ISSN 2321 – 7359 EISSN 2321 – 7367

discovery Cover the uncovered

Medical Science

Obesity - the new epidemic: an Indian perspective

Jahan Ara M[1*], Sailaja K[2]

1. Asst.Prof.in Zoology, Osmania College for Women, Kurnool – A.P., India
2. Asst.Prof. in General Surgery, Rangaraya Medical College, KAKINADA – A.P., India

*Corresponding author: Mail: Jahanara.phd@gmail.com, mobile no: (+91)9866153569

Received 11 June; accepted 23 July; published online 01 August; printed 16 August 2013

ABSTRACT

Obesity is a major global public health hazards in the 21st century. Obesity is a part of the spectrum of malnutrition with excess weight gain for the given age and habitus. In India all ages and both sexes have a high prevalence of obesity. Obesity invites disability disease and premature death. Obesity leads to breathlessness on exertion and predisposes a person to diseases like atherosclerosis, high blood pressure, stroke diabetes, gall bladder diseases, varicose veins and osteoartheritis of weight bearing joints. South East Asian countries including India once considered poor and developing countries also presently facing the epidemic of obesity as a health problem. This needs extensive studies to evaluate the incidence of obesity and its causative role for increased morbidity and mortality. Prevalence of obesity in children, adolescence and pregnant woman has greatly influenced that diseases pattern of general population, which needs urgent consideration due planning for the treatment and prevention of obesity. Health care programs need to include public awareness regarding obesity as a causative factor for chronic diseases like diabetes, cardio-vascular problem and cancers.

Keywords: Obesity, risk factor, Indian, physical activity, diet, cardiovascular disease, diabetes mellitus.

1. INTRODUCTION

Obesity rates are rising worldwide and affecting both the developed and developing countries. WHO recognized obesity as a global epidemic. Obesity is a state in which there is a generalized accumulation of excess adipose tissue in the body, leading to more than 20% of the desirable weight. Over weight is a condition where the body is 10-20% greater than the mean standard weight for age, height and sex. Obesity is based on the degree of excess fat, obesity is acomplex multifactorial chronic disease developing from interactive influences of numerous factors-social behavioral, psychological, metabolic, cellular and molecular. It is defined by body mass index (BMI) and further evaluated interms of fat distribution via the waist-hip ratio and total cardiovascular risk factors. BMI is closely related to both percentage body fat and total body fat (Chopra et al. 2002). Normal BMI ranges between 18.5 and 25. An average BMI of a population should be between 21 or 22. BMI less than 18.5 denotes chronic under nutrition.

BMI	*Classification*
<18.5	Under weight
18.5-24.9	normal weight
25.0-29.9	over weight
30.0-39.9	Grade I obesity
35.0-39.9	Grade II obesity
>40.0	Grade III Obesity

BMI is calculated by dividing the subjects mass by the square of his or her height.
Metric BMI = Kilograms / meters^2.

WHO estimates that approximately 1.2 billion people in the world are overweight, of which atleast 300 millions are obese. The waist circumference and waist to hipratios are useful for estimation of central obesity. The central obesity is directly correlated with chronic degenerative diseases, especially metabolic syndrome. A ratio of more than 0.9 among men and more than 0.8 in women is associated with increased risk of several chronic diseases in Asian Indians. The waist circumference cut off levels for Asian Indians are 80cm for women and 90cm for men. Genetic Inheritance probably influences 50-70%, a person's chance of becoming fat, within families the chance is 80% if both parents are obese and 50% if one parent is obese. More than 20genes and 12 chromes have been implicated in obesity. A mutation in the human gene for the B_3 receptor in adipose tissue involved in lipolysis and thermogenesis markedly increase the risk of obesity. Many genes play a role in energy homeostasis (UFP1, UCP_2, UCP_3) food intake regulation [MC_3R, MC4R, CCKAR], appetite (NPYRS) and ultimately obesity (ASIP, CPE, LEP, LEPR, TUB, POMC)in mammals. People with two copies of the FTO gene (fat mass and obesity associated gene) have been found on an average to weight 3.4kg more and have a 1.67 bold greater risk of obesity compared to those without the risk allele. Obesity can occur at any age in either sex. Present study has shown more females are found to be overweight due to hormonal predisposition (kuriyan, 1998).

In present date obesity is found during middle age when physical activity decreases without corresponding decrease in food consumption. Another interesting point noted in present study is 38.7% obese are also found to be having hypothyroidism. The energy required by a person for basic metabolic processes is relatively constant throughout the day. The growth spurt during childhood and adolescence needs extracalories, pregnant and lactating women also belong to such a special group who need extra calories. A normal healthy human being with average physical activity will not require additional calories. A normal healthy human being with average physical activity will not require additional calories but most people consume daily intake of

Jahan Ara et al.
Obesity - the new epidemic–an Indian perspective,
Medical Science, 2013, 1(2), 34-38,
http://www.discovery.org.in/md.htm

calories through two or three meals and more than one snacks. If the intake of metabolic fuels is consistently greater than energy expenditures, the surplus is stored largely as triacylglycerol in adipose tissue, leading to the development of obesity and its health hazards. Obesity gene is expressed in the fat cells and codes for the protein 'Leptin'. It acts as a hormone in the hypothalamus. It promotes negative energy balance by suppressing appetite and increasing the energy expenditure. People with genetic defect of leptin show signs of poor appetite, control, constant hunger and eat more and may gain weight. The concentration of leptin in blood is usually proportional to the amount of stored fat. Leptin is regarded as a body weight regulatory hormones, it binds to a specific receptors in the brain and functions as a lipostat. When the fat stores in the adipose tissue are adequate, leptin levels are high, this signals to restrict the feeding behaviour and limit fat deposition. Leptin stimulates lipolysis and inhabits lipogenesis, any genetic defect in leptin or its receptor will lead to obesity. 'Leptin' and 'ghrelin' are considered to be complementary in their influence on appetite, with ghrelin produced by the stomach modulating short-term appetitive control i.e… to eat when the stomach is empty and to stop when the stomach is stretched. While leptin and ghrelin are produced peripherally, they control appetite through their actions on the central nervous system, they act on the hypothalamus, a region of the brain central to the regulation of food intake and energy expenditure. There are several circuits within the hypothalamus and ventromedial hypothalamus (VMH) the brain's feeding and satiety centres respectively. The arcuate nucleus contains two district groups coexpresses neuropeptide Y(NPY) and agouti-related peptide (AgRp) and has stimulatory inputs to the LH and inhibitory inputs to the VMH. The second group coexpresses pro-opiomelanocortin (POmc) and cocaine and amphetamine – regulated transcript (CART) and has stimulatory inputs to the VMH and inhibitory inputs to the LH. Consequently WPY/AgRp neurons stimulate feeding and inhibit satiety, while POMC/CART neurons stimulate satiety and inhibit feeding. Both groups of arcuate nucleus neurons are regulated in part by leptin. Leptin inhibits the NPY/AgRp group while stimulating the POMC/CART group. Thus a deficiency in leptin signaling, either via leptin deficiency or leptin resistance leads to over feeding and may account for some genetic and acquired forms of obesity (Bonded et al. 2002).

Insulin effects in the central nervous system to inhibit food intake acts in the periphery to ensure the efficient storage of incoming nutrients. Insulin and leptin are released and circulate in the blood stream at levels that are proportionate to body fat content. Insulin stimulates leptin production, which acts centrally to reduce energy intake and increase energy expenditure. Decreased insulin and leptin production during the consumption of high fat diets could help contribute to the obesity promoting effects of dietary fat. The formation and utilization of reserves of triacylglycerol and glycogen and the extent to which tissues take up and oxidize glucose are largely controlled by the hormones insulin and glucagon (Ramachandran et al. 1986). Obesity located in the central abdominal parts of the body is statistically associated with a number of metabolic derangements such as insulin resistance, hyperinsulinemia, non insulin dependent diabetes mellitus, hyperlipidimia and hypertension. The mechanism might be due to the lipolytically sensitive abdominal depots providing excess free fattyacids to a muscle tissue that has a decreased capacity for their oxidation. The excessive exposure of tissues to fattyacids impairs insulin function and increases blood insulin levels, ultimately reduced insulin sensitivity making a person insulin resistant. This is inturn result in hyperinsulinemia which might be starting point for most of the other metabolic derangements. Majority of Indians suffer from diabtes due to insulin resistance. Trunkal obesity is associated with high blood insulin levels which also increases the activity of lipoprotein lipase and fat storage and may indirectly cause over eating. Fat cells size increase (hypertrophy) in case of upperbody obesity, where as lower body fat diposition is by increase in number of cells (hyperplasia). Reducing the number of cells in the lowerbody hyperplasia depot is difficult compared to reduce trunkal obesity where there is increase in size of adipocytes. Heredity plays a role in fat distribution; particularly abdominal obesity is a risk factor for increased mortality, hypertension, type-2 diabetes mellitus, hyperlipidemia, hyperglycemia, hypercholesterolemia premature atherosclerosis and various endocrine dysfunctions. Malnutrition in earlylife is believed a play a role in the rising rates of obesity in the developing world. Endocrine changes that occur during periods of malnutrition may promote of fat once more food energy becomes available (Goldberg et al. 2001). The thrifty gene hypothesis was proposed by James V.Neel in 1962, while studying diabetes but the idea soon expanded to encompass obesity. According to this theory, the thrifty genotype would allow individuals to fatten more quickly during times of abundance, so intimes of famine, they survive better. But in modern societies with constant abundance in food, this genotype makes people fatter. The result is wide spread chronic obesity and related health problems like diabetes. The problem of obesity with respect to its prevalence, health consequences aetiology and preventions are discussed.

2. MATERIALS AND METHODS

The urban slum colonies of Kurnool situated in Kurnool Andhra Pradesh was selected for the study. A total of subjects (134 males and 166 females) were randomly selected with the help of questionnaire cum interview technique from the areas Khander, Chidambaram, Kuruva Fortline and Laxminagar respectively, the project work was completed within a period of one year April 2012 to March 2013 - 134 men and 166 women included children were interviewed from high, middle and low income groups of the five areas. All the subjects were fully informed about the purpose of the study. Those detected as having any disease were provided with complete medical investigation and management in the referral hospital. A detailed questionnaire incorporating demographic profile, relevant sympotoms (tobacco and alcohol consumption) food frequency questionnaire was administered. A complete physical examination was performed on all the subjects. Blood pressure was recorded in sitting position after 5min. rest with a mercury sphygnaomanometer according to the standard guidelines. If one abnormal reading was observed, a second reading was also recorded.

2.1. Anthropometric Measurements

For the assessment of obesity height and weight measurement were taken on each subject using quetelet index. The practical and clinical definition of obesity is based on body mass index (BMI) therefore the value of BMI was calculate for each subject BMI=weight (kg) / Height $(M)^2$. The suggested clinical limits of BMI by 'WHO' were utilized for the assessment malnutrition. Waist circumstance was measured midway between iliac crest and lowermost margin of the ribs and the girth was measured at the maximum circumference of buttocks. Waist circumference is the most practical tool to evaluate a patient's abdominal fat before and during weight loss treatment. High risk waist circumference taken for men > 40 inches (>102cm) women; >35 inches (>88cm).

2.2. Bio Chemical Samples and Analysis

A fasting venous blood sample was obtained after anthropometry and physical examination for blood glucose and lipid and thyroid profiles Estimation of total cholesterol (TC) serums triglycerides (TG) and high density lipoprotein cholesterol (HDL-C), Low density lipoprotein cholesterol was performed on the sample drawn after 12hrs over weight fact. TC was estimated with the ferric chloride method. The method described by Rosenberg and Gottfried was used for the determination of TG. After precipitation of very low-density lipoprotein cholesterol and Low-density lipoprotein cholesterol (LDL-C) from the serum by phosphotungstic and megnisium chloride, the supernatant was taken and HDZ-C estimation performed by the method described for TC. The

Jahan Ara et al.
Obesity - the new epidemic–an Indian perspective,
Medical Science, 2013, 1(2), 34-38,
http://www.discovery.org.in/md.htm

Table 1
Profile of Various Measures of Obesity; Male – 134 - Female – 166

S.No.	Types of Obesity	Male	Female	Total	Total Percentage %	Male %	Female%
1	Normal	26	06	32	10.7%	19.4%	3.6%
2.	Over weight (OW)	26	24	50	16.7%	19.4%	14.4%
3.	Grade-I (G-1)	24	74	98	32.7%	17.9%	44.6%
4.	Grade-II (Gr-II)	32	32	64	21.3%	23.8%	19.3%
5.	Grade-III (G-III)	26	30	56	18.7%	19.4%	18.1%
6.	WHR	14	80	94	31.3	10.4%	48.2%

Table 2
Percentage prevalence of various measures of Obesity; Male – 134 - Female – 166 = Total: 300

S.No.	Variables	Male	Female	Total	Total Percentage %	Male %	Female%
1	Heredity Obese	66	58	124	41.3	49.3%	34.9%
2.	Health Hazards Obese	14	32	46	15.3%	10.5%	19.3%
3.	Rich diet obese	66	64	130	43.3%	49.3%	38.6%
4.	Type-2 Diabeties (DB)	48	48	96	32%	35.8%	28.9%
5.	Hypertension (B.P)	66	78	144	48%	49.3%	47%
6.	Hypothyroidism (HT)	52	64	116	38.7%	38.8%	38.6%
7.	Hyperchdesterimea (HC) (Trighycerides)	60	66	123	41%	44.8%	39.8%
8.	LDL	62	58	120	40%	42.2%	34.9%
9.	HDL	41	59	100	33.3%	30.6%	35.5%
10.	Cardiac Incidence (CI)	36	22	58	19.3%	26.9%	13.3%
11.	Ostearthritis (OA)	44	82	126	42%	32.8%	49.4%
12.	Obstructive sleep apnea (OSA)	22	32	54	18%	16.4%	19.3%

value of LDL-C was calculated by using Fried Wald's equation. Estimation of T_3 (Total Triiodothyronine), Total thyroxine (Tu) and thyroid stimulating hormone (TSH) was also performed on the sample drawn after 12hrs overnight fact. T_3 and T_4 was estimated by competitive chemiluminescent Immuno Assay and TSH was estimated by Ultra sensitive sand wich chemicuminescent Immuno Assay.

2.3. Definitions

Obesity was defined as BMI > 25kg/m^2 WHR of >0.95 for males and >0.80for femals was considered to indicate abdominal obesity. Persistent elevation of blood pressure >140/90mm Hg was defined as hypertension. Dyslipidemias were defined by the Criteria laid down by national cholesterol Education program. Adult Treatment Panel. Type 2 diabetes mellitus and impaired fasting glucose were diagnosed according to the diagnostic criteria of the American Diabetic Association. TSH more than 5µ I.U. / litre is diagnosed as hypothyroidism.

2.4. Statistical Methods

Data were entered in observation Table 1& 2. For both anthropometric and biochmicalf parameters, percentage were noted. Mean and standard deviation then summarized the variable combined Geometric mean and Harmonic mean applied to compare the difference in various anthropometric parameters amongst males and females.

3. OBSERVATIONS

In this study 300 subjects (134 males and 166 females) had complete records of clinical profile and Biochemical tests (Figures 1-5). The anthropometric profile of the study population has been shown in Table 1. By the measurement of BMI alone. Obesity was more prevalent in females than males. Males were 19.4% and females were 14.4% followed by obese grade I males were 17.9% and females were 44.6%. In addition 23.8% were grade II males and 19.3% females. The overall prevalence of grade III males were 19.4% and females were 18% respectively. The mean ± standard deviation BMI was enumerated 34.9 in male and 42.8 in female (Table 1). The statistical analysis witnessed that the mean value of BMI were difference between male and female. The overall prevalence of WHR was higher in females than males. High WHR was observed in 48.2% of the females and 10.4% of the males. Prevalence of heredity obese was observed more in males 38.8% than in females 33.4%. In present study health hazards obese were more in females 19.3% than males 10.5% but incase of higher economic status obese were significantly higher in males 42.3% than females 37.3%. Impaired fasting glucose IFG was observed more in females as compared to males (Table 2). Type-2 diabetes was observed in 35.8% and 28.9% of males and females respectively, the over all subjects being 32% prevalence of hypertension was 49.3% in males and 47% in females (Table 2). Hyper cholesterolaemia was observed in high number males 44.8% and 39.8% females and similarly equally high number of subjects had high LDL-C level 42.2% males and 34.9% females (Table 2). Further 30.6% males and 35.5% females subjects showed low levels of HDL-C (Table 2). Prevalence of hypothyroidisim in females were 38.8% almost equal with males 38.6%. In the present study it is interesting point noted that most of the obese were due to hypothyroidism. The prevalence of joint pains was more 49.4% in females than in male 32.8%. In females it was noticed to be high even in the younger age group and the magnitude of increase was mostly observed in highest grade II obese.

4. PREVENTIVE MEASUREMENTS

It's easier to prevent obesity than to cure it. In addition to a healthy life style, people can help prevent overweight and obesity by balancing a healthy diet with enough physical activity. It is important to create balance between the food eaten (Energy IN) and physical activity (Energy out). Prevention should begin in early childhood. Obesity is harder to treat in adults than it is in children. The control of obesity centres round weight reduction. This can be achieved by dietary changes increased physical activity and a combination of both. If the person is at risk of becoming obese, currently over-weight or at a healthy weight, can take steps to prevent unhealthy weight-gain and related health problems. Regularly monitoring weight can help the person detect small weight gains before it become big problems. Regular exercise need to get 30 minutes of moderate intensity activity, which include fast walking and swimming per day to prevent weight gain obsese should avoid saturated fat and limit sweets and alcohol and should take low-calorie, nutrient dense foods such as fruits, vegetables and whole grain. Yoga is a uniquely Indian sustain of health living. In addition to improving physical activity Yoga can help in preventing stress. Stress has been linked to diabetes, obesity and cardiovascular diseases meditation may even reduce cortisol and catecholamine responses. Stress has been shown to be linked to overheating and a sedentary life and alleviating stress is definitely good for health. However, implantation of yoga should take place in

Jahan Ara et al.
Obesity - the new epidemic–an Indian perspective,
Medical Science, 2013, 1(2), 34-38,
http://www.discovery.org.in/md.htm

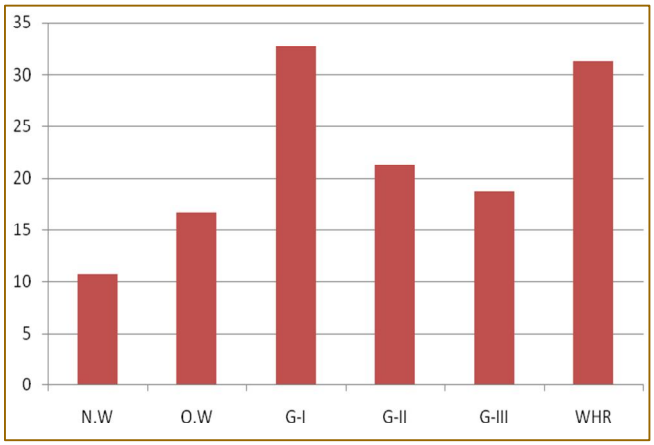

Figure 1

Total Percentage of different categories obese; N.W=Normal Weight – O.W – Over weight; GI-Grade I, G-II, GradeII; G-III, Grade-III

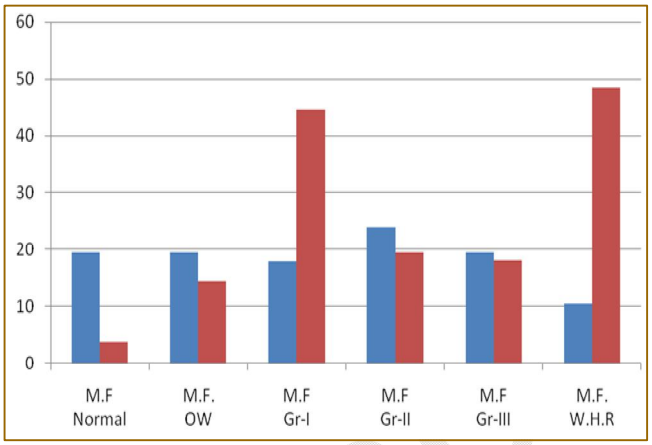

Figure 2

Male and Female percentage of different obese

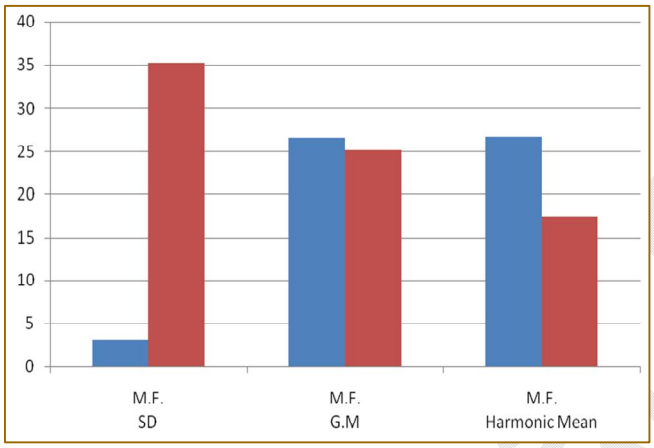

Figure 3

Standard Deviation, Geometric mean & Harmonic mean of Male and Female Obese; S.D= Standard Deviation: G.M-Geometric Mean, H.M. Harmonic Mean

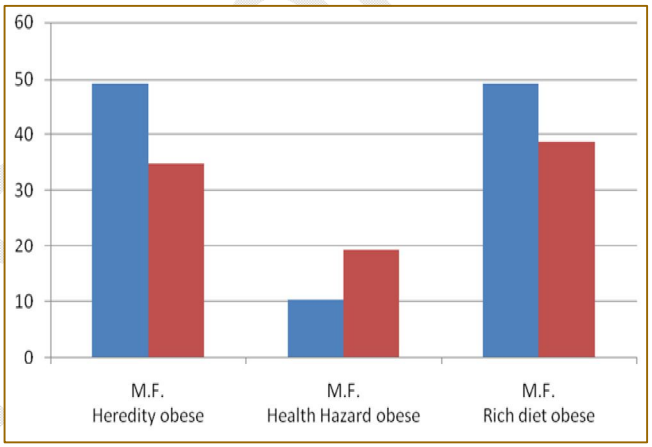

Figure 4

Percentage of different categories obese

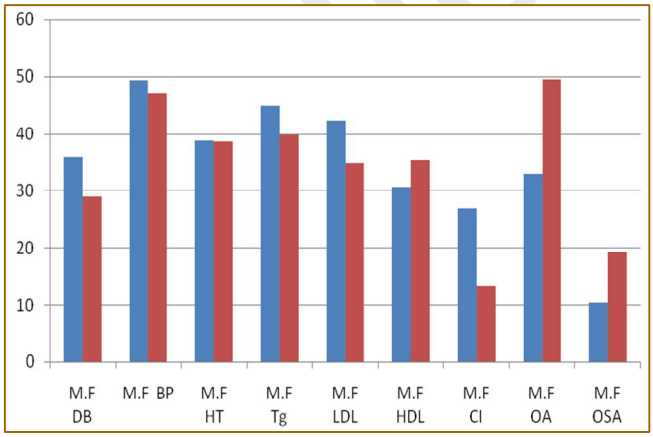

Figure 5

Percentage Prevalence of various measures of obesity; Db: Diabetic, BP: Blood Pressure; HT: Hypothyriodism; Tg-Triglycerides; LDL-Low density Lipids; HDL-High density Lipid; CI:cardiac Incidence;OA; Osteoartheritis

the schools and colleges of India. Indian folkdance system Indian dances are physically energy consuming and help to prevent obesity. Antiobesity drugs may be taken to reduce appetite or inhibit fat absorption but it is not much advisable.

5. DISCUSSION

In the present study the high prevalence of dyslipidaemia was striking. In male 44.8% and 31.8% females subjects had hypercholestermeia in this area, approximately 41% of the subjects suffering with high levels of total cholesterol in the current study overweight and obesity was associated with many diseases particularly heart diseases 26.9% in male and 13.3% in female, type 2 diabetes ranged was 35.8% in male 23.9% in female respectively. In the present study 16.4% males and 19.3% females were observed breathing difficulties during sleep obstructive sleep apnea. Osteoarthrities suffers were 19.3% and 16.4% in females and males were recorded. Obesity is the 22nd century epidense of the human society man tapped the agricultural and industrial resources of the earth thus changed the EARTH a better place to live in invention of antibiotics and vaccines gave victory over most of the diseases, with the result excess availability of food grains and better health brought OBESITY to the forefront. Apart from developed nations developing countries of south-east Asia also joined the OBESITY CLUB (Troiano et al.1998). In this backdrop study of obesity its prevalence, causes and effects is very relevant. Obesity is named the syndrome X, the long standing effects of obesity seem to involve every organ system that is phylogenetically evolved in Human beings. Eating habits and cooking methods are personal but obesity

Jahan Ara et al.
Obesity - the new epidemic–an Indian perspective,
Medical Science, 2013, 1(2), 34-38,
http://www.discovery.org.in/md.htm

has its roots in them. Obesity and the resultant disease spectrum of Hypertension, Type-2 Diabetes. Dyslipidemia with cardiac or / an neurological events contribute to the major share of HEALTH CARE facilities and thus the economy of the state. Obesity predisposes to clo-rectal carcinoma in males, breast and uterine cancers in females. So, we put forward this study to the general public to raise an alarm against this GLOBAL DISEASE OBESITY.

6. PROPOSALS

1. Causes and effects of obesity should be included in natural sciences at the primary education level.
2. Adolescent obesity is the fore runner of adult obesity, hence the ill effects of obesity must be popularized and games and sports regular exercise must be stressed for growing children.
3. All persons who crossed 35 years must be covered under screening programme for obesity at their workplace public or private for awareness and early detection of obesity.
4. The relatives and attendants of patients with hypertension, Type-2, diabetes, heart attacks, stroke and paralysis, joint replacement at early age should be offered counseling against obesity and lack of exercise.

Health care professionals including doctors and nursing staff should be trained regarding diet, nutrition exercise modalities and management of obesity in a non-cosmetic non commercial manner for prevention of complications of obesity.

ACKNOWLEDGEMENT

Authors would like to first of all thank Dr.K.Sultan Shahabaz and Dr.N.S.kareemunnisa, for performing various investigations and Dr.Pancy Prardhana for performing quality control of various biochemical tests; we would also like to thank Mr. Maqsood Javed who provided invaluable guidance throughout the study. We are particularly grateful to Mrs. B. Sandhyalaxmi, Mrs Asha for their statistical support Mrs. Kubriya Shabnam for editing the manuscript and the population of the slum community is appreciated. Special Thanks, to all the persons who provided blood for our project work.

REFERENCES

1. Alisha A, Pandey, RM Rama Devi, J.High prevalence of diabetes, obesity and dyslipidaemia in Urban slum population in northern India. *IJ of obesity*, 2001, 25(11), 1722-1729
2. Bender DA, Bender AE: Nutrition: A Reference Hand book, oxford University press 1997, 482-486
3. Bjorntorp P. Visceral fat accumulation; the missing link between psychosocial factors and cardiovascular disease. *J. Intern Med* 1991, 230, 195-201
4. Bonder DA. Introduction to Nutrition and metabolism 3rd edition Taylor & Francis London 2002, 132
5. Brosnan JT. Comments on the metabolic needs for glucose and the role of gluconeogensis. *Eureopean Journal of clinical nutrition* 1999, 53(1). SL07-SIII
6. Chopra M. Galbrath S.Darton Hill I. A global response to a global problem: the epidemic of over nutrition, Bull WHO 2002, 80, 953-8
7. Connolly V, Unwin N, Sherriff P.Bilons R, Kelly W. Diabetes prevalence and socio economic status; a population based study showing increased prevalence of type II diabetes in the deprived area. *J.Epidemiol Community Health* 2000, 54, 173-177
8. ContentoI, Balch GI, Bronner YL, et al. The effectiveness of nutrition education and implications for nutrition education policy, programs and research, a review of research. *J Nutr Educ*. 1995, 27, 284-418
9. Demeisil. The concept of energy – rich phosphate compound, water transform ATPases and entropy energy. *Arch Biochemistry, Biophysics* 1993, 30c, 287, pp 88
10. Eaton S et al. Multiple biochemical effects in the pathogenesis of fatty diver, *Eur J.Clin Invert* 1997, 27, 719
11. Freedom PS, serdula MK, percy CA Ballew C, White L. Obesity levels of lipids and glucose and smoking anog Navajo adolescents. *Jutr* 1997, 127, 1205-75
12. Goldberg IJ, Merked. M. Lipoprotein, Lipase, physioglogy biochemistry and molecular biology, *Front Bioscience* 2001, 6, D388
13. Goldberg IJ, Merkel M. Lipoprotein Lipase; Physiology, biochemistry and molecular biology. *Front Bioscience* 2006, 6, 388
14. Gopinath N Chadha, SL, Sood AK et al. Epidemiological study of hypertensives in young delhi urban population Ind. *J.Med. Res*. 1994, 99, 32-37
15. Gupta OP, Joshi MH. Dave SK, Prevalence of diabetes in India. *Adv Metab Disord* 1978, 9, 147-165
16. Howard BV, Lee ET, Cowan LD, et al coronary heart diseases prevalence and its relation to risk factors in American Indians; the strong heart study. *An J.Epidemiol* 1995, 142, 254-68
17. Hill JO, Peters JC. Environmental contribution to the obesity epidemic science, 1998, 280, 1371-1374
18. Klain M. Coulehan JL, Arenea VC et al. More frequent diagnosis of acute myocardial infarction among Nevajo Indians. *Am J.Public Health* 1988, 78, 1351-2
19. Kosuri M, Sridhar GR. Yoga Practice in diabetes improves physical and psychological outcomes. *Metab syndr Relet Discord* 2009, 7, 515-7
20. Kuriyan R, Petracchi C, Ferro – Luzzi A, Shetty PS, Kurpad AV. Validation of expedient methods for measuring body composition in Indian adults. *Ind JMed Res* 1998, 107, 37-45
21. Merril AH, sandhoff K. Sphingolipids; metabolish and cell signaling In: Biochmeistry of Lipids, Lipoproteins and membrane, 4th ed. Vance DE, VancJE (Eds) Elsevier 2002, 217
22. MC Phail LC. Glycerolipid in signal transduction in: Biochemistry of Lipids, Lipoprotiens and membrane 4th edition vance DE, Vance JE (Eds) Elsevier 2002 pp.196
23. Millar WJ, Wigle DT. Socio-economic disparities in risk factors for cardio vascular diseases. *CMAJ* 1986, 134, 127-132
24. Patandia S, Bots ML, Abel R, Valenburg HA. Impaired glucose intolerance and diabetes mellitus in a rural population in south india. *Diabetes Res clin pract* 1994, 24, 47-53
25. Ramachandran A, Jali MV, Mohan V, Snehalatha C. High prevalence of diabetes in an Urban Population in south India. *Br Med J.* 1986, 297, 587-590
26. Ramaiya K, Kodali VRR, Alberti, KGMM. Epidemiology of diabetes in Asians of the Indian subcontinent. *Diebetes Metab Rev*.1990, 6, 125-146
27. Srilakshmi B, Dietetics. 5th edition pp.173-188
28. Troiano RP. Flegal KM overweight children and adolescents description, epidemiology, and demographics. *Pedoiatrices* 1998, 101, 497-504