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Study the anthropometric parameters for obese cases beside dietary habits, blood test and sex hormones that affect Fertility

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Abstract

Obesity is defined as an abnormal or excessive accumulation of body fat and currently is one of the most public health problems. The objective of the study is to determine the relation between human obesity and several related parameters that may affect fertility in both sexes, two hundred adult(100)males and (100) females aged 21 to 25 years were volunteers for the study. A questionnaire was completed on eating habits, focusing on the number of daily meals, meal composition, and sedentary lifestyle habits. An anthropometric assessment was also performed, including weight, height, BMI, skin-fold thickness, waist and arm circumference. Also, some sexual male and female hormones were determined. The results revealed that the mean age of males were 23± 1.45 y and for females were 23±2.05y. Calories,total fat, carbohydrate, phosphorus,potassium, vitaminA,B2 and niacin together with cholesterol intake by obese volunteers were higher than the recommended dietary allowances. Both genders had BMI more than 40; BMI of females was higher than males.The mean value of cholesterol, triglycerides and VLDL-c for obese males and females were higher than normal range. Obese male showed an increase in serum levels of leptin, prolactin PRL estrogen and E2, accompanied with marked reduction in serum testosterone (T),dehydroepiandrosterone (DHEA) and T/ Estrogen E2 ratio.

Obesity in females showed reduction in the levels of progesterone and estrogen while it caused an increase in luteinizing hormone,follicle-stimulating hormone, leptin and prolactin. Therefore, it was important to keep attention to obesity as an etiology of male and female fertility and change the lifestyle of young obese female and male.

Introduction

Obesity is one of the most concerned public health problem of contemporary society; it is multifactorial in origin, including genetics and family environment, (being related to cultural, eating, and sedentary habits). Obesity involves an abnormal and excessive fat accumulation that negatively affects the health of the body. According to the World Health Organization ***(WHO),*** if the body mass index (BMI) equals to or is greater than 30 kg/m2, it is considered obesity ***(Jungheimet al., 2012*)**.Obesity brings out many problems such as social, psychological, demographic, and health problems. It is related to increased health risks. Approximately 44% of the problems are subjects to diabetes, 23% may suffer from ischemic heart disease, and between 7% and 41% to may complain of various malignancies associated with obesity ***(Tong and Xu, 2014)***. Infertility is a medical condition in both sex characterized by a diminished or absent ability to produce offspring and the risk factors can be genetic, environmental or related to lifestyle. One of the most common and well documented risk factors for infertility in both men and women is obesity. Males and females differ in terms of how and where they store body fat, the hormones they secrete in proportion to their fat, and the way their brains respond to signals that regulate

food intake and body weight ***(Agarwal et al., 2015 and Broughton & Moley, 2017)***. It led to abnormal hormone issues affecting reproductive processes for both women and men. Abnormal hormone signals, as a result of excess weight, negatively impact ovulation in women and sperm production in men. Hormone irregularities in men affect stimulation of the testicles that inhibit sperm production. Excess fat causes the male hormone, testosterone, to be converted into estrogen, and those estrogens decrease testicle stimulation. Researchers from Reproductive Biology Associates reported that a high BMI in men correlates with reduced testosterone levels. Men with high BMIs were found to have an abnormal semen analysis.

(Rehman et al., 2018) found that overweight men were 11 percent more likely to have a low sperm count and 39 percent more likely to have no sperm in their ejaculate**.**In women obesity, it can cause the overproduction of insulin, which may cause irregular ovulation as polycystic ovarian syndrome (PCOS). PCOS is a specific medical condition associated with irregular menstrual cycles, anovulation (decreased or stopped ovulation) ***(Mittal et al., 2013)***. Obese women have higher levels of a hormone called leptin, which is produced in fatty tissue. This can disrupt the hormone balance and lead to reduced fertility. Leptin levels may negatively influence the neuroendocrine regulation of reproduction, with a threshold level being permissive to normal reproduction ***(Li et al., 2018)***. Conversely, elevated leptin levels may negatively influence normal ovarian function and/or embryo development and viability. In obese women, gonadotropin secretion is affected because of the increased peripheral aromatization of androgens to estrogens. The insulin resistance and hyperinsulinemia in obese women leads to hyperandrogenemia. The sex hormone-binding globulin (SHBG), growth hormone (GH), and insulin-like growth factor binding proteins (IGFBP) are decreased and leptin levels are increased. Thus, the nutriregulation of the hypothalamic-pituitary-gonadal (HPG) axis deteriorates***(Traviesoet al., 2019)***.The purpose of this research is to explore the links between obesity and male and female fertility.

Volunteers and methods

Research design

The volunteers for current study were chosen randomly form obese women and men attending at the clinic in National Nutrition Institute located in Cairo. The total sample size was 200 (100 men and 100 women). Their ages ranged between 21-25 years.The present study started in January 2021 and ended in January 2022.

Design of work

Before starting the work an approval was obtained from the ethical committee. Interviews were held with the obese men and women using three forms of questionnaires.They designed after trial on pilot studyA-for social and demographic data.B-for health history and anthropometric measurements. C-the third one for the dietary habits study ***(Rasanen, 1979)***.

Socio-economic and demographic data

The socio-economic and demographic data included age, sex, education level and total income sources were collected by questionnaire through an interview.

Health History

Health history include current health status and recent complaints and disease,

Anthropometric measurements

The anthropometric measurements Body weight, height, arm circumference and waist circumferencewere measured. Body mass Index(BMI) was obtained by calculating weight in kilograms/ square height in meters(kg / m2) The volunteers are considered obese since body mass index was greater than 30 ***(Garrow and Webster, 1985)***.

Dietary Habits

Information about dietary habits of studied persons were collected during an interview with patients and including method of meals cooking, number of daily meals, snacks,salt, sugar preference and source of nutritional information.

Daily dietary data

The 24 hours recall method and dietary history were used.The dietary history included all food items, the amount and frequency of consumption. The 24-hour recall include all food items taken during the last 24 hours in a semi quantitative method.

Assessment of nutrient intake from food consumption

The daily food intake had been assessed from the data collected by the 24-hour recall method for seven days. Portion size had been converted into equivalent weights. Kinds and amounts of the consumed food per meal had been tabulated. Nutrient values were derived from standard reference tables ***Food and Nutrition Board (2008) and NNI Food Composition Table's in Cairo (2006)***. ***(Recommended Dietary allowances (RDA, 2010)***

Assessment of biochemical parameters

Serum testosterone (T) and dehydroepiandrosterone (DHEA) levels were evaluated, according to the methods of ***Tietz (1995)*** and ***Longcope (1996)*** using kits provided by Rock Diagnostics GmbH-D-68298. Estrogen (E2) and prolactin (PRL) were estimated by Enzyme linked Flurorescent Assay (ELFA) technique using kits of Biomerieux, as described by ***Dupont et al. (1991)*** and ***Sapin & Simon (2001)***. Leptin was estimated by ELISA technique using commercially available kit (DRG instruments, Gmbh, Germany), according to the method of **Considine and Siha *(1996)***. Serum T/E2 ratio was calculated as T in ng/ml divided by E2 in ng/ml ***(Talalay, 1962)***. Serum (TG), (TC), (HDL-c), (LDL-c) and (VLDL-c) were determined according to the methods described by ***Fossati and Principe (1982), Richmond (1973), Allain (1974), Castelli et al., (1977), Lee and Nieman (1996).***For female hormones,the procedure described in the hormone assay kits was used according to the principle highlighted by ***Tietz (1995)*** for prolactin, estradiol and progesterone while that of ***Uotila et al. (1981)*** was used for luteinizing and follicle stimulating hormones.

Statistical analysis

The first step done in order to analyze the collected data was to tabulate all the raw values for each variable, and for obese females and males and their significances were also identified ***(SPSS, 2008)***.

Results and discussion

The socio-demographic characteristics of those participating in the study are shown in Table 1. There were 100 (50%) males and 100 (50%) females. All of them were single. Only 45% of the sample lived in rural areas whereas 55% lived in urban regions 35% had secondary education and 65% had academic education. For parental income, the majority of sample had income between 3000 to less than 5000 EP (60%), 23.5% had income between 5000 to less than 7000 EP and the rest of the group (16.5%) had more >= 7000 EP. Concerning family history of obesity, 25% and 35% of sample had obese father or mother respectively, 25% had obesity for both parents while the lowest percentage (15%) had obese grandparents.

The higher risk for obesity was due to the unhealthy dietary practices (i.e., a diet high in fat and low in fruits and vegetables) and sedentary lifestyles. According to a report from the Center for Disease Control and Prevention, adults living in rural counties are more likely to be obese than adults living in urban counties. Obesity prevention is important regardless of where the person live, but it may be more difficult in some areas than in others ***(De Silva et al., 2015)***. This was also consistent with a previous study that had a higher prevalence of obesity among those who had higher education , urban, high income and high social status. ***Drewnowski et al.(2014*)** study, census tract level home values and college education were more strongly associated with obesity than household incomes. For each in median home values, the census tract obesity prevalence was 2.3% lower. The three socioeconomic status factors together explained 70% of the variance in census tract obesity prevalence. There was a pattern that the risk of obesity was greater among city residents with higher education. It seems possible that the education level may be complicating the relationship between dietary behavior and obesity. On the one hand, residents with a higher education level are more likely to endorse health ideals such as a healthier diet or physical activities to preserve a good body image, and linked to a lower prevalence of obesity among city residents, and the result was consistent with previous studies. On the other hand, a higher education level may be associated with clerical work or increased sitting time among township residents and rural residents, which one might expect would increase the risk of obesity; thus, we could not find the effect of education level on the risk of obesity in a township and rural area. In addition, this inconsistency between city and township residents and rural area residents was similar to the opinion that an initial increase from low social economic status to mid-level social economic status was associated with worse health outcomes and behaviors; however, the continued increase from mid-social economic status to high social economic status saw returns to healthy outcomes and behaviors ***(Misra et al.,2019)*.**

Several factors can play a role in gaining and retaining excess weight. These include diet, lack of exercise, environmental factors, and genetics. Obesity occurs in families according to a clear inheritance pattern caused by changes in a single gene. The most commonly implicated gene is MC4R, which encodes the melanocortin 4 receptor. Changes in MC4R that diminish its function are found in a small fraction (<5%) of obese people in various ethnic groups. Family health history reflects the effects of shared genetics of obesity and environment among close relatives***(Qasim, 2018 and Meyre&Choquet, 2020)***.

Table (1)

Socio-economic and demographic data of the studied sample

Socio-economic and demographic data		Number (No)	Percent %
Sex	Male	100	50
	Female	100	50
Marital Status	Single	200	100
	Married	0	0
Residence	Rural	90	45
	Urban	110	55
Education level	Secondary	70	35
	Academic	130	65
Parental income (EGP)	3000 to < 5000	120	60
	5000 to < 7000	47	23.5
	> 7000	33	16.5
Familymembers with history of obesity	Father	50	25
	Mother	70	35
	Both of them	50	25
	Grandparents	30	15

The mean age and sample pocket money was tabulated in table(2) which showed that the mean age of sample was 23± 1.45 y for male and23±2.05y for female. The mean pocket money was 228.9±12.93 EP for a week in case obese male and for female, the mean value was210.6±10.33. From this table, it could be observed that the mean pocket money for each gender was about 219.75 EGP and this was a main factor effect on food choice and the amount of food which eat especially out of home like high caloric snacks ***(De Silva et al., 2015)***. The results showed that the amount of pocket money received by children was significantly associated with few variables like age, gender and total income of the family. ***Bonke (2013)*** studied young Danish children, it showed similar results where income of parents was related to amount of pocket money received.

Table (2)

Socioeconomic status for studied group

Category	Male Mean+SD	Female Mean+SD
Age (Year)	23^{a}±1.45	23^{a}±2.05
Pocket money (EGP)/week	228.9^{a}±12.93	210.6^{b}±10.33

The eating habits of obese male and female status are shown in Table 3. Obese participants differed by sex in food habits status, 59% and 53%from male and female respectively had dinner as the main meal. 55% of the obese females eat 4 meals, whereas 39% for obese males. Furthermore, 83% and 73% obese female and obese males respectively used to eat meals at irregular times. Most of males (30 %) used to sleep 4 h. while 37% females used to sleep 5h.The majority of females and males used to eat outside their houses (83 and 72% respectively). 74% of females and 61% males often eat their meals while watching T.V and the most of the males and the females (68 and 76%) used to eat their meals in ten minutes time.68% from males and 60% obese females like starchy and sugary foods as favorite one and the majority used to eat fast food more than 2 times per week (42% male and 49% female). Most of them preferred quantity rather than quality of food. As shown in the same table, the most frequently consumed drinks by both groups were carbonated beverage (43 and 45% for males and

females respectively), followed by caffeine drinks, (20% each) followed by fruits juice, water (17% & 15%) and the highest used herbs drinks (5 and 4% respectively). The highest percentages of girls ate chocolate as the favorite snacks contrary to males who like spicy snacks.Recent hypotheses in the scientific community suggest the current obesity epidemic is being driven largely by environmental factors (e.g., high energy/high fat foods, fast food consumption, television watching, "super-sized" portions, etc.) rather than biological ones. Obesity is generally caused by eating too much and activity too little. If you consume high amounts of energy, particularly fat and sugars or starchy food, but do not practice exercise and physical activity,the surplus energy will be stored by the body as fat.Studies have shown that over the past four decades, consumption of food eaten away from home has also risen alarmingly. It is well known that eating out of homes may lead to excess calorie intake and increases the risk of obesity because of the increased energy density of foods. This consumption could increase the risk of obesity and obesity-related diseases as a major public health issue ***(Seravalle& Grassi,2017 and Popkin et al., 2020)***.

Table (3)

Food habits status for the studied group

Habits stas		Male (100)		Female(100)	
		Frequency	Percent %	Frequency	Percent%
The main meal	Breakfast	16	16	20	20
	Lunch	25	25	27	27
	Dinner	59	59	53	53
Number of meals per day	1	7	7	5	5
	2	20	20	12	12
	3	25	25	21	21
	4	39	39	55	55
	More	9	9	7	7
Eating meals at irregular times	Yes	73	73	83	83
	No	27	27	17	17
Number of hours of sleep	4	30	30	16	16
	5	25	25	37	37
	6	17	17	30	30
	7	10	10	7	7
	8	10	10	6	6
	More than 8	8	8	4	4
Eat outside home	Yes	72	72	83	83
	No	28	28	17	17
Eating period	≤10 min	32	32	24	24
	>10 min	68	68	76	76
Eat while watching television	Often	61	61	74	74
	Occasionally	29	29	20	20
	Never	10	10	6	6
The favorite food	Meat products	12	12	25	25
	Sugary and starchy foods	68	*68*	60	60
	Fruit and vegetables	10	10	8	8
	Dairy	10	10	7	7

Habits stas		Male (100)		Female(100)	
		Frequency	Percent %	Frequency	Percent%
	products				
Frequency of eating fast food	Never	6	6	7	7
	Once a week	`24	`24	21	21
	More than 2 times/week	42	42	49	49
	1–2 times/month	23	23	23	23
Do you distinguish foods, especially in the family	Quantity	68	68	60	60
	Type	32	32	40	40
The favorite drinks	Carbonated drinks	43	43	45	45
	water	15	15	21	21
	Fruit juice	17	17	10	10
	Caffeine drinks	20	20	20	20
	Herbs drink	5	5	4	4
The favorite snacks	Biscuits	11	11	8	8
	Cakes	13	13	17	17
	Candy	17	17	21	21
	Chocolate	21	21	29	29
	Spicy snacks	28	28	17	17
	Puffed food	10	10	4	4

The results of table (4) represent the means and standard deviations of nutrients intakes compared with RDA . It could be noticed that Calories, Total fat, Carbohydrate, Phosphorus, potassium, vitaminA,B2, cholesterol and niacin intake by obese female and male were higher than the recommended dietary allowances while , total protein, vitamin E and vitamin B12 were little higher in obese females than recommended dietary allowances . The results of this study showed that animal fat, potassium, Zinc, Vitamin "A",Vitamin "C, and folate were significantly higher in obese male than female . There is no significant difference between the both gender in Total fat, Phosphorus,Plant iron, Vitamin "B2", Vitamin "B6" and Niacin.Severe calcium deficiency together with low vitamin D induce osteomalcia in adults***. (Gaman and Sheringtion, 1990)***.

According to ***Whitney and Rolfes (2011),*** excess of certain minerals (Ca, Fe, P, K& Na) may cause intoxication. For (Ca) symptoms are constipation, interference with other minerals and increased risk of urinary stone formation and kidney dysfunction For (Fe): nausea, vomiting, diarrhea, rapid, heartbeat, weak pulse, and shock, Excess (P) may draw calcium out of the body in being excreted, for (K): Muscular weakness and vomiting Exess (Na): may cause edema and acute hypertension For Zinc: it lowers, the body's Copper content which lead to degeneration of heart muscle accelerate atherosclerosis, vomiting, diarrhea, fever exhaustion reduced hemoglobin production and possibly renal failure.Hence , considerable increase of minerals intake than RDA may be also dangerous.

Table (4)

Means and standard deviations of nutrients intakes compared with RDA(2010)

Nutrients intake	Male (100)		Female (100)		LSD
	Mean ±SD intake	% RDA	Mean ±SD intake	% RDA	
Calories	3036.25 ^{b}±32.94	116.78	3297.75 ^{a}±70.87	164.88	22.50
Animal protein	19.83 ^{b}±11.85		23.71 ^{a}±5.46		3.97
Plant protein	34.37 ^{b}±9.34		43.64 ^{a}±10.96		7.85
Total protein g	54.20 ^{b}±11.63	96.78	67.35 ^{a}±16.87	120.26	6.79
Animal fat	92.28 ^{a}±10.97		68.46 ^{b}±7.89		5.606
Plant fat	37.01 ^{b}±14.51		56.55 ^{a}±10.90		7.334
Total fat g	129.29 ^{a}±9.27	167.90	125.01 ^{a}±15.02	162.35	11.843
Carbohydrate g	413.95 ^{b}±22.78	127.36	475.82 ^{a}±11.77	211.47	24.196
Fiber g	12.973 ^{b}±3.60	43.24	19.236 ^{a}±4.24	76.94	5.525
Calcium mg	501.90 ^{b}±15.19	50.19	569.11 ^{a}±18.57	56.91	31.353
Phosphorus mg	869.154 ^{a}±23.63	124.16	888.85 ^{a}±17.87	126.98	28.046
Animal iron	4.58 ^{b}±0.11		6.61 ^{a}±2.54		0.043
Plant iron	9.65 ^{a}±1.85		10.24 ^{a}±1.001		1.306
Total iron mg	14.23 ^{b}±3.80	88.94	16.85 ^{a}±5.35	93.61	0.586
Sodium mg	3884.05 ^{b}±14.51	168.87	4853.95 ^{a}±20.12	211.04	27.405
Potassium mg	3517.835 ^{a}±33.660	175.892	2877.335 ^{b}±55.51	143.87	26.227
Zinc mg	10.44 ^{a}±2.13	94.90	6.31 ^{b}±4.33	78.87	3.323
Magnesium mg	376.93 ^{b}±19.70	94.23	433.72 ^{a}±21.94	135.53	29.841
Vitamin "A"mcg	977.63 ^{a}±17.20	108.62	707.21 ^{b}±24.61	101.03	11.306
Vitamin "D"IU	103.58 ^{b}±10.94	17.26	202.27 ^{a}±11.34	33.71	2.775
Vitamin "E"mg	13.76 ^{b}±3.93	91.73	17.06 ^{a}±2.44	113.73	2.946
Vitamin "C"mg	45.85 ^{a}±9.16	50.94	37.31 ^{b}±5.12	49.74	6.945
Vitamin "B1"mg	0.62 ^{b}±0.09	51.67	0.98 ^{a}±0.01	81.67	0.071
Vitamin "B2"mg	1.78 ^{a}±1.057	136.93	1.81 ^{a}±2.011	146.54	0.40
Vitamin "B6"mg	1.14 ^{a}±0.47	87.69	1.16 ^{a}±0.95	89.23	0.49
Vitamin "B12"µg	2.07 b±1.43	90	2.58 ^{a}±1.04	112.17	0.041
Folatemcg	269.17 ^{a}±6.93	67.29	240.84 ^{b}±13.09	60.21	9.640
Cholesterol mg	522.72 ^{b}±12.50	174.24	602.84 ^{a}±18.65	200.94	13.642
Niacin mg	16.58 ^{a}±5.22	103.62	16.72 ^{a}±7.38	104.5	0.347

All results are expressed as mean± SD. Means followed by different superscripts within columns are significantly different (P≤0.05).

Data of table (5) explain means and standard deviations of the anthropometric measurements for obese male and female compared with the reference standard ***(Jellife, 1966)***. The average waist circumference (WC) of male as well as female was significantly higher than controls 128.86 ± 2.98vs. 163.33 ±3.21 ($p \leq 0.05$) respectively. Similarly, the average height among obese of both genders was

nearly identical to the controls (169.67 ±3.64 cm among males and 159.38 ±5.11 among females, respectively). The average BMI was different among obese male and obese female compared to controls (167 % and 224.02 %) ($p \leq 0.05$), whereas, the females had a significantly higher average BMI compared to males. The average Arm Circumference and Waist Circumference were higher for males and females than controls, however, the female had significantly higher average Arm Circumference and Waist Circumference compared to male. The average Triceps Skin Fold Thickness(mm) for both genders were significantly higher than controls while the meantriceps SkinFold Thickness of male was significantly higher than female.

Body mass index (BMI), and waist circumference (WC) are both effective indicators of obesity. Body Mass Index (BMI) is defined as body weight in kilograms divided by height in meters squared BMI is most commonly used to measure body fatness ***Balkau et al., (2007).***BMI when equal to or greater than 40.0 is severs obesity ***(Dong et al., 2011).*** From the obtained results, both of gender had BMI more than 40, the mean BMI of females was higher than that of males.

Table (5)

Means and standard deviations of anthropometric measurement compared with the standard

Anthropometric measurement	Male		Female	
	Mean±SD	%ofnormal	Mean±SD	%ofnormal
Weight (kg)	120.65 ^{b}±5.89	172.35	143.37^{a}±10.99	238.95
Height(cm)	169.67 ^{a}±3.64	100.120	159.38 ^{b}±5.11	99.936
Body Mass Index (W/H2)	41.75 ^{b}±2.65	167	56.004 ^{a}±4.02	224.02
Arm Circumference (cm)	33.93 ^{b}±2.44	130.5	37.24 ^{a}±2.66	134.607
Triceps Skin Fold Thickness(mm)	25.82 ^{b}±2.33	215.16	31.79 ^{a}±8.21	176.61
Waist Circumference (cm)	128.86 ^{b}±2.98	184.12	163.33 ^{a}±3.21	233.32

All results are expressed as mean± SD. Means followed by different superscripts within columns are significantly different (P≤0.05).

Table (6) shows the laboratory parameters tested for obese cases. The mean value of cholesterol, triglycerides and VLDL-c for obese males and females were higher than normal range while hemoglobin, was lower in both gender than normal range ***(Bunn et al, 2016)***. While fasting blood glucose for female were higher than normal. Hemoglobin mean levels were significantly lower in females' than the obese males (P≤ 0.05) and both are lower than normal. There is no significant different between the both groups in HDL-c levels and both are low. Studies have reported that obesity has an adverse effect on iron metabolism. Obesity is characterized by chronic, low-grade, systemic inflamation and anemia of chronic disease with elevated serum ferritin and decreased level of serum iron, transferrin saturation and hemoglobin. Obesity may disrupt iron homeostasis, resulting in iron deficiency anemia. The association between obesity and iron deficiency may be due to increased hepcidin levels mediated by chronic inflammation. Hepcidin is a small peptide hormone that functions as a negative regulator of intestinal iron absorption **(*Chooi et al., 2019 and Cappellini et al., 2020)***.being overweight raises the risk for type 2 diabetes, heart disease, and stroke. It can also increase the risk of high blood pressure, unhealthy cholesterol, and high blood glucose (sugar)

Obesity causes increased levels of fatty acids and inflammation, leading to insulin resistance, which in turn can lead to type 2 diabetes. Type 2 diabetes, known as non-insulin dependent diabetes, is the most common form of diabetes and accounts for approximately 90% of diabetic cases. The altered glucose homeostasis is caused by faulty signal transduction via the insulin signaling proteins, which

results in decreased glucose uptake by the muscle, altered lipogenesis, and increased glucose output by the liver. The mechanisms by which visceral obesity results in insulin resistance appear to be related to excess lipid accumulation in liver. This may be due to excess fatty acids from visceral adipose tissue draining into the portal vein ***(Hutton and Davidson, 2010)***The lipid abnormalities in patients who are obese include elevated serum triglyceride, VLDL, apolipoprotein B, and non-HDL-C levels. The increase in serum triglycerides is due to increased hepatic production of VLDL particles and a decrease in the clearance of triglyceride rich lipoproteins. People who are overweight or obese often have higher than normal levels of triglycerides. All these conditions may increase your risk for developing heart disease or of having a heart attack or stroke. The liver synthesizes TG-rich lipoproteins called very low density lipoproteins (VLDL), which increase postprandially when food derived TG and FFA reach the liver . The assembly of VLDL is almost identical to the synthesis of chylomicrons, but apo B100 is the structural protein of VLDL and its remnants, *i.e.*, intermediate density lipoproteins (IDL) and low-density lipoproteins (LDL***) (Atilgan et al., 2019)***.

Table(6)

The mean value of the laboratory tests conducted on the studied cases.

Variables	Male	Normal range	Female	Normal range
	Mean±SD		Mean±SD	
Hemoglobin (g/dl)	12.43 [a] ±0.89	13.2 to 16.6	10.11 [b] ±1.34	11.6 to 15
Fasting blood glucose (mg/dl)	170.54 [b] ±4.38	100 to 125	180.76 [a] ±4.76	100 to 125
Cholesterol (mg/dl)	200.34 [a] ±4.21	Less than 170	191.47 [b] ±10.78	Less than170
Triglycerides(mg/dl)	230.14 [a] ±11.95	Less than 150	219.13 [b] ±38.57	Less than150
LDL (mg/dl)	116.54 [b] ±5.34	less than 70	122.02 [a] ±16.04	less than 60
VLDL (mg/dl)	46.03±7.99 [a]	Less than 30	43.83 [b] ±9.04	Less than 30
HDL (mg/dl)	37.77 [a] ±2.91	close to 50	35.62 [a] ±6.12	close to 60

All results are expressed as mean± SD. Means followed by different superscripts within columns are significantly different (P≤0.05).

The hormonal profile of obese male are presented in table (7). Obese male showed an increase in serum levels of leptin,prolactinPRL and estrogen E2, accompanied with marked reduction in serum testosterone(T), dehydroepiandrosterone (DHEA) and T/ Estrogen E2 ratio. In particular, men with increased BMI are significantly more likely to be infertile than normal weight. Most studies have focused on alterations in the hormonal profiles as the main cause of infertility. It was noted that the increase in E2 levels in obese males is due to increased conversion of (T) owing to high aromatase availability with excess of adipose tissue. Consequently, a relation was found between the rise in aromatase activity and the increased (E2)with decline of both (T)and(T/E2) ratio, which is particularly responsible for developing infertility in the obese males ***(Roth et al., 2008)***.Increased E2 production in obese men leads to reduction in serum DHEA which is important for producing male androgens. Current decline of DHEA could be therefore relevant to the observed reduction of T levels in the present obesity model. Reduction of T may corroborate also to higher PRL levels with obesity ***(Hofny et al., 2010)***. PRL is a hormone produced by the pituitary gland in mammals. It is a key hormone in controlling milk production, however apart from lactation, PRL is closely involved in several physiological actions, such as reproduction. Synthesis and release of PRL is controlled by dopaminergic inhibition mechanism, however an altered dopaminergic system may present in obese men with rise of PRL secretion. Excessive PRL concentration is correlated with hypogonadism, impotence and infertility through interfering with production of FSH and LH which in turn will affect the testicular function with decrease in T release

(Bellentani et al., 2011). Leptin is a protein hormone that is synthesized and secreted by adipocytes. Its physiological role is to regulate appetite and body weight, but due to excess adipose tissue in the obese subjects' levels of leptin are often elevated leading to adverse effects, particularly on the male fertility.The most important of these effects is that increased levels of leptin may act as inhibitory signal for T synthesis through membrane receptors on testicular Leydig cells. Leptin receptors are also present on the plasma membrane of sperms, suggesting that leptin may directly affect sperm production independent of changes intestosterone production. So, elevated BMI and fat accumulation may lead to lowered T production indicating a negative relation between leptin and serum T ***Atilgan et al., (2019)***.

Table (7)

Hormonal profile of obese male

Serum parameters	Normal range	Male (Mean ±SD)
Testosterone(nmol/L)	10 to 35	2.49 ± 0.44
DHEA µg/dL	65 to 380	60.39 ± 6.23
Estradiol (pg/mL)	10 – 40	41.42 ± 0.83
T/E2 ratio	0.19	0.10 ± 0.004
Leptin (ng/ml)	0.5 - 12.2	31.42 ± 0.73
Prolactin (ng/ml)	less than 20	26.53 ± 1.96

As shown in table (8), the hormonal profile of obese female changes on comparing to normal levels.

The increase in body weight showed reduction in the levels of progesterone and estrogen while it increased the luteinizing hormone,follicle-stimulating hormone, Leptin and Prolactin. The quantity and distribution of body fat affect the menstrual cycle through a range of hormonal mechanisms Obese women have higher levels of the hormone leptin, which is produced in fatty tissue. This can disrupt the hormone balance and lead to reduced fertility. The risk of subfecundity and infertility, conception rates, miscarriage rates, and pregnancy complications are increased in the obese women. They have poor reproductive outcomes in natural as well as assisted conception and negatively impacting further development. In addition to reproductive hormones, obesity induced elevations in insulin, glucose, or free fatty acids, and changes in adipokines appear to impact the developmental competence of the oocyte. In obese women, gonadotropin secretion is affected because of the increased peripheral aromatization of androgens to estrogens. The insulin resistance and hyperinsulinemia in obese women leads to hyperandrogenemia. The sex hormone-binding globulin (SHBG), growth hormone (GH), and insulin-like growth factor binding proteins (IGFBP) are decreased and leptin levels are increased. Thus, the neuro-regulation of the hypothalamic-pituitary-gonadal (HPG) axis deteriorates. These alterations may explain impaired ovulatory function and so reproductive health. Obesity may impair reproductive functions by affecting both the ovaries and endometrium. The levels of luteinizing hormone (LH), and rostenedi-one,estrone, insulin, triglycerides, and very low density lipo-protein are increased and high density lipoprotein levels are decreased in obese women.

Leptin also inhibits LH-stimulated estradiol production by the granulosa cells. The other effect of leptin on reproductive functions is the regulation of early embryo cleavage and development. This may explain the poor reproductive outcomes in obese women. Adiponectin is the most common circulating

protein synthesized by adipose tissue. In obese women, unlike the other adipose tissue hormones, adiponectin levels decrease ***(Tong &Xu ,2014 and Travieso et al., 2019)***

Table (8)

Hormonal profile of obese female

Serum Parameters	Normal range	Female(Mean ±SD)
Progesterone pg/mL	50 - 170	34.60 ± 0.18
Estrogen pg/mL	30 to 400	24.90 ± 0.13
Iuteinizing hormone IU/L	5-25	26.03 ± 0.75
Follicle-stimulating hormone mlU/mL	less than 7.0	8.18 ± 0.003
Leptin (ng/ml)	0.5 - 15.2	33.06 ± 0.61
Prolactin (ng/ml)	less than 25	± 0.69

Conclusion

Obesity is one of the health Problem among both human sexes. Containing intake of energy-dense foods that are high in fat and sugars and a trend toward decreased physical activity, as a result of increasingly sedentary nature of work, changing modes of transportation and increasing urbanization. Significant quantities of data show a relationship between obesity diabetes mellitus, high cholesterol, atherosclerosis, anemia and the male or female hormones that affected their fertility. The author recommend that managing and preventing obesity by changing their food lifestyle and increase the physical effort.

References

Allain C Charles , Lucy S Poon, Cicely S G Chan, W Richmond, Paul C (1974) :
Fu Clinical Chemistry, Volume 20, Issue 4, Pages 470–475,
https://doi.org/10.1093/clinchem/20.4.470

Abinaya, K.R. and Pavitra, R.(2014):
Management of obesity and its related disease by herbal drugs. Int. J. Ayurvedic. Herb Med.,4:1470e9.

Agarwal, A.; Mulgund, A.; Hamada, A. and Chyatte, M.R. (2015):
A unique view on male infertility around the globe. Reprod. Biol. Endocrinol., 13: 37.

Atilgan, D.; Markoc, F.; Boztepe, O. and Suha-Parlaktas, B.(2019):
The effect of diet induced obesity on testicular tissue and serum oxidative stress parameters. Acta. Urol. Esp.,36(3):153e9.

Balkau, J.E. Deanfield, J.P. Despres, J.P. Bassand, K.A. Fox, S.C. Smith Jr., P. Barter, C.E. Tan, L. Van Gaal, H.U. Wittchen, C. Massien, S.M 9i (2007):
Haffner International Day for the Evaluation of Abdominal Obesity (IDEA):
A study of waist circumference, cardiovascular disease, and diabetes mellitus in 168,000 primary care patients in 63 countries

Bellentani, F.F.; Fernandes, G.S. and Perobelli, J.E.(2011):
Diet-induced obesity in rats leads to a decrease in sperm motility. Reprod. Biol. Endocrinol.,11:9e32.

Bonke, J.(2013):
Do Danish children and young people receive pocket money? The Rockwool Foundation Research Unit, 57.

Broughton, M.R. and Moley, K.H.(2017):
Obesity and female infertility: potential mediators of obesity's impact. Fertil. Steril., 107 (4) : 840-847

Bunn HF, Haney DN, Gabbay KH, Gallop PM (2016) .
Further identification of the nature and linkage of the carbohydrate in haemoglobinA1c. BiochemBiophys Res Commun. 67:103–9.

Castelli WP, Cooper GR, Doyle JT, Garcia-Palmieri M, Gordon T,Hames C, Hulley SB, Kagan A, McGee D, Vicic W, Zukel WJ(1977):
Distribution of triglyceride and total LDL and HDL cholesterol in several populations: The cooperative lipoprotein phenotyping study. J Chronic Dis,in press

Cappellini, Maria .Domenica, Fabio Nascimbeni, Simonetta Lugari, Elena Cassinerio, Irene Motta, Alessia Cavicchioli, Annalisa DallaSalda, Serena Bursi, Salvatore Donatiello and Vincenzo Spina (2020):
Liver steatosis is highly prevalent and is associated with metabolic risk factors and liver fibrosis in adult patients with type 1 Gaucher disease. Liver international WILEY, DOI: 10.1111/liv.14640 ,August 2020

Cosidine, R.V. and Siha, M.K.(1996):
Serum immunoreactive-leptin concentrations in normal weight and obese humans**.** N. Engl. J. Med., 334 : 292-295.(Chooi Yu Chung ,

Cherlyn Ding and Faidon Magkos 2019:
The epidemiology of obesity, Jurnal of Metabolism,Volume 92, March, Pages 6-10

De Silva, A.; De Silva, S. and Haniffa, R.(2015):
A cross sectional survey on social, cultural and economic determinants of obesity in a low middle income setting. Int. J. Equity Health, 14:6.

Drewnowski, A.; Rehm, C.D. and Arterburn, D. (2014):
The geographic distribution of obesity by census tract among 59767 insured adults in King County, WA. Int. J. Obes., 38:833–9.

Dong Dong, Xiaojian Shao, Naiyang Deng, Zhaolei Zhang (2011):
Nucleic Acids Research, Volume 39, Issue Pages 403–413, https://doi.org/10.1093/nar/gkq844

Dupont,A.; Dupont,B.; Cusan,L.; Tremblay, M. and Rioux, D.(1991):
Comparative endocrinological and clinical effects of percutaneous estradiol and oral conjugated estrogens asreplacement therapy in menopausal women**.** Malturitas, 13: 297-311.

Food composition tables, National Nutrition Institute,Cairo,A.R.E (2006)

Food and Nutrition Board (2008):
A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). The National Academies Press, Washington, D.C.ISBN0-309-08537-3.

Fossati, P and Prencipe, L(1982):
Clinical Chemistry, 28, 2077-2080

Garrow, J.S. and Webster, J.(1985):
Quetelet's index (W/H2) as a measure of fatness. *Int. J. Obes.*, 9(2):147–153.

Gaman, P.M. and Sherrington, K.B. (1990)
The Science of Food. An Introduction to Food Science, Nutrition and Microbiology. 3rd Edition, Pergamon Press, Oxford and New York, 104-115

Hofny, E.R.; Ali, M.E.; Abdel-Hafez, H.Z.; Kamal, D.;Mohamed, .E.E. and Abd El-Azeem, H.G.(2010):
Semen parameters and hormoal profile in obese fertile and infertile males. Fertil. Steril.,94:581e4.

Hutton JC, Davidson HW.(2010)
Cytokine-induced dicing and splicing in the β-Cell and the immune response in Type 1 Diabetes. Diabetes.;59(2):335–336.

Jellife, D. B.(1966):
Assessment of nutritional status of Community.Wld. Hlth. Org.Mono Ser. 53.Jungheim ES,

Lee R, Nieman D (1996)
Nutritional assessment, 2nd edn. Mosby, St Louis

Li, Y.; Lin, H.; Ping, P.; Yang, D. and Zhang Q.(2018):
Impact of central obesity on women with polycystic ovary syndrome undergoing in vitro fertilization. Biores. Open Access, 7 (1) : 116-122,

Longcope,C.(1996):
Dehydroepiandrosterone metabolism. J. Endocrinol., 150 : 125-127.

Meyre, D. and Choquet, H.(2020):
Genetics of Obesity: What have we Learned? *Curr.Genomics*,12(3):169-79.

Misra, A.; Sharma, R. and Gulati, S.(2019):
National Dietary Guidelines Consensus Group. Consensus dietary guidelines for healthy living and prevention of obesity, the metabolic syndrome, diabetes, and related disorders in Asian Indians. DiabetesTechnol.Ther., 13:683–94.

Mittal B, Awasthi S, Agarwal CG, Srivastava N, Prakash J . K121Q ENPP1/PC-1 gene polymorphism is associated with insulin resistance in a North Indian population. J Genet 2013.
Dec;92(3):571-576. 10.1007/s12041-013-0287-2

Popkin, B.M.; Du, S. and Green, W.D. (2020):
Individuals with obesity and COVID-19: A global perspective on the epidemiology and biological relationships. Obes Rev.,21(11):e13128.

Qasim A (2018):.
Obesity reviews : An official journal of the International Association for the Study of Obesity , 19(2) 121-149.

Rasanen, L. (1979):
Nutrition survey of Finnish rural children. VI Methodological study comparing the 24-hour recall and the dietary history interview. American Jamal of Clinical Nutrition, 32: 2560-2567.

Recommended Dietary Allowances: RDA(2010):

10th Edition. *Washington, DC: The National Academies Press.*

Rehman, R.; Abidi, S.H .and AlamF.b(2018):

Metformin, oxidative stress, and infertility: A way forward. Front. Physiol., 9 (1722):345-356.

Richmond W (1973) :

Clinical Chemistry, Volume 19, Issue Pages 1350–1356,
https://doi.org/10.1093/clinchem/19.12.1350

Roth, M.Y.; Amory, J.K. and Page, S.T.(2008):

Treatment of male infertility secondarytomorbid obesity. Nat. Clin. Pract. Endocrinol. Metab.,4:415e9. Richmond W : Clinical Chemistry, Volume 19, Issue 12, 1 December 1973, Pages 1350–1356, https://doi.org/10.1093/clinchem/19.12.1350

Sapin, R. and Simon, C.(2001):

False hyperprolactinemia corrected by the use of heterophillic antibody-blocking agent**.** Clin. Chem., 47 : 2184-2185.

Seravalle, G. and Grassi, G. (2017):

Obesity and hypertension. Pharmacol. Res.,122:1–7.

SPSS (2008):

Statistical Package for Social Science, Computer Software, IBM, SPSS Ver. 16.0 in 2008., SPSS Company, London, UK.

Talalay,P.(1962):

Hydroxysteroid dehydrogenase. Colowick, Kaplan (Eds.), Methods in enzymology, Academic Press, 512-516.

Tietz, N.W. (1995):

Clinical guide to laboratory tests(3rd ed), WB Saunders Co, Philadelphia, Pa, 186-188.

Tong, Q. and Xu, Y. (2014):

Central leptin regulation of obesity and fertility. Curr. Obes. Rep., 1: 236-44.

Travieso, J.L.; Carson, K.R. and Moley, K.H.(2019):

Obesity and reproductive functions. Obstet. Gynecol. Clin. North Am., 39: 479-93.

Uotila, M.; Ruoslathi, E.andEnvall, E.(1981):

Two-site sandwich enzyme immunoassay with monoclonal antibodies to human alphafetoprotein. J. Immunol. Methods, 42:11-15.Whitney, Eleanor Noss and Sharon Rady. Rolfes, Understanding Nutrition. Australia ; Belmont, CA, Wadsworth, Cengage Learning, 2011.

Whitney, Eleanor Noss and Sharon Rady. Rolfes (2011) :

Understanding Nutrition. 12 edition Australia ; Belmont, CA, Wadsworth, Cengage Learning,

دراسه المقاييس الجسمانيه لحالات السمنه والعادات الغذائيه وبعض الاختبارات المعمليه في الدم والهرمونات الجنسيه التي تؤثر علي الخصوبه

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الملخص العربي

الهدف من هذه الدراسة هو تحديد العلاقة بين السمنة البشرية والعديد من العوامل ذات الصلة التي قد تؤثر على الخصوبة عند كلا الجنسين. تُعرَّف السمنة بأنها تراكم غير طبيعي أو مفرط للدهون في الجسم ، وهي حاليًا واحدة من أكثر قضايا الصحة العامة إثارة للقلق ، حيث إنها مرتبطة بمجموعة واسعة من الأمراض والاضطرابات الخطيرة. تم إجراء الدراسه علي مائتي متطوع ومتطوعة من البالغين (100ذكور)و (100إناث) تتراوح أعمارهم بين 21 و 25 عامًا. تم إكمال استبيان حول عادات الأكل ، مع التركيز على عدد الوجبات اليومية ، وتكوين الوجبة ، وعادات نمط الحياة الخاملة. كما تم إجراء تقييم القياسات البشرية ، بما في ذلك الوزن والطول ومؤشر كتلة الجسم وسمك ثنيات الجلد والخصر ومحيط الذراع. كما تم تحديد بعض الهرمونات الجنسية الذكرية والأنثوية. أظهرت النتائج أن متوسط عمر الذكور كان 23 ± 1.45 سنة وللإناث 23 ± 2.05 سنة. كانت السعرات الحرارية ، والدهون الكلية ، والكربوهيدرات ، والفوسفور ، والبوتاسيوم ، وفيتامين A ، و B2 ، والنياسين مع تناول الكوليسترول من قبل المتطوعين الذين يعانون من السمنة المفرطة أعلى من البدلات الغذائية الموصى بها. كان مؤشر كتلة الجسم لكلا الجنسين أعلى من 40 ، وكان مؤشر كتلة الجسم للإناث أعلى من مؤشر كتلة الجسم للذكور. كان متوسط قيمة الكوليسترول والدهون الثلاثية و VLDL-c للذكور والإناث المصابين بالسمنه أعلى من المعدل الطبيعي. أظهر الذكور المصابيين بالسمنه زيادة في مستويات مصل اللبتين و PRL و E2 ، مصحوبة بانخفاض ملحوظ في هرمون التستوستيرون (T) و dehydroepiandrosterone (DHEA) ونسبة T / Estrogen E2. أظهرت السمنة لدى الإناث انخفاضًا في مستويات هرمون البروجسترون والإستروجين بينما تسبب زيادة في الهرمون اللوتيني والهرمون المنبه للجريب واللبتين والبرولاكتين. لذلك كان الاهتمام بالسمنة من المسببات المسببة لخصوبة الذكور والإناث وتغيير نمط حياة الشبان المصابين بالسمنه إناثا وذكورا.