10.1007/s00394-014-0740-x](#) PMID: [25052543](#)
34. Aounallah-Skhiri H, El Ati J, Traissac P, Gartner A, Ben Gharbia H, Hsairi M, et al. Prevalence of metabolic syndrome and associated behavioural factors in Tunisian adolescents. *Obesity Facts*. 2015; 8 (Suppl. 1).
35. Bhalavi V, Deshmukh PR, Goswami K, Garg N. Prevalence and correlates of metabolic syndrome in the adolescents of Rural Wardha. *Indian journal of community medicine: official publication of Indian Association of Preventive & Social Medicine*. 2015; 40(1):43.
36. Smitha F, Meenakshi J, Padma R, Multani S. Survey and evaluation of various epidemiological factors in a multiethnic Diabetic Population in Ras Al-Khaimah, UAE. *Indian Journal of Pharmacy Practice*. 2011; 4(1).
37. Hamer M, Stamatakis E, Steptoe A. Effects of substituting sedentary time with physical activity on metabolic risk. 2014; 46(10): 1946–1950. doi: [10.1249/MSS.00000000000000317](#) PMID: [24674977](#)
38. Ahmed HM, Blaha MJ, Nasir K, Rivera JJ, Blumenthal RS. Effects of physical activity on cardiovascular disease. *The American journal of cardiology*. 2012; 109(2):288–95. doi: [10.1016/j.amjcard.2011.08.042](#) PMID: [22011559](#)
39. Kant A, Whitley M, Graubard B. Away from home meals: associations with biomarkers of chronic disease and dietary intake in American adults, NHANES 2005–2010. *International journal of obesity*. 2015; 39(5):820–7. doi: [10.1038/ijo.2014.183](#) PMID: [25319744](#)
40. Elmagd MA, Mossa AH, Sami MM, El-Marsafawy TS, Al Jadaan O, Eldin MS. The Impact of Physical Activity on the Academic Performance among Medical and Health Sciences Students: A Cross Sectional Study from RAKMHSU-Ras Alkhaimah-UAE. *International Journal of Physical Education, Sports and Health* 2015; 2(1): 92–95.

41. Al-Daghri NM. Extremely high prevalence of metabolic syndrome manifestations among Arab youth: a call for early intervention. *European journal of clinical investigation*. 2010; 40(12):1063–6. Epub 2010/07/14. doi: [10.1111/j.1365-2362.2010.02341.x](https://doi.org/10.1111/j.1365-2362.2010.02341.x) PMID: [20624169](https://pubmed.ncbi.nlm.nih.gov/20624169/).
42. Al-Daghri NM, Al-Attas OS, Alokail MS, Alkharfy KM, Sabico SL, Chrousos GP. Decreasing prevalence of the full metabolic syndrome but a persistently high prevalence of dyslipidemia among adult Arabs. *PLoS One*. 2010; 5(8):e12159. Epub 2010/08/24. doi: [10.1371/journal.pone.0012159](https://doi.org/10.1371/journal.pone.0012159) PMID: [20730053](https://pubmed.ncbi.nlm.nih.gov/20730053/); PubMed Central PMCID: [PMCPmc2921394](https://pubmed.ncbi.nlm.nih.gov/PMC/PMC2921394/).
43. Mabry RM, Reeves MM, Eakin EG, Owen N. Gender differences in prevalence of the metabolic syndrome in Gulf Cooperation Council Countries: a systematic review. *Diabetic medicine: a journal of the British Diabetic Association*. 2010; 27(5):593–7. Epub 2010/06/12. doi: [10.1111/j.1464-5491.2010.02998.x](https://doi.org/10.1111/j.1464-5491.2010.02998.x) PMID: [20536958](https://pubmed.ncbi.nlm.nih.gov/20536958/).
44. Guo S. Insulin signaling, resistance, and metabolic syndrome: insights from mouse models into disease mechanisms. *Journal of Endocrinology*. 2014; 220(2):T1–T23. doi: [10.1530/JOE-13-0327](https://doi.org/10.1530/JOE-13-0327) PMID: [24281010](https://pubmed.ncbi.nlm.nih.gov/24281010/)
45. Jain J, Rao T, Desmukh P, Banait S. Prevalence and Correlates of Metabolic Syndrome in Young Population: A Cross Sectional Study. *J Diabetes Metab*. 2015; 6(503):2.
46. Kim D-i, Kim JY, Lee MK, Lee H-D, Lee J-W, Jeon JY. The relationship between fitness, BMI and risk factors of metabolic syndrome among university students in Korea. *The Korean Journal of Obesity*. 2012; 21(2):99–107.