Scholars Academic Journal of Biosciences (SAJB) **ISSN 2321-6883 (Online)**
Sch. Acad. J. Biosci., 2015; 3(11):922-930 **ISSN 2347-9515 (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublisher.com

Research Article

Studying the effects of socio-demographic factors and medical history of recurrent infections, chronic diseases, smoking and drugs intake on obesity prevalence in adults

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Abstract: This study aims at studying the effect of socio-demographic factors and medical history of recurrent infections, chronic diseases, smoking habits and chronic use of drugs on obesity prevalence in adults. This work was applied on 50 obese subjects visited to the Nutrition Center of Marjan hospital in Babylon province from October 2014 to January 2015. Obese subjects consisted of 39 females and 11 males with age range 20-60 years. This study involved also 30 apparently health subjects (normal weighted) as controls, which consisted of 21 males and 9 females with age range 22-50 years. Body mass index for each subject was estimated, socio-demographic factors and medical history of recurrent infections, chronic diseases, smoking and chronic use of drugs also have been determined in a special questionnaire. In socio-demographic factors, the age, residence, occupational state, educational level were significantly associated with obesity prevalence ($p<0.05$), while gender was highly significant associated with obesity ($p<0.001$), marital status not associated ($p>0.05$). In medical history, recurrent infections and chronic diseases were a highly significant associated with obesity ($p<0.001$), and chronic use of drugs was significantly associated with obesity ($p>0.05$). smoking habits non- significantly associated with obesity, and increasing of pack-year unit paralleled to decreasing of BMI.The results provide a good correlation between socio-demographic factors and obesity; and medical history of chronic diseases, recurrent infections, chronic uses of drugs, smoking habits and obesity.
Keywords: Body mass index, obesity.

INTRODUCTION

Obesity is a French word comes to English. In Latin the verb obedere means (over eat) , obesitas means (very fat) [1]. It is defined as a chronic disease in which body fat has been accumulated in excessive level, which leads to bring adverse effects, for assessment of obesity should be calculate a person's weight (in kilograms) divided by square of his\her height (in meter) in law known as Body Mass Index (BMI). The person considered as obese when his\her BMI equal or above to 30. The obesity can be categorized in concerning to the value of BMI, if it is smaller than 18.5 kg/m^2 defined as underweight, but if the BMI in the range of (18.5 – 24.9) kg/m^2 known as normal weight, with the range of (25–29.9)kg/m^2 defined as overweight, the person will be obese if his\her BMI 30 or more. Also, obesity can be classified in to three classes; class I if the BMI lies in the range of (30-35) kg/m^2, and so class II in the range (35-40) kg/m^2, class III if the BMI equal 40 or above called morbid obesity [2]. Obesity increases rates of various diseases such as heart diseases, diabetes mellitus (type II), obstructive sleep apnea, and some types of cancer and osteoarthritis [3]. It is considered preventable cause worldwide increasing rate of death in adult, also it is showed as a most health problem in 21st country [4].

The epidemiology of obesity has been described in numerous areas of the world. The highest percentage of obesity has been indicated in the Pacific Islands, while the lowest percent has been indicated in Asia. In Europe and North American are generally with high percentages of obesity, while in Africa and Middle Eastern countries the rates are variable [5]. The obesity prevalence around the world has been monitored by the world health organization through the global database on body mass index. The analysis of data that involved in the database are identified from the literature or from a wide network of collaborators. The database has collected information comprising approximately 86 percent of the adults' populations of the world. A total of (1.6) billion individuals suffering from overweight, and approximately four hundred million individuals adults were obese. During (2015-2016) approximately 700 million adults will be obese and at least 2.3 billion will be overweight. According to the data from the Global Database on body mass index, there are wide variations in the prevalence of obesity throughout the world, ranging from India, where 1% or less of the

population is obese, to the Pacific ocean Islands, where the prevalence of obesity can reach to 80 percent in some regions [6]. Flegal *et al*.; [7] mentioned that in USA about 64.1 percentages of women, 72.3 percentage of men of 68 percentage of adults were either obese or overweight. Moreover, about 33.8% of adults with obesity dividing into 33.2% of men plus 35.5% of women. Kant *et al*., used nutritional records from four successive national health and nutrition examination survey (NHANES) studies involving (39,094) adults subjects of the American population to confirm that the progressive drifts in the increase of the amount and density of energy in consumed foods of adults corresponding to the elevation of obesity prevalence in the united state population [8].

The main cause of obesity is the excessive food energy intake and lack of physical activity or training [9]. Other causes that attributed to genetic factor and medications and lifestyle [10]. Diet play a significant role in process of obesity incidence, where in united states since 1971 to 2000 the calories average that consumed everyday by women increased to 1.400 kcal/day, most of this increasing in calories caused by carbohydrate consumption rather than fat, during the same period the obesity rates increased from 14.5 to 30.9 percentage [11]. The type of fat in causing weight gain is very important than amount of it, where, in Nurse's health study found that increased consumption of saturated fat causes with increasing weight, but increased consumption of monounsaturated and polyunsaturated fat was not [12].

MATERIALS &METHODS

Samples:

In this study a total of 50 obese adult consists of 43 females and 7 males, athletes and diabetic subjects were excluded. Their ages ranged from (20-60) years. Socio-demographic characteristics for each one have been taken which were:- sex, age, marital state, residency, educational state; and history of smoking, recurrent infections, chronic diseases, and drugs in a special questionnaire. These patients were visited the center of nutrition in Marjan hospital in Babylon province during the period October 2014 to January 2015. The controls a total of 30 healthy subjects (normal weight) consisting of 21 male and 9 female were involved in control group, diabetic patients also excluded, their ages ranged from (22-50) years.

Ethical approval:

Agreement of all subjects in case & control groups has been taken prior the study. Moreover, the study design was approved by Research Ethical Committee in college of Medicine / Babylon University.

Estimation of Body Mass Index (BMI):

BMI of all case and control subjects was measured, the weight of each subject has been taken by personal balance without shoes and heavy clothes, and the standing height also was measured by tap measure, then body mass index estimated according to WHO, by the following equation [13]:

$$\text{Body Mass Index (BMI)} = \text{weight (in kilograms)}/\text{height (in meter)}^2.$$

Statistical Analysis:

Statistical analysis was performed using Statistical Package for Social Science Software (SPSS, version 22). Variables were described using frequency distribution and percentage for the subjects according to their characteristics and mean; standard deviation (SD) for continuous variable, also Chi-square test was used for the assessment of association between the variables studied. The p-value of less than 0.05 was statistically significant, and highly significant for p-value of less than 0.001.

RESULTS & DISCUSSION

Socio-demographic Factors:

***Age*:**

The distribution of study groups according to age group in this study shows (46%) of obese subjects were in age group (31-40) year, and (70%) of control subjects were in age group (20-30) year. As showed in table-1, regarding to age group, it has a significant association with obesity (p=0.028). This result lead to that non-obese individuals younger than obese others, and obesity prevalence noted to be increased with age increasing, where overall among obese adults group aged 20-40 years appear to be increased with age. This finding agree with Iranian study that showed obesity increased with age in the range 20-49 years, after 50 years it is decreased, and explained this reduction in obesity after that year as a result of alteration in energy expenditures, food eating, appetite and composition of body that happen with age increasing [14]. But, Kaiser *et al*.; [15] explained this reduction resulted from some factors linked to aging such as sarcopenia & cachexia, and loss of bone mineral density may have a role in weight loss.

Gender:

In this study, a total of (50) obese adults were included, consisted of 39 (78%) female, and 11 (22%) male, whereas (30) control distributed as 9 (30%) female, 21 (70%) male as showed in tabl-1. This result lead to that obese female ratio higher than that of males with a highly significant association (p<0,001) and (OR= 0.121). This finding agrees with Garawi *et al.*; study [16]. The higher prevalence of female than male in obese group can be explained by behavioral difference between them, and physiological differences in hormones thought the live [17]. Moreover, Kanter

and Caballero [18] indicated to the increasing of obesity prevalence in females because females have low physical activity compared with males. WHO mentioned that in 2014 about 13 percent of the world's adult populations were obese, divided to 57.7% female and 42.3% male. Moreover, women are more likely to be obese than men, where in Africa, Eastern Mediterranean and south East Asia, female had doubled obesity incidence of male [19].

Residence:

The present study shows that from obese adults group who participate in current study (54%) lived in urban area, and (46%) lived in rural area with a significant association between obesity & residence (p=0.043), (OR=0.357), (table-1). This finding agree with study in Morocco showed that obesity significantly higher in urban than that in rural [20]. The current result can be explained by the sedentary life in urban, and most of participants who lived in rural area working in farming, agree with study in Ghana indicate to that reduction of obesity percentage in rural area resulted from jobs of rural inhabitants, where often they work manually with physical activity and effort [21], disagree with Christie *et al*.; [22] that showed in USA obesity percent in rural area was (39.6%) that higher than that in urban (33.4%), and explained the cause of this elevation returns to difference in environment variables, and high fatty diet (HFD) which may contribute in obesity increasing in the rural area.

Marital Status:

In regards to marital status, this study showed that 27 (54%) of obese group were married and the others 23 (46%) were not married subdivided to 10 (20%) single, 6 (12%) widow, and 7 (14%) divorce. In control (non-obese) group, 13 (43.3%) were married and 17 (56.7%) were single. This result as showed in table -1 demonstrates that there is non-significant association between obesity and marital status (P= 0.356), (OR=1.535), agree with the study in Greece demonstrates there is non-significant association between obesity and marital status [23]. The present study showed that in obese group the percentage of married individuals higher than that of singles, and the causes maybe return to that married individuals have less activity and exercise than singles. Moreover, single obese adults were younger than who are obese married, concordant with Janghorbani *et al*.; [24] who indicated that obese married subjects in Iran accounts (74.7%) of obese adults group, and obese married individuals were older than singles. Rhazi *et al*.; disagree; they proved that a significant association between obesity and marital status in Morocco, suggested that married females more predisposed to obesity than married males [20].

Occupational Status:

In this study, 23 (46%) subjects of obese group were employees, and 27 (54%) were not employed, while in the control group, 23 (76.7%) were employee, and the remained 7 (23.3%) as not employed, as illustrated in table-1). This result demonstrates that in obese group the high percentage was of not employed (54%), but the higher percentage in control group was of employees (76.7%) with a significant association between obesity and employment status (P=0.007), (OR=0.259), agree with Bonauto *et al*.; who showed that obesity significantly associated with employment status, non-employed subjects more predisposed to be obese than that employees because of physical activity during work that maybe has an effect on obesity prevalence [25]. Shaikh *et al*.; study [26] disagree, indicated that employment status had no effect on obesity predominance, by showing that obesity was also high in employed subjects that work with computers an mathematical jobs away from physical activity.

Educational Level:

In regards to educational levels, present study showed that in obese group 20 (40%) were with low educational level (illiterate & primary level), 14 (28%) moderate (secondary) level, and 16 (32%) with high education level. But, in control group 2 (6.7%) with low education, 7 (23.3%) of moderate level, and 21 (70%) of high education level, as illustrated in table-1.

This result showed a significant association between educational level and obesity (p=0.002) in two groups, agree with Reynolds *et al*., study [27] who mentioned that obesity prevalence tend to be decreased in high educational level in American population, and showed about every one year of education reduces the obesity percent by 2-9%.Moreover, In Spain, showed by higher education obesity in Spain population declined by (1.4%) in females, (7.3%) in males [28]. Another study in Malaysia, showed that increasing period of education has a role in the decreasing possibility of obesity in Malaysia population, explained that schooling has a role in guidance to choice health food and avoiding that facilitate weight increasing [29].Tzotzas *et al*.; disagree by showed there is no significant association between educational level and obesity prevalence in Grecian population indicated to there is no association between Grecian schooling and obesity incidence [30].

Table-1: Distribution of Study Groups According to Socio-demographic Factors

Variable	Obese (N=50) No. (%)	Control (N=30) No. (%)	Total	X^2	P- value	OR	95% C.I.
Age group							
20-30 years	18(36%)	21 (70%)	39 (48.75%)	9.11	0.028	-	-
31-40 years	23(46%)	7 (23.3%)	30 (37.5%)				
41-50 years	7 (14%)	2 (6.7%)	9 (11.25%)				
51-60 years	2 (4%)	0 (0.0%)	2 (2.5%)				
Gender							
Female	39 (78%)	9 (30%)	48 (60%)	18	<0.001	0.12	0.043-0.338
Male	11(22%)	21 (70%)	32 (40%)				
Residence							
Urban	27 (54%)	23 (76.66%)	50 (62.5%)	4.11	0.043	0.35	0.130-0.983
Rural	23 (46%)	7 (23.33%)	30 (37. 5%)				
Marital status							
Married	27 (54%)	13 (43.3%)	40 (50%)	0.85	0.356	1.53	0.617-3.819
Single, Widow, Divorce	23 (46%)	17 (56.7%)	40 (50%)				
Occupation							
Employee	23 (46%)	23 (76.7%)	46 (57.5%)	7.21	0.007	0.26	0.094-0.714
Not-Employed	27 (54%)	7 (23.3%)	34 (42.5%)				
Educational level							
Low	20 (40%)	2 (6.7%)	22 (27.5%)	13.58	0.002	-	-
Moderate	14 (28%)	7 (23.3%)	21 (26.25%)				
Higher education	16 (32%)	21 (70%)	37 (46.25%)				

Smoking Habits, Chronic Diseases, Re-current Infection, and Chronic Drug Intake:

Smoking Habits:

This study showed that for (50) obese adult group, 13 (26%) were current smoker, 4 (8%) ex-smoker, and 33 (66%) no smoker, while for (30) control group, 5 (16.7%) were current smoker, 1 (3.3%) ex-smoker, and 24 (80%) no smoker. As showed in Table - 2. This result demonstrates there is non-significant association between smoking habits and obesity (P=0.388), the percent of smoker subjects in obese group was (26%) lower than that of obese ones who non-smoker (66%), this result agree with Auchincloss *et al.*; study in USA showed that obesity has inverse relationship with smoking by showing that current smokers were account (16.3%) of obesity group, non-smoker account (43.8%), and the remained (39.9%) were ex-smoker [31]. Moreover, Bakhshi *et al.*; [32] showed in cross sectional study that obesity has inverse relationship, where they found that the percentage of obese smoker individuals lower than non-smoker in Iranian population, explained the cause returns to energy expenditure be high during smoking, and nicotine has a role in reduction of appetite. This study also shows the relation of intensity of smoking (pack-year) and BMI also was an inverse relationships in figure (3.1). intensity of smoking (pack-year) defined as amount of smoked packs or cigarette along period (years) of smoking, calculated as (pack-year= number of packs/day × duration of smoking in years, or pack-year= number of "cigarette/day"/20 × duration of smoking in years) [33]. This finding agree with Gonseth *et al.*, study [34] who confirmed long duration of smoking causes loss of weight, indicated to there are certain materials have been added to cigarette by manufactory of tobacco act as inhibitors for appetite, hence lead to decrease in weight.

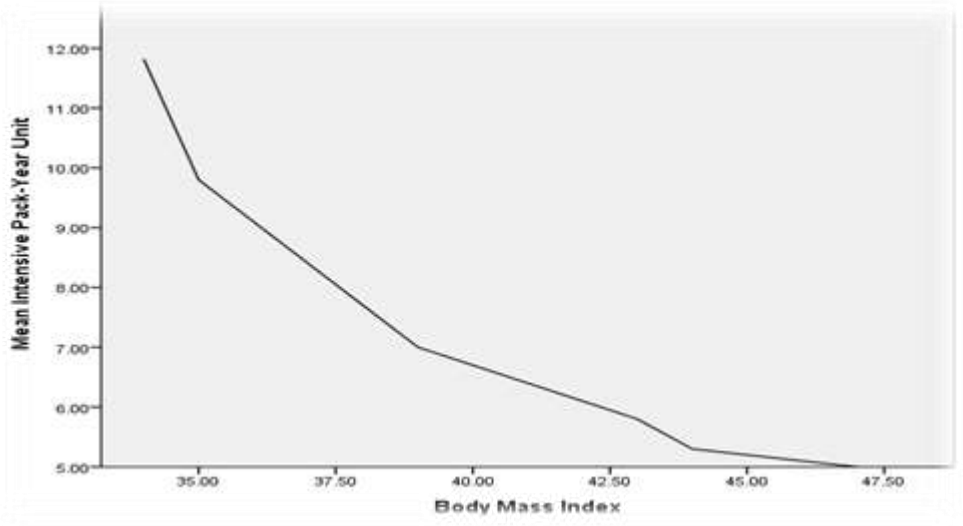

Fig-1: Relationship of Intensive of Smoking (pack-year) and BMI

The smoking affects immune system in different forms according to that showed by Abd study [35] that indicate immunoglobulins IgA, IgG antibodies reduced in smokers as compared with that non-smokers, but IgM was elevated in smokers, in addition to C3 and C4 complement factors. O'Leary *et al*.; [36] study showed that smokers individuals with mycobacterium tuberculosis infection have lower secretion of tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ) in comparison with non-smokers infected.

Chronic Diseases:

Present study showed that 33 (66%) of obese adults group have a chronic diseases (42% hypertension, 28% asthma, 6% ischemic heart diseases (IHD), and other diseases with low percent such as gallstone 4%, irritable bowel syndrome 2%, arthritis 2%, and cystitis 2%), the remained 34% reported no such history, some subjects have mix chronic diseases. Fig-2 shows distribution of obesity according to chronic diseases. In control group there are only 7 (23.33%) who have a chronic diseases (13.33% hypertension, 6.66% asthma, and only one 3.33% irritable bowel syndrome), and the most 76.7% reported not such history of chronic diseases. As showed in (table 3.3), there is a high significant association between obesity and chronic diseases (P=<0.001), (OR=3.378). This finding agree with Sjostrom *et al*.; [37] who mentioned that hypertension is the chronic disease that has a significant link with obesity, they showed 51% of obese individuals reported history of hypertension. Moreover, interaction between obesity and hypertension is recognized both in children and adults [38]. World health organization also mentioned that hypertension was one of the complications resulted from obesity [2]. The mechanism by which obesity lead to hypertension under search, but there are studies suggest some mechanisms, one of these represents that hypertension influenced by the secretion of many inflammatory adipokines especially from white adipose tissue (WAT) in visceral fat depots, some of these adipokines enhance endothelial vasomotor tone through the secreting of rennin, angiotensinogen, and angiotensinogen II which are similar in action to those of renal-angiotensinogen system (RAS) causing hypertension by vasoconstriction and activation of sympathetic nervous system [39].

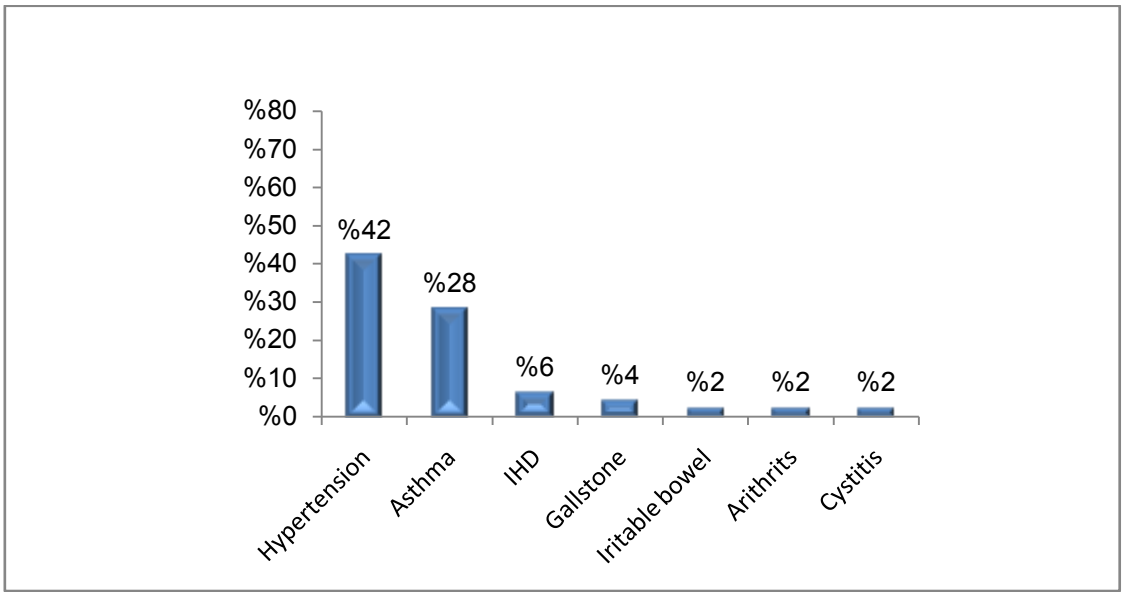

Fig-2:Distribution of Obese Persons According to Chronic Diseases

Regarding to asthma, Beuther and Sutherland [40] indicated that incidence of asthma was higher by 38 percent in overweight patients, and by 92 percent in obese patients than normal weight, with no dissimilarities among females and males. Many studies have been performed on asthma, and most of these studies showed that the incidence of asthma frequently related to obesity, persons with higher BMI considered to be more threatening to asthma disease, and this effect was strongest in women particularly as a result of the elevation of adiposity in women. Moreover, asthmatic obese patients who achieved weight loss by diet or bariatric surgery are established with developing in their lung function and reduction in asthma symptoms [41]. The mechanism of asthma incidence in obese subjects explained by Dixon *et al*.; [42] study who mentioned that many immune mediators are secreted by adipose tissue (adipokines) such as IL-10, IL-6, adiponectin, C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-alpha). The last one TNF-alpha has been shown in adipocytes and it is directly related to the size of body fat, it has a role in pathogenesis of asthma, where it is increased in asthma and considered as a stimulator for the production of IL-4, IL-6 (Th2 cytokines) in bronchial epithelium, and this led to increased levels of these cytokines in asthmatic patients. seasons), and indicated to that severe obesity considered as a significant predictor of influenza complications. Beside, a study by Louie *et al*., indicated that 50 percent of the Californians obese adults hospitalized with the 2009 H1N1 infection [45]. There are some factors related to obesity explained by Ashburn *et al.*; [46] facilitate the threatening of respiratory infection such as decreased pulmonary volumes, pulmonary restriction, ventilation-perfusion mismatching, obstructive sleep apnea (OSA), the risk of pulmonary embolism, and dysregulated immune response in the lung. In addition, one of studies on animal suggests diet-induced obese mice have reduced and postponed expression of pro-inflammatory cytokines, reduced natural killer (NK) cells cytotoxicity, and weakened dendritic cell (DC) activity during influenza infection [47].

Ischemic heart disease also has been found linked with obesity as one of the obesity complications, Nordestgaard *et al*.; [43] mentioned that every 4 kg/m2 elevate in BMI suggested (26%) increase in IHD risk.

Recurrent Infections:

This study showed that 48 (96%) of obese adults group have a recurrent infection (50% who have recurrent with respiratory infection, 20% gastrointestinal tract (GIT) infection , 18 % recurrent with skin infection, 18% with eye infection, 14% with urinary tract infection (UTI)), some subjects have recurrent with mix infections, (figure 3.3). In control group, from (30) subjects there are only 11 (36.7%) have a recurrent infection (16.66% recurrent with respiratory infection, 13.33% with GIT), one subject have mix recurrent infection. There is a highly significant association between obesity and recurrent infection (P=<0.001), (OR=41.455) as in table (3.3).Agree with Kowng *et al*.; [44] who showed the association between obesity and respiratory infection, they found obesity (BMI > 30) to be independently linked to an improved risk of recurrent infection for the period of seasonal influenza activity (over 12 influenza

Chronic Drugs Intake:

This study showed there are 17 (34%) of obese adults group have a history of drug intake (12% with history of Tenormine (atenolole) intake, 10% of Metoprolol intake, 8% of steroid drug intake, 4% of non-steroid anti-inflammatory drugs (NSAID) intake, and others with Antihistamine 2%, Ventolin (inhaler) 2%, and Ketofen 2%, (figure-3), some persons have

mix of drug intake. In control group (non-obese), only 4 (13.33%) have a history with drug intake, (10% of Tenormine intake, and 3.33% Metoprolol).There is a significant association between obesity and chronic use of drugs (P=0.042) and (OR=3.348) as in table-2.

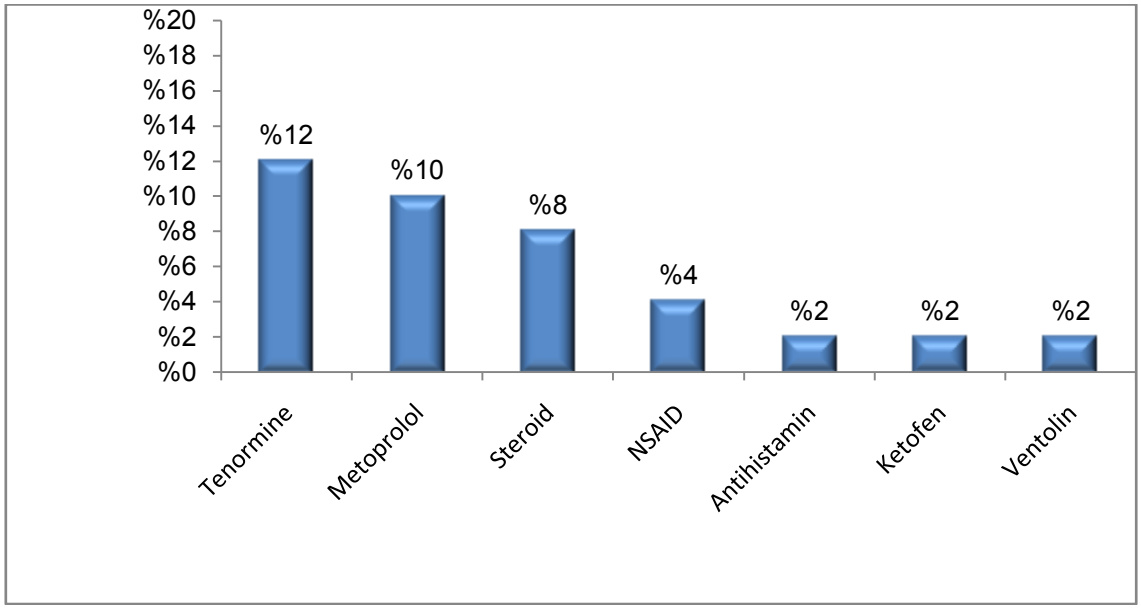

Fig-3: Dstribution of Obese Persons According to History of Drugs Intake

The high percent was of Beta-blockers drug (Tenormine Metoprolol) as anti-hypertension drugs, agree with Lee *et al.*; [48] who mentioned that the average of weight increasing was highly significant in hypertensive patient with chronic treatment with beta-blocker drugs in compared with not treated. Moreover, they concluded that these drugs because increasing in weight by its role in reducing energy expenditures. Also, a study by Karasoy *et al.*; in Denmark refers to that chronic uses of beta-blockers drugs increased in women with increasing of body mass index of them [49]. These drugs also have altering effects on immune system, Shaw *et al.*; [50] showed subjects with chronic uses of BBs (beta-blockers) drug chronic uses have altering in HLA-DR expression on T lymphocytes and CD107 expression on cytotoxic Tcells.

Table -2: Distribute of Study Groups According to Smoking, Medical History of Chronic Diseases, Recurrent Infections, and Chronic Drugs Intake

Variable	Obese (N=50) No. (%)	Control (N=30) No. (%)	Total	X^2	P-Value	OR	95% CI
Smoking habits Current smoker Ex-smoker No smoker	13 (26%) 4 (8%) 33 (66%)	5 (16.7%) 1 (3.3%) 24 (80%)	18 (22.5%) 5 (6.25%) 57 (71.25%)	1.895	0.388	-	-
History of Chronic diseases Yes No	33 (66%) 17 (34%)	7 (23.3%) 23(76.7%)	40 (50%) 40 (50%)	13.65	<0.001	3.378	2.280-17.842
History of re-current infections Yes No	48 (96%) 2 (4%)	11 (36.7%) 19 (63.3)	59 (73.75%) 21 (26.25%)	34.09	<0.001	41.455	8.39-204.82
Chronic drug intake Present Absent	17 (34%) 33 (66%)	4 (13.3%) 26 (86.7%)	21 (26.25%) 59 (73.75%)	4.137	0.042	3.348	1.004-11.166

References:

1. World Health Organization. Obesity and overweight. 2011; Fact sheet N 311.
2. World Health Organization. Obesity and overweight. 2014; Fact sheet N 311.
3. Haslam DW, James WP. "Obesity". Lancet 2005; 366 (9492): 1197–209.
4. Barness LA, Opitz JM, Gilbert-Barness E; "Obesity: genetic, molecular, and environmental aspects". American Journal of Medical Genetics 2007; 143A (24): 3016–34.
5. Prentice AM; The emerging epidemic of obesity in developing countries. International Journal of Epidemiology 2006; 35:93–99.
6. World Health Organization. Obesity and overweight Archived from the original. 2010; Retrieved January 10, 2009.
7. Flegal KM, Carroll MD, Ogden CL, Curtin LR; Prevalence and Trends in Obesity among US Adults, 1999-2008. JAMA. 2010; 303(3):235-241.
8. Kant AK and Graubard BI; Secular trends in patterns of self-reported food consumption of adult Americans: NHANES 1971-1975 to NHANES 1999–2002-3. Am J Clin Nutr. 2006; 84:1215–23.
9. Lau DC, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E; "2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children summary". CMAJ. 2007; 176 (8): S1–13.
10. Bleich S, Cutler D, Murray C, Adams A; "Why is the developed world obese?".Annu Rev Public Health 2008; 29: 273–95.
11. Wright JD, Kennedy-Stephenson J, Wang CY, McDowell MA, Johnson CL; "Trends in intake of energy and macronutrients—United States, 1971–2000". MMWR Morb Mortal Wkly Rep. 2004; 53 (4): 80–2.
12. Field AE, Willett WC, Lissner L, Colditz GA; Dietary Fat and Weight Gain among Women in the Nurses' Health Study. Obesity 2007; Vol. 15 No. 4. 967.
13. World Health Organization. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. 1995. Geneva.
14. Bakhshi E, Koohpayehzadeh J, Seifi B, Rafei A, Biglarian A, Asgari F, *et al.*; Obesity and Related Factors in Iran: The STEPS Survey, 2011. Iran Red Crescent Med J. Iran Red Crescent Med J. 2015; 17(6): e22479.
15. Kaiser R, Winning K, Uter W, Volkert D, Lesser S, Stehle P, *et al.*; Functionality and mortality in obese nursing home residents: an example of 'risk factor paradox'?. J Am Med Dir Assoc. 2010; 11(6):428–35.
16. Garawi F, Devries K, Thorogood N, Uauy R; Global differences between women and men in the prevalence of obesity: is there an association with gender inequality?. European Journal of Clinical Nutrition 2014; 68 1101-1106.
17. Tsai AG, Fabricatore AN, Wadden TA, Higginbotham AJ, Anderson A, Foreyt J, *et al.*; Readiness redefined: A behavioral task during screening predicted 1-year weight loss in the look AHEAD study. Obesity.2014;Volume 22, Issue 4, pages 1016–1023.
18. Kanter R, Caballero B; Global gender disparities in obesity: a review. Adv Nutr. 2012; 3(4):491–8.
19. World Health Organization. Obesity and overweight. 2015; Fact sheet N 311.
20. El Rhazi K, Nejjari C, Zidouh A, Bakkali R, Berraho M, Barberger Gateau P; Prevalence of obesity and associated socio demographic and lifestyle factors in Morocco. Public Health Nutrition 2011; 14(1), 160–167.
21. Amoah AG; Socio demographic variations in obesity among Ghanaian adults. Public Health Nutrition 2003; 6(8): 751–757.
22. Befort CA, Nazir N, Perri MG; Prevalence of Obesity among Adults from Rural and Urban Areas of the United States: Findings from NHANES (2005–2008). J Rural Health 2012; 28(4): 392–397.
23. Panagiotakos DB, Pitsavos C, Chrysohoou C, Risvas G, Kontogianni MD, Zampelas A, *et al.*; Epidemiology of overweight and obesity in a Greek adult population: the ATTICA Study. Obes Res. 2004; 12(12):1914-1920.
24. Janghorbani M, Amini M, Rezvanian H, Gouya MM, Delavari A, Alikhani S, *et al.*; Association of body mass index and abdominal obesity with marital status in adults. Arch Iran Med. 2008; 11(3):274-81.
25. Bonauto DK, Lu D, Fan ZJ; Obesity prevalence by occupation in Washington State, Behavioral Risk Factor Surveillance System. Prev Chronic Dis. 2014; 11:130219.
26. Shaikh RA, Sikora A, Siahpush M, Singh GK; Occupational Variations in Obesity, Smoking, Heavy Drinking, and Non-Adherence to Physical Activity Recommendations: Findings from the 2010 National Health Interview Survey. American Journal of Industrial Medicine 2015; 58:77–87.
27. Reynolds SL, Hagedorn A, Yeom J, Saito Y, Yokoyama E, Crimmins EM; A Tale of Two Countries-the United States and Japan: Are Differences in Health Due to Differences in Overweight?. J Epidemiol. 2008; 18(6):280-290.

28. Artajelo FR, Garcia EL, Gutiérrez-Fisac JL, Banegas JB, Urdinguio PL, Rojas VD; Changes in the prevalence of overweight and obesity and their risk factors in Spain 1987-1997. Prev Med. 2002; 34:72-81.
29. Tan AK, Dunn RA, Samad MI, Feisul MI; Socio demographic and Health–Lifestyle Determinants of Obesity Risks in Malaysia. Asia-Pacific Journal of Public Health 2011; 23(2) 192–202.
30. Tzotzas T, Vlahavas G, Papadopoulou SK, Kapantais E, Kaklamanou D, Hassapidou M; Marital status and educational level associated to obesity in Greek adults: data from the National Epidemiological Survey. BMC Public Health 2010; 10:732.
31. Auchincloss AH, Mujahid MS, Shen M, Michos ED, Whitt-Glover MC, Diez Roux AV; Neighborhood Health-Promoting Resources and Obesity Risk (the Multi-Ethnic Study of Atherosclerosis). Obesity 2013. Vol.21, 621-628.
32. Bakhshi E, Seifi B, Biglarian A, Mohammad K; Changes in Body Mass Index across Age Groups in Iranian Women: Results from the National Health Survey. Journal of Nutrition and Metabolism. 2012; Article ID 848403, 9 pages.
33. Prignot J; Quantification and chemical markers of tobacco-exposure. Eur J Respir Dis. 1987; 70:1–7.
34. Gonseth S, Jacot-Sadowski I, Diethelm PA, Barras V, Cornuz J; The tobacco industry's past role in weight control related to smoking. Eur J Public Health 2012; 22(2):234–7.
35. Abd FG; Studying of immunoglobulins, C3, C4 and IL- 8 concentrations in serum smokers and nonsmokers. Journal of Kerbala University 2008; Vol. 6 No.3 p16-19.
36. O'Leary SM, Coleman MM, Chew WM, Morrow C, McLaughlin AM, Gleeson LE, *et al.*; Cigarette smoking impairs human pulmonary immunity to Mycobacterium tuberculosis. Am J Respir Crit Care Med. 2014; 190(12):1430-6.
37. Sjöström L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, *et al.*; Lifestyle, diabetes and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004; 351:2683.
38. Kotsis V, Stabouli S, Papakatsika S, Rizos Z, Parati G; Mechanisms of obesity-induced hypertension. Hypertension Research 2010; 33: 386–393.
39. Chinetti G, Fruchart JC, Staels B; Peroxisome proliferator-activated receptors and inflammation: from basic science to clinical applications. Int J Obes Relat Metab Disord. 2003; 27 Suppl 3:S41-5.
40. Beuther DA, Sutherland ER; Overweight, obesity, and incident asthma: a meta-analysis of prospective epidemiologic studies. Am J Respir Crit Care Med. 2007; 175(7):661-6.
41. McLachlan CR, Poulton R, Car G, Cowan J, Filsell S, Greene JM, *et al.*; Adiposity, asthma, and airways inflammation. J Allergy Clin Immunol. 2007; 119(3):634-9.
42. Dixon AE, Shade DM, Cohen RI, Skloot GS, Holbrook JT, Smith LJ, *et al.*; Effect of obesity and clinical presentation and response to treatment in asthma. J Asthma 2006; 43:553-8.
43. Nordestgaard BG, Palmer TM, Benn M, Zacho J, Tybjaerg-Hansen A, Davey Smith G, *et al.*; The Effect of Elevated Body Mass Index on Ischemic Heart Disease Risk: Causal Estimates from a Mendelian Randomisation Approach. PLoS Med. 2012; 9(5): e1001212.
44. Kwong JC, Campitelli MA, Rosella LC; Obesity and respiratory hospitalizations during influenza seasons in Ontario, Canada: a cohort study. Clin Infect Dis. 2011; 53: 413–421.
45. Louie JK, Acosta M, Samuel MC, Schechter R, Vugia DJ, Harriman K, Matyas BT; A novel risk factor for a novel virus: obesity and 2009 pandemic influenza A (H1N1). Clin Infect Dis 2011; 52:301–312.
46. Ashburn DD, De Antonio A, Reed MJ; Pulmonary system and obesity. Crit Care Clin. 2010; 26: 597–602.
47. Smith AG, Sheridan PA, Tseng RJ, Sheridan JF, Beck MA; Selective impairment in dendritic cell function and altered antigen-specific CD81 T-cell responses in diet-induced obese mice infected with influenza virus. Immunology 2009; 126:268–79.
48. Lee P, Kengne AP, Greenfield JR, Day RO, Chalmers J, Ho KK; Metabolic sequelae of β-blocker therapy: weighing in on the obesity epidemic?. International Journal of Obesity 2011; 35: 1395–1403.
49. Karasoy D, Bo Jensen T, Hansen ML, Schmiegelow M, Lamberts M, Gislason GH, *et al.*; Obesity is a risk factor for atrial fibrillation among fertile young women: a nationwide cohort study. Euro pace 2013; 15(6):781-6.
50. Shaw SM, Coppinger T, Waywell C, Dunne L, Archer LD, Critchley WR, *et al.*; The Effect of Beta-Blockers on the Adaptive Immune System in Chronic Heart Failure. Cardiovascular Therapeutics 2009; 27: 181–186.