REVIEW ARTICLE

Obesity and Disease Association: A Review

***Tanjila Tahmin Sarnali, Md. Moyenuddin PK**

Dr. Tanjila Tahmin Sarnali, Lecturer, Pathology,
Anwer Khan Modern Medical College, Dhanmondi, Dhaka
Md. Moyenuddin PK, Biochemist, Modern Diagnostic Centre Limited

***Corresponding Author**

ABSTRACT

Obesity is a major public health problem which is not only confined to developed countries but has now become an important public health problem in developing countries like Bangladesh also. High calorie intake in diet due to increased consumption of refined sugars, sweetened beverages, vegetable oils, chunk and fast food and lack of physical activity, absence of play ground (open-spaces), sedantery life style all play a role in the development of obesity. Excess adiposity also known as obesity and excess body weight are associated with increased association with different types of diseases like type 2 diabetes, dyslipidemias, cardiovascular disease, hypertension and cancer. This review article highlights the pathogenesis, disease association with obesity.

Key words: Obesity, Disease Association, Public Health Problem

Introduction

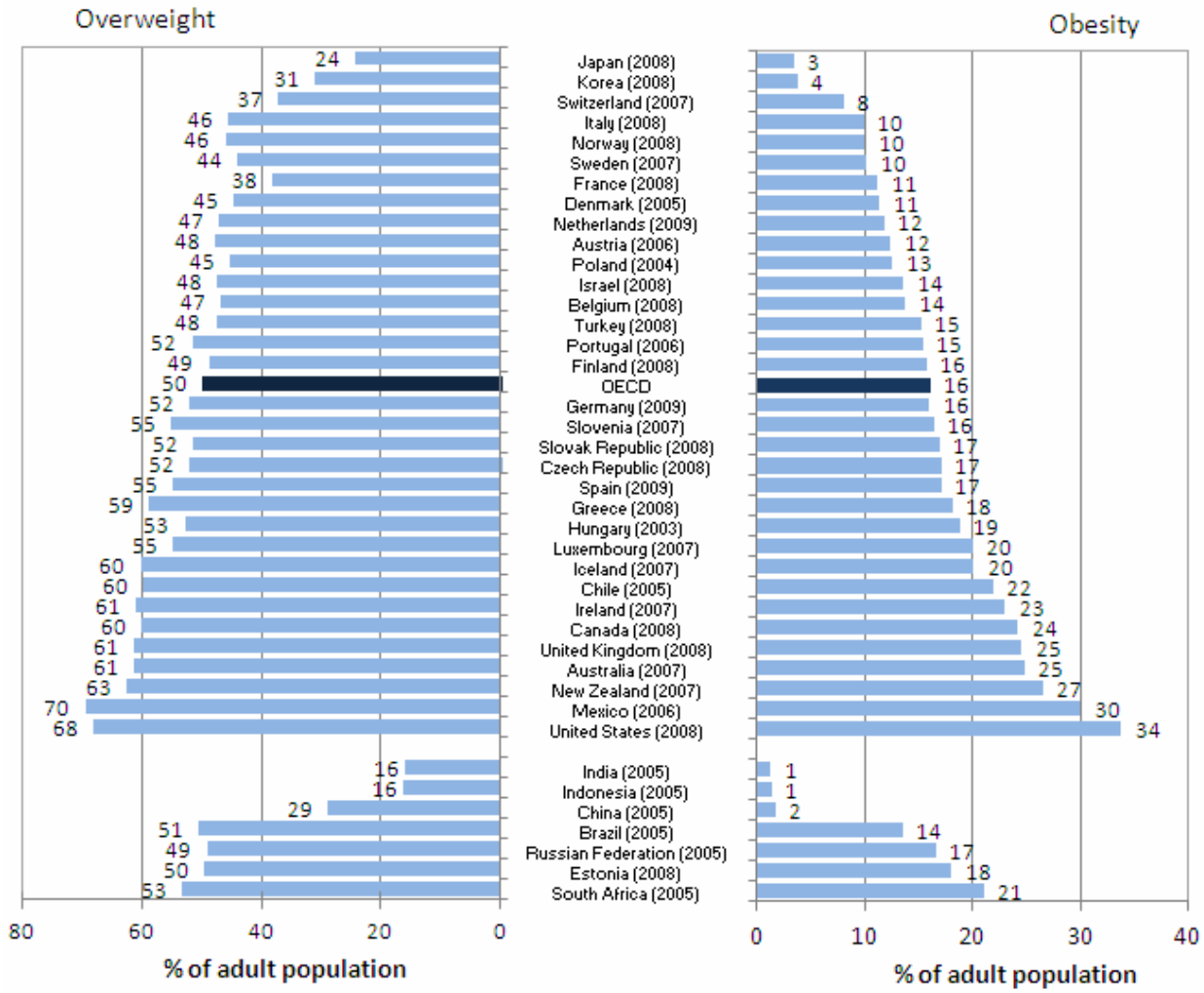
Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems[1,2]. Body mass index (BMI), a measurement which compares weight and height, defines people as overweight (pre-obese) if their BMI is between 25 kg/m^2 and 30 kg/m^2, and obese when it is greater than 30 kg/m^2.[3] BMI is calculated by dividing the subject's mass by the square of his or her height, typically expressed either in metric or US "customary"- unit [4].

Metric: BMI = kilograms / $meters^2$
US customary and imperial: BMI=lb $703/in^2$ where lb is the subject's weight in pounds and in is the subject's height in inches.

- Any BMI ≥ 35 or 40 is severe obesity
- A BMI of ≥ 35 or 40–44.9 or 49.9 is morbid obesity
- A BMI of ≥ 45 or 50 is super obesity

BMI	Classification
< 18.5	underweight
18.5–24.9	normal weight
25.0–29.9	overweight
30.0–34.9	class I obesity
35.0–39.9	class II obesity
≥ 40.0	class III obesity

The following Graph gives the overweight and obese incidence among people of the world. Available data from most of the countries showed the incidence of overweight between 16% (India) to 70% (mexico) and the incidence of obesity between 3% (Japan) to 34% in USA which is an alarming situation. As no data is available for Bangladesh a true estimate of the incidence of overweight and obesity cannot be made. However it is assumed that this incidence will be not low but high due to increasing urbanization, change of food habits to chunk and high energy food, lack of physical activity and sedantery lifestyle[5].



Pathogenesis of Obesity

The pathogenesis of obesity is complex and involves humoral and neuronal mechanisms that control apetite and satiety. These stimulations respond to genetic, nutritional, environmental and psychological signals and triggers centers in hypothalamus. The nueurohumoral mechanism that regulate energy balance is divided into three components. The peripheral or afferent systems that create signals from various sites. These include leptin and adiponectin that are produced by fate cells, ghrelin from stomach and peptide YY from Ileum and colon and insulin from pancreas. Leptin (meaning thin in Greek Leptos) is a 16kd hormone synthesized by fat cells is the product of ob gene. The leptin receptor (OB-R) is the product of diabetic gene (dg) and belongs to type I cytokine receptor that includes gp130, Granulocyte CSF, IL 6, 2 receptors. Genetically deficient mice in leptin fail to sense fat stores, overeat and gain weight. Adiponectin stimulate fatty acid oxidation causing a decrease in fat mass. In addition to leptin and adiponectin adipose tissue continuously produces cytokines like TNF, IL 1,6,18, chemokines and steroid hormones that create a chronic subclinical inflammatory state (Asymptomatic) that includes high level of CRP. Ghrelin is produced in stomach and in arcuate nucleus of hypothalamus and is the only known gut hormone that increases food intake (orexigenic effect). The arcute nucleus in the hypothalamaus process and integrate signals to generate different signals through two subsets. The first order of neurons

include POMC (Promelanocortine) and CART (Cocaine and amphetamine regulated transcripts neurone and second order neurons including Neuropeptide Y and AgRP (Agouti related peptide). The effector system carries signals generated in the second order of neurons of hypothalamus to control food intake and expenditure. POMT and CART increases energy expenditure and weight loss by producing alpha melanocyte stimulating hormone (MSH) and the activation of melanocortin receptors 3 and 4 (MC3/4) in second order of neurons. NYP/Ag RP neurons promote food intake and weight gain through activation of Y1/5 receptors in secondary neurons[6].

Review of literatures and discussion

Obesity increases the likelihood of various diseases, particularly heart disease, type 2 diabetes, breathing difficulties during sleep, certain types of cancer, and osteoarthritis[6]. Obesity is most commonly caused by a combination of excessive dietary calories, lack of physical activity, and genetic susceptibility, although a few cases are caused primarily by genes, endocrine disorders, medications or psychiatric illness. Evidence to support the view that some obese people eat little yet gain weight due to a slow metabolism is limited; on average obese people have a greater energy expenditure than their thin counterparts due to the energy required to maintain an increased body mass.

Association of Obesity and Diseases

There is increased association of obesity and diseases. These include Diabetes, Hypertension, Osteoarthritis, Pancreatitis, Cholelithiasis and dyslipidemia which are discussed below[7].

Type1 Diabetes-There is overall evidence for an association between childhood obesity, higher BMI, increased risk of subsequent type1 diabetes.

Type2 diabetes- insulin resistance and hyper insulinaemia. Weight loss associated with improvement. Excess insulin retain Na, expansion of blood volume, production of excess nor epinephrine ,smooth muscle proliferation - hallmark of Hypertension[9,10,11].

Osteoarthritis- marked obesity predisposes to degenerative joint disease. Cumulative effect of wear and tear on joint due to obesity, greater the burden of fat greater the trauma to joints with time[12].

Gall stone- 6 times more common in obese than non obese. Increased total cholesterol, increased biliary excretion and cholesterol in bile, cholesterol rich gall stones[13].

Nonalcoholic steatohepatitis- adolescents and adult who are obese and have type2 diabetes. Fatty change accompanied by inflammation lead to fibrosis.

Dyslipidemia- increased risk of CAD due to hyper TG, Low HDL Syndrome X-distinctive metabolic syndrome-abdominal obesity, insulin resistance, hyper TG, low HDL, HTN, increased risk of CAD.

Thrombosis- increases the risk of is chaemic stroke. Abdominal obesity is associated with increased risk of thrombosis[14].

Cancer- increased BMI and mortality in cancer esophagus, colon, rectum, liver and NHL[15,16].

Hypoventilation syndrome- respiratory anomalies, increased sleep both at night and day. Apnic pauses during sleep, polycythaemia and eventually RHF[17].

Obesity & Systemic Disease Association

GIT-increased gall stone, pancreatitis, Gastrointestinal reflux disease, Nonalcoholic fatty liver disease, abdominal hernia[18,19].

Endocrine & Metabolic system-Increased type 2 diabetes, insulin resistance, IGT, dyslipidemia.

Cardiovcascular system -increased thromboembolism, Hypertention, Coronary artery disease, Chronic heart failure, Pulmonary hypertention, Asthma[20, 21].

Female Ginital tract/gynaecological-Menstrual abnormality, infertility, carcinoma[22,23].

Eye-Cataract

Musculoskeletal system-Osteoarthritis, Gout, Lowbackpain

Postoperative-Atelactasis, Pneumonia, Deepvein thrombosis, Pulmonary embolism

Genitourinary-Stress urinary incontenience[24].

Neurological-idiopathic inherited

Conclusions

It can be concluded from the above reviewed literatures that obesity is an important public health problem in both developing and developed countries. It increases morbidity and mortality in different disease association both among child and adults and in both sexes. However strict diet control in relation to height and weight of the individual age, physical activity, less intake of chunk and high calorie food and increased consumption of vegetables, antioxidants, polyunsaturated fat will decrease the incidence of obesity. A social movement involving all strata of people like People representatives, government and private health organizations, mass media, teachers, Imams could play a vital role to curb this menace.

References

1. Haslam DW, James WP (2005). "Obesity". *Lancet* 366 (9492): 1197–209
2. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L (January 2003). "Obesity in adulthood and its consequences for life expectancy: A life-table analysis" (PDF). *Ann. Intern. Med.* 138 (1): 24–32
3. Gray DS, Fujioka K (1991). "Use of relative weight and Body Mass Index for the determination of adiposity". *J Clin Epidemiol* 44 (6): 545–50.
4. http://hobpages.com/hub/obesity. cause-effect-and treatment.
5. http://www.hive health media.com/world-obesity-status-2010/
6. Robbins pathologic basis of disease. Editor: Kumar, Abbas, Fausto, ster. 8th edition 2010. Publisher: Elsevier India private limited. page: 438-444.
7. Verbeeten KC, Elks CE, Daneman D, Ong KK .Association between childhood obesity and subsequent Type 1 diabetes: a systemic review and meta-analysis. Diabetic Med. 2011 Jan; 28(1): 10-8.
8. Zachary Bloomgarden (2003). "Prevention of Obesity and Diabetes". *Diabetes Care* 26 (11): 3172–3178..
9. Qin L, Knol MJ, Corpeleijn E, Stolk RP. Does ohysical activity modify the risk of obesity for type 2 diabetes: a review of epidemiological data. *Eur J Epidemiol.* 2010; 25(1): 5-12.
10. Reis AF, Hauache OM, Velho G. Vitamin D endocrine system and the susceptibility to diabetes, obesity and vascular diease. A review of evidence. Diabetes Metab. 2005 Sep; 31(4 pt 1): 318-25.
11. Basen- Engquist K, Chang M. Obesity and cancer risk: :Recent review and evidence. *Curr Oncol Rep.* 2011; 13910; 71-76.
12. Tukker A, Visscher T, Picavet H (April 2008). "Overweight and health problems of the lower extremities: osteoarthritis, pain and disability". *Public Health Nutr* 12 (3): 1–10.
13. Bray GA (2004). "Medical consequences of obesity". *J. Clin. Endocrinol. Metab.* 89 (6): 2583–9.
14. Darvall KA, Sam RC, Silverman SH, Bradbury AW, Adam DJ (February 2007). "Obesity and thrombosis". *Eur J Vasc Endovasc Surg* 33 (2): 223–33
15. Johnson IT, Lund EK; Review article:nutrition, obesity and colorectal cancer. Aliment Pharmacol Ther. 2007 jul 15; 26(2): 161-81.
16. Basen- Engquist K, Chang M. Obesity and cancer risk: recent review and evidence. Curr Oncol Rep. 2011; 13910; 71-76.
17. Poulain M, Doucet M, Major GC, *et al.* (April 2006). "The effect of obesity on chronic respiratory diseases: pathophysiology and therapeutic strategies". *CMAJ* 174 (9): 1293–9**.**
18. Anand G, Katz PO (2008). "Gastroesophageal reflux disease and obesity". *Rev Gastroenterol Disord* 8 (4): 233–9.
19. Shoelson SE, Herrero L, Naaz A (May 2007). "Obesity, inflammation, and insulin resistance". *Gastroenterology* 132 (6): 2169–80.
20. Reis AF, Hauache OM, Velho G.Vitamin D endocrine system and the susceptibility to diabetes, obesity and vascular disease. A review of evidence. *Diabetes Metab.* 2005 Sep; 31 (4 pt 1): 318-25.
21. Darvall KA, Sam RC, Silverman SH, Bradbury AW, Adam DJ (February 2007). "Obesity and thrombosis". *Eur J Vasc Endovasc Surg* 33 (2): 223–33.
22. Maruthur NM, Bolen SD, Brancati FL, Clark JM. The association of obesity and cervical cancer screening: a systemic review and meta-analysis. Obesity (Silver spring). 2009 Feb ;17(2): 375-81.
23. Cohen SS, Palmieri RT, Nyante SJ, Koralek DO,Kim S, Bradshaw P, Olshan AF. Obesity and screening for breast, cervical, and colorectal cancer in women:a review. Cancer. 2008 May 1; 112(9): 1892-904.
24. Obligado SH, Gldfarb DS. The association of nephrolithiasis with hypertension and obesity: a review. *Am J Hypertens*. 2008 Mar; 21(3): 257-64.