AMERICAN UNIVERSITY OF BEIRUT

OBESITY AND ITS ASSOCIATION WITH SOCIOECONOMIC, DIETARY, LIFESTYLE, AND SLEEP CHARACTERISTICS IN LEBANESE URBAN ADULTS

by
LARA MOHAMMED ITANI

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LARA MOHAMMED ITANI

Approved by:

Dr. Lara Nasreddine, Associate Professor — Advisor
Nutrition and Food Sciences

Dr. Farah Naja, Associate Professor — Member of Committee
Nutrition and Food Sciences

Dr. Hani Tamim, Associate Professor — Member of Committee
Internal Medicine

Date of thesis defense: April 25th, 2016.

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AN ABSTRACT OF THE THESIS OF

Lara Mohammed Itani for Master of Science
Major: Nutrition

Title: Obesity and its Association with Socioeconomic, Dietary, Lifestyle, and Sleep Characteristics in Lebanese Urban Adults.

Background and objectives: The worldwide prevalence of obesity is increasing in developed and developing countries, reaching alarming levels in several parts of the world. Eastern Mediterranean is no exception to this trend, with the prevalence of obesity in its countries reaching high levels, exceeding at times those reported from developed countries. This study aims at assessing the prevalence of obesity amongst Lebanese adults residing in the Greater Beirut Area, and investigating the association of obesity with socioeconomic, lifestyle, dietary, and sleep characteristics.

Methods: A representative cross sectional survey was conducted, in 2015, on Lebanese adults aged 18 years and older (n=501). A multi0component questionnaire inquiring about: socio-economic, lifestyle, dietary, and sleep characteristics was administered to study participants in a face – to – face interview. Dietary intake was assessed using an eighty - six -item FFQ, and sleep patterns were evaluated using the Berlin Questionnaire. Anthropometric measurements (weight, height, and waist circumference) were obtained using standardized techniques. Statistical analysis was done using SPSS 20 for Windows (SPSS Inc, Chicago, IL). Obesity was defined as BMI $\geq$ 30 kg/$m^{2.}$ The association of obesity with socioeconomic, lifestyle, dietary, and sleep correlates was examined using univariate and multivariate regression analyses.

Results: Findings of the current study estimated the prevalence of obesity at 41.5% in Lebanese urban adults, with significantly higher estimate of obesity among females (70.7% for females vs. 29.3% for males; P<0.05). Obesity also showed significant associations with higher age, and lower SES (assessed by lower income and education status), lower physical activity levels, lower sleep duration and the presence of obstructive sleep apnea (OSA). When looking at dietary intake data, there was no association of obesity with energy and macronutrient intakes. Findings of the multivariate logistic regression showed a significantly higher risk of obesity for those: aged $\geq$ 40 years old (OR=2.24; CI=1.05 – 4.80), those who slept for short duration (< 7 hours) (OR=1.8; CI=1.099 – 3.30), and those who had OSA (OR =4.8; CI=3.40 – 6.80). Significantly lower odds of obesity were reported for those who smoke (OR=0.56; CI=0.37 – 0.85) and those with a university degree (OR=0.33; CI=0.13 – 0.82).

Conclusion: This study documented a high prevalence of obesity among Lebanese urban adults living in the Greater Beirut Area (41.5%). Findings of this study

showed a high prevalence of obesity among the study population (41.5%). This prevalence rate was higher than those reported in some MENA regions (Morocco, Tunisia, Algeria, Iran, Palestine, and Oman) and European countries (France, Italy, and England). This study has also identified specific factors associated with obesity in this population group, including higher age, lower SES, and low physical activity levels. In addition, this study is the firstto explore and demonstrate an association of sleep duration and sleep patterns with body weight and the risk of obesity amongst adults in Lebanon and the Eastern Mediterranean Region. Taken together, this study's findings call for culture-specific intervention strategies for the promotion of physical activity, healthy lifestyle and sleep patterns amongst Lebanese adults, underline the importance of building culturally – appropriate, community-based interventions to help increase health awareness in Lebanon, especially among low SES individuals

CONTENTS

ILLUSTRATIONS

TABLES

ABBREVIATIONS

%	Per Cent
/	Per
&	And
±	Plus or Minus
=	Equal
<	Less than
>	greater than
≤	Less than or equal
≥	Greater or equal
AUB	American University of Beirut
% BF	Percent Body Fat
BIA	Bioelectrical Impedance Analysis
BMI	Body Mass Index
CDC	Center for Disease and Control Prevention
CI	Confidence Interval
Ci	Crowding Index
CRF	Corticotropin-releasing Factor
CVD	Cardiovascular Diseases
d	Day
DEXA	Dual Energy X-Ray
DL	Dislipidemia
DM	Diabetes Mellitus
et al.	And Others
EE	Energy Expenditure

FFQ	Food Frequency Questionnaire
g	Gram
HTN	Hypertension
Ht	Height
IDF	International Diabetes Foundation
IL-6	Interleukin 6
IR	Insulin Resistance
IRB	Institutional Review Board
Kcal	Kilocalorie
Kg	Kilogram
m^2	Square meter
MENA	Middle East and North Africa
MetS	Metabolic Syndrome
METS	Metabolic Equivalents
n	Number
NCD	Non-Communicable Diseases
PA	Physical Activity
REM	Rapid Eye Movement
UAE	United Arab Emirates
UK	United Kingdom
USA	United State of America
USD	US Dollar
vs.	Versus
SES	Socio-economic Status
SPSS	Statistical Package for Social Science
TG	Triglyceride
WC	Waist Circumference

WHR	Waist- to - Hip Ratio
WHO	World Health Organization
Wt	Weight

To My

Beloved Parents

CHAPTER I

INTRODUCTION

The worldwide prevalence of obesity has nearly doubled between 1980 and 2008 and has been increasing ever since (WHO, 2014). According to the World Health Organization (WHO), more than 1.9 billion adults, 18 years and older, were overweight in 2014, of whom 600 million were obese. Accordingly, the WHO estimated that 39% of the adult world population are overweight and 13% are obese (WHO, 2014).

Obesity is a public health problem that causes economic, physcosocial and critical medical obstacles for both developed and developing countries (Magee, 2008). Obesity is an important risk factor for non-communicable diseases (NCD) such as: coronary heart disease, diabetes, osteoarthritis, and metabolic related disorders (Kopelman, 2000). It is typically caused by an energy imbalance where energy input exceeds energy expenditure (EE). This causes excess body fat to accumulate to the extent that it may exert negative effects on health (WHO, 2014).Several factors can lead to positive energy balance and obesity, including: environment, genetics, and behavior (CDC, 2009). Dietary intakes and lifestyle-related factors including physical activity are recognized as important modulators of the energy balance equation and the risk of obesity. In addition, over the past decade, the rise in obesity has been increasingly linked to a reduction in sleep duration (Beccuti & Pannain, 2011). Sleep is an important part of the human biology where it plays an important role in determining human behavior. Sleep is important for three major body functions: 1). It protects the body at night when sensory capacity are down-regulated, 2). It gives the brain the time needed to gather important memories and experiences for learning, 3). It serves as energy

restoration period from daytime activities (Gerber, Brand, Holsboer-Trachsler & Pühse, 2010). The adequacy of sleep duration has been reported to exert an effect on weight regulation by possibly influencing eating patterns and appetite regulation (Kim, DeRoo & Sandler, 2011; Knutson, 2012). Growing evidence also show that sleep is an important modulator of glucose metabolism and neuroendocrine function. Sleep deprivationmay, therefore, alter metabolic and endocrine functions, including changes in appetite regulating hormones and a decrease in glucose tolerance(Beccuti & Pannain, 2011). In addition, obstructive sleep apnea (OSA), which causes "recurrent episodes of obstruction of the upper airway leading to sleep fragmentation and intermittent hypoxia during sleep" (Drager, Togeiro, Polotsky & Lorenzi-Filho, 2013), has been associated with increased risk of obesity.

Evidence on the link between obesity and sleep has suggested from studies conducted in adults in the USA and European countries (Cappuccio, Taggart, Kandala& Currie, 2008). However, this association has not been identified in the Middle- East, a region that is characterized by one of the highest rates of obesity worldwide. In Lebanon, a study done by Nasreddine *et al*. documented a significant escalating trend in obesity over the past decade, whereby the prevalence of adult obesity was found to increase from 17.4% to 28.2%with the odds of obesity being two times higher in 2009 compared to 1997 (Nasreddine, Naja, Chamieh, Adra, Sibai & Hwalla. 2012).

Studies investigating factors associated with obesity in Lebanon are scarce, but available evidence suggests a nutrition transition in developing countries due to a higher pace of urbanization, modernization, and affluence as well as a dramatic shift in dietary habits and lifestyle (Chamieh, Moore, Summerbell, Tamim, Sibai & Hwalla, 2015; Sibai, Hwalla, Adra & Rahal, 2003). Gaining greater insight into factors that are associated with obesity within the country's context is crucial for the development of

effective public health programs which aim to prevent and manage obesity *(Chamieh et al*., 2015; Nasreddine *et al*., 2014).The high disease burden of obesity in Lebanon highlights the need for rigorous investigations of its determinants, context-specific patterns and associated factors. The present study aims at examining the prevalence and correlates of obesity in a representative sample of adults living in Beirut, the capital of Lebanon.

The specific objectives of the study are to:

- Determine the prevalence of obesity in Lebanese adults living in Beirut.
- Examine the association of obesity with socio-demographics, lifestyle, and dietary factors.
- Investigate the association of obesity with sleep duration and sleep patterns.

CHAPTER II

LITERATURE REVIEW

A. Overview of Obesity

The reasons behind the growing epidemic of obesity are complex and multifactorial. However, the main cause is the growing imbalance of energy input and EE that leads to the hyperplasia and hypertrophy of adipocytes (Otto & Lane, 2005). It is a major health burden which can contribute to several non-communicable diseases (NCD) such as cardiovascular disease (CVD), diabetes, hypertension (HTN), and other metabolic – related disorders (Table 1). Major causes of morbidity and mortality have been found to be liked to NCDs. In the Middle East, NCD represents more than 60% of the total annual death (Musaiger, 2011).

Many factors such as genetics, environment, and behavior can lead to obesity (CDC, 2009). Although the contribution of genetics and environment to the etiology of obesity varies from one study to the other, it has been shown that 30 – 40 % can be attributed to genetics and approximately 70% to environment (Pi-Sunyer, 2002). Increased consumption of energy dense foods high in sugar and saturated fat, and reduced physical activity (PA) may lead to obesity (WHO, 2011a). In a given population, it has also been suggested that some people are genetically predisposed to develop obesity. However, the expression of the genotype may occur under certain environmental conditions, such as sedentary lifestyles and high fat diets (Stunkard, 1988).

Table 1. Metabolic abnormalities and disorders associated with obesity

• Insulin resistance/ hyperinsulinemia	• Osteoarthritis
• Type 2 diabetes	• Stroke
• Hypertension	• Asthma
• Dyslipidemia	• Sleep apnea
• Coronary heart disease	• Breathing difficulties
• Gallbladder disease	• Complications of pregnancy
• Cancer (prostate, endometrial, uterine, cervical, ovarian, colon, kidney, gallbladder, and postmenopausal breast)	• Menstrual irregularities
• Premature death	• Hirsutism
	• Increases surgical risk
	• Psychological distress

Source: Pi-Sunyer, F.X. (2002). "The obesity epidemic: pathophysiology and consequences of obesity". *Obesity Research* 10(S12), 97S-104S.

In addition to excess body weight, the pattern of fat distribution may also increase the risk of these disorders.

B. Assessment of Obesity

Both direct and indirect methods have been used to assess overweight and obesity. Direct methods are used extensively in research to assess body fat *(Barreira et al*., 2012). Such methods include: multi-frequency bioelectrical impedance analysis (BIA), dual energy X-ray (DEXA), the magnetic resonance imaging, and underwater weighing (densitometry). For practical reasons, indirect methods have been suggested for clinical and epidemiological settings. Such methods include anthropometric measurements such as body mass index (BMI), waist circumference (WC) and skin fold thickness (Dehghan, Akhtar-Danesh& Merchant, 2005; Lobstein, Baur, & Uauy, 2004).

1. Body Mass Index

According to the World Health Organization (WHO), the most commonly used method to classify overweight and obesity is the BMI. BMI is calculated using the following equation: $\frac{Weight\ (kg)}{Height\ (m2)}$ (WHO, 1995). Body fat is suggested to increase as the BMI value increases (Han, Sattar & Lean, 2006).

Studies have shown how BMI can be positively associated with adiposity, mortality, and cardiovascular risk factors (Barreira *et al*., 2012; Taylor *et al*., 2010). Most relevant literature has defined overweight as a BMI between 25.0 and 29.9 kg/m^2 and obesity as a BMI of 30.0 kg/m^2 or above (Table 2). At an individual level, BMI has not been shown to accurately measure body fat in muscular, short, or tall people (Lobstein *et al.*, 2004). However, at a population level, BMI has been recognized as a useful method by the WHO to measure the obesity prevalence and its associated health risks (WHO, 2000).

Table 2. Health risk classification according to BMI

Classification	**Body Mass Index Category[a] (kg/m^2)**	**Risk of Developing Health Problems[b]**
Underweight	< 18.5	Increased
Normal	18.5-24.99	Least
Overweight	25.0 – 29.99	Increased
Obese	≥ 30	≥ 30
Class I	30.0 – 34.9	High
Class II	35 – 39.9	Very high
Class III	≥ 40	Extremely high

Source: [a] *(WHO, 1995; WHO, 2000; WHO, 2004)*
[b] *(Tchernof & Després, 2013)*

2. Waist Circumference

Since BMI is not entirely reflective of body fat, other studies have proposed WC as a simple and practical measurement. WC has been found to correlate better with visceral fat deposits than with BMI as it measures the absolute amount of abdominal fat. The main limitation in WC is that there is no universal cutoff point for gender and various ethnic-groups. As per the International Diabetes Foundation (IDF), abdominal obesity is defined as a WC ≥90 cm in men and ≥ 80 cm in women for Asia- Pacific countries (IDF, 2005). Table 3 shows the stratified WC according to gender and ethnic groups. People with a large WC are more likely to suffer from health risks, including shortness of breath, features of metabolic syndrome (such as hypertension, dyslipidemia, and diabetes), as well as poor quality of life (Han *et al*., 2006).

Table 3. Ethnic-specific values for central obesity according to waist circumference by organization

Country/ethnic group	**Waist Circumference[a] (in cm)**		**Organization[b] (Reference)**
	Men	**Women**	
Europids	≥ 94	≥ 80	IDF
Asian	≥ 90	≥ 80	IDF & WHO
Chinese	≥ 90	≥ 80	Cooperative Task Force
Japanese	≥ 85	≥ 90	Japanese Obesity Society
Eastern Mediterranean and Middle East (Arab) populations	≥ 94	≥ 80	IDF
Sub – Saharan African	≥ 94	≥ 80	IDF

Source: [a] *(IDF, 2005)*
[b] *(Alberti et al., 2009)*

Based on the IDF, The European data for WC cutoff points are assigned for the Mediterranean and Middle Eastern population as no specific data is available for this ethnic group.

3. Percent Body Fat

Percent body fat (% BF) is defined "as the proportion of individual fat mass over body weight" (Zeng, Dong, Sun, Xie& Cui, 2012). Some studies show that % BF is more accurate when it comes to assessing body composition as compared to BMI (Houtkooper, Lohman, Going & Howell, 1996; Ling *et al*., 2011). According to the Asian BMI criteria and U.S. National Institutes of Health criteria, it has been stated that obesity corresponds to % BF >25 in males and >35 in females (Wen, David Cheng, Tsai, Chan, Hsu, Hsu& Eriksen, 2009). However, it has also been suggested that no linear relationship exists between BMI and % BF, where a high % BF does not always mean a high BMI, and vice versa (Adams *et al*., 2007; Meeuwsen, Horgan& Elia, 2009).

% BF is usually assessed using indirect methods like BMI and skinfold thickness measurements or direct methods like: DEXA and BIA.Several regression equations are suggested in the literature for the purpose of calculating the percent body fat from skinfold measurement. In epidemiological studies, skinfolds are often used the most to assess total body fat. The most common skinfold measurements include: the upper arm (biceps and triceps), above the iliac crest (suprailiac), and under the scapula (subscapular). The main disadvantage of this method is that subjects need to be partially undressed. In obese subjects, the measurement errors are larger since they are rather difficult to measure (Deurenberg & Yap, 1999).

DEXA and BIA are two frequently used methods for the quantification of body composition. The DEXA technique is widely applicable clinically as it allows concurrent quantifications of soft tissue body composition and bone mineral content. It has been validated against several reference standards and has been accepted as being a precise and accurate technique in assessing body composition (Prior *et al*., 1997).

However, one of its main limitations is its high cost. Furthermore, the DEXA procedure also requires subjects to remain motionless which may cause a burden to some patients (Ling *et al*., 2011).

Another technique used to assess body fat is the BIA. It has been shown to be non-invasive, simple, easily accessible, and requires minimal intra and inter-observer variability. It gives reliable and reproducible results with less than 1 % error on repeated measurements (Diaz, Villar, Immink & Gonzales, 1989; Segal *et al*., 1991). A study done by Heitmann on adults (n = 139) compared the difference between some % BF measurements such as: skinfold thickness measurements, anthropometrics like BMI and WC, and BIA. All measurements predicted body fat equally as well; however the BIA was shown to have significantly lower variability of estimates, making it more accurate than other methods (Heitmann, 1990).

C. Prevalence of Obesity

The prevalence of obesity varies from one country to the other due to different factors such as lifestyle and socioeconomic status (SES) (Chamieh *et al*., 2015). A global study done in 2013 showed that worldwide obesity prevalence rose by 27.5% for adults and 47.1% for children between the years 2008 to 2013. Overall BMI of 25 kg/m^2 or greater between 1980 and 2013 has increased from 28.8% to 36.9% in men and from 29.8% to 38% in women (Ng *et al*., 2014). The prevalence of obesity in many developed countries is high estimated at 33% in the U.S., 26.9% the United Kingdom, 26.6% in Spain, 19.6% in Italy, 18.6% in Sweden, and 18.2% in France (Ng *et al*., 2014).

High obesity prevalence among adults in the Arab countries has also been documented. It has been estimated, according to the WHO NCD Arab country profile,

that 31.4% of men and 54.2% of women are either overweight or obese. The lowest prevalence was found in Oman (20.9%), while increasing in the following countries: United Arab Emirates (32.7%), Bahrain (32.9%), Saudi Arabia (33%), Qatar (33.2%), and Kuwait (42%) having the highest obesity prevalence of them all (WHO, 2011b).

In Lebanon, a trend analysis done by Nasreddine *et al*. (2012) highlighted an alarming increase in obesity prevalence among Lebanese adults aged ≥ 20 years. The study showed how obesity rates among adults have almost doubled from the year 1997 to 2009 from 17.4% to 28.2% (Nasreddine *et al*., 2012). Gender differences in obesity have also been documented in Lebanon whereby women aged more than 50 years showed higher prevalence rates than men (Chamieh *et al*., 2015).

D. Pathophysiology of Obesity

A curvilinear relationship between BMI and medical difficulties has been observed. Such complications occur when BMI ranges between 30 – 40 kg/m^2 (Bray, 1992). It has been currently accepted that obesity promotes a state of low – grade inflammation caused by adipocyte hypertrophy, hypoxia, and oxidative stress via the production of excessive pro-inflammatory cytokines such as: TNFα, IL-6, plasminogen activator inhibitor-1 (PAI-1) (Van Greevenbroek, Schalkwijk& Stehouwer, 2013). Insulin- sensitizing anti- inflammatory hormones such as adiponectin may also decrease due to excess visceral fat deposits. Several studies highlighted the association of obesity with IR, hyperglycemia, hypertension (HTN), dyslipidemia (DL), and diabetes mellitus DM, thus all leading to cardiovascular and metabolic diseases (Figure 1). Some studies suggested excess visceral fat to be associated with dysregulation of adipokines (such as leptin, ghrelin, and adiponectin), which may have an important role in the pathophysiology of obesity and CVD (Diaz-Melean *et al*., 2013).

It has been shown that a high energy intake may lead to an increase in triglyceride (TG) storage in the adipose tissue. Findings suggest that the size of the body fat cell increases as body weight increases. When the maximum size has been reached, more adipocyte cells are required to accommodate for the increase in TG. This increase in TG leads to an increase in cholesterol production. High cholesterol production may increase cholesterol secretion in the bile which can increase gallstone formation, causing the development of gallbladder disease. High TG levels may also reduce high density lipoprotein (HDL) cholesterol which may account for an elevated risk for CVD in obese patients (Bray, 1992).

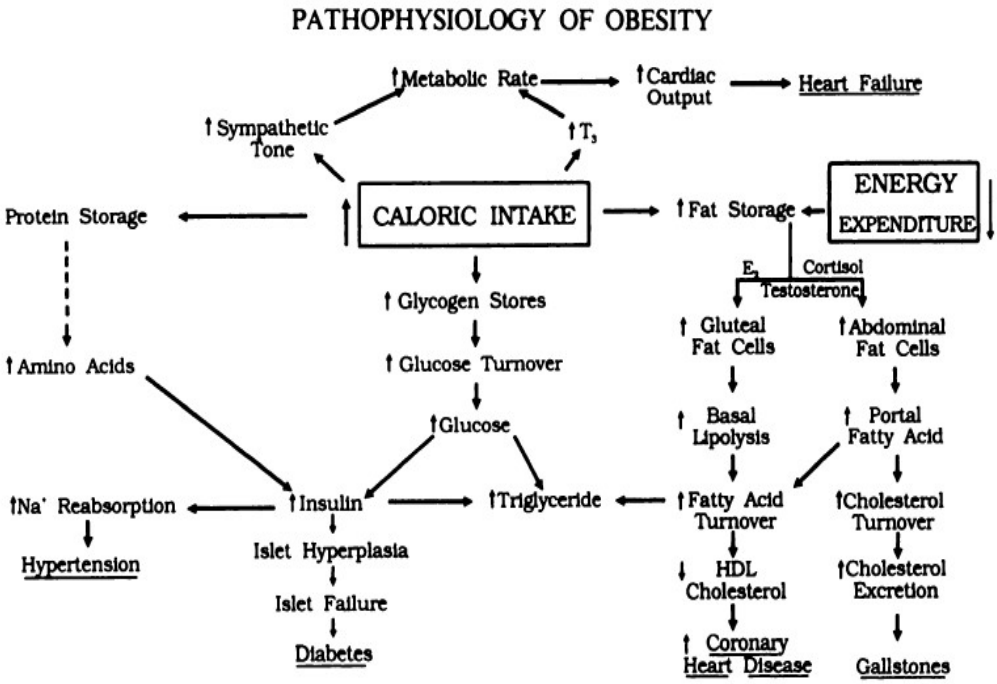

Fig. 1. Pathophysiology of obesity - consequences of increased energy intake and reduced PA
Source: G.A. Bray, (1992). "Pathophysiology of obesity". *The American Journal of Clinical Nutrition* 55(2), 488S-494S.

1. Obesity and Insulin Resistance

Several studies have suggested how a reduction in insulin sensitivity may be associated with obesity through increased secretion of non- esterified fatty acids (NEFA), glucose in circulation, specific hormones, pro-inflammatory cytokines, glycerol, and other factors linked to the development of IR. Intra- abdominal or visceral fat has been shown to have the closest link to IR. Enhanced IR and hyperinsulinemia may lead to pancreatic failure, causing DM (Pi-Sunyer, 2002).

An increase in inflammatory cytokines (TNF, IL-6, CRP) may decrease insulin signaling causing an increase in glucose in circulation. An increase in food intake occurs due to a decrease in glucose uptake; thus leading to: weight gain, a decrease in the inhibition of hepatic glucose production and efficiency of glucose uptake in the muscles causing hyperglycemia, and an increase in lipolysis in the adipose tissue causing an increase in NEFA. All those mechanisms may result in the development of IR (Figure 2).

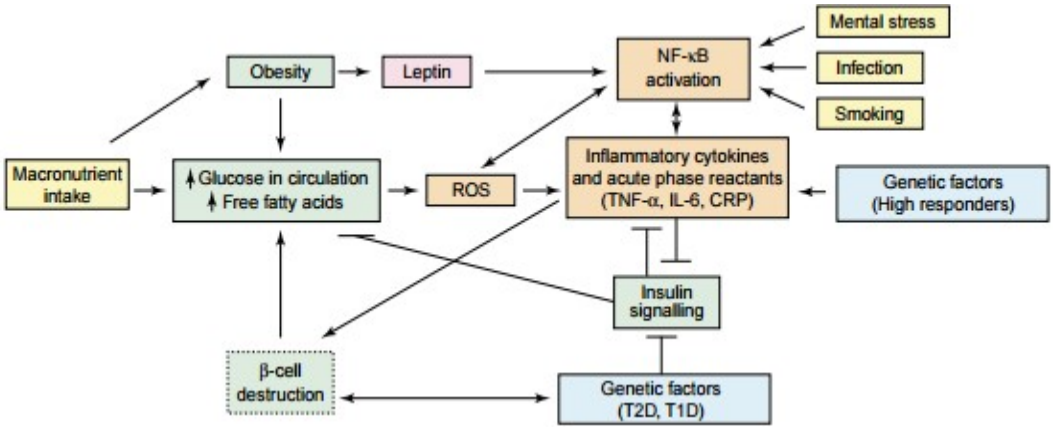

Fig. 2. The induction of reactive oxygen species (ROS) generation and inflammation (NF-kB activation) by macronutrient intake, obesity, free fatty acids, leptin, infection, smoking, mental stress and genetic factors. Interference with insulin signaling (insulin resistance) leads to hyperglycemia and pro-inflammatory changes.
*Source:*Dandona, P., Aljada, A. & Bandyopadhyay, A. (2004). "Inflammation: the link between insulin resistance, obesity and diabetes". *Trends in Immunology* 25(1), 4-7.

A theory, known as the Portal Theory, suggests how excess visceral fat may stimulate the release of NEFA which may lead to IR via the liver. High NEFA levels may increase hepatic glucose production, eventually causing glucose intolerance. It may also lead to an increase in hepatic very low density protein (VLDL) triglyceride secretion which potentially impairs postprandial lipid metabolism. Excess fatty acids may compete with glucose for substrate oxidation. This competition results in the accumulation of glucose-6-phosphate, thus causing a decrease in glucose uptake by the muscle and the adipose tissue, further contributing to IR.

2. Obesity and Displipidemia

It has well been stated that IR and hyperinsulinemia may cause several alterations in the lipid profile (Marinou, Tousoulis, Antonopoulos, Stefanadi, & Stefanadis, 2010). Excessive NEFA levels, as previously discussed, may result in increased hepatic lipogenesis which may cause high TG, VLDL, and ApoB production (Bamba & Rader, 2007). IR and hyperinsulinemia in obese individuals may impair lipoprotein lipase activity and enhance cholesteryl ester transfer protein (CETP) causing a reduction in HDL levels.

The figure below shows the schematic description of proposed relationships between IR to insulin-mediated glucose disposal, compensatory hyperinsulinemia, and multiple consequences causing increased TG, decreased HDL, and increased blood pressure; leading to coronary heart disease(Reaven, 1995).

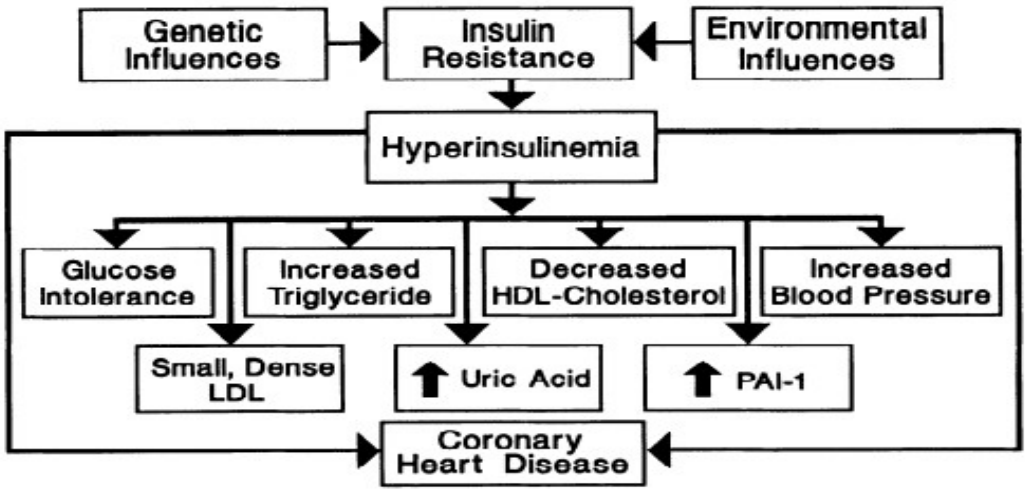

Fig. 3. The suggested pathophysiology of hyperinsulinemia leading to dyslipidemia
*Source:*Reaven, G.M. (1995). "Pathophysiology of insulin resistance in human disease". *Physiological Reviews* 75(3), 473-486.

3. Obesity and Hypertension

A linear relationship has also been seen between hypertension and BMI. In the Nurses' Health Study, 41,541 subjects were studied to determine the risk of developing hypertension with increasing BMI (Ascherio *et al*., 1996). The relative risk was 4.8 for those with a BMI higher than 32 kg/m^2. Similarly, subjects with a large WC were twice as likely to develop hypertension (Okosun, Chandra, Choi, Christman, Dever & Prewitt, 2001). Development of hypertension in obese subjects is caused by several mechanisms but it is mainly characterized by an increase in vascular volume. Those mechanisms include: changes in Na+/ H+ - ATPase activity, increased renal sodium and water absorption, changes in growth factor – mediated structural changes, and activation of the sympathetic nervous system (Pi-Sunyer, 2002).

4. Obesity and Neuroendocrine Factors

The adipose tissue is an endocrine and paracrine organ responsible for the

production of bioactive molecules (IL-6 and TNF- α) and immunomodulators (leptin and adiponectin). Studies have suggested excess visceral fat to be associated with the dysregulation of certain adipokines such as leptin, adiponectin, and ghrelin. Adipokines are important for energy homeostasis and may contribute to IR, HTN, DM, thrombosis, and inflammation (Diaz-Melean *et al*., 2013).

- ***Leptin***

Leptin is produced in proportion to fat stores and is an important appetite regulator where it suppresses food intake and increases EE. Leptin has been found to increase in obese individuals causing leptin resistance, which is "the failure of high levels of leptin in obese individuals to suppress feeding and prevent or mitigate obesity" (Myers, Cowley, & Münzberg, 2008). Leptin resistance has been linked to the disruption of several mechanisms in obese individuals. It induces CRP expression, which has been found to be an independent risk factor for metabolic and cardiovascular disease, and increases oxidative stress which contributes to the development of atherosclerosis (Diaz-Melean *et al*., 2013). It has also been shown to cause HTN by activating the sympathetic nervous system and reducing nitric oxide levels responsible for vasodilation. This causes an increase in blood pressure eventually leading to HTN.

- ***Adiponectin***

Adiponectin, also synthesized by the adipose tissue, is inversely associated with CVD. High levels of adiponectin play an important cardioprotective role which is exerted through multiple pathways. Some of these pathways include: reducing the expression of pro-inflammatory cytokines, increasing cholesterol efflux from macrophages, and enhancing insulin sensitivity. Adiponectin levels have been found to be low in individuals with excess visceral fat, thus increasing the risk of CVD (Diaz-Melean *et al*., 2013).

- ***Ghrelin***

Ghrelin, an appetite stimulating hormone, is produced by the stomach and is important for energy balance. Its secretion depends largely on the nutritional status. Studies have shown plasma ghrelin levels to be inversely correlated with BMI. Data revealed ghrelin levels to be significantly decreased in obese and overweight subjects but remain low after food intake compared to lean subjects (Tschöp, Weyer, Tataranni, Devanarayan, Ravussin & Heiman,2001; Williams, Grill, Cummings& Kaplan, 2006). This demonstrates a smaller post – prandial reductions in ghrelin levels following a meal in obese subjects, leading to a reduced feeling of satiety and increased food intake, thus causing weight gain (Daghestani, 2009; Le Roux, Patterson, Vincent, Hunt, Ghatei & Bloom 2005).

Orexigenic hormones including neuropeptide Y, melanin-concentrating hormone, agouti-related protein, and orexin are all stimulated upon ghrelin release. It has been shown to have an inhibitory effect on anorexigenic hormones such as pro-opiomelanocortin, corticotropin-releasing factor (CRF).

5. Obesity and Metabolic Syndrome

The metabolic syndrome (MetS) is a complex cluster of inter-related risk factors for CVD and DM. The MetS is closely linked with excessive adiposity. The pathophysiology of MetS seems to be attributable to IR and excess NEFA flux which may increase the inflammatory response in the body (Eckel, Grundy, & Zimmet, 2005). Risk factors leading to the metabolic syndrome include: HTN, DL (high TG and low HDL), increased fasting glucose and central obesity (Alberti *et al*., 2009). Individuals with metabolic syndrome are five times more likely to develop DM. It is important to note that the pathways related to the metabolic syndrome have not yet been fully

identified.

E. Factors Associated with Obesity

Obesity and weight gain result from the cumulative effect of a positive energy balance. Determinants known to cause weight gain include but are not limited to: diet and physical inactivity, family history of obesity, genetics, education, SES, etc. (Zhang & Hu, 2012). More recently, a role for sleeping patterns and duration has also been suggested in the etiology of obesity.

1. Diet

It has been shown that the strongest evidence for an increased risk of obesity is a diet high in fat or low in fiber(Lindström, Peltonen, Eriksson, Louheranta, Fogelholm, Uusitupa & Tuomilehto, 2006). When compared to protein or carbohydrate, dietary fat is more readily stored in the body as fat with minimal energy cost required to convert it. Moreover, the proportion of carbohydrate in the diet varies reciprocally with fat. Evidence relating the intake of sugar to weight change is inconsistent and may be partly due to the various sources of sugar found in the diet including milk, fruit, as well as "added" sugar (Hill & Prentice, 1995). Moreover, a smaller contribution to total energy is found in protein when compared to fat and carbohydrates. In some observational studies, a high protein intake has been correlated with a decrease in weight gain due to an increase in satiety cues (Westerterp-Plantenga, Lejeune, Nijs, Van Ooijen & Kovacs, 2004).

High fiber intake has been shown to assist in weight loss and is negatively correlated with BMI and weight gain when compared to a low fiber intake (Howarth, Saltzman& Roberts, 2001; Koh-Banerjee *et al.*, 2004; Liu, Willett, Manson, Hu, Rosner

& Colditz, 2003). Fiber has been shown to increase satiety and decrease total energy intake (Pereira & Ludwig, 2001). It delays gastric emptying and the rise in postprandial glucose and insulin responses. It has also been found to impact gut hormones involved in appetite regulation, such as cholecystokinin.

Some evidence shows the importance of other specific foods like fruits and vegetables which have a modest protective effect on obesity (He, Hu, Colditz, Manson, Willett & Liu, *2004;* Kahn, Tatham, Rodriguez, Calle, Thun & Heath , 1997). Consuming moderate amounts of nuts with the diet was also shown to cause lower body weight (Sabaté, 2003). In addition, a positive correlation between sugar – rich beverages and poor satiety and weight gain has also been found (Malik, Schulze & Hu, 2006; Mattes & Rothacker, 2001).

2. Physical Activity

In addition to diet, PAalso plays an important role in maintaining energy balance and weight control. Rapid increasing rates of obesity reflect both a lack of energy balance and a decrease in EE. Therefore, it is reasonable to assume that individuals with high EE are less likely to gain weight over time, compared with those who have low EE (Haskell *et al*., 2007). Sedentary behavior increases the risk of several chronic diseases like CVD, DM, HTN, etc. (Owen, Leslie, Salmon & Fotheringham, 2000). Recent recommendations focus on engaging in at least 30 minutes of moderate - intensity physical exercise, preferably all days of the week (Pate *et al*., 1995).

A global study done by Hallal *et al*. studied the PAof 122 countries. Results showed the overall prevalence of physical inactivity worldwide for adults to be 31% (Hallal *et al*., 2012). Wide variations in the prevalence of physical inactivity were seen in different regions: U.S (43%), Eastern Mediterranean (43.2%), Europe (34.8%),

Western Pacific (33.7%), Africa (27.5%), and South – East Asia (17%) (Hallal *et al*., 2012). A study done in Lebanon found the prevalence of physical inactivity among adults to be 52.1%(Sibai *et al*., 2003). These rates have been found to be higher than those reported in the U.S and European Union.

3. Sleep as a Risk Factor for Obesity

Sleep is an integral part of human survival as it occupies about one third of adult life. The definition of normal human sleep is described as "a state of perceptual disengagement from and unresponsiveness to the environment" (Acebo, Sadeh, Seifer, Tzischinsky, Hafer & Carskadon, 2005). Sleep plays an important role in contributing to better physical and mental health. The importance of sleep to survival has been demonstrated in several human studies where a lack of a good sleep results in a variety of cognitive and negative health issues. Several findings have found a link between sleep duration and mortality (Kripke, Garfinkel, Wingard, Klauber & Marler, 2002). According to Nieminen *et al*., adequate and quality sleep are important for the sustainability of body homeostasis, the secretion of growth hormone, and thus the development of the child (Nieminen, Löppönen, Tolonen, Lanning, Knip & Löppönen, 2002).

Insufficient sleep has been found to be linked with different types of health problems including: obesity, altered emotions, and impaired cognitive function (Nixon *et al*., 2008; Patel & Hu, 2008; Touchette, Petit, Séguin, Boivin, Tremblay & Montplaisir, 2007). As previously stated, it has been recently suggested that the rise in obesity may be linked to a reduction in sleep duration. A study done by National Sleep Foundation (NSF) concluded that adults today sleep on average of 6 hours and 40 minutes on weekdays and 7 hours and 25 minutes on weekends, while in 1960 they

slept on average of 8.5 hours (Leproult & Van Cauter, 2010). According to NSF, a reduction in the hours of sleep (for 8 hours or more on weekends) has been observed in the U.S., where the sleeping rate has decreased from 61% in 2001 to 52% in 2002 and 49% in 2005.

The below section provides a brief overview on sleep, its measurements, as well as the stages of sleep and the accepted views regarding sufficient sleep duration. This section is then followed by another one focusing on the link between sleep and obesity. It is important to note that the precise physiological functions of sleep and the adequacy of sleep remain unclear and needs further investigation.

F. Physiology of Sleep

It was previously believed that brain activity diminishes during sleep. Recent findings, however, have suggested that sleep is a highly complex behavior accompanied by recurring alterations in the brain activity. It has been studied that 4-5 sleep cycles occur in one night of sleep; each cycle consist of approximately 90 minutes in duration. Sleep has been traditionally divided into two main stages: the rapid – eye movement (REM) sleep and non-REM (NREM) sleep. Both stages differ in their physiological and neurological functioning e.g. heart rate, sympathetic nerve activity and respiration, brain activity, and muscle toning (Acebo *et al*., 2005). Prior to that phase, brief periods of wakefulness occur in 60 to 90 minutes sleep cycles (Carksdon MA, 1994).

The NREM sleep is subdivided into 3 stages: N1, N2, and N3, each representing the depth of the sleep (Silber *et al*., 2007). Stage 1 (N1) NREM serves as a transitional phase between wakefulness and sleep. Stage 2 (N2) is considered somewhat deeper than stage 1 (N1), while stage 3 (N3) occurs mostly during the first third of the night and then reduces later in the sleep cycle. It is known as the slow wave sleep and is

significant in the recovery from the previous day (Silber *et al*., 2007). After NREM phases finishes, it shifts into REM sleep which occurs in the latter parts of the sleep.

Rhythms, known as the circadian rhythms, are generated by neural structures in the hypothalamus (Dunlap, Loros& DeCoursey, 2004). They are known to regulate the sleep- wake cycle, nutritional intake, modulate physical activity, and control several bodily functions (hormone secretion, heart rate, or temperature). It has been suggested that the duration of sleep changes with age (Figure 4). Newborn babies may sleep up to 18 hours per day, and the duration begins to decline throughout childhood and adolescence, reaching up to 7 to 8 hours of sleep in adulthood. The NSF recently developed age-specific guidelines for sleep duration according to systematic review of the scientific literature relating sleep duration to health, performance, and safety. NSF recommends 7 to 9 hours of sleep for young adults (aged 18 – 25 years) and adults (aged 26 – 64 years), and 7 to 8 hours for older adults (aged ≥ 65 years) (NSF, 2015).

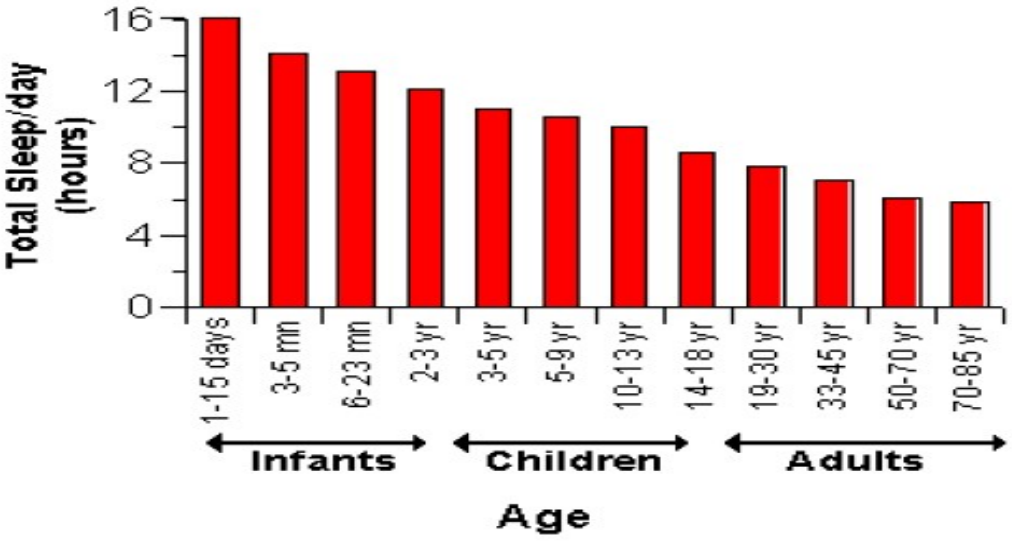

Fig. 4. Total sleep duration according to age in humans
*Source:*Arora, T. (2012). "Sleep and its association with metabolic function across the lifespan". University of Birmingham.

1. Sleep and its Measurements

Methods used to collect data on sleep and its disorders include subjective tools such as sleep logs, self – reported questionnaires, and sleep diaries, and objective tools such as polysomnography (PSG) and actigraphy. When using subjective assessment tools, there is a trade-off between quality and quantity of data collected due to individual burden (Iwasaki, Iemura, Oyama & Matsuishi, 2010). It is recommended that the survey should take less than 20 minutes to prevent individuals from carelessly responding (Rothman, 1998) .Well-known sleep questionnaires include: the Epworth Sleepiness Scale, Pittsburg Sleep Quality Index, and the Berlin Questionnaire (Lauderdale, Knutson, Yan, Rathouz, Hulley, Sidney & Liu, 2006). It is important to note that subjective sleep tools are easy for subjects to complete and are cost efficient. They may capture vital data such as sleepiness, tiredness, and sleep quality (Lauderdale *et al*., 2006).

The gold standard for measuring sleep is the use of the PSG. PSG is used in a research setting in a sound proof, completely dark, and climatized room. It involves several electrodes attached to an individual while sleeping overnight. It may provide information about the quality of sleep through different physiological measurements. Such measurements include: cardiac monitoring, snoring, and respiratory monitoring (Jafari B, 2010). PSG can provide the following results: sleep period time, duration of each stage, number of arousals, sleep efficiency, wake time after sleep onset, and the total sleep time. One main disadvantage of PSG is the fact that it may disturb normal sleep patterns, especially in children (Iwasaki *et al*., 2010). Furthermore, actigraphy has also been able to measure sleep. Actigraphy can measure a subject's movement activity though a wrist- worn device. Examples of actigraphy devices include the following: Actical, ActiTrainer, ActiSleep, Actiwatch, and Sleepwatch. The device can be worn for

up to one week. One main disadvantage of the actigraph is that it makes it hard to study individuals with movement related sleep disorders as is it cannot tell the difference between being awake without movement and sleeping without movement (Yi, Shin& Shin, 2006). It also cannot provide information about sleep quality and depth (Miwa, Sasahara & Matsui, 2007). Table 4 provides the advantages and limitations of the various sleep techniques discussed above.

Table 4. The advantages and limitations of the various techniques used to measure sleep

Sleep measure	**Advantages**	**Limitations**
Questionnaire	• Inexpensive • Quick to administer • Can be administered to large populations • Less labour intensive • Some have been validated against objective sleep techniques • Can enquire about numerous sleep parameters	• Subjective • Recall bias • Variable response rates • Inaccurate for detecting sleep disorders • Only provides cross sectional data • May have missing data • May report time in bed (TIB) rather than total sleep time (TST)
Sleep/time diary	• Can obtain longitudinal data • Can obtain detailed • information on other sleep variables (time in bed [TIB], total sleep time [TST], nighttime awakenings, naps, sleep quality) • Inexpensive • Quick to administer • Allow data collection from large samples • Less labour intensive (researcher)	• Fatigue • Recall • Failure to complete or return • May have missing data • Labour intensive (participant) • Requires participant motivation for completion
Polysomnography (PSG)	• Accurate for various sleep parameters • Objective • Defines sleep architecture • Determines brain activity and other physiological measures • Can determine sleep disorders • Can be combined with other physiological measures (hormone sampling under controlled conditions)	• Expensive • May not capture usual sleep due to equipment and/or environment (1st night effect) • Requires experienced technicians to score • Invasive • Uncomfortable • Unsuitable for long-term assessment • Inter/intra observer variation

"Table 4 –*Continued*"

Sleep measure	Advantages	Limitations
Actigraphy	• Objective • Can be used in free-living conditions • Can provide data over prolonged period • Inexpensive compared to PSG • Non-invasive	• Does not determine sleep architecture • Only provides sleep estimates • Some are not waterproof • No physiological measures to determine sleep • Needs accompanying accurate sleep diary • May overestimate sleep because of inactivity • Several different software and cut-points for analysis

*Source:*Arora, T. (2012). "Sleep and its association with metabolic function across the lifespan". University of Birmingham.

2. Causes and Consequences of Sleep Restriction

Sleep restriction has become globally prevalent due to certain influences such as: environment, diet, health conditions, smoking, caffeine and alcohol consumption, technology use, and work shifts. In addition, other factors that influence sleep include: age, sex, and physical and psychological health. A review done by Banks & Dinges (2007) suggested that sleep restriction in adults may lead to several negative health effects such as: elevated blood pressure, hormonal changes (reduced leptin and increased ghrelin), altered inflammatory markers, reduced glucose tolerance, reduced PAand increased energy intake, all leading to an increase in obesity and mortality (Figure 5).

A poll done by NSF (2011) concluded that 95% of the respondents reported to use technology at least one hour before going to bed for a few nights per week. Using techonology (such as cell phones, computers, televisions, and videogames) before going to bed has been shown to provide stimuli that keeps people awake past their natural bed time. A study done by Goel *et al*. (2009) showed the effects of sleep deprivation on the

body, such as increased reaction time and decreased ability to retain information properly and to hold attention (Goel, Rao, Durmer& Dinges, 2009).

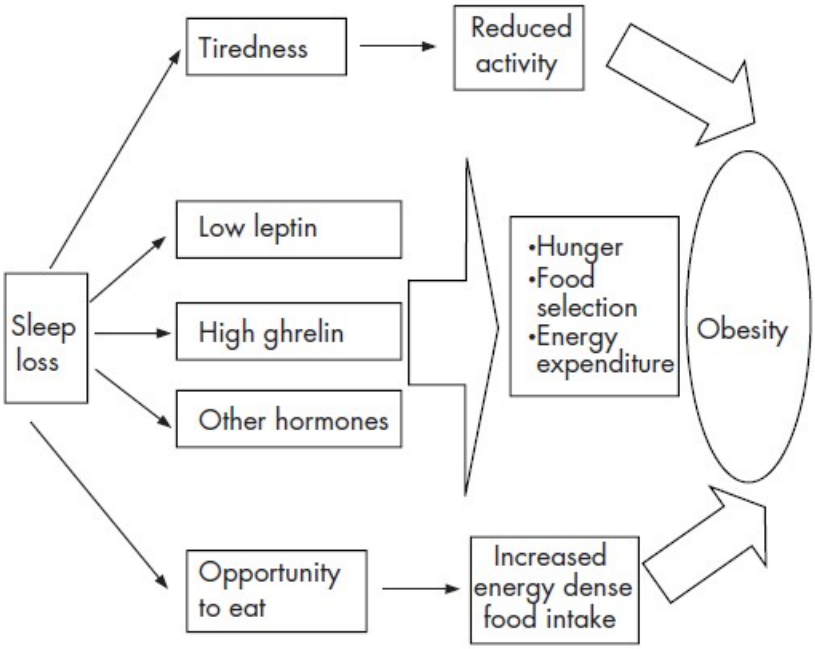

Fig. 5. The potential mechanisms through which short sleep duration could result in obesity
*Source:*Hosseini Araghi, M. (2014). "The association between sleep and obesity and its impact on health and wellbeing". University of Birmingham.

A recent study done in Australia on 20,000 young adults (aged 17 to 24 years) found that short sleep duration was linearly associated with continuous psychological stress (Glozier *et al*., 2010). The Centers for Disease Control (CDC) found that people who slept less than 6 hours per day, in the US, were more likely to engage in risky health behaviors than people who slept for 7 to 8 hours (Schoenborn & Adams, 2010).

G. Overview of Sleep Restriction and Obesity

1. Determinants of Chronic Sleep Restriction

Unlike acute total sleep deprivation which is the absence of sleep for a minimum of 24 hours, chronic sleep restriction is more common and is often defined as "habitual sleep durations that are less than 7 hours, and more than 4 hours, a night"(Dinges, Rogers & Baynard, 2005). Chronic sleep restriction has been currently identified as a health concern because it is associated with several health conditions such as depression, HTN, DM, obesity, and increased mortality (Heslop, Smith, Metcalfe, Macleod & Hart, 2002; Hublin, Partinen, Koskenvuo& Kaprio, 2007).

A combination of different factors can potentially affect sleep duration: socio-demographic, behavioral, environmental, health, and genetics (Magee, Huang, Iverson & Caputi, 2010). Socio-demographic factors like lower education level, lower income, and increased age has all been identified as strong predictors of chronic sleep restriction. Smoking, excessive alcohol intake, reduced PA, long working hours, increased television viewing has also been found to be associated with short sleep duration. People with chronic diseases like diabetes and CVD, mental conditions like depression and stress may also reduce sleep duration (Krueger & Friedman, 2009; Magee, Iverson & Caputi, 2009).

There are different processes though which chronic sleep restriction could contribute to obesity. Those pathways include: 1). glucose regulation, 2). neuroendocrine and metabolic pathways, and 3). waking behavior including eating and PA (Figure 6).

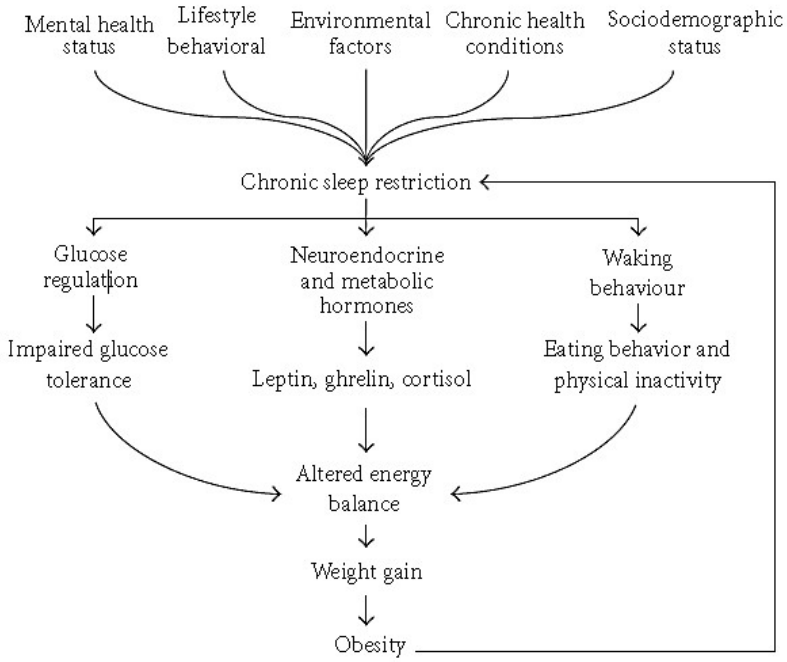

Fig. 6. Schematic representation of the pathways linking chronic sleep restriction to obesity
*Source:*Magee, C.A., Huang, X.-F., Iverson, D.C. & Caputi, P. (2010). "Examining the pathways linking chronic sleep restriction to obesity". *Journal of Obesity*.

2. Obesity and Sleep Disorders

The International Classification of Sleep Disorders listed more than 80 different sleep disorders dividing into 8 categories. Those categories include sleep disordered breathing/apnea, insomnia, and sleep- related movement disorders. All these disorders result in a reduced sleep duration and quality, thus causing weight gain and adiposity.

3. Obesity and Obstructive Sleep Apnea

One of the major risk factor causing the development and progression of obstructive sleep apnea (OSA) is obesity. It has been shown that the connection between OSA and obesity involves a two – way relationship, one is the implications of

OSA contributing to obesity and the other is the contribution of obesity to OSA. Studies have found obese subjects to be twice more likely to develop OSA than those who are normal weight (Caples, 2013). The Wisconsin Sleep Cohort found the strongest covariate of OSA to be the neck circumference, where the regional distribution of fat in the neck is more accurate than overall weight or BMI.

OSA has been found to impact different mechanisms in the body including: 1). changes in hormones related to weight control (like leptin, ghrelin, and adiponectin), satiety and obesity associated with impaired lipid and glucose levels, thus increasing inflammation and atherosclerosis, 2). changes in EE, 3). increase in calorie intake and preference for energy dense foods (especially carbohydrates and fat). Alterations in sleep duration leading to fatigue and a decrease in PA has also been observed (Figure 7) (Hargens, Kaleth, Edwards & Butner, 2013).

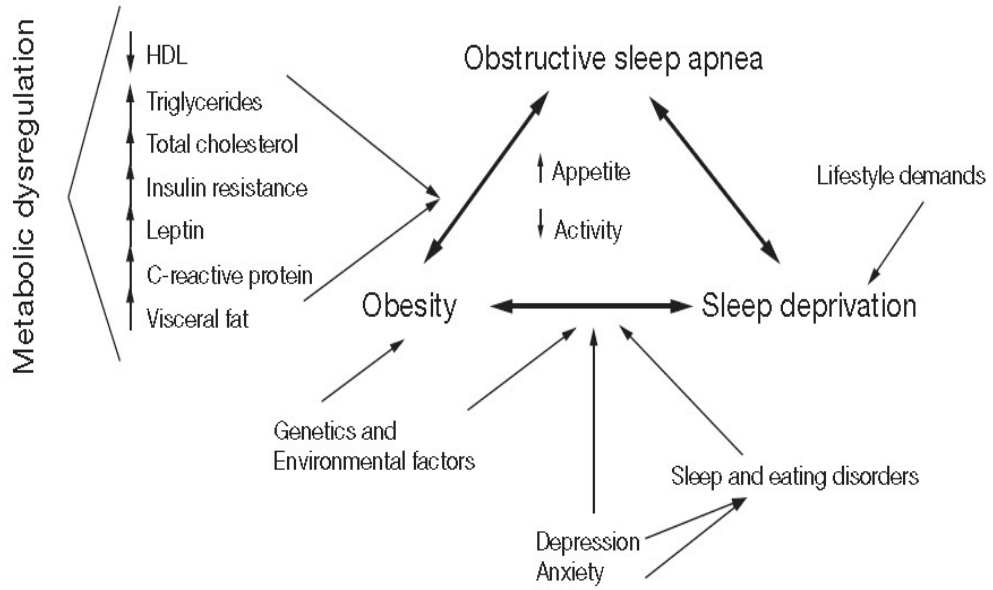

Fig. 7. Interactions between sleep apnea, obesity, sleep curtailment/deprivation and metabolic parameters
Source: Caples, S.M. (2013). "Sleep and Obesity". In: C.A. Kushida (Ed.), *Encyclopedia of Sleep*. Waltham: Academic Press, 408-412.

- *Sleep, Appetite and Energy Intake*

Evidence suggests altered sleep duration to increase hunger, appetite, and consumption of foods that are dense in carbohydrates and calories (Spiegel, Tasali, Penev& Van Cauter, 2004). A study on adolescents (n= 210) where dietary intake was assessed by a 24 hour recall and sleep was assessed by actigraphy indicated that subjects who slept for less than 8 hours were more likely to consume more calories from fats compared to those who slept for more than 8 hours (Weiss, Xu, Storfer-Isser, Thomas, Ievers-Landis & Redline, 2010). Similar findings suggest sleep restriction to be associated with a higher energy intake due to an increase in snacking (Nedeltcheva, Kessler, Imperial& Penev, 2009).

- *Sleep and Energy Expenditure*

Several findings indicate a functional role of sleep in the regulation of energy metabolism. Insufficient sleep has been shown to contribute to metabolic dysfunction as it reduces EE. A study conducted on healthy lean adult men (n= 14) showed a decrease in resting and postprandial EE when sleep deprived (Benedict *et al*., 2011). In another randomized crossover design, adult subjects were asked to sleep 4 hours and 15 minutes for two nights and 8 hours and 15 minutes for another two nights. After the first night of sleep restriction, PA levels significantly decreased during the day where less time was spent engaging in intense activity than when compared to usual sleep opportunity (Schmid *et al*., 2009).

3. Obesity and Insomnia

According to the NSF, insomnia is a complicated condition in which the person has a difficulty in falling asleep or staying asleep. Individuals with insomnia feel dissatisfied with their sleeping experiences which may often cause: fatigue, difficulty

concentrating, low energy, decreased work performance, and mood disturbances. It has been stated that the pathophysiology of insomnia might exacerbate due to a hyper-arousal state caused by CRF. It may initially start from some psychosocial factors which increase the arousal of the hypothalamic–pituitary–adrenal (Hargens *et al*., 2013).

Research shows that obese individuals are significantly more likely to report having sleeping difficulties or insomnia than non-obese individuals. Insomnia has been found to play an important role in leading to overconsumption of energy, thus causing weight gain. It increases the person's desire for high fat high sugar foods as well as their ability to store fat in the abdominal region through the elevation of glucocorticoid hormones like cortisol.

CHAPTER III

MATERIALS AND METHODS

A. Study Population

A secondary analysis of a cross-sectional titled the "Assessment of BPA levels and their association with the health status among the Lebanese population" was conducted in Beirut, Lebanon. A random sample of adult Lebanese subjects residing in Greater Beirut was recruited for the study through the support of "Information International S.A.L" which is a research and consultancy firm based in Beirut - Lebanon.

Inclusion criteria in the main study included the following*:* Lebanese adults, residing in Greater Beirut Area and aged more than 18 years old. Subjects were excluded based on the following:plastic or chemical factory workers, pregnant and lactating women, patient on dialysis, and other vulnerable groups (mental disabled people).

The random selection of the study participants was based on a multistage probability sampling, where the strata were the districts of Central Administrative Beirut. The second stage included the selection of neighborhoods within each of the selected areas in a way as to represent the make-up of the areas, followed by the selection of households based on systematic random sample in each selected neighborhood according to the estimated number of buildings in the neighborhood, and finally sampling a primary respondent within each household based on the most recent birthday.

B. Ethical Considerations

The study received the approval of the Institutional Review Board at the American University of Beirut (AUB). All subjects provided written consent prior to enrollment in the study (Appendix I and Appendix II). The patients signed the consent form after CITI certified field workers visited the respondents in their residence to explain the study aims and methodology.

Data collection was performed in a manner that ensures the confidentiality of the individuals.

C. Data Collection

A total of 501 participants, based on a rate of 10 participants per working day over 5 working days per week (Monday through Friday), excluding official holidays, agreed to participate in the study were invited to visit AUB for data collection. Subjects were instructed to fast overnight and those on regular medication were requested to bring their medications with them when visiting AUB on the assigned date.

Data collection took place at the Department of Nutrition and Food Sciences (NFSC) in the Faculty of Agricultural and Food Sciences at AUB by trained field workers, dietitians, and phlebotomists.

The interviewer asked about the total number of adults aged 18 years and over living in the household, and chose the one with the most recent birthday (at the date of the interview) to be the main respondent (excluding pregnant and lactating women as well as residents with mental disabilities). If the selected person was not at home, one follow-up was conducted before declaring a non-response. This method ensured that everyone had an equal chance of inclusion, with no one allowed to self-select into the sample. The name, date of birth, availability on week days and telephone number of the

potential participant were recorded for further follow up to specify the exact dates for taking them to AUB.

Data collection was conducted through face to face interviews using a multi-component questionnaire, and anthropometric assessment. The multicomponent questionnaire is presented in Appendix III and included the following sections:

1. Demographic and Socioeconomic Characteristic

- Age, gender, marital status, residence and previous travel, education, occupation, monthly income (expressed in U.S dollars).

2. Medical History

- Diseases such as: coronary artery disease, HTN, DM, DL, thyroid disease, cancer and other diseases (stroke, arthritis, chronic bronchitis or emphysema, and liver disease)
- Use of medications

3. Lifestyle Characteristics

- Smoking status and patterns, alcohol and coffee intake

4. PA Assessment

In the present study, the short form of the International Physical Activity Questionnaire (IPAQ) was used to assess physical activity (IPAQ, 2004; Maddison et al., 2007). The IPAQ short form is an instrument designed mainly for the assessment of PA among adults (age range of 15 – 69 years). It inquires about three specific types of activity: walking, moderate – intensity, and vigorous – intensity activity. Moderate PA

included activities like carrying light loads, bicycling at a regular pace, or tennis ; vigorous PA included activities such as heavy lifting, aerobics, or fast bicycling; and moderate PA included activities such as carrying light loads, bicycling at a regular pace, or tennis.

In order to compute the total score, the summation of the duration (in minutes) and frequency (days) of walking, moderate – intensity and vigorous – intensity activities was needed. These scores are expressed in MET –minutes/week:

- Walking MET-minutes/week = 3.3 * walking minutes * walking days at work
- Moderate MET-minutes/week = 4.0 * moderate-intensity activity minutes * moderate-intensity days at work
- Vigorous MET-minutes/week = 8.0 * vigorous-intensity activity minutes * vigorous-intensity days at work
- Total Work MET-minutes/week =sum of Walking + Moderate + Vigorous MET-minutes/week scores at work

The total PA for each subject was calculated using the MET-minutes/ week procedure described above. As stated by the Guidelines for Data Processing and Analysis of the IPAQ, very low (less than 10 MET - minutes/day) and very high activity (more than 180 MET - minutes/day) were recoded respectively into 0 and 180 minutes. Total METs with more than 960 minutes per day were labeled as outliers and then removed. The IPAQ sitting question is an additional indicator of time spent in sedentary activities and is not included as part of any summary score of physical activity.

Based on the above, individuals are assigned to three levels of PA: low, moderate, and high. Those levels were divided into three categories:

- *Category 1: Low*

This is the lowest level of physical activity. Those individuals who do not meet criteria for Categories 2 or 3 are classified as 'low'.

- *Category 2: Moderate*

The pattern of activity to be classified as 'moderate' is for either of the following criteria:

a) 3 or more days of vigorous-intensity activity of at least 20 minutes per day

OR

b) 5 or more days of moderate-intensity activity and/or walking of at least 30 minutes per day

OR

c) 5 or more days of any combination of walking, moderate-intensity or vigorous intensity activities achieving a minimum Total PA of at least 600 MET-minutes/week.

Individuals meeting at least one of the above criteria would be defined as accumulating a moderate level of activity.

- *Category 3: High*

A separate category labeled 'high' can be computed to describe higher levels of participation in PA. The two criteria for classification as 'high' are:

a) Vigorous-intensity activity on at least 3 days achieving a minimum Total PA of at least 1500 MET-minutes/week

OR

b) 7 or more days of any combination of walking, moderate-intensity or vigorous-intensity activities achieving a minimum Total PA of at least 3000 MET-minutes/week.

5. Sleep Apnea, Insomnia, and Sleep Habits

- *Sleep apnea* was assessed using a validated Berlin Assessment Questionnaire (Sharma et al., 2006). The Berlin Assessment Questionnaire is a series of eleven questions broken into three categories. Its main purpose is to determine if the individual is at risk for sleep apnea and to assess the subject's sleep habits.

    - The first category asks the following questions: *Do you snore? If yes, how loud? How often? Has your snoring ever bothered other people? Has anyone noticed that you stop breathing during your sleep?*

    - The second category asks the following questions: *How often do you feel tired or fatigued after your sleep? During your waking time, do you feel tired fatigued or not up to par? Have you ever nodded off or fallen asleep while driving a vehicle? If yes, how often does this occur?*

    - The third category includes the following: *Do you have high blood pressure? Or if BMI is > 30 kg/m^2.*

Questions were then scored depending on how respondents answer. If a person scored high on at least two of the three categories listed above, they were classified as being at a "high" risk for sleep apnea.

Other than the Berlin Questionnaire, additional questions were used to assess sleep duration and insomnia.

- *Sleep duration* for both weekend and weekdays were categorized according to the National Sleep Foundation. A duration of <7 hours per day of sleep was considered short, between 7-8 hours per day was considered medium, and ≥ 8 hours per day of sleep was considered long.

- *Sleepingdifficulties* were assessed using the following three questions:

    - *Do you have trouble falling asleep? Do you wake up during the*

night and have difficulty resuming sleep? Do you wake up too early in the morning and be unable to resume sleep? All three questions included the following answers: never, rarely (once/month), sometimes (2-4 times/ month), frequently (5 – 15/ month), and almost always (16 – 30 times/ month).

- In order to obtain the sleep difficulties score, "never" and "rarely" were coded as "0" and "sometimes", "frequently", and "almost always" were coded as "1". The sleep difficulties score was categorized into a "yes" if the sum of one of the three questions was ≥ 1, and "no" if the sum of all the three questions was a "0".

- *Insomnia* variable was identified using the sleep difficulties score along with the following questions: *How often do you feel tired or fatigued after you sleep? During your waking time do you feel tired, fatigued or not up to par?* Answers to those two questions included: never or nearly never, 1 – 2 times a month, 1 – 2 times a week, 3 – 4 times a week, and nearly every day. The answers: 1- 2 times a week, 1- 2 times a month, or never or nearly never were coded as "0" and nearly every day and 3-4 times a week were coded as "1".

The third question asks if the person has ever *"nodded off or fallen asleep while driving a vehicle?* Answers included: "yes" with a coding of "1" and "no" with a coding of "0".

The summation of those score along with the sleep difficulties score allowed us to obtain the insomnia score. If the sleep difficulties score was equal to 1 and the summation of the three questions was also equal to 1, then the subject was categorized as having insomnia.

6. Anthropometric Measurements and Blood Pressure

Weight, height, and WC were measured using standardized techniques in the nutrition research unit at the department of NFSC at AUB. Weight was taken, in light clothing, using a calibrated body composition analyzer (Inbody 3.0, Biospace Co. Ltd, Korea) and height was taken with a with a portable wall stadiometer (Seca 213, Germany). Measurements were repeated twice and the mean of the two values were used.

WC was measured using a plastic, inelastic measuring tape to the nearest 0.5cm (Seca 201, Germany) by locating the upper hip bone and the right upper iliac crest. The measuring tape was placed around the abdomen, in a plane parallel to the floor, at the level of the iliac crest without compressing the abdomen. The measurement was made at the end of a normal expiration. It was repeated twice (David York, 2000) and the mean of the two values was calculated and used.

Body fat was estimated using the Bioelectrical Impedance Analysis (BIA) technique (Inbody 3.0, Biospace Co. Ltd, Alpha-Tec s.a.r.l.). Sitting blood pressure and heart rate were also measured twice after ten minutes of rest with a standard digital sphygmanometer.

7. Dietary Assessment

Dietary assessment was conducted using a quantitative Food Frequency Questionnaire (FFQ). The use of FFQ has been reported to provide a relatively simple and effective way of obtaining individual food consumption data (Willett, 1998) and was found to be adaptable and appropriate for use in assessing the usual intake and dietary patterns in the Lebanese population (Hammami, Moghames, Shoaib, Nasreddine, Hwalla & Naja, 2015). One of the main advantages of the FFQs is the fact

that they are capable of capturing data that go beyond the time limits of conventional dietary survey and are thus able to reflect eating habits in the long term (Hammami *et al*., 2015).

The quantitative FFQ consisted of a list of 86 food items "as normally consumed". A reference portion, expressed in household measures or grams, was specified for each food item in the FFQ. The individual was asked to estimate the number of times per day, week, month or year he/she consumed this particular food product and the amount usually eaten per food item by making comparisons with the specified reference portion. The FFQ was categorized into 9 different groups:

1) *Bread and cereals:* all types of bread, pasta, rice, and cereals

2) *Dairy products:* low fat or full fat milk, yogurts, and cheeses

3) *Fruits and fruit juices*: fresh, canned, and dried fruits

4) *Vegetables*: cooked vegetables, vegetables stews, and raw salads

5) *Meat and meat alternatives*: legumes (cooked or dried lentils, beans, chickpeas, etc.), nuts, meat, poultry, fish, eggs, organ meats and sausages.

6) *Addedfatsand oils:* vegetable oil (corn/sunflower/soya), olive oil, olives, butter, ghee, mayonnaise, and tahini.

7) *Sweets and desserts:* sugar, jam, ice cream, cakes, chocolate bars, and Arabic sweets.

8) *Beverages*: water, coffee, tea, sodas (regular and diet), and alcoholic drinks.

9) *Miscellaneous:* any item not included in the above categories

The FFQ has been designed to include culture - specific recipes and dishes by a panel of nutritionists and was tested to check for cultural sensitivity and clarity. Nutritionist Pro software was used to estimate the participants' intakes of energy and macronutrients.

D. Statistical Analysis

Statistical analysis was done using SPSS 20 for Windows (SPSS Inc, Chicago, IL). The level of significance was set at a p-value ≤0.05. A database application using Microsoft Access (Corp., Redmond, WA, USA) was used for data management of nutritional data. This analysis module determines individual and mean consumption values per food item and per food group (g /day), average daily intake per individual, per sex group (g/day) and per age group (g/day), and the percentage of consumers per food item and per food group. Nutritionist Pro software was also used to estimate the participants' intakes of energy and macronutrients.

Descriptive analysis was conducted for different variables, using means with standard deviations for continuous variables, and frequencies and percents for categorical variables. Inferential statistics was carried out to assess the associations between different factors using either the independent t-test or the chi-square test. Correlation analysis using Pearson's correlation coefficient was performed between continuous variables. A binary variable was created for the purpose of classifying the study participants into obese and non – obese (including normal and overweight subjects). This variable was then used in the binary logistic regression to estimate odds ratios of obesity depending on different covariates. Univariate analysis was then performed to assess the association of obesity with different covariates, were the percent of obese served as the dependent variable and a number of baseline categorical characteristics served as independent variables. The estimated odds ratio (OR) and 95% confidence interval (CI) were obtained for each variable. Significance was defined when the 95% CI did not include 1. Multivariate linear regression analysis, using forced - entry method, was carried out to identify predictors of obesity. Multivariate analysis using logistic regression was then conducted to determine the overall association

between obesity and several covariates, and was generated by inclusion of significant variables at the univariate analysis. A p-value≤0.05 will be used to indicate significance in all cases.

CHAPTER IV

RESULTS

A. Baseline Characteristics

A total of 501 participants agreed to participate in the study. The characteristics of the study population are summarized in the tables below. They are mainly presented separately according to BMI (obese vs. non-obese).

Prevalence of overweight and obesity are shown in Table 5 for the total sample (n = 501). Obesity prevalence was estimated at 41.5% for obese subjects and 34.1% foroverweight subjects (Table 5).

Table 5. Prevalence of Overweight and Obesity of the Study Subjects (n=501)

BMI[a]	**Criteria[b](kg/m^2)**	**n (%)**
Normal	18.5 – 24.9	122 (24.4)
Overweight	25 – 29.9	171 (34.1)
Obese	≥ 30	208 (41.5)

[a] Body Mass Index
[b] World Health Organization, 2010

2. Anthropometric Characteristics

Anthropometric characteristics of the study sample are shown in Table 6 for the total sample (n = 501) and separately for obese and non- obese. The total sample had a mean BMI of 28.9 ± 5.4, percent body fat of 28.6 ± 11.5, WC of 95.3 ± 13.9, and a waist - to - hip ratio of 0.97 ± 0.008. Mean estimates of percent body fat of obese (38.7 ± 8.8) were significantly higher compared to non-obese (p=<0.0001). The same was observed for elevated waist circumference (106.17 ± 10.7; p=<0.0001) and waist – to –

hip ratio (1.02 ± 0.08, p=<0.0001).

Table 6. Anthropometric Measurements of the Study Subjects According to BMI Classification (n=501)

Anthropometrics	**Total** (n= 501)	**Non Obese** BMI<30 kg/m^2 n= 293(58.5%)	**Obese** BMI≥30 kg/m^2 n= 208(41.5%)	**P- value**[a]
Mean ± SDor n (%)[b]				
BMI[c]	28.9 ± 5.4	25.3 ± 3.06	34.06 ± 3.5	**<0.0001****
% Body Fat[d](n=499)	28.6 ± 11.5	21.5 ± 6.9	38.7 ± 8.8	**<0.0001****
Waist Circumference (in cm)	95.3 ± 13.9	87.7 ± 10.5	106.17 ± 10.7	**<0.0001****
Normal WC: Males: < 94 cm Females: <80 cm	118 (23.6)	117 (39.9)	1 (0.5)	**<0.0001****
Elevated WC[e] Males: ≥94 cm Females: ≥80 cm	383 (76.4)	176 (60.1)	207 (99.5)	
Waist-to-Hip Ratio (WHR)[f]	0.97 ± 0.008	0.93 ± 0.70	1.02 ± 0.08	**<0.0001****

[a] p-value is derived from Pearson Chi-Square for all categorical variables and from independent samples T-test for all continuous variable.
[b] Percentages are within column
[c] BMI: Body Mass Index.
[d] Body fat % was derived using Inbody machine.
e The classification criteria for waist circumference was defined according to International Diabetes Federation (IDF) standardized criteria (IDF, 2005).
f The classification criteria for waist – to – hip ratio was defined according to World Health Organization (WHO) standardized criteria (WHO, 2011b).
** p <0.001: highly significant

3. Socio-Demographic and Lifestyle Characteristics

Baseline socio-demographic and lifestyle characteristics of the study sample are presented in Tables 7 and 8, respectively. Overall, 35.7% of the study sample were males while 64.3% were females (p= 0.012) with a mean age of 45.3 ± 14.9 years. Most of the participants had a crowding index ≥ 1 persons/ room (79.6%). Approximately 91% of the study population earned an income of less than 2000 USD and have reached secondary education level or below (82.1%), indicating a low SES. Low intensity PA was seen in almost half of the study population (47.7%) compared to high intensity PA

(21.2%).

Table 7. Socio- demographic Characteristics of the Study Subjects Classified based on Adiposity Status (n=501)

Characteristics	**Total** (n= 501)	**Non Obese** BMI<30 kg/m^2 n= 293(58.5%)	**Obese** BMI≥30 kg/m^2 n= 208(41.5%)	**P- value**[a]
Mean ± SDor n (%)[b]				
Age in years(n=501)	45.3 ± 14.9	41.9 ± 16.3	47.2 ± 13.8	**<0.0001****
18 – 24	61 (12.2)	48 (16.4)	13 (6.3)	**<0.0001****
25 – 29	36 (7.2)	29 (9.9)	7 (3.2)	
30 – 39	82 (16.4)	56 (19.1)	26 (12.5)	
≥ 40	322 (64.3)	160 (54.6)	162 (78)	
Gender (n = 501)				
Male	179 (35.7)	118 (40.3)	61 (29.3)	**0.012***
Female	322 (64.3)	175 (59.7)	147 (70.7)	
Marital Status[c] (n= 499)				
Married	332 (66.5)	185 (63.6)	147 (70.7)	0.098
Unmarried	167 (33.5)	106 (36.4)	61 (29.3)	
Income[d] (n= 453)				
<600$	153 (33.8)	72 (27.4)	81 (42.6)	**0.02***
600–999.9$	170 (37.5)	100 (38)	70 (36.8)	
1000-2000$	90 (19.9)	64 (24.3)	26 (13.7)	
>2000$	40 (8.8)	27 (10.3)	13 (6.8)	
Education[e] (n = 498)				
Illiterate	54 (10.8)	24 (8.2)	30 (14.5)	**0.007****
Up to Intermediate	263 (52.8)	147 (50.5)	116 (56)	
Secondary	92 (18.5)	55 (18.9)	37 (17.9)	
Technical diploma	35 (7)	23 (7.9)	12 (5.8)	
University degree	54 (10.8)	42 (14.4)	12 (5.8)	
Crowding Index[f] (n = 495)				
≥ 1 person/room	394 (79.6)	242 (83.4)	152 (74.1)	**0.011***
<1 person/room	101 (20.4)	48 (16.6)	53 (25.9)	

[a] p-value is derived from Pearson Chi-Square for all categorical variables and from independent samples T-test for all continuous variable.
[b] Percentages are within column.
[c] Single includes divorced and widowed.
[d] Income is expressed in terms of U.S. dollars.
[e] Up to intermediate includes: primary and elementary school.
[f] Crowding index was calculated as the No. of persons living in the household per the No. of bedrooms.
* p ≤0.05: significant
** p <0.0001: highly significant

Table 8. Lifestyle Characteristics of the Study Subjects Classified based on Adiposity Status (n=501)

Lifestyle Factors	**Total** (n= 501)	**Non Obese** BMI<30 kg/m^2 n= 293(58.5%)	**Obese** BMI≥30 kg/m^2 n= 208(41.5%)	**P- value**[a]
Mean ± SDor n (%)[b]				
Smoker (n= 501)				
No	285 (56.9)	151 (51.5)	134 (64.4)	
Yes	216 (43.1)	142 (48.5)	74 (35.6)	**0.004***
Number of cigarettes/day	45.98 ± 149.22	42.96 ± 143.22	51.74 ± 160.90	0.685
Nargileh (n= 501)				
No	359 (71.7)	202 (68.9)	157 (75.5)	0.11
Yes	142 (28.3)	91 (31.1)	51 (24.5)	
Coffee drinker (n= 501)				
No	98 (19.6)	64 (21.8)	34 (16.3)	0.12
Yes	402 (80.4)	229 (78.4)	174 (83.7)	
Alcohol Drinker (n= 501)				
No	406 (81)	222 (75.8)	184 (88.5)	**<0.0001****
Yes	95 (19)	71 (24.2)	24 (11.5)	
Physical activity[c]**(n=501)**				
Met-minutes of heavy work per week	8.26 ± 31.8	9.01 ± 32.23	7.21 ± 31.20	0.53
Met-minutes of Moderate work per week	9.64 ± 30.6	13.31 ± 35.56	4.47 ± 20.77	**0.001***
Met-minutes of Walking per week	72.72 ± 65.30	74.16 ± 66.02	70.7 ± 64.4	0.56
Total exercise (minutes/ day)	107.62 ± 79.50	112.17 ± 83.16	73.47 ± 5.63	0.15
Total Met-minutes from all three categories per week	2042.67 ± 2063.75	1888.24 ± 2038.88	2146.85 ± 2077.90	0.207
Sedentary time (minutes/day)	291.9 ± 176.07	280.81 ± 176.21	307.50 ± 175.11	0.096
Levels of physical activity (n=501)				
Low-intensity	239 (47.7)	130 (44.4)	109 (52.4)	
Moderate- intensity	156 (31.1)	90 (30.7)	66 (31.7)	**0.041***
High- intensity	106 (21.2)	73 (24.9)	33 (15.9)	

[a] p-value is derived from Pearson Chi-Square for all categorical variables and from independent samples T-test for all continuous variable.
[b] Percentages are within column.
[*] p ≤0.05: significant
[**] p <0.0001: highly significant

Alcohol consumption of the study population was relatively low (19%). Forty three percent of the study sample reported to smoke cigarettes. The proportion of obese was significantly higher with higher age (p= <0.0001), female gender (p= <0.0001),

lower income (p=0.02) and education status (p= 0.007), and low intensity physical activity (p=0.041). The proportion of obesity was significantly higher in those who do not smoke cigarettes (p=0.004) and do not drink alcohol (p= <0.0001).

4. Sleep Patterns and Sleep Duration amongst Study Subjects

Sleep duration and patterns of the study subjectsare shown in Table 9 for the total sample (*n* = 501) and separately for obese and non-obese. Almost half of the study population reported to sleep less than 7 hours per day on weekdays (51.1%) and weekends (66.1%). A significantly higher proportion of obese was found among those with shorter sleep duration during weekdays (56.7%; p= 0.019). A high risk of sleep apnea was reported for 31.3% of the study sample of which 54.9% were obese (p=<0.0001).

Table 9. Sleep Duration and Patterns of the Study Subjects According to BMI Classification (n=501)

Characteristics	**Total** (n= 501)	**Non Obese** BMI<30 kg/m^2 n= 293(58.5%)	**Obese** BMI≥30 kg/m^2 n= 208(41.5%)	**P- value**[a]
Mean ± SDor n (%)[c]				
Sleep duration on weekdays (in hours) (n= 501)				
Short: <7	256 (51.1)	138 (47.1)	118 (56.7)	**0.019***
Medium: 7 – 8	98 (19.6)	55 (18.8)	43 (20.7)	
Long: ≥ 8	147 (29.3)	100 (68)	47 (22.6)	
Sleep duration on weekends (in hours) (n= 500)				
Short: <7	331 (66.1)	182 (62.1)	149 (71.6)	0.069
Medium: 7 – 8	92 (18.4)	58 (19.8)	34 (16.3)	
Long: ≥ 8	78 (15.6)	53 (18.1)	25 (12)	
Sleep Apnea Score[b] **(n = 432)**	1.10 ± 0.88	0.71 ± 0.78	1.68 ± 0.70	**<0.0001****
Sleep Apnea[b] **(n=432)**				
Low Risk	297 (68.8)	219 (84.6)	78 (45.1)	**<0.0001****
High Risk	135 (31.3)	40 (15.4)	95 (54.9)	

"Table 9 – *Continued*"

Characteristics	**Total** (n= 501)	**Non Obese** BMI<30 kg/m^2 n= 293(58.5%)	**Obese** BMI≥30 kg/m^2 n= 208(41.5%)	**P- value**[a]
Sleep Difficulties (n= 501)				
Never	123 (24.6)	75 (25.6)	48 (23.1)	0.66
Rarely (once/month)	33 (6.6)	16 (5.5)	17 (8.2)	
Sometimes (2-4/ month)	63 (12.6)	34 (11.6)	29 (13.9)	
Frequently (5-15/month)	59 (11.8)	36 (12.3)	23 (11.1)	
Almost always (16-30/ month)	223 (44.5)	132 (45.1)	91 (43.8)	
Insomnia (n=501)				
No	286 (57.1)	171 (58.4)	115 (55.3)	0.49
Yes	215 (42.9)	122 (41.6)	93 (44.7)	

[a] p-value is derived from Pearson Chi-Square for all categorical variables and from independent samples T-test for all continuous variable.
[b] Score obtained from Berlin Questionnaire: *High risk*: if there are 2 or more categories where the score is positive, *Low risk*: if there is only 1 or no categories where the score is positive.
[c] Percentages are within column
[*] p ≤0.05: significant
[**] p <0.001: highly significant

5. Dietary Energy and Macronutrient Intakes

Energy and macronutrient intakes of the study sample are presented in Table 10 for the total sample (*n* = 501) and separately for obese and non-obese. Mean energy intake for both categories amounted to 3319.97 ± 1594.23 kcal/day, of which 41.2% was derived from fat. The average contributions of protein and carbohydrates to energy intake were 12.6% and 46.9%, respectively.

Table 10. Mean intake for energy and macronutrients selected according to BMI Classification (n= 501)

Dietary Intake	**Total** (n= 501)	**Non Obese** (n= 293)	**Obese** (n= 208)	**P-value**[a]
Mean ± SDor n (%)[b]				
Energy (Kcal) (n= 486)	3319.97 ± 1594.23	3438 ± 1627.15	3159.53 ± 1537.94	0.056
Carbohydrates (g/day) (n= 487)	381.05 ± 178.23	394.19 ± 177.34	363.21 ± 178.31	0.058

"Table 10 – *Continued*"

Dietary Intake	**Total** (n= 501)	**Non Obese** (n= 293)	**Obese** (n= 208)	**P-value**[a]
Carbohydrate (% of total Kcal)	46.84 ± 8.9	46.85 ± 8.5	46.83 ± 9.34	0.98
Protein (g/day) (n= 483)	101.42 ± 50.21	106.28 ± 53.04	94.75 ± 45.33	**0.012***
Protein (% of total Kcal)	12.47 ± 2.83	12.4 ± 2.6	12.6 ± 3.09	0.48
Fat (g/day) (n= 493)	155.72 ± 85.33	162.9 ± 88.6	145.76 ± 79.7	**0.028***
Fat (% of total Kcal)	41.14 ± 8.88	40.98 ± 8.1	41.35 ± 9.83	0.65

[a] p-value is derived from independent samples T-test for all continuous variable
[b] Percentages are within column
* p ≤0.05: significant

B. Univariate Logistic Regression Association between Obesity and Baseline Covariates (Socio-demographics, Lifestyle, Anthropometrics, Energy Intake, and Sleeping Behaviors)

1. Socio-Demographic and Lifestyle Characteristics of Participants in Predicting the Risk of Obesity

Simple binary logistic regression was carried out to show the association ofobesity with baseline covariates such as socio-economic status, lifestyle and energy intake, and sleep behaviors. Subjects were divided into two groups based on their adiposity status. The bivariate logistic regression results are presented in Table 11.

The logistic regression showed significant associations between all tested socio-demographic variables and the risk of obesity. The odds of obesity were significantly higher in those aged between 30-39 years old (OR= 1.71; CI =0.80 – 3.70) and more than 40 years old (OR=3.74; CI = 1.95 – 7.17) when compared to those aged 18 – 24 years. The odds of obesity were also higher amongst females (OR= 1.63; CI= 1.11 – 2.40) when compared to males. An income greater than 2000 USD and a university level of education significantly decrease the odds of obesity (OR= 0.43, CI=

0.20 – 0.9) vs. (OR= 0.3; CI= 0.09 – 0.53). A lower CI (<1 person/room) significantly increases the odds of obesity by 1.7 (p=0.012) when compared to a CI of ≥1person/room.

Table 11. Univariate Logistic Regression between Socio- demographic Characteristics of the Study Subjects and Obesity (n=501)

Variables	OR (95% CI)	Significance
Age in years(n=501)		
18 – 24	Ref	
25 – 29	0.89 (0.32 – 2.50)	0.36
30 – 39	1.71 (0.80 – 3.70)	**<0.0001****
≥ 40	3.74 (1.95 – 7.17)	**<0.0001****
Gender (n = 501)		
Male	Ref	
Female	1.63 (1.11 – 2.40)	**0.012***
Income (n= 453)		
<600$	Ref	
600–999.9$	0.62 (0.40 – 0.97)	**0.035***
1000-2000$	0.36 (0.20 – 0.63)	**<0.0001****
>2000$	0.43 (0.20 – 0.9)	**0.023***
Education(n = 498)		
Illiterate	Ref	
Up to Intermediate	0.63 (0.35 – 1.14)	0.13
Secondary	0.54 (0.27 – 1.062)	0.54
Technical	0.42 (0.17 – 1.007)	0.052
University	0.3 (0.09 – 0.53)	**0.001****
Crowding Index (n = 495)		
≥ 1 person/room	Ref	
<1 person/room	1.7 (1.13 – 2.73)	**0.012***

* p ≤0.05: significant
** p <0.0001: highly significant

2. Lifestyle and Energy Intake Characteristics of Participants in Predicting the Risk of Obesity

Subjects were divided into two groups based on their BMI status. Univariate logistic regression between lifestyle and energy intake characteristics and obesity is shown in Table 12. There are significant associations between smokers, alcohol drinkers, and physical activity with risk of obesity.

Table 12. Univariate Logistic Regression between Lifestyle and Energy Intake Characteristics of the Study Subjects and Obesity (n=501)

Variables	OR (95% CI)	Significance
Smoker (n= 501)		
No	Ref	
Yes	0.59 (0.40 – 0.85)	**0.004***
Nargileh (n= 501)		
No	Ref	
Yes	0.72 (0.48 – 1.077)	0.110
Coffee drinker (n= 501)		
No	Ref	
Yes	1.43 (0.90 – 2.27)	0.13
Alcohol Drinker (n= 501)		
No	Ref	
Yes	0.40 (0.25 – 0.67)	**<0.0001****
Levels of physical activity (n=501)		
Low-intensity	Ref	
Moderate- intensity	0.89 (0.58 – 1.31)	0.52
High- intensity	0.54 (0.33 – 0.87)	**0.012***
Energy (Kcal)	1.000 (1.000 – 1.000)	**0.02***

* $p \leq 0.05$: significant
** $p \leq 0.0001$: highly significant

The univariate logistic regression showed that smoking and alcohol drinking significantly decrease the odds of obesity (OR=0.59; CI=0.40 – 0.85 and OR=0.40; CI= 0.25 – 0.67), respectively. Having a high level of physical activity significantly decreases the odds of obesity when compared to low level of physical activity (OR= 0.54; CI=0.33 – 0.87).

3. Sleep Patterns and Behavior of Participants in Predicting the Risk of Obesity

Subjects were divided into two groups based on their adiposity status. Univariate logistic regression between sleep pattern and behavior and obesity is shown in Table 13.

Table 13. Univariate Logistic Regression between Sleep Variables of the Study Subjects and Obesity (n=501)

Variables	OR (95% CI)	Significance
Sleep Apnea Score	4.92 (3.57 – 6.8)	**<0.0001****
Sleep Apnea		
Low Risk	Ref	
High Risk	6.66 (4.25 – 10.5)	**<0.0001****
Sleep Duration (weekend)		
Long	Ref	
Medium	1.50(0.87 – 2.60)	0.14
Short	1.75(1.008 – 3.04)	**0.04***
Sleep Duration (weekday)		
Long	Ref	
Medium	1.82(1.190 – 2.78)	0.06
Short	1.66(0.98 – 2.82)	**0.006****
Insomnia		
No	Ref	
Yes	1.13 (0.79 – 1.63)	0.45
Sleep Difficulties		
Never	Ref	
Rarely	1.66 (0.77 – 3.60)	0.20
Sometimes	1.33 (0.72 – 2.46)	0.36
Frequently	0.99 (0.53 – 1.89)	0.10
Almost always	1.07 (0.68 – 1.69)	0.75

* $p \leq 0.05$: significant
** $p < 0.0001$: highly significant

The regression showed a significant association between sleep apnea and sleep duration (weekdays and weekends) with risk of obesity. Having sleep apnea significantly increased the odds of obesity (OR=4.92; CI=3.57 – 6.8) and (OR=6.66; CI=4.25 – 10.5). The univariate logistic regression also indicated that smoking and alcohol drinking significantly decrease the odds of obesity (OR=0.59; CI=0.40 – 0.85) and (OR=0.40; CI= 0.25 – 0.67). Whereas having insomnia increases the odds of obesity, but without statistical significance.

4. Association between Obesity and Sleep Apnea (Multivariate Regression)

The association between obesity and sleep apnea among obese and non-obese

participants is found in Table 14. After adjusting for other variables including: age, gender, energy intake, physical activity, education, CI, smoking, and drinking alcohol; findings of the logistic regression showed significantly higher prevalence of obesity among those with OSA (OR=6.2; CI=3.8-10.06). Smoking remained negatively associated with obesity after adjusting for other variables, whereby the odds of obesity were lower in those who smoke compared to nonsmokers (OR=0.53; CI=0.32 – 0.87).

Table 14. Multivariate Logistic Regression Describing the Associations between Sleep Apnea and the risk of Obesity in the Study Sample (n =501)

Variables	**OR (95% CI)**	**Significance**
Age in years		
18 – 24	Ref	
25 – 29	0.80 (0.22 – 2.63)	0.70
30 – 39	1.32 (0.50 – 3.54)	0.58
≥ 40	2.06 (0.86 – 4.93)	0.11
Gender		
Males	Ref	
Females	1.008 (0.56 – 1.80)	0.98
Total Energy (Kcal/day)	1.000 (1.000 – 1.000)	0.77
Sleep Apnea		
No	Ref	
Yes	6.2 (3.8 – 10.06)	**<0.0001****
Physical Activity		
Low	Ref	
Moderate	1.4 (0.82 – 2.33)	0.23
High	0.68 (0.37 – 1.30)	0.22
Education		
Illiterate	Ref	
Up to Intermediate	0.93 (0.42– 2.06)	0.90
Secondary	0.79 (0.31 – 1.98)	0.60
Technical	0.74 (0.24 – 2.32)	0.60
University	0.40 (0.13 – 1.11)	0.07
Crowding Index		
>=1 person per room	Ref	
<1 person per room	1.22 (0.7 – 2.22)	0.50
Smoker		
No	Ref	
Yes	0.53 (0.32 – 0.87)	**0.012***
Alcohol Drinker		
No	Ref	
Yes	0.70 (0.34 – 1.44)	0.33

* p ≤0.05: significant
** p<0.0001: highly significant

5. Association between Obesity and Sleep Apnea Score (Multivariate Regression)

The association between obesity and sleep apnea score among obese and non-obese participants is found in Table 15. In agreement with findings presented in Table 14, findings of the multivariate regression showed significantly higher risk of obesity in those with a high sleep apnea score (OR= 4.8; CI=3.40 – 6.80). Moreover, lower odds of obesity were reported for those who smoke (Or=0.5; CI= 0.28 – 0.83) when compared to those who do not smoke.

Table 15. Multivariate Logistic Regression Describing the Associations between Sleep Apnea Score and the risk of Obesity in the Study Sample (n =501)

Variables	**OR (95% CI)**	**Significance**
Age in years		
18 – 24	Ref	
25 – 29	0.51 (0.133 – 1.99)	0.34
30 – 39	1.08 (0.38 – 3.08)	0.9
≥ 40	1.61 (0.632 – 4.10)	0.32
Gender		
Males	Ref	
Females	1.12 (0.607 – 2.06)	0.78
Total Energy (Kcal/day)	1.000 (1.000 – 1.000)	0.60
Sleep Apnea Score	4.8 (3.40 – 6.80)	**<0.0001****
Physical Activity		
Low	Ref	
Moderate	1.45 (0.82 – 2.54)	0.20
High	0.77 (0.40 – 1.51)	0.45
Education		
Illiterate	Ref	
Up to Intermediate	1.09 (0.47 – 2.60)	0.84
Secondary	0.90 (0.33 – 2.40)	0.82
Technical	0.95 (0.28 – 3.20)	0.93
University	0.65 (0.20 – 2.05)	0.46
Crowding Index		
>=1 person per room	Ref	
<1 person per room	1.20 (0.61 – 2.20)	0.67
Smoker		
No	Ref	
Yes	0.5 (0.28 – 0.83)	**0.008***
Alcohol Drinker		
No	Ref	
Yes	0.68 (0.32 – 1.50)	0.33

* p ≤0.05: significant
** p <0.0001: highly significant

6. Association between Obesity and Sleep Duration during Weekdays (Multivariate Regression)

The association between obesity and sleep duration during weekdays among obese and non-obese participants is found in Table 16. Based on a multivariate model that included: age, gender, energy intake, physical activity, education, CI, smoking, and drinking alcohol; findings of the multivariate logistic regression showed a significantly higher risk of obesity for those: aged ≥ 40 years old (OR=2.24; CI=1.05 – 4.80), slept for shortduration (<7 hours) (OR=1.8; CI=1.099 – 3.30), and those with a CI of < 1 person/ room (OR=1.90; CI=1.14 – 3.12). Significantly lower odds of obesity were reported for those who smoke (OR=0.56; CI=0.37 – 0.85) and those with a university degree (OR=0.33; CI=0.13 – 0.82).

Table 16. Multivariate Logistic Regression Describing the Associations between Sleep Duration (Weekdays) and the risk of Obesity in the Study Sample (n =501)

Variables	OR (95% CI)	Significance
Age in years		
18 – 24	Ref	
25 – 29	0.80 (0.27 – 2.40)	0.70
30 – 39	1.08 (0.46 – 2.60)	0.90
≥ 40	2.24 (1.05 – 4.80)	**0.04***
Gender		
Males	Ref	
Females	1.14 (0.70 – 1.86)	0.60
Total Energy (Kcal/day)	1.000 (1.000 – 1.000)	0.60
Sleep duration on weekdays (in hours)		
Long: ≥ 8 hours	Ref	
Medium: 7 – 8 hours	1.56 (0.98 – 2.48)	0.08
Short: <7 hours	1.8 (1.099 – 3.30)	**0.04***
Physical Activity		
Low	Ref	
Moderate	0.97 (0.62 – 1.51)	0.93
High	0.61 (0.36 – 1.04)	0.06
Education		
Illiterate	Ref	
Up to Intermediate	0.90 (0.48 – 1.70)	0.75
Secondary	0.88 (0.41– 1.87)	0.74
Technical	0.72 (0.27 – 1.93)	0.50
University	0.33 (0.13 – 0.82)	**0.01***
Crowding Index		
>=1 person per room	Ref	
<1 person per room	1.90 (1.14 – 3.12)	**0.01***
Smoker		
No	Ref	
Yes	0.56 (0.37 – 0.85)	**0.006***
Alcohol Drinker		
No	Ref	
Yes	0.61 (0.33 – 1.15)	0.13

* p ≤0.05: significant

CHAPTER V

DISCUSSION

Obesity is a growing health concern because of its adverse impacts on metabolism, blood pressure, and the risk of chronic illnesses like DM, CVD, gastrointestinal disease, cancer, and respiratory disease (Cheung, Machin, Karlberg & Khoo, 2004; Gunturu & Ten, 2007; Morrison, Friedman& Gray-McGuire, 2007; Weiss *et al.*, 2004). Family history of obesity and genetic factors are amongst the recognized non-modifiable determinants of obesity risk. However, obesity is increasingly linked to an obesogenic environment characterized by high consumption of energy dense foods and physical inactivity. More recently, large population studies from the U.S. and Europe have suggested a potential role of unhealthy sleeping patterns and short sleep duration (Gupta, Mueller, Chan& Meininger, 2002; Hasler *et al.*, 2004; Kripke *et al.*, 2002). This study aimed at investigating the prevalence of obesity and its association with sociodemographics, dietary intake, lifestyle, and sleep patterns in a sample of Lebanese urban adults aged 18 years and older.

Findings of the current study estimated the prevalence of obesity (i.e., BMI ≥ 30 kg/m^2) (WHO, 2008) at 41.5% in Lebanese urban adult, with a significantly higher prevalence in women (70.7%) compared to men. When these estimates are compared to those reported by the most recent national survey conducted in 2008/2009, an increasing trend in the prevalence of obesity is noted, whereby obesity rates have increased from 28.2% in 2008/2009 to 41.5% in 2014. This increasing trend has been previously suggested by Nasreddine *et al*. (2012) who showed that the prevalence rates of adult obesity have increased from 17.4% in 1997% to 28.2% in 2009 in Lebanon (Nasreddine

et al., 2012). It is, however, important to keep in mind that the estimates of obesity prevalence as assessed by the present study may not be directly comparable to those reported by previous national surveys given that the current study's population is representative of the Greater Beirut area and not of Lebanon as a whole. The high prevalence of adult obesity in the urban setting of Beirut is in line with the literature, whereby several studies have found a higher risk of obesity in those living in urban areas compared to rural areas (Ebrahim *et al*., 2010; Ezzati *et al*., 2005; Popkin, 1999). This higher risk of obesity has been attributed to increased consumption of saturated fats and sugars, and decreased PA associated with urbanization and westernization (Fall, 2001).

When compared to data from other countries in the Eastern Mediterranean Region, current prevalence rates of obesity in Lebanese urban adults were found to exceed those reported from Morocco (31.2%) (Berraho, El Achhab, Benslimane, Rhazi, Chikri & Nejjari, 2012) Tunisia (37 % in women and 13 % in men) (Atek *et al*. 2013), Algeria (30.1 % in women and 9.1 % in men) (Atek *et al*. 2013), and Iran (22.5% in women and 10.5% in men) (Ayatollahi & Ghoreshizadeh, 2010)., Palestine (37.2% in women and 21 % in men) (Abu-Rmeileh, Husseini, Capewell, & O'Flaherty, 2013), and Oman (36.9% in women and 20.6% in men), while being very similar to those reported from Syria (43%) (Al Ali, Rastam, Fouad, Mzayek & Maziak, 2011), Saudi Arabia (40%) (Al-Daghri, Al-Attas, Alokail, Alkharfy, Yousef, Sabico & Chrousos, 2011), and Qatar (54.7% in women and 44% in men) (Ng *et al*., 2014). When comparing obesity rates as estimated in this study to those reported from European countries, the prevalence of obesity amongst Lebanese urban adults were found to be higher than that reported from than Italy (8.2% in women and 7% in men), France (12.7% in women and 11.7% in men), England (15.5% in women and 24.8% in men), and other European

countries (Ng *et al*., 2014). However, inter-country comparison in obesity rates may be limited by the different age groups and dates of the surveys and the fact that, as mentioned earlier, the prevalence of obesity as assessed by the present study may not be representative of Lebanese adults as a whole.

In the present study, a significantly higher risk of obesity was found among those aged ≥ 40 years old (OR= 5.7; CI= 3.12 - 10.23) vs. younger adults. Comparable findings depicting the relationship between age and obesity were also observed in a national study conducted on Lebanese adults where women aged ≥ 50 years old were more likely to be obese compared to younger age groups (Chamieh *et al*., 2015). Parallel to our findings, a study conducted in Morocco on women aged 64 and men aged 45–54 years old (El Rhazi, Nejjari, Zidouh, Bakkali, Berraho & Gateau, 2011) also found a positive association between obesity and age. Similar findings were reported by other studies from Kuwait and Yemen (Al-Sharafi & Gunaid, 2014; Ahmed *et al*., 2012; El-Hazmi & Warsy, 2002). Data from large population studies conducted in the USA and in several European countries also showed that mean body weight and BMI gradually increase during adult life reaching peak values at 50 –59 year of age in both men and women (Flegal, Carroll, Kuczmarski & Johnson, 1998; Kuskowska-Wolk & Rössner, 1990; Mokdad, Bowman, Ford, Vinicor, Marks & Koplan, 2001). The reason behind this association lies within the considerable changes that occur in body composition as the individual age. With aging there is a greater relative increase in intra-abdominal fat, which may contribute to as state of IR (Villareal, Apovian, Kushner& Klein, 2005). Another plausible explanation is the decrease in EE due to loss of fat free mass and increase in sedentary behavior (El Rhazi *et al*., 2011).

A gender differential in the prevalence of obesity was observed in this study, with the prevalence of obesity being significantly higher in women compared to men

(OR=1.63; CI=1.11 – 2.40). Even though studies conducted in several developed countries including Europe and USA do not document gender disparities in the prevalence of adult obesity, our findings are similar to those reported from other Middle Eastern and North African countries (Aida Turrini& Ngo, 2009). A national cross sectional study conducted in Tunisia on more than 5000 subjects showed that BMI was higher among women (28.4%) vs. men (25.3%) as was obesity (37% versus 13.3%; OR= 3.8, CI= 3.1 – 7.4), and abdominal obesity (42.6% versus 15.6; OR= 4.0, CI= 3.3 – 4.8) (El Ati *et al*., 2012). Comparable findings were also reported from Morocco (Benjelloun, 2002; El Rhazi *et al*., 2011; Mokhtar *et al*., 2001), Algeria (Aida Turrini & Ngo, 2009) and Egypt (El-Zanaty & Way, 2009). It is well recognized that women have more body fat than men. Ultrasound measurements have shown that women are more prone to having larger amounts of fat due to an increase in adipose tissue thickness in certain regions (Sjöström, Smith, Krotkiewski & Björntorp, 1972). Moreover, in Iran, and perhaps indicative of other Middle Eastern and North African countries, sedentary Iranian females were more likely to consume non-nutritive snacks and more sweets than their male counterparts, leading to weight gain and obesity (Azizi, Azadbakht & Mirmiran, 2005; Hosseinpanah, Barzin, Eskandary, Mirmiran & Azizi, 2009). Another pathway proposed by Wang *et al*. suggests that women, due to their household roles, are more in charge of preparing meals, and thus of potentially consuming more food. This higher food intake is frequently coupled with a decrease in PA and an increase in sedentary behavior (Wang *et al*., 2004).

Findings of the present study suggest a positive association between obesity and low SES as assessed by education level and income. Similar to our findings, Chamieh *et al*. (2015) have reported lower odds of obesity in Lebanese adults with high SES (Chamieh *et al*., 2015). Even though several studies conducted in Europe, North

America, and few Middle Eastern countries have reported an increase in obesity risk with higher SES (Musaiger, 2011), our findings of an inverse association between obesity and SES are in line with findings reported from developed countries such as the USA (Sánchez-Vaznaugh, 2009) and Spain (García-Álvarez *et al*., 2007). Our findings are also in agreement with those reported from some developing countries and countries in nutrition transition such as Korea (Yoon, Oh & Park, 2006) and Iran (Hajian-Tilaki & Heidari, 2010). Monteiro *et al.* have argued that obesity in the developing world can no longer be considered solely a disease of groups with higher SES (Monteiro, Moura, Conde & Popkin, 2004). Strong evidence from population-based studies in industrial areas indicated that low SES is a risk factor for obesity (Haas, Lee, Kaplan, Sonneborn, Phillips& Liang, 2003; Organization, 1998; Stunkard, 1996). One possible explanation for the difference in SES-obesity relationship in developed and developing countries is the influence SES has on people's lifestyles such as diet and physical activity. Two plausible arguments may explain the link between low SES and obesity. The first focuses on the obeseogenic environments that low SES individuals usually live in which promotes energy dense foods and dissuades PA participation (Reidpath, Burns, Garrard, Mahoney& Townsend, 2002). The other argument focuses on the inability of low SES individuals to afford healthy foods (e.g. fruits and vegetables) that helps them maintain a normal body weight (Drewnowski & Specter, 2004). Additionally, a higher education level was also found to be associated with better nutrition knowledge *(*Klohe-Lehman *et al*., 2006).

In this study, smoking was found to decrease the risk of obesity in adults (OR=0.53; CI= 0.32 – 0.87), even after adjusting for confounding factors in the multivariate model. In line with our study, numerous cross- sectional studies indicate lower BMI in smokers compared to non-smokers (Flegal, Carroll, Ogden& Curtin,

2010; Huot, Paradis & Ledoux, 2004; Shimokata, Muller& Andres, 1989; Ward, Klesges& Vander Weg, 2001). For example, a national study conducted by Flegal *et al*. in the years 1988 to 1991 on U.S. adults (n= 5247), reported a positive association between smoking cessation and increased prevalence of overweight (Flegal, Troiano, Pamuk, Kuczmarski& Campbell, 1995). One theory behind this is the fact that nicotine acutely increases EE and reduces appetite which is why gain weight is frequently observed after smoking cessation (Hofstetter, Schutz, Jéquier& Wahren, 1986; Ward *etal*., 2001; Williamson, Madans, Anda, Kleinman, Giovino & Byers, 1991).

When looking at dietary intake data, there was no association of obesity with energy and macronutrient intake. This finding is in agreement with several other studies where no association was found between dietary intakes and obesity in adults (Hill & Davies, 2001; Schoeller, 1995; Trabulsi & Schoeller, 2001). One of the possible explanation is the underreporting that obese subjects tend to adopt in dietary assessment. The more respondents require or consume, the more difficult it is to report consumption accurately perhaps because of social pressure to consume less or remembering more foods or bigger portion sizes is challenging (Subar *et al*., 2003). It is important to note that the study's findings document a high intake of dietary fat (41.35%), which is in agreement with previous studies conducted in Lebanon (Chamieh *et al*., 2015).

The present study has also shown that almost half of the study population to have a low PA level (47.7%), which is in line with findings reported by a recent national study conducted amongst Lebanese adults (45.5%), using the same measurement tool (IPAQ) (Chamieh *et al*., 2015). Data on trends in PA in the MENA region is scarce. According to a review by Sibai *et al*.(2010), the prevalence of physical inactivity in the MENA region ranged between 21.6% and 86.8%, with Lebanon being

on the upper end of the range (68.7%) (Sibai, Nasreddine, Mokdad, Adra, Tabet & Hwalla, 2010). Physical inactivity has been associated with modernization and industrialization, which have led to a reduction in EE and an increase in sedentary behaviors, particularly in urbanized populations (Popkin, 1999). Physical inactivity may lead to several risk factors including IR, microvascular dysfunction, and is often significantly associated with weight gain, obesity, and MetS (Strasser, 2013; Zhu, St-Onge, Heshka & Heymsfield, 2004). Findings of the present study suggest a negative association between obesity and PA, whereby subjects engaging in high intensity PA had significantly lower odds of obesity (OR= 0.54; 95 % CI: 0.33 – 0.87). However, this association lost its significance after adjustment for other confounders in the multivariate model.

Studies investigating the relationship between short sleep duration, sleep patterns, and obesity are scarce in the Eastern Mediterranean region. This study is the first to explore the association of sleep duration and sleep patterns with body weight and the risk of obesity in the Lebanese adult population. Data from this study showed that more than half of the study population reported to sleeping less than the recommended 7 hours per day on weekdays (51.1%) and weekends (66.1%). Those who slept for less than 7 hours on weekdays were significantly more obese than those who reported to sleep more than 7 hours per day (OR= 1.75; CI= 1.008 – 3.04). The relationship between sleep duration and the odds of obesity remained significant after adjustment for potential confounders. These findings are in agreement with several other studies reporting an association with short sleep duration and BMI (Gangwisch, Malaspina, Boden-Albala& Heymsfield, 2005; Hasler *et al*., 2004; Vioque, Torres & Quiles, 2000). Results from a longitudinal study done on U.S. adults aged ≥ 20 years old (n = 13,742) (Ford, Li, Wheaton, Chapman, Perry & Croft, 2014) reported an inverse linear

relationship between sleep duration and adiposity, where 36.6% of those who slept for < 7 hours had higher abdominal obesity (OR= 1.10; CI= 1.03 – 1.16) (Ford *et al*., 2014). Other studies conducted in European countries like France, Germany, and Portugal also demonstrated the interrelationship between short sleep duration and weight gain in adults (Patel & Hu, 2008). A number of biological mechanisms have been proposed to link sleep duration and obesity (Taheri, 2006). For instance, it has been suggested that the number of hours of sleep is an important factor in regulating energy balance, appetite, and weight maintenance (Horne, 2008; Taheri, Lin, Austin, Young & Mignot, 2004).

This study further documented, in a cross sectional design, an association between OSA and obesity risk in adults. This association remained significant after adjusting for potential confounders in the multivariate model, with the odds of obesity being six times higher in subjects reporting OSA compared to those without OSA. When comparing the current prevalence of OSA in Lebanese urban adults to those reported from other countries, the prevalence of OSA in Lebanon was found to be higher than those reported from UAE (21%) and Jordan (16.8%), using the same measurement tool to assess OSA (Berlin Questionnaire). However, the prevalence of OSA amongst Lebanese adults appears to be lower than estimates reported from the USA (43.3%) and Europe (43.5%) (Netzer, Hoegel, Loube, Netzer, Hay, Alvarez-Sala & Strohl, 2003).

Since this study has a cross sectional design, the association between sleep apnea and obesity risk may be bidirectional. It has been shown that the connection between OSA and obesity involves a two – way relationship, one is the implications of OSA contributing to obesity and the other is the contribution of obesity to OSA (Hargens*et al*., 2013). Obesity may contribute to the development of OSA by

influencing the upper airway and altering its function and structure; causing negative effects on respiratory drive. Changes in cytokines (leptin, tumor necrosis factor, IL-6) due to obesity have a negative impact on neuromuscular control of the upper airway (Calvin, Albuquerque, Lopez-Jimenez& Somers, 2009). In addition, IR is also associated with OSA severity, independent of body weight, and may be linked with sleep deprivation (Caples & Somers, 2005). Location of fat deposition, especially in the neck and viscera, may also contribute to OSA susceptibility (Tuomilehto, Seppä & Uusitupa, 2013). The role of OSA in contributing to obesity is less established. As described by Ong *et al*., OSA changes sleep duration causing a reduction in EE and overall PA due to lethargy and daytime sleepiness, and an increase in the consumption of energy dense foods; thus increasing total caloric intake (Ong, O'Driscoll, Truby, Naughton & Hamilton, 2013).

The findings of this study must be considered in light of the following limitations. First, because of its cross sectional design, the present study does not allow for a causal inference regarding the observed associations. The second limitation of this study is that there may have been a selection bias on gender distribution (64.3% womenvs. 35.7% men) and on low SES individuals due to recruitment methods. The gender distributiondoes not seem to represent the national ratio of male to female population in Lebanon (MOH, 2009). However, although gender and low SES groups were overestimated because of selection bias, it is important to note that this should not have affected the associations as much as the prevalence, which was estimated at 41.5%. Moreover, even though the prevalence of obesity estimate may have been overestimated in the present study, available evidence suggests that obesity is on the rise in the Lebanese population (Chamieh et al., 2015). The third limitation is a differential bias related to the method we used to collect information regarding dietary

intakes. The FFQ relies on subject recall which raises concerns regarding the possibility of recall bias among participants who may have either under – or over – reported their dietary intake (Kushi, 1994). Under – reporting dietary intake and over – reporting PA have been observed in many studies, (Maddison, Mhurchu, Jiang, Vander Hoorn, Rodgers, Lawes& Rush, 2007; Mendez *et al*., 2011). However, it is worth mentioning that the FFQ was not self – completed but was conducted by research nutritionists who went through extensive training prior to data collection in order to minimize interviewer errors. Similarly, inter-observer measurement error in anthropometric assessment was minimized by extensive training and follow up to maintain quality of measurement among all research nutritionists.

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CHAPTER VI

CONCLUSION AND RECOMMENDATIONS

Obesity is a problem of growing concern worldwide, as it predisposes to a broad range of medical (such as HTN, DM, DL, IR, etc.) as well as psychosocial problems. The etiology of obesity is not fully understood but it is believed to be determined by complex interactions between genetic, environmental, and behavioral factors. Factors known to cause weight gain include but are not limited to: diet and physical inactivity, family history of obesity, genetics, education, SES, etc. (Zhang & Hu, 2012).More recently, sleep deprivation has been suggested to increase the risk of weight gain and to play a role in the current epidemic of obesity.

This cross-sectional study was conducted to assess the prevalence of obesity among Lebanese urban adults residing in the Greater Beirut area, to examine the association of obesity with socio-demographic, lifestyle, and dietary factors, and to investigate the association of sleep duration and patterns with obesity. In this population, our data revealed age (≥ 40 years old), low SES, low PA, short sleep duration and the presence of OSA to be significant predictors of obesity in our study sample.

Findings of this study showed a high prevalence of obesity among the study population (41.5%). This prevalence rate was higher than those reported in some MENA regions (Morocco, Tunisia, Algeria, Iran, Palestine, and Oman) and European countries (France, Italy, and England). Our findings, therefore, underline the importance of building culturally – appropriate, community-based interventions to help increase health awareness in Lebanon, especially among older adults (≥ 40 years) and low SES

individuals. It is crucial to understand the importance of a healthy balanced diet and PA in the prevention of lifestyle – related diseases, regardless of genetic susceptibility. Strategies to improve the environment within which individual behavioral decisions are made, such as those related to cost of healthy foods, food availability, and access to PA opportunities, must be considered. Those responsible in the public health sector, like policy makers and commissioners of health services, should tailor their resources and efforts to tackle obesity in the Lebanese adult population.

The present study also supports an association between suboptimal sleeping patterns with obesity. Sleep deprivation has been shown to contribute to obesity by influencing neuroendocrine hormones (leptin and ghrelin), thus affecting food intake and EE (Taheri *et al.*, 2004). Our data showed that almost half of the study population who reported to sleep less than the recommended 7 hours on weekdays (51.1%) and weekends (66.1%) were obese. Furthermore, the odds of obesity were found to be six times higher in subjects reporting OSA compared to those without OSA. Sleep appears to be one of the multifactorial factors that contribute to obesity. Because there are many aspects of sleep that appear to impact weight, and because the interactions between sleep and obesity are often bidirectional and complex, there is a need to better understand the association between the sleep and weight gain.

This study is the first to explore the association of sleep duration and sleep patterns with body weight and the risk of obesity in the Lebanese urban adult population, residing in the Greater Beirut area. However, further research is needed in Lebanon and Eastern Mediterranean countries to further confirm our findings. Longitudinal studies for monitoring sleep duration and energy balance is necessary to better understand the effect of adequate sleep in fighting against obesity. From a public health perspective, it is crucial to integrate sleep awareness among Lebanese adults as it

has recently been shown to play an important role in the obesity epidemic. There is little risk in encouraging healthy sleep and efforts to educate individuals regarding the deleterious effects of sleep deprivation on long – term health and well – being should be considered.

APPENDIX I

PARTICIPANT CONSENT FORM (ARABIC)

أسس الموافقة على الإشتراك في دراسة تتعلق بالأبحاث الجينية

تقييم مستويات ثنائي الفينول أ عند اللبنانيين وتقييم إرتباطه بالوضع الصحي لهم

رقم البروتوكول: IM.HT.03
الباحث: د. هاني تميم
العنوان: شارع القاهرة- بيروت – لبنان
تلفون : 01350000 ext: 5453
المكان الذي سوف تتم فيه الدراسة: المركز الطبي في الجامعة الأميركية في بيروت (AUBMC)

أنت مدعو(ة) للمشاركة ببحث علمي سريري سيجرى في الجامعة الأميركية في بيروت. الرجاء أن تأخذ(ي) الوقت الكافي لقراءة المعلومات التالية بتأن قبل أن تقرر(ي) إذا كنت تريد(ين) المشاركة أم لا. بإمكانك طلب إيضاحات أو معلومات إضافية عن أي شيء مذكور في هذه الإستمارة أو عن هذه الدراسة ككل.

إن الهدف من دراستنا هو قياس مستويات ثنائي الفينول أ (BPA) في عينة تمثل السكان اللبنانيين المقيمين في بيروت الكبرى، وتقييم إرتباط المستويات بمختلف الأمراض . كما نود أن نرى أيضا إذا كانت مستويات BPA تتغير مع مرور الوقت في كل شخص. ستتألف هذه الدراسة من مرحلتين، المرحلة الأولى عند بدء الدراسة والثانية بعد سنتين للمتابعة. سنقوم بتسجيل ما يقارب 500 مشارك في الدراسة التي ستتم في المركز الطبي في الجامعة الأميركية في بيروت (AUBMC) حيث سيتمّ حصر استخدام هذه الموافقة الموقّعة ومعها البيانات التي يتمّ جمعها لغايات هذه الدراسة من دون أي استخدام آخر.

ال BPA مادة كيميائية مصنّعة تتعارض مع الهرمونات الطبيعية في الجسم. ومن الممكن العثور عليها في زجاجات من البلاستيك وحاويات المياه والزجاجات وأكواب الأطفال، والحاويات البلاستيكية، والبطانة الداخلية لعلب الطعام والمشروبات. قد يتناول البشر ال BPA إذا انتقل من الحاوية البلاستيكية إلى الطعام أو الشراب في ظل ظروف معينة. ويرتبط استهلاك ال BPA بالآثار الصحية الضارة بما في ذلك أمراض القلب وارتفاع ضغط الدم، ومرض السكري، والتغيرات في الكولسترول، والدهون الثلاثية، ومستوى هرمونات الغدة الدرقية. من الممكن أن مادة ال BPA تؤثر أيضا على المواد الجينية (DNA).

سيقوم الباحثون الميدانيون أصحاب شهادة (CITI) العاملون في شركة "الدولية للمعلومات" (Information International) المتعاقد معها استخدام الطريقة المباشرة لتعيين المشاركين. وسوف يقومون بزيارة المشاركين في مكان إقامتهم لشرح أهداف الدراسة وطريقة التنفيذ. ثم تأخذ موافقة المشاركين وسيتم إعطاء تفاصيل عن تاريخ ووقت الدراسة. و سيتم تسجيل اسم المشارك وتاريخ الميلاد، و أيام الأسبوع المتوافر فيها للمشاركة ورقم هاتف لاتاحة المجال للمتابعة وتحديد التاريخ الدقيق لنقلهم الى المركز الطبي في الجامعة الأميركية في بيروت (AUBMC) وسوف تشمل كل زيارة 10 مشاركين سوف يقومون بالإجراءات المبينة أدناه.

إن مشاركتكم تعني أنّكم ستقابلون شخصاً مؤهّلاً يجري معكم دراسة تتضمّن العديد من الأسئلة حول الوضع الديمغرافي والاجتماعي والاقتصادي (العمر، والجنس، وموقع السكن، والتعليم، والمهنة والدخل)، ونمط الحياة (التدخين، الكحول، القهوة والنشاط البدني)، والحالة الصحية (التاريخ الطبي والأدوية)، والعادات الغذائية (الاستمارة الغذائية). وعلاوة على ذلك، سوف تخضعون لاختبار بدني لقياس الوزن والطول ومحيط الخصر وضغط الدم، ومعدل ضربات القلب. بالإضافة على ذلك سيتم فحص مستوى السكر بالدم بواسطة الاصبع، ويتضمن وخزة صغيرة واحدة في الاصبع لاخذ أقل من نقطة دم واحدة لإجراء الفحص. كما يطلب منكم الخضوع لسحب الدم الاختبارات الجينية المحددة (الحامض النووي) والفحوصات المخبرية (بما في ذلك مخزون السكر(HbA1c)، نسبة السكر الصباحي في الدم ، الكرياتينين، الدهون، هرمونات الغدة الدرقية (TSH)، خمائر الكبد (SGPT و GGT)، الانسولين، الكرياتينين البولية، الزلالي، فيتامين د (25 OH vit D), الكورتيزول، الليبتين، البرولاكتين، الببتيد C. وعلاوة على ذلك، سيتم جمع البول لقياس مستويات ال BPA. وسوف تنجز هذه الفحوصات المخبرية مجانا، ولكن في وقت لاحق أثناء الدراسة.

خلال زيارتك، من المتوقع أن تكون مدة الانتهاء من الإجراءات خلال اليوم الواحد حوالي ساعة ونصف فقط، مقسمة بين 30 دقيقة لسحب الدم وجمع البول, و 60 دقيقة لملء الاستمارات لكل مشارك .ومن المتوقع أن تستغرق الزيارة مدة أقصاها 3 ساعات، بالنظر إلى أن سيكون هناك مشاركين آخرين يمرون بنفس العملية.

بعد حوالي سنتين من الزيارة الأولى، سيتم الاتصال بكم هاتفياً لدعوتكم إلى استكمال الجزء الثاني من الدراسة وذلك من خلال زيارة المركز الطبي في الجامعة الأميركية في بيروت (AUBMC) والقيام بنفس الإجراءات التي قمتم بها في الزيارة الأولى.

على الرغم من أن أي دراسة قد تترافق مع مخاطر لا يمكن التنبؤ بها، هذه الدراسة تحمل الحد الأدنى من المخاطر. لا تحمل أي من عمليات جمع البيانات أية مخاطر على المدى الطويل، وسوف يتم سحب الدم ضمن ظروف وقاية صحية صارمة وحجم الدم الإجمالي المطلوب هو 20 سم مكعب. ومن الآثار الجانبية الضئيلة التي من المحتمل أن تصيبكم: ألم معتدل، نزف محدود، رضّة خفيفة في موضع إدخال الإبرة. وقد تحدث في بعض الأحيان حالات إغماء أو دوار خفيف، ولكنّها لا تدوم عادةً أكثر من دقائق قليلة.

ستقدم نتائج جميع الاختبارات التي أجريت مجاناً للمشاركين وذلك عبرالإتصال بهم وتزويدهم بنتائج الفحوصات المخبرية عند انتهائها. وعلاوة على ذلك، سيتم تعويض المشاركين عن نفقات التنقل بمبلغ 30,000 ليرة لبنانية عند وصولهم إلى المركز الطبي في الجامعة الأميركية في بيروت (AUBMC)، كما سيزود المشتركون بوجبة الفطور في ذات اليوم.

إذا وافقت على الإشتراك بهذا البحث سوف تبقى المعلومات سرية. وحدهم الأطباء ودائرة الأخلاقيات والمحققين في المؤسسات العامة يمكنهم الإطلاع على النتائج بناءً لأمر قانوني فقط.

سيتم تخزين كافة البيانات والعينات البيولوجية التي تم جمعها بطريقة سرية. وستتخذ جميع التدابير لضمان عدم حدوث أي خرق لخصوصية المشتركين. وعلاوة على ذلك، سيتم تخزين ما تبقى من عينات الدم والبول بشكل آمن إلى أجل غير مسمى في مختبر الدكتورة ناتالي زغيب خويري في المركز الطبي في الجامعة الأميركية في بيروت (AUBMC). إذا اخترتم سحب موافقتكم من الدراسة، سيتم تدمير العينات الخاصة بك.

بناءً على طلبكم، سوف نزودكم بنتائج الفحوصات الجينية وشرح أهميتها لكم. . سيتم الابقاء على سرية المعلومات.

أود أن أعرف ما إذا كنت على استعداد للمشاركة في هذه الدراسة. لديك الحق في قبول أو رفض المشاركة. في حال رفض المشاركة، لن يكون هنالك أي خسارة للمنافع التي يقدمها المركز الطبي التابع للجامعة الأميركية في بيروت (AUBMC). كما يحق لكم الانسحاب من هذه الدراسة في أي وقت من دون خسارة المنافع التي يقدمها المركز الطبي التابع للجامعة الأميركية في بيروت (AUBMC). أيضاً, يحق للباحث انهاء مشاركتك بهذه الدراسة.

أوافق على المشاركة في هذه الدراسة والإجراءات المحدّدة أعلاه.
نعم_____ لا_____

أوافق على أن يتم التواصل معي للدراسات المستقبلية
نعم_____ لا_____

أوافق على أن يتم التواصل معي إذا كانت نتائج الفحوصات الجينية ذات أهمية طبية
نعم_____ لا_____

استخدام ما تبقى من عينات الدم والبول للدراسات المستقبلية
نودّ تخزين ما تبقى من عينات الدم والبول لاستخدام محتمل في دراسات مستقبلية. للقيام بذلك، قد يكون هناك في المستقبل متعاونين في الجامعة الأميركية في بيروت، أو في المؤسسات الأخرى في لبنان و/أو خارج لبنان. لن يتمّ أي عمليات وخز إضافية. وسيتمّ "ترميز" عيّنات الدم المخزّنة. *(تشير عبارة "ترميز" إلى قابلية التعريف والتعقّب.لا يتمّ تعريف عينات الدم لغايات الأبحاث، ولكن يمكن ربطها بمصدرها عبر استخدام الرموز؛ إلاّ أنّ الباحث المسؤول أو المشرف الأساسي هو الوحيد الذي يحق له الحصول على اللائحة التي تحدّد الرمز الخاص بكل مريض).*

أوافق على أن يتم استخدام ما تبقى من عينات الدم والبول للدراسات المستقبلية
نعم_____ لا_____

يمكن مشاركة عينات دمكم المرمّزة مع باحثين آخرين لدراسات ذات صلة. ولن يعرف هؤلاء الباحثون هويتكم.

أوافق على مشاركة عيّنات دمي المرمّزة مع باحثين آخرين لإجراء دراسات ذات صلة.
نعم_____ لا_____

إقرار المريض بالمشاركة في البحث:

أنا الموقع أدناه وبعد أن اطلعت واستوعبت كل جوانب هذا البحث وأجبت عن كل أسئلتي أوافق بملئ إرادتي على المشاركة في هذه الدراسة وأنا على علم تام بأنني أستطيع الإتصال بالدكتور هاني تميم على الرقم 01350000 المقسم 5453 أو بأي من ممثليه الضالعين بهذه الدراسة وذلك إذا أردت توجيه أي سؤال، كما أنني أعلم أنه فيما لو أن أسئلتي لم يجاوب عليها بطريقة مقنعة يمكنني الإتصال بأحد أعضاء لجنة الأخلاقيات على المقسم 5445. كما إنني أعلم أنه يمكنني الإنسحاب من المشاركة في هذه الدراسة في أي وقت شئت حتى بعد التوقيع على هذه الوثيقة وإن العناية التي أتلقاها لن تتأثر بهذا الإنسحاب وإنني سوف أزود بنسخة عن هذه الوثيقة.

_____ _____
إسم المريض أو ممثله القانوني/قريبه أو وصيه — التوقيع

_____
التاريخ و الساعة

_____ _____
إسم الشاهد — التوقيع
التاريخ و الساعة

إقرار الباحث باستلام التعهد بالإشتراك:

لقد أطلعت بالتفصيل على التعهد بالإشتراك في البحث مع_____ (إسم المريض، ممثله القانوني، قريبه، وصيه)، وأفهمت المريض الغاية من هذه الدراسة ومن أخطارها وفوائدها. لقد أجبت المشترك على جميع الأسئلة التي تقدم بها بوضوح تام وتعهدت له بإعلامه عن أي تغيير يطرأ في موضوع هذا البحث.

_____ _____
التوقيع — إسم الباحث أو ممثل المشترك

_____
التاريخ و الساعة

Institutional Review Board
American University of Beirut
17 FEB 2014
APPROVED

APPENDIX II

PARTICIPANT CONSENT FORM (ENGLISH)

Consent to participate in a genetic research study

Assessment of BPA levels and their association with the health status among Lebanese population

Protocol number: IM.HT.03
Investigator: Dr. Hani Tamim
Address: American University Hospital
Hamra Street
Beirut, Lebanon
Phone: (01) 350 000 ext: 5453

Site where the study will be conducted: AUBMC

You are being asked to participate in a clinical research study conducted at the American University of Beirut. Please take time to read the following information carefully before you decide whether you want to take part in this study or not. Feel free to ask the representative of the contracted company if you need more information or clarification about what is stated in this form and the study as a whole.

The aim of our study is to measure Bisphenol A (BPA) levels in a representative sample from the Lebanese population residing in Greater Beirut, and to assess if it is related to different diseases. We also would like to see if BPA measures change over time in any person. This study will be composed of 2 stages; at baseline and a 2-year follow up. We will be recruiting approximately 500 subjects and study will be conducted at AUBMC whereby this informed consent along with the data collected will be used for this study only.

BPA is a synthetic chemical that interferes with the natural hormones in the body. It can be found in plastic bottles and water containers, baby bottles and toddler cups, plastic ware, the inner lining of food cans and beverages. Humans may ingest BPA if it leaches from the plastic container into the food or drink under certain conditions. Consumption is associated with adverse health effects including heart disease, high blood pressure, diabetes, changes in cholesterol, triglycerides, and thyroid levels. BPA can also affect the expression of DNA material, called 'epigenetic effect.

The CITI certified field workers employed by the contracted company (Information International) will use the direct approaching method to recruit the cohort. They will visit the respondents in their residence to explain the study aims and method of implementation. Then the respondents will be consented and given the details of the date and time of the study. The name, date of birth, availability on week days and telephone number of the potential participant will be recorded for further follow up to specify the exact date for taking them to AUBMC. Each visit will include 10 participants who will complete the procedures described below.

Participating in this study means that you will sit with a certified research assistant who will conduct a survey which includes multiple questions about the demographic and socioeconomic status (Age, gender, location, education, occupation, income), lifestyle (smoking, alcohol, coffee, physical activity),

health status (medical history and medication), and dietary habits (Food Frequency Questionnaire). Moreover, you will undergo a physical exam to measure weight, height, waist circumference, blood pressure, and heart rate. Moreover, your blood sugar will be checked by a fingerstick, which means a very small prick will be done to your finger to get less than a drop of blood to do the test. You will also be asked to have blood withdrawn for specific genetic testing (DNA methylation) and clinical laboratory tests (including HBA1c, fasting blood sugar, creatinine, lipid profile, TSH, SGPT, GGT, fasting insulin, urinary creatinine, microalbuminuria, 25 OH vit D, Cortisol, leptine, C-peptide, prolactin). Moreover, urine will be collected for measuring BPA levels. These tests will be done free of charge, but will be done at a later time during the study.

During your visit, the duration for completing the procedures is expected to be for around an hour and a half over one day only, divided between 30 minutes for blood withdrawal and urine collection and 60 minutes for filling the surveys for each participant. Your total visit time to AUBMC is expected to be for a maximum of 3 hours, given that there will be other participants undergoing the same process.

After around 2 years from the baseline visit, you will be contacted by phone to be invited to complete the second part of the study (2-year follow-up stage) by visiting the AUBMC and going through the same process as the one described at baseline.

Although any study may be associated with any unforeseeable risk, this proposal has minimal risk. None of the data collection measures bare any long term hazards, and all blood withdrawal will be done under sterile hygienic conditions and the total volume required is 20 cc. Possible side effects include mild pain, bleeding, bruising at the site of the needle insertion. Fainting or light-headedness can sometimes occur, but usually last only a few minutes.

The results of all tests conducted will be freely provided to the participants by calling them and providing them with the results of the test upon its completion. Moreover, the participants will be compensated for travel expenses with 30,000 LBP upon arriving to AUBMC. In addition, we will provide the participants with breakfast the same day.

If you agree to participate in this research study, the information will be kept confidential. Unless required by law, only the study doctor and designee, the ethics committee and inspectors from governmental agencies will have direct access to your information collected.

All data and biological samples collected will be stored in a confidential manner. These measures will all be conducted ensuring there is no breach of participants' privacy. Moreover, the remaining blood and urine samples will be stored securely indefinitely in Dr. Nathalie Zgheib Khoueiry's laboratory at the AUBMC. If you elect to withdraw your consent for the study, your samples will be destroyed.

You may ask that we provide you with the genetic results and explain their significance to you. The information will be kept confidential.

I would like to know if you would be willing to participate in this study. You have the right to accept or decline participation. Refusing to participate will not involve any loss of benefits offered in the future by AUBMC. Moreover, you are entitled to withdraw from the study at any time without any loss of benefits offered by AUBMC at any time.

I agree to participate in this study and the procedures explained above.

YES ..... NO.....

I agree to be contacted for future studies

YES ..... NO.....

I would like to be contacted if the genetic test results are significant

YES ..... NO.....

Using remaining blood and urine for other future studies

We would like to keep the remaining blood and urine samples for potential use in other future studies. To do so, there might be future collaborators at AUB, at other institutions in Lebanon and/or outside Lebanon. There will be no extra prick. The stored blood and urine samples will be coded (*"Coded" means identifiable, traceable. Blood* and urine *samples that are unidentified for research purposes but can be linked to their source through the use of codes; however, the principal investigators or VMP will be the only ones to have the list linking patients to the codes assigned.)*

I agree to permit the use of the remaining blood and urine sample for future studies

YES ..... NO.....

Your coded blood and urine samples may be shared with other investigators for related studies. These investigators will not know your identity.

I agree to have my coded blood and urine samples shared with other investigators for related studies.

YES ..... NO.....

APPENDIX III

ARABIC QUESTIONNAIRE

تقييم مستويات الBPA وارتباطها بالحالة الصحية بين السكان اللبنانيين

الاسم:	الحروف الاولي للإسم:	رقم المشارك:
رقم الهاتف:		التاريخ:

العوامل الديموغرافية:

تاريخ الميلاد:	الجنس: □ذكر □ أنثى
الحالة الاجتماعية: □ متزوج □ أعزب □ أرمل □ مطلق □ خاطب	

الاجتماعية والاقتصادية:

هل كنت مقيم خارج لبنان خلال العام الماضي : نعم كلا	
إذا كانت الإجابة بنعم، المكان _____ المدة _____	
مكان الإقامة_____	
طبيعة العمل_____	
ما هو دخلك في الأسرة	□ <600$ □ 600-999$ □ 1000-2000 $ □ >2000$ □ لا أعلم □ رفضت الإجابة
ما هو أعلى مستوى تعليمي أكملته؟	□ لم ألتحق بالمدرسة □ المرحلة الإبتدائية □ المرحلة المتوسطة □ المرحلة الثانوية □ دبلوم تعليم تقني/فنّي □ شهادة جامعية □ رفضت الإجابة
ما هو عدد الاشخاص الذين يسكنون في منزلكم (بما في ذلك الأقارب, أفراد العائلة أو الخدم الذين يسكنون معك بشكل جزئي)؟	
كم عدد الغرف في منزلكم (باستثناء المطبخ والحمامات والممرات والكراج والشرفات المفتوحة)؟	

نمط الحياة:

تاريخ التدخين			
السجائر	هل تدخن(ي) السجائر حاليا ؟	□ نعم □ كلا	إذا كان الجواب نعم، كم سيجارة في اليوم؟_____ منذ متى؟_____
	إذا لا، هل أنت مدخن(ة) سجائر سابق(ة)؟	□ نعم □ كلا	إذا كان الجواب نعم، متى توقّفت؟_____
النرجيلة / الشيشة	هل تدخن(ي) النرجيلة حاليا ؟	□ نعم □ كلا	إذا كان الجواب نعم، كم نرجيلة في اليوم؟_____ منذ متى؟_____
	إذا لا، هل أنت مدخن(ة) نرجيلة سابق(ة)؟	□ نعم □ كلا	إذا كان الجواب نعم، متى توقّفت؟_____
الكحول			
هل تشرب الكحول حاليا؟		□ نعم □ كلا	إذا كانت الإجابة نعم، حدد النوع_____ كم كوب في اليوم؟_____ منذ متى؟_____
هل كنت تشرب الكحول سابقا		□ نعم □ كلا	إذا كان الجواب نعم، متى توقّفت؟_____
القهوة			
هل تشرب القهوة حاليا؟		□ نعم □ كلا	إذا كان الجواب نعم، كم كوب في اليوم؟_____
النشاط البدني			
خلال السبعة أيام الماضية، كم مرة مارست الأنشطة البدنية القوية مثل رفع الأوزان الثقيلة، والتمارين الرياضية، أو ركوب الدراجات بسرعة لفترة لا تقل عن 10 دقائق أو أي نشاط يتطلب الجهد البدني الشاق ويسبب صعوبة بالتنفّس)؟			_____ أيام في الأسبوع □ لم أمارس الأنشطة البدنية القوية كم من الوقت قضيت على المعدل لممارسة الأنشطة البدنية القوية؟ _____ساعات _____ دقائق؟ خلال ال 3 أشهر الماضية، كم عدد الأسابيع التي مارست بها الأنشطة البدنية القوية؟ _____ أسابيع
خلال السبعة أيام الماضية ، كم مرة مارست الأنشطة البدنية المعتدلة مثل رفع الأوزان الخفيفة، أو ركوب الدراجات، أو ممارس رياضة التنس أو أي نشاط يتطلب الجهد البدني المعتدل ويسبب صعوبة خفيفة بالتنفّس (لا تشمل المشي) ؟			_____ أيام في الأسبوع □ لم أمارس الأنشطة البدنية المعتدلة كم من الوقت قضيت على المعدل لممارسة الأنشطة البدنية المعتدلة ؟ _____ساعات _____ دقائق؟ خلال ال 3 أشهر الماضية، كم عدد الأسابيع التي مارست بها الأنشطة البدنية المعتدلة ؟ _____ أسابيع
خلال السبعة أيام الماضية ، كم مرة مارست رياضة المشي لفترة لا تقل عن 10 دقائق؟ و هذا يشمل المشي في المنزل و مكان العمل و المشي للتنقل اليومي أو الرياضة أو المتعة			_____ أيام في الأسبوع □ لم أمارس الأنشطة البدنية المعتدلة كم من الوقت قضيت على المعدل لممارسة رياضة المشي؟ _____ساعات _____ دقائق؟ خلال ال 3 أشهر الماضية، كم عدد الأسابيع التي مارست رياضة المشي؟ _____ أسابيع
خلال السبعة أيام الماضية ، ما هي الفترة الزمنية التي أمضيتها جالسا؟ و هذا يشمل الجلوس وراء مكتب أو خلال زيارة الأصدقاء أو الجلوس للقراءة أو مشاهدة التلفاز أو السفر على متن حافلة			_____ساعات _____ دقائق؟ خلال ال 3 أشهر الماضية، كم عدد الأسابيع التي اتبعت فيها هذا الكم من الوقت جالسا ؟ _____ أسابيع

التّاريخ الطبي:

مرض الشريان التاجي:

هل لديك أي من أفراد الأسرة الذين تم تشخيصهم بمرض الشريان التاجي أو ماتوا فجأة؟	□ نعم □ كلا إذا كان الجواب نعم حدد من:_____	في أي سن: _____
هل قيل لكم من قبل طبيب أنكم أصبتم بنوبة قلبية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل خضعت لعملية تمبيل (قسطرة) شرايين القلب؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل تم وضع رصور (الدعامة)؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل خضعت لعملية جراحية لتغيير شرايين القلب؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____

ارتفاع ضغط الدم:

هل قيل لكم من قبل طبيب أو أحد العاملين في مجال الرعاية الصحية أن لديكم ارتفاع ضغط الدم؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل خضعت لقياس ضغط الدم من قبل الطبيب أو أحد مقدمي الرعاية الصحية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى: حدد النتيجة:_____
هل تخضعون لأي علاج لارتفاع ضغط الدم؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد النوع: □ تعديل نمط الحياة □ الأدوية:_____

داء السكري:

هل قيل لكم من قبل طبيب أو أحد العاملين في مجال الرعاية الصحية أنكم تعانون من ارتفاع نسبة السكر في الدم أو من مرض السكري؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل خضعت لقياس نسبة السكر في الدم من قبل طبيب أو العاملين في مجال الرعاية الصحية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____ حدد النتيجة:_____
هل تخضعون لأي علاج لارتفاع السكر في الدم أو لمرض السكري؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد النوع: □ تعديل نمط الحياة □ الأدوية:_____

ارتفاع مستوى الدهون في الدم:

هل قيل لكم من قبل طبيب أو أحد العاملين في مجال الرعاية الصحية أنكم تعانون من ارتفاع نسبة الكولسترول أو الدهون الثلاثية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل خضعت لقياس الكولسترول من قبل طبيب أو العاملين في مجال الرعاية الصحية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____ حدد النتيجة:_____
هل تخضعون لأي علاج لارتفاع ارتفاع مستوى الدهون في الدم؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد النوع: □ تعديل نمط الحياة □ الأدوية:_____

مرض الغدة الدرقية:

هل قيل لكم من قبل طبيب أو أحد العاملين في مجال الرعاية الصحية أنكم تعانون من مرض الغدة الدرقية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____ حدد طبيعة المرض:_____
هل خضعت لقياس هرمونات الغدة الدرقية من قبل طبيب أو العاملين في مجال الرعاية الصحية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____ حدد النتيجة:_____
هل تخضعون لأي علاج لمرض الغدة الدرقية؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد النوع: □ تعديل نمط الحياة □ الأدوية:_____
هل لديك أي من أفراد الأسرة الذين تم تشخيصهم بمرض الغدة الدرقية؟ (أب، أم، اخ، أخت، جد، جدة)	□ نعم □ كلا	إذا كان الجواب نعم حدد من:_____

تاريخ أمراض السرطان:

هل قيل لكم من قبل طبيب أو أحد العاملين في مجال الرعاية الصحية أنكم تعانون من مرض السرطان ؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____ حدد طبيعة المرض:_____
هل تخضعون لعلاج كيمياني أو أي علاج آخر لمرض السرطان؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد النوع: _____
هل لديك أي من أفراد الأسرة الذين تم تشخيصهم بمرض السرطان؟ (أب، أم، اخ، أخت، جد، جدة)	□ نعم □ كلا	إذا كان الجواب نعم حدد من:_____ حدد طبيعة المرض:_____

تاريخ الكسور:

هل عانيت من أيّ كسر في العظم؟	□ نعم □ كلا
إذا كان الجواب نعم	حدد أين:_____ العمر عند حصول الكسر:_____ كيف تم الكسر؟ (الوقوع من إرتفاع، حادث سير) _____

أمراض أخرى:

هل قيل لكم من قبل طبيب أو أحد مقدمي الرعاية الصحية أن لديك أي من التالي:		
السكتة الدماغية	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
التهاب المفاصل	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
التهاب الشعب الهوائية المزمن أو انتفاخ الرئة	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
أمراض الكبد	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____

هل تعاني من أمراض أخرى؟

زيارة طبيب الأسنان:

هل قمت بزيارة طبيب الأسنان في العام الماضي؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____
هل تم وضع الحشوات في العام الماضي ؟	□ نعم □ كلا	إذا كان الجواب نعم، حدد متى:_____

الأدوية: (إذا لم تتوفر الأدوية الرجاء الاتصال بالمشترك)

الاسم (العلامة التجارية و الاسم العام)	الجرعة	تاريخ بدأ الاستعمال

مراجعة عامة:

هل شعرت بتغيير في الوزن خلال ال 3 أشهر الماضية؟		□ الوزن مستقر □ وزن مفقود:_____كلغ □ وزن مكتسب:_____ كلغ
للنساء فقط	متى كانت آخر دورة شهرية؟_____	
	هل أنت في مرحلة: □ قبل انقطاع الطمث □ بعد انقطاع الطمث	إذا في مرحلة قبل انقطاع الطمث: □ الدورة الشهرية منتظمة □ الدورة الشهرية غير منتظمة
هل تعانين من:	□ حب الشباب □ الشعرانية	

عادة النوم

1 - كم ساعة تنام في الليل في أيام الأسبوع؟

4 ساعات أو أقل		5 إلى 6 ساعات		6 إلى 7 ساعات		7 إلى 8 ساعات		8 إلى 9 ساعات		9 ساعات أو أكثر

2 - كم ساعة تنام في الليل في أيام عطلة نهاية الأسبوع ؟

4 ساعات أو أقل		5 إلى 6 ساعات		6 إلى 7 ساعات		7 إلى 8 ساعات		8 إلى 9 ساعات		9 ساعات أو أكثر

3 - هل تشعر أنك لا تحصل على قسط كاف من النوم؟

أبدا		نادرا (يوم واحد في الشهر)		أحيانا (2-4 أيام في الشهر)		كثيرا (5-15 يوم في الشهر)		تقريبا دائما (16-30 يوم في الشهر)	

4 – هل تواجه او صعوبة خلودك عند مصاعب للنوم ؟

أبدا		نادرا (يوم واحد في الشهر)		أحيانا (2-4 أيام في الشهر)		كثيرا (5-15 يوم في الشهر)		تقريبا دائما (16-30 يوم في الشهر)	

5 - هل تستيقظ خلال الليل وتجد صعوبة في العودة إلى النوم؟

أبدا		نادرا (يوم واحد في الشهر)		أحيانا (2-4 أيام في الشهر)		كثيرا (5-15 يوم في الشهر)		تقريبا دائما (16-30 يوم في الشهر)	

6 - هل تستيقظ في الصباح الباكر جدا وتكون غير قادر على متابعة النوم؟

أبدا		نادرا (يوم واحد في الشهر)		أحيانا (2-4 أيام في الشهر)		كثيرا (5-15 يوم في الشهر)		تقريبا دائما (16-30 يوم في الشهر)	

7 - هل قال لك الطبيب أن لديك حالة توقف التنفس أثناء النوم؟

		لا أعرف		لا		نعم	

8 -هل تشخر؟

		لا أعرف		لا		نعم	

9 -إذا كنت تشخر, كيف يمكن أن تصف ارتفاع صوت شخيرك؟

د .مرتفع جدا يمكن سماعه من الغرف المجاورة		ج .أعلى من الكلام		ب .بنفس درجة ارتفاع الكلام		أ .أعلى بقليل من صوت التنفس	

10 - إذا كنت تشخر, كم مرة يتكرر شخيرك؟

ه .لا يحدث		د .مرة إلى مرتين بالشهر		ج .مرة إلى مرتين بالأسبوع		ب. 3- 4 مرات بالأسبوع		أ .تقريبا كل يوم	

11 - إذا كنت تشخر, هل سبق وأن سبب شخيرك الإزعاج للآخرين؟

		لا أعرف		لا		نعم	

12 - هل لا حظ أي شخص أنك توقف التنفس أثناء النوم؟

ه .لا يحدث		د .مرة إلى مرتين بالشهر		ج .مرة إلى مرتين بالأسبوع		ب. 3- 4 مرات بالأسبوع		أ .تقريبا كل يوم	

13 - كم مرة تشعر بالتعب أو الإرهاق عند الاستيقاظ من النوم؟

ه .لا يحدث		د .مرة إلى مرتين بالشهر		ج .مرة إلى مرتين بالأسبوع		ب. 3- 4 مرات بالأسبوع		أ .تقريبا كل يوم	

14 - هل تحس بالتعب أو الإرهاق أثناء ساعات اليقظة؟

ه .لا يحدث		د .مرة إلى مرتين بالشهر		ج .مرة إلى مرتين بالأسبوع		ب. 3- 4 مرات بالأسبوع		أ .تقريبا كل يوم	

15 - هل سبق أن نعست أو نمت خلال قيادة السيارة أو الانتظار؟

				لا		نعم	

16 - إذا كانت الإجابة نعم، كم مرة يحدث هذا؟

ه .لا يحدث		د .مرة إلى مرتين بالشهر		ج .مرة إلى مرتين بالأسبوع		ب. 3- 4 مرات بالأسبوع		أ .تقريبا كل يوم	

إستفتاء حول وتيرة إستهلاك الطعام

الاسم:	الحروف الاولى للإسم:	رقم المشارك:

إستبيان وتيرة إستهلاك الطعام. يرجى منك التفكير بالنمط الغذائي الخاص بك الذي اتّبعته خلال **العام السابق**. الرجاء تحديد الكمية المتناولة عادةً **في اليوم** أو **الأسبوع** أو **الشهر** لكل من المواد الغذائية التالية

Code	الطعام	مرجع حجم الحصّة	حجم الحصّة	في اليوم	في الأسبوع	في الشهر	نادراً/أبداً
	مثال: أرز، أبيض، مطبوخ	Side A/ Page 5	1/2 A 11		3		
	جبن (غني بالدسم/صفراء)	حصة واحدة = مثلث/مربع	3 B1, thick 2	1			
	بقول: عدس، فاصولياء، حمّص، الخ، مطبوخة	Side A or B Side A/Page 4	1.5 cups		1		
1	**الخبز والحبوب**						
1.1	خبز أبيض	رغيف خبز عربي كبير/					
		رغيف خبز عربي وسط/					
		خبز فرنجي (baguette)					
		توست وسط					
1.2	خبز أسمر أو مصنوع من القمح الكامل	رغيف خبز عربي كبير/					
		رغيف خبز عربي وسط/					
		خبز فرنجي (baguette)					
		توست وسط					
1.3	تنور / مرقوق	رغيف					
1.4	حبوب الفطور، عادي/نخالة/ سكر	Side A					
		(علبة صغيرة 35g)					
1.5	منتوجات الكعك	Finger size: small/long Round kaak: small/medium Page 13					
1.6	أرز، أبيض، مطبوخ	Side A/ Page 5					
1.7	معكرونة، سادة، مطبوخة	Side A/ Page 5					
1.8	قمح، كامل، مطبوخ/برغل	Side A/ Page 5					
1.9	أرز/ معكرونة مصنوع من القمح الكامل	Side A/ Page 5					
2	**مشتقّات الحليب**						
2.1	حليب قليل الدسم (٢ % دهون)	Side A					
2.2	حليب كامل الدسم	Side A					
2.3	لبن قليل الدسم /خالٍ من الدسم	Side A					
		عيران					
2.4	لبن كامل الدسم	Side A					

Code	الطعام	مرجع حجم الحصّة	حجم الحصّة	في اليوم	في الأسبوع	في الشهر	نادراً/أبداً
		عيران					
2.5	جبن (غني بالدسم/صفراء)	حصة واحدة = مثلث/مربع Side A or B					
2.6	جبن (قليل الدسم/لايت/بيضاء)	حصة واحدة = مثلث/مربع Side A or B					
2.7	لبنه، عادي	Side A					
2.8	لبنه، لايت/ خالية الدسم	Side A					
3	**الفاكهة والعصائر**						
3.1	الحمضيّات: برتقال ,غريفون	حبّة واحدة وسط / Side A					
3.2	فاكهة ذات اللون الأصفر أو البرتقالي الداكن (درّاق، خوخ، الخ)	حبّة واحدة وسط / Side A					
3.3	فراولة	10 فراولة/ Side A					
3.4	عنب	10 عنب/ Side A					
3.5	فاكهة أخرى: موز، / تفاح، طازج	حبّة واحدة وسط					
3.6	فاكهة مجفّفة: زبيب ، تمر مشمش	زبيب (1 ملعقة طعام)، تمر/ مشمش (حبّة واحدة)					
3.7	عصير فاكهة طازج	Side A					
3.8	مشروبات بطعم الفاكهة: تنكة/بلاستيك	Side A/تنكة					
3.9	مشروبات بطعم الفاكهة: ممعبّاة في زجاجات /كرتونة	كرتونة / زجاجة صغيرة Side A					
3.10	فاكهة معلّبة	Peach/ apricot = ½ fruit, Pineapple = 1 slice					
4	**الخضار**						
4.1	سلطة خضراء: خس، فلفل أخضر، خيار،نعنع..	Side A/ Page 8					
4.2	خضار ذات اللون الأخضر أو الأصفر الداكن (سبانخ، هندبة، ملوخيّة، جزر...)	Side A/ Page 4					
4.3	بندورة، طازجة، حجم وسط	10 cherry/ حبّة واحدة					
4.4	ذرة / بازلاء خضراء، مطبوخة	Side A/ Page 4					
4.5	ذرة / بازلاء خضراء، معلّبة	Side A/ Page 4					
4.6	بطاطا مشويّة/ مسلوقة/ مهروسة	Side A/ حبّة واحدة					
4.7	قرع، كوسى، باذنجان/ مطبوخ	Side A/ 1 med. stuffed					
4.8	قرنبيط/ ملفوف/ بروكولي	Side A/ Page 4					
4.9	خضار أخرى معلّبة (بالميتو- فطر-هليون)	Side A/ Page 4					
4.10	عصير خضار طازج: بندورة/ جزر	Side A					
5	**اللحوم وبدائلها**						
5.1	بقول: عدس، فاصوليا، حمّص..مطبوخة/ غير معلبة	Side A/ Page 4					
5.2	بقول معلّبة (فول/ فاصوليا ...) تنك-زجاج	Side A/ Page4					

Code	الطعام	مرجع حجم الحصّة	حجم الحصّة	في اليوم	في الأسبوع	في الشهر	نادراً/أبداً
		عيران					
2.5	جبن (غني بالدسم/صفراء)	حصة واحدة = مثلث/مربع Side A or B					
2.6	جبن (قليل الدسم/لايت/بيضاء)	حصة واحدة = مثلث/مربع Side A or B					
2.7	لبنه، عادي	Side A					
2.8	لبنه، لايت/ خالية الدسم	Side A					
3	**الفاكهة والعصائر**						
3.1	الحمضيّات: برتقال ,غريفون	حبّة واحدة وسط / Side A					
3.2	فاكهة ذات اللون الأصفر أو البرتقالي الداكن (درّاق، خوخ، الخ)	حبّة واحدة وسط / Side A					
3.3	فراولة	10 فراولة/ Side A					
3.4	عنب	10 عنب/ Side A					
3.5	فاكهة أخرى: موز، / تفاح، طازج	حبّة واحدة وسط					
3.6	فاكهة مجفّفة: زبيب ، تمر مشمش	زبيب (1 ملعقة طعام)، تمر/ مشمش (حبّة واحدة)					
3.7	عصير فاكهة طازج	Side A					
3.8	مشروبات بطعم الفاكهة: تنكة/بلاستيك	تنكة/Side A					
3.9	مشروبات بطعم الفاكهة: ممعبّأة في زجاجات /كرتونة	كرتونة / زجاجة صغيرة Side A					
3.10	فاكهة معلّبة	Peach/ apricot = ½ fruit, Pineapple = 1 slice					
4	**الخضار**						
4.1	سلطة خضراء: خس، فلفل أخضر، خيار،نعنع..	Side A/ Page 8					
4.2	خضار ذات اللون الأخضر أو الأصفر الداكن (سبانخ، هندبة، ملوخيّة، جزر...)	Side A/ Page 4					
4.3	بندورة، طازجة، حجم وسط	حبّة واحدة/ 10 cherry					
4.4	ذرة / بازلاء خضراء، مطبوخة	Side A/ Page 4					
4.5	ذرة / بازلاء خضراء، معلّبة	Side A/ Page 4					
4.6	بطاطا مشويّة/ مسلوقة/ مهروسة	حبّة واحدة/ Side A					
4.7	قرع، كوسى، باذنجان/ مطبوخ	Side A/ 1 med. stuffed					
4.8	قرنبيط/ ملفوف/ بروكولي	Side A/ Page 4					
4.9	خضار أخرى معلّبة (بالميتو- فطر-هليون)	Side A/ Page 4					
4.10	عصير خضار طازج: بندورة/ جزر	Side A					
5	**اللحوم وبدائلها**						
5.1	بقول: عدس، فاصوليا، حمّص..مطبوخة/ غير معلبة	Side A/ Page 4					
5.2	بقول معلّبة (فول/ فاصوليا ...) تنك-زجاج	Side A/ Page4					

APPENDIX IV

ENGLISH QUESTIONNAIRE

Assessment of BPA levels and their association with the health status among Lebanese population

Name:	**Initials:**	**Study ID number:**
Tel number:		**Date:**

Demographic Factors:

Date of birth:	**Gender:** □ Males □ Females
Marital status: □ Married □ Single □ Widow □ Divorced □ Engaged	

Socioeconomic:

Have you lived outside Lebanon for the past year: □ No □ Yes	
If yes, where_____ and for how long _____	
Which area do you live?	
What do you work?	
What is your income per family:	□ <600$ □ 600- 999.9$ □ 1000-2000$ □ >2000$ □ I don't know/ Not sure □ I prefer not to answer
What is your highest level of education?	□ No schooling □ Primary school □ Intermediate school □ Secondary school □ Technical diploma □ University degree □ I prefer not to answer
What is the total number of individuals living in your house? (Including relatives, family members and maids that frequently live with you on a semi-permanent basis)	
How many rooms are there in your house? (Excluding kitchens, bathrooms, hallways, balconies, and garage)	

Lifestyle:

Smoking history			
Cigarette	Do you currently smoke cigarettes?	□ No □ Yes	If yes, how many cigarettes/day? Since when?
	If no, are you a previous cigarette smoker?	□ No □ Yes	If yes, when did you stop?
Narghileh	Do you currently smoke narghileh?	□ No □ Yes	If yes, how many narghileh/day? Since when?
	If no, are you a previous narghileh smoker?	□ No □ Yes	If yes, when did you stop?
Alcohol			
Do you currently drink alcohol?		□ No □ Yes	
		If yes specify type? Since when?	How many glasses/week?
Previous drinker?		□ No □ Yes	If yes, when did you stop?
Coffee			
Do you currently drink coffee?		□ No □ Yes If yes how many cups/day?	
Physical activity			
During the last 7 days, on how many days did you do **vigorous** physical activities like heavy lifting, aerobics, or fast bicycling for at least 10 minutes (or any activity that take hard physical effort and make you breathe harder than normal)?		---------- days/week □ None - How much time in total did you usually spend on one of those days doing vigorous physical activities? _____ hours _____ minutes? - How many weeks did you spend doing vigorous physical activities during the last 3 months? ----------weeks	
During the last 7 days, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or tennis or any activity that take hard physical effort and make you breath harder than normal)? Do not include walking.		---------- days/week □ None - How much time in total did you usually spend on one of those days doing moderate physical activities? _____ hours _____ minutes? -How many weeks did you spend doing moderate physical activities during the last 3 months? ----------weeks	
During the last 7 days, on how many days did you **walk** for at least 10 minutes at a time? This includes walking at work and at home, walking to travel from place to place, and any other walking that you did solely for sport, exercise or leisure.		---------- days/week □None - How much time in total did you usually spend walking on one of those days? _____ hours _____ minutes? -How many weeks did you spend walking during the last 3 months? ----------weeks	
During the last 7 days, how much time in total did you usually spend sitting on a week day? This includes time spent sitting at a desk, visiting friends, reading traveling on a bus or sitting or lying down to watch television.		_____ hours _____ minutes? -How many weeks have you been spending the same time in terms of sitting during the last 3 months? ----------weeks	

Medical History:

Coronary artery disease:

Do you have any family member who has been diagnosed with coronary artery disease or died suddenly?	□ No □ Yes If yes: specify who	At what age:
Have you been told by a doctor that you had a heart attack?	□ No □ Yes If yes when:	
Did you undergo cardiac catheterization?	□ No □ Yes If yes when:	
Was a stent placed?	□ No □ Yes If yes when:	
Did you have coronary heart bypass surgery?	□ No □ Yes If yes when:	

Hypertension:

Have you been told by a doctor or a health care worker that you have high blood pressure?	□ No □ Yes If yes when:
Have you had your blood pressure measured by a doctor or a health care worker?	□ No □ Yes If yes when? What was it?
Are you taking any treatment for high blood pressure?	□ No □ Yes If yes specify: □ Life style modifications □ Drugs:

Diabetes Mellitus:

Have you been told by a doctor or a health care worker that you have raised blood sugar or diabetes?	□ No □ Yes If yes when:
Have you had your blood sugar measured by a doctor or a health care worker?	□ No □ Yes If yes when? What was it?
Are you taking any treatment for high blood sugar or diabetes?	□ No □ Yes If yes specify: □ Life style modifications □ Drugs:

Dyslipidemia:

Have you been told by a doctor or a health care worker that you have raised cholesterol or triglycerides?	□ No □ Yes If yes when:
Have you had your cholesterol measured by a doctor or a health care worker?	□ No □ Yes If yes when? What was it?
Are you taking any treatment for dyslipidemia?	□ No □ Yes If yes specify: □ Life style modifications □ Drugs:

Thyroid disease:

Have you ever been told by a doctor or a health care worker that you have thyroid disease?	□ No □ Yes If yes when? What was the disease?
Have you had your thyroid hormones measured by a doctor or a health care worker?	□ No □ Yes If yes when? What was it?
Are you taking any thyroid drug?	□ No □ Yes If yes specify:
Do you have any family history of thyroid disease? (Parents, siblings and grandparents)	□ No □ Yes If yes specify who:

Cancer history:

Have you ever been told by a doctor or a health care worker that you have cancer?	□ No □ Yes If yes when? What was the disease?
Are you taking any chemotherapy or other drug for cancer?	□ No □ Yes If yes specify
Do you have any family history of cancer? (Parents, siblings and grandparents)	□ No □ Yes If yes specify the disease: Specify who:

Fracture history:

Did you ever sustain a fracture?	□No □Yes
If yes:	Where? Age at onset? How did it happen? (fall from height, accident…)?

Other diseases:

Have you been told by a doctor or a health care worker that you have any?	
Stroke?	□No □Yes If yes when:
Arthritis?	□No □Yes If yes when:
Chronic bronchitis or emphysema?	□No □Yes If yes when:
Liver disease?	□No □Yes If yes when:

Do you have any other illnesses?

Dentist visits:

Have you visited any dentist in the past year?	□ No □ Yes If yes when:
Did you have any fillings done in the past year?	□ No □ Yes If yes when:

Medications (if not brought, call the participant later)

Name (brand and generic)	Dose	Date started

Review of system:

Do you have any weight changes during the last 3 months?		□ Stable weight □ Lost weight How many Kgs? □ Gained weight How many Kgs?
For women:	When was your last menstrual period?	
	Are you□ premenopausal □ postmenopausal	If premenopausal do you have □ Regular mences □ Irregular mences
	Do you have? □ Acne □ Hirsutism	

Sleep Habits and Berlin questionnaires

1- How many hours do you sleep per night on weekdays?					
4 hrs or less	5 to 6 hrs	6 to 7 hrs	7 to 8 hrs	8 to 9 hrs	9 hrs or more
2- How many hours do you sleep per night on weekends?					
4 hrs or less	5 to 6 hrs	6 to 7 hrs	7 to 8 hrs	8 to 9 hrs	9 hrs or more
3- Do you feel that you are not getting enough sleep?					
Never	Rarely (1 / month)	Sometimes (2-4 / month)	Frequently (5-15 /month)	Almost Always (16-30 / month)	
4- Do you have Trouble falling asleep?					
Never	Rarely (1 / month)	Sometimes (2-4 / month)	Frequently (5-15 /month)	Almost Always (16-30 / month)	
5- Do you wake up during the night and have difficulty resuming sleep?					
Never	Rarely (1 / month)	Sometimes (2-4 / month)	Frequently (5-15 /month)	Almost Always (16-30 / month)	
6- Do you wake up too early in the morning and be unable to resume sleep?					
Never	Rarely (1 / month)	Sometimes (2-4 / month)	Frequently (5-15 /month)	Almost Always (16-30 / month)	
7- Did your doctor tell you that you have sleep apnea?					
Yes	No				
8- Do you snore?					
Yes	No	Don't Know			
9- If you snore, your snoring is?					
a. Slightly louder than breathing	b. As loud as talking	c. Louder than talking	d. Very loud-can be heard in adjacent rooms		
10- If you snore, how often do you snore?					
a. Nearly every day	b. 3-4 times a week	c. 1-2 times a week	d. 1-2 times a month	e. Never or nearly never	
11- If you snore, has your snoring ever bothered other people?					
Yes	No	Don't Know			
12- Has anyone noticed that you quit breathing during sleep?					
a. Nearly every day	b. 3-4 times a week	c. 1-2 times a week	d. 1-2 times a month	e. Never or nearly never	
13- How often do you feel tired or fatigued after you sleep?					
a. Nearly every day	b. 3-4 times a week	c. 1-2 times a week	d. 1-2 times a month	e. Never or nearly never	
14- During your waking time do you feel tired, fatigued or not up to par?					
a. Nearly every day	b. 3-4 times a week	c. 1-2 times a week	d. 1-2 times a month	e. Never or nearly never	
15-Have you ever nodded off or fallen asleep while driving a vehicle?					
Yes	No				
16- If yes, how often does this occur?					
a. Nearly every day	b. 3-4 times a week	c. 1-2 times a week	d. 1-2 times a month	e. Never or nearly never	

FOOD FREQUENCY QUESTIONNAIRE

Name of the participant: ---------- Initials:---------- Study ID number: ----------

*Please think about your eating patterns during **the past year**. Please indicate your usual intake of each of the following food items per day, week, or month. Please be as precise as you can in your recall.*

Code	Food item	Reference Portion	Serving Size	Day	Week	Month	Rarely/Never
	Examples:						
	Rice, white, cooked	*A side*	*½ A 11*		*3*		
	Cheese, regular	*B side/ Thickness*	*B1 / Th. 2*	*4*			
	Legumes, canned (beans, peas)	*Side A/ Page 4*	*1.5 cups*		*2*		
1	**Bread and Cereals**						
1.1	Bread, white	1 large Arabic loaf					
		1 medium Arabic loaf					
		1 French baguette					
		1 pain de mie/ toast					
1.2	Bread, brown	1 large Arabic loaf					
		1 medium Arabic loaf					
		1 French baguette					
		1 pain de mie/ toast					
1.3	Traditional breads(markouk/tannour)	1 loaf					
1.4	Breakfast cereals, regular/ sugar coated/ chocolate/ bran	Side A					
		Carton (35 g)					
1.5	Kaak	Finger size Small round / Page 13					
1.6	Rice, white, cooked	Side A/ Page 5					
1.7	Pasta/ Noodles, plain, cooked	Side A/ Page 5					
1.8	Wheat/ Bulgur, cooked	Side A/ Page 5					
1.9	Rice/Pasta/Cereals, whole grain	Side A / Page 5					
2	**Dairy Products**						
2.1	Milk, skim/low-fat (0-2%)	Side A					
2.2	Milk, whole-fat	Side A					
2.3	Yogurt, fat-free/low-fat	Side A					
		Bottled ayran					
2.4	Yogurt, whole-fat	Side A					
		Bottled ayran					

2.5	Cheese, regular / yellow	Side A Side B / Thickness Cube/ triangular portion					
2.6	Cheese, low fat / white	Side A Side B / Thickness Cube/ triangular portion					
2.7	Labneh, regular	Side A					
2.8	Labneh, low fat	Side A					
3	**Fruits and Fruit Juices**						
3.1	Citrus orange/ grapefruit	Side A / 1 medium					
3.2	Peach, plum, prunes	Side A / 1 medium					
3.3	Strawberries	Side A / 10 strawberries					
3.4	Grapes	Side A / 10 grapes					
3.5	Banana/ Apples	Side A / 1 medium					
3.6	Dried Fruits	Raisins= 1 tbsp Dates: 1 portion Apricots: 1 portion					
3.7	Fruit juice, fresh	Side A					
3.8	Fruit juice, canned	1 can					
3.9	Fruit juice, bottled	1 bottle/ carton					
3.10	Fruits, canned	Peach/ apricot = ½ fruit Pineapple = 1 slice					
4	**Vegetables**						
4.1	Salad, green: lettuce, mint, cucumber, green pepper, rocket, purslane, etc.	Side A/ Page 8					
4.2	Dark green or deep yellow (spinach, Swiss Chard, Jew's mellow, carrots..)	Side A/ Page 4					
4.3	Tomatoes, fresh	1 medium / 10 cherry					
4.4	Corn / Green peas, fresh	Side A/ Page 4					
4.5	Corn/ Green peas, canned	Side A/ Page 4					
4.6	Potatoes, baked / boiled/ mashed	Side A / 1 medium					
4.7	Zucchini/ Eggplants, cooked	Side A/5 med. stuffed					
4.8	Cauliflower/ Cabbage/ Broccoli	Side A/ Page 4					
4.9	Other canned vegetables (Mushroom, palmetto, asparagus, etc.)	Side A/ Page 4					
4.10	Vegetable juice, fresh	Side A					
5	**Meat and Meat Alternatives**						
5.1	Legumes: lentils, beans, chickpeas, etc., dried, cooked	Side A/ Page 4					
5.2	Legumes, canned (beans, peas)	Side A/ Page 4					

5.3	Nuts & seeds: walnuts, peanuts, almonds, sunflower seeds, etc.	Side A/ Page 4 Pre-packed small bag					
5.4	Red meat, beef/ lamb/goat	Side A/ Ground					
		Steak - Side B/ Thickness					
5.5	Poultry	Leg/thigh/breast/wings Side B					
5.6	Fish/ Seafood, fresh	Side B/ Thickness					
		Shrimp: 1 medium					
		Calamari: 1 medium					
		Crab: 1 medium					
5.7	Fish, canned (tuna, sardines)	1 large can/ 1 small can Page 19					
5.8	Eggs	1 medium					
5.9	Organ meats (liver, kidney, brain)	Side B/ Thickness					
5.10	Luncheon meats (mortadelle, turkey, salami, ham, etc.)	Side B/ Thickness Regular slice					
5.11	Sausages, makanek, uncanned	Side B/ Thickness Makanek size					
5.12	Sausages, makanek, hotdogs, canned	Hotdog size Makanek size Side B/ Thickness					
6	**Added Fats and Oils – Salads/ Cooking / Fries**						
6.1	Vegetable oil, corn/ sunflower/ soya	Side A					
6.2	Olive oil (including with thyme)	Side A					
6.3	Olives	5 olives					
6.4	Butter	Side A					
6.5	Ghee	Side A					
6.6	Mayonnaise	Side A					
6.7	Tahini	Side A					
7	**Sweets and Desserts**						
7.1	Cakes / Cookies/ Doughnuts / Muffins/ Croissant / Biscuits	Side B / Thickness Page 14-15-16					
7.2	Ice cream	1 scoop/ 1 stick/ Page 9					
7.3	Chocolate bar	1 medium					
7.4	Sugar, honey, jam, molasses, chocolate spread	Side A					
7.5	Arabic sweets Baklava, maamoul, knefe	Side B					

8	**Beverages**						
8.1	Soft drink, regular	Side A / 1 can (330 mL)					
8.2	Soft drink, diet	Side A / 1 can (330 mL)					
8.3	Turkish coffee	Side A					
8.4	Instant coffee / Tea	Side A					
8.5	Cocoa / Hot chocolate	Side A					
8.6	Beer	Side A / 1 bottle					
8.7	Wine, red / white/ blush	Side A					
8.8	Liquor, whiskey/ vodka/ gin/ rum	Side A					
8.9	Water	Side A / Bottle (0.5 L)					
9	**Miscellaneous**						
9.1	Manaeesh, zaatar/ cheese	1 regular / 1 bouché Page 17- 18					
9.2	French fries	Side A Page 4					
9.3	Potato chips / Tortilla	XS/ S/ M/ L/ XL bag Page 20					
9.4	Falafel, without bread	1 medium falafel					
9.5	Shawarma	1 medium sandwich					
9.6	Burgers (beef, chicken, fish)	1 medium burger					
9.7	Pizza	Side B / Thickness					
9.8	Canned/ Pre-packed soups	Side A / Page 3					
9.9	Ketchup	Side A					
9.10	Mustard	Side A					

10.1. How many times do you ***season your food with a tomato-based sauce*** (tomato, onion, garlic and simmered with olive oil)?
---------- number of times per day / week / month?

10.2. Do you actually consume chicken or turkey meat ***instead of*** veal, pork, hamburger, or sausage?
------ Yes ------ No

* Are there any other foods/supplements that you regularly consume **[at least once per week]** and that were not mentioned in the FFQ list above?

Food Item	Usual serving size	Frequency of intake per week

Dietary Habits Questionnaire

1. Do you know what Bisphenol A (BPA) is? ----- No ----- Yes
2. Are you aware of BPA free bottles / plastic containers (Tupperware)? ----- No ----- Yes

		Always (6-7 times/week)	Most of the times (4-5 times/week)	Few times (2-3 times/week)	Rarely (1x/week to 2x/month)	Never	Don't know
3	Do you store foods in plastic containers?						
4	Do you heat foods in plastic containers?						
5	Do you make sure that the plastic containers you use are BPA-free?						
6	Do you heat foods that are wrapped in cling film?						
7	Do you drink bottled water?						
	7.1 From plastic- bottled water: ---------- cups/day 7.2 From water cooler: ---------- cups/day						
8	Do you reuse bottled water?						
9	Do you drink from bottles you left in your car?						
10	Do you eat outside home (snacks, restaurants, bars)?						
11	Do you order delivery foods?						
12	Do you purchase soft drinks in cans and/or plastic bottles?						
13	Do you consume canned tomato paste?						

24-Hours Dietary Recalls

Date: (dd/mm/yyyy) ----/----/--------

Day of the week: ----------

Time	Food eaten	Amount	Method of preparation

*** Was yesterday a usual eating day?**

- Yes
- No, please specify ----------

- When was the last meal taken?

Lab work data collection

Name of the participant: ---------- Initials:---------- Study ID number:----------

Test	Unit	Result
Hba1c		
LDL		
SGPT		
Urinary creat		
FBS		
HDL		
GGT		
Spot microalbumin		
Fasting insulin		
Triglycerides		
CRP		
Creatinine		
Total cholesterol		
TSH		
25OHvit D		
Cortisol		
C-peptide		
Prolactin		
Leptin		

Physical Exam Form

Name of the participant: ---------- Initials:---------- Study ID number: ----------

		Results النتائج	Healthy ranges النطاقات الصحية
Body weight (kg الوزن			
Height (cm): الطول			
BMI: مؤشر البدانة			*18.5-24.9 kg/m²*
Waist circumference (cm): قياس دائرة الخصر			نساء *<80 cm*, رجال *<94 cm*
Body fat (kg): نسبة الدهون في الجسم			نساء *<32%;* رجال *<25%*
Muscle mass (kg): نسبة العضل في الجسم			نساء *24-30 %:* رجال *33-40%*
Waist to hip ratio: قياس محيط الأوراك			نساء *<0.9,* رجال *<0.85*
Heart rate: قياس نبض القلب			*60-100 bpm*
Blood Pressure – Measurement # 1 قياس ضغط الدم 1			
Systolic blood pressure (mmHg):	العالي		*120 mmHg*
Diastolic blood pressure(mmHg):	الواطي		*80 mmHg*
Blood Pressure – Measurement # 2 قياس ضغط الدم 2			
Systolic blood pressure (mmHg):	العالي		*120 mmHg*
Diastolic blood pressure(mmHg):	الواطي		*80 mmHg*

Time of urine collection	
Time of blood withdrawal	

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