The American Journal of Surgery®

The American Journal of Surgery 184 (2002) 4S–8S

The extent of the problem of obesity

Paul E. O'Brien, M.D.*, John B. Dixon, M.B.B.S.

Monash University Department of Surgery and the Alfred Hospital, Commercial Road, Melbourne, Victoria 3181, Australia

Abstract

The prevalence of obesity is increasing worldwide. In the United States, in 1999, 27% of adults had a body mass index >30 kg/m^2, almost double the prevalence of 20 years earlier. The estimated mortality from obesity-related diseases in the United States is approximately 300,000 annually and growing. In the future, mortality related to obesity is expected to exceed that of smoking. Numerous diseases are caused or made worse by obesity. These include type 2 diabetes; hypertension; dyslipidemia; ischemic heart disease; stroke; obstructive sleep apnea; asthma; nonalcoholic steatohepatitis; gastroesophageal reflux disease; degenerative joint disease of the back, hips, knees, and feet; infertility and polycystic ovary syndrome; various malignancies; and depression. Type 2 diabetes is perhaps the most visible obesity-related problem. Present in at least 14 million Americans, it leads to serious complications and premature death. It is largely caused by obesity, and is generally cured by weight loss. The quality of life of the obese is markedly reduced, and the costs to health care systems are great. Preventive programs have yet to affect the rising prevalence. An effective solution is needed.

Obesity is steadily and inexorably becoming the greatest health problem in the developed world. It has recently been estimated that 1.1 billion people are overfed and overweight, a number that rivals the number who are underfed and underweight [1]. Overweight becomes the disease of obesity when excess fat has accumulated to the extent that it may adversely affect health. This point is most commonly defined by the body mass index (BMI). Although a BMI ≥25 can be associated with a reduced life expectancy and a risk of exacerbating many diseases, it is now usual to consider BMI of 30 as the cutoff—the point at which the accumulation of fat is a major health hazard [2].

The majority of adults in the United States, Australia, and most of Western and Eastern Europe are overweight (BMI >25), and more than 20% are obese [3]. The most recent National Health and Nutrition Examination Survey (NHANES) from the US Centers for Disease Control and Prevention [4] indicated that 61% of adults in the United States in 1999 were overweight and that 27% were obese. Shown in Fig. 1 is the rise in obesity through three survey periods. In less than 20 years, the number of obese Americans has nearly doubled.

Throughout the world, in both developed and developing nations, a similar pattern is occurring [3]. In Russia, 54% of adults are overweight [3]. In Brazil, the figure is 36% [3], and in Malaysia, 27% [3]. Even China has a developing problem. A recent survey of adults in urban Shanghai reported that 29.5% were overweight, and 4.3% were obese [5].

After millions of years spent evolving a genetic structure to survive the life of the hunter-gatherer, our health and our lives are now threatened by a lifestyle characterized by ready access to copious amounts of attractive food and little need for physical activity. Foods high in fat, carbohydrates, and, in particular, simple sugars are booming. The US Department of Agriculture reports that the US per capita intake of sugar was 152.4 lb in 2000, an increase of more than 20% in the last 14 years. We do not even need to get out of the chair to order the food delivered or to change the channel on the television.

Obesity is our worst pathogen

Obesity is damaging our health, reducing the quality of our lives, and leading us to a premature death. The economic costs to the individual and to the health care system are enormous [6,7].

Numerous diseases are either caused or made worse by

* Corresponding author: Tel.: +61 3 9903 0608; fax: + 61 3 9510 3365

E-mail address: paul.obrien@med.monash.edu.au

0002-9610/02/$ – see front matter
PII: S0002-9610(02)01172-8

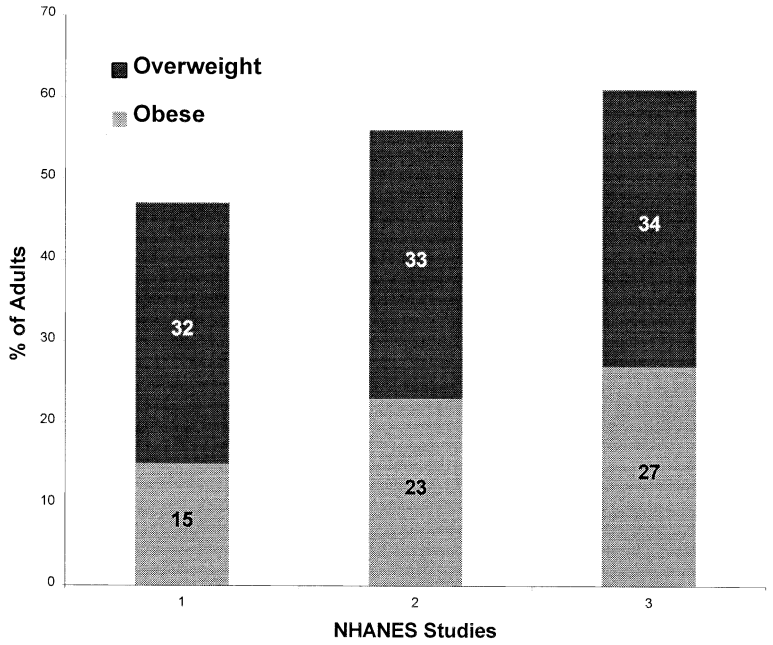

Fig. 1. The increasing prevalence of overweight and obesity in the United States, based on National Health and Nutrition Examination Surveys in 1976–80 (*1*), 1988–94 (*2*), and 1999 (*3*) [4]. NHANES = National Health and Nutrition Examination Survey.

Table 1
Some medical sequelae of obesity

Type 2 diabetes	Osteoarthritis: knees, hips, feet
Hypertension	Lower back pain
Dyslipidemia	Infertility
Ischemic heart disease	Polycystic ovary syndrome
Stroke	Obstetric complications
Cardiomyopathy	Fetal abnormalities
Obesity-hypoventilation syndrome	Venous thromboembolic disease
Pulmonary hypertension	Depression
Asthma	Cancer: breast, bowel, endometrium, prostate
Obstructive sleep apnea	Venous and stasis ulcers
Gallstones	Accident prone
Nonalcoholic steatohepatitis	Intracranial hypertension
Urinary incontinence	Gout
Gastroesophageal reflux	Skin disorders: acanthosis nigricans, acne, interigo, hirsutism

obesity (Table 1). We comment here on a selection of these to emphasize the increased prevalence and the clinical problem. On the top of the list is type 2 diabetes, an epidemic paralleling the epidemic of obesity [8].

The "diabesity" epidemic

Type 2 diabetes is the paradigm of obesity-related disease. In most cases, it exists because of the obesity, and, in most cases, it will disappear with weight loss. It is common, generates multiple serious complications, and is lethal.

Most patients with type 2 diabetes are overweight, and about half are obese [9]. In the Nurses' Cohort Study, from a base BMI of 21, the risk of developing type 2 diabetes was 5 times greater at BMI 25, 35 times greater at BMI 30, and 93 times greater at or above BMI 35 [10,11]. It is estimated that there are now more than 14 million type 2 diabetics in the United States. Given the morbidity and mortality that diabetes causes in young and middle-aged adults, this disease alone should elicit a call for action against the rising epidemic of obesity.

The metabolic syndrome

The metabolic syndrome describes a cluster of insulin resistance and hyperinsulinemia, dyslipidemia, essential hypertension, type 2 diabetes, and an increased risk of cardiovascular events [12]. A principal feature of this syndrome is central obesity. The primary pathogenic process or pathway

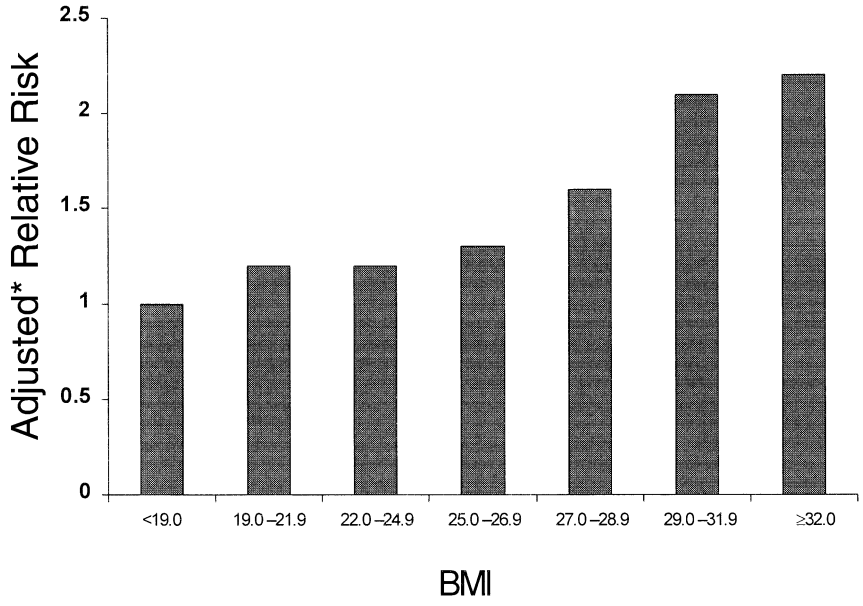

Fig. 2. Relation of body mass index (BMI) and adjusted relative risk of mortality in the Nurses' Health Study [37]. The risk of obesity is modified by a number of factors, including the location of body fat (central abdominal, or android, is worse than peripheral, or gynoid), age (the risk decreases with age), and sedentary lifestyle [3]. (Adapted with permission from N Engl J Med [37].) *Adjusted for age in 5-year categories, smoking (never, former, or current; 1–14,15–24, or ≥25 cigarettes per day), menopausal status, oral contraceptive and postmenopausal hormone use (never, past, or current), parental history of myocardial infarction before the age of 60 (yes or no), alcohol intake (in 5 categories), saturated fat intake (in quintiles), and physical activity (in 5 categories). $P < 0.001$.

remains obscure but probably involves the effects of an increased free fatty acid load on the hepatic uptake of insulin, leading to systemic hyperinsulinemia and hyperlipidemia, skeletal muscle insulin resistance, and eventual pancreatic beta cell failure.

The dyslipidemia of obesity and the metabolic syndrome include elevated triglycerides, depressed levels of high-density lipoprotein cholesterol, and an increase in small, dense, low-density lipoprotein (LDL) particles. The latter have increased concentrations of apolipoprotein B (apo-B), and are considered to be highly atherogenic [13]. The triad of hyperinsulinemia, high apo-B, and small, dense LDL particles is associated with a 20-fold increase in the risk of coronary heart disease [14].

The metabolic syndrome is associated with, and possibly causes, at least 2 other common and important diseases of obesity. The first is polycystic ovary syndrome, which is characterized by ovulatory dysfunction, infertility, androgen excess, and reduced sex hormone binding globulin, all probably driven by insulin resistance and hyperinsulinemia [15]. The second is nonalcoholic steatohepatitis, an increasingly common cause of abnormal liver function [16,17] and now the most common cause of nonalcoholic, nonhepatitis-C cirrhosis. For BMI values above 35, the odds ratio for abnormal liver function is greater than 5. An analysis of the NHANES III data showed that 30% of men and 40% of women who were obese had some evidence of fatty liver disease [18].

Respiratory diseases

Obstructive sleep apnea (OSA) is characterized by episodes of apnea during sleep as a result of intermittent pharyngeal obstruction due to soft tissue enlargement and reduced tone in the muscles controlling the tongue and soft palate. It occurs particularly in those with central (android) obesity. For both men and women whose BMI exceeds 40, it is at least 10 times as common as the community norm of 2% to 4% [19]. OSA leads to daytime drowsiness and psychosocial and cognitive dysfunction. The drowsy driver is now recognized as a major contributor to road deaths [20].

Another important form of sleep-disordered breathing is obesity-hypoventilation syndrome. Less common but more pathogenic than OSA, it leads through chronic hypoxia and hypercapnia to pulmonary hypertension and right heart failure [21,22].

Asthma has only recently been recognized as an important comorbidity of obesity. There is an increased prevalence and severity of asthma in obese children. Unger showed that the prevalence of both obesity and asthma in children was rising, and the 30% prevalence of asthma in obese children was significantly greater than the 5% to 12% incidence reported in that community as a whole [23]. We have found the prevalence of asthma in our obese patients receiving laparoscopic adjustable gastric banding with the LAP-BAND (INAMED Health, Santa Barbara, CA) to be double the matched community norm of 12% [24]. Both

OSA and gastroesophageal reflux could be factors in its pathogenesis.

Gastroesophageal reflux disease

Gastroesophageal reflux is a common problem, with an estimated 20% prevalence in the community; approximately 7% have a form of the disease severe enough to require daily medication [25]. Obesity is regarded as an important contributing factor to the disease. Study of the frequency of gastroesophageal reflux symptoms in the obese indicates a major increase in prevalence—to between 37% and 72% [26]. Objective measures of abnormal reflux by 24-hour pH monitoring and abnormal pressures at the lower esophageal sphincter on manometry support the clinical findings of increased prevalence [27].

Cardiovascular disease

Increased body fat increases oxygen consumption through increased tissue mass and metabolic demands. Cardiac output, stroke volume, and total blood volume increase. The increase in systemic vascular resistance leads to hypertension, and the increase in preload and afterload leads to left ventricular dilatation and hypertrophy [21]. Atherosclerosis develops in association with the dyslipidemia of obesity. The combination results in increased prevalence of myocardial infarction and stroke, two of the major causes of premature death in the obese [28–31].

Pregnancy

Pregnancy in severely obese women is associated with increased risks and costs. These women suffer an increased incidence of complications during pregnancy, including hypertension, preeclampsia, late fetal death, and gestational diabetes [32]. There is a higher risk of induction of labor, primary cesarean section, and perioperative morbidity. Their infants are more likely to have fetal growth abnormalities, macrosomia, and intrauterine growth retardation, and are more likely to require admission to a neonatal intensive care unit [33]. The infants also may be at greater risk of developmental abnormalities, including neural tube defects. Duration of hospital stay and overall cost are strongly related to maternal weight [34].

Mortality

Based on the analysis of five prospective cohort studies, Allison and coworkers estimated that between 275,000 and 325,000 Americans die each year from obesity-related diseases [35]. It has been estimated that approximately 400,000 Americans die each year from smoking-related diseases. With a significant decrease in cigarette smoking, it can be anticipated that, in the near future, obesity will achieve the distinction of being the most important single pathogen causing death in our communities [36].

Multiple studies have described the exponential relationship between increasing BMI and the relative risk of dying prematurely. As an example, Fig. 2 shows the relative risk reported in the Nurses' Health Study in 1995 [37].

Conclusion

Obesity is steadily becoming our worst pathogen. It is a side effect of an evolving lifestyle of copious food and relative inertia, a lifestyle that is becoming a part of the culture of our young and a part of the growth of our economies. It will not be easy to change. Obesity causes serious comorbidities, shortens life expectancy, reduces quality of life, and increases health care costs. An effective solution that is broadly acceptable is clearly needed.

References

[1] Gardner G, Halweil B. Underfed and overfed: the global epidemic of malnutrition. Washington, DC: Worldwatch Institute, 2000.
[2] National Institutes of Health. The practical guide: identification, evaluation and treatment of overweight and obesity in adults (NIH Publication Number 00-4084). Bethesda, MD: National Institutes of Health, National Heart, Lung, and Blood Institute, and North American Association for the Study of Obesity, 2000, 1–94.
[3] World Health Organization. Obesity: preventing and managing the global epidemic. Geneva: World Health Organization, 1998.
[4] National Center for Health Statistics. Prevalence of overweight and obesity among adults. Atlanta, GA: National Center for Health Statistics, Centers for Disease Control and Prevention. Available at: www.cdc.gov/nchs/products/pubs/pubd/hestats/obese/obse99.htm. Accessed August 4, 2002.
[5] Jia WP, Xiang KS, Chen L, et al. Epidemiological study on obesity and its comorbidities in urban Chinese older than 20 years of age in Shanghai, China. Obes Rev 2002;3:157–165.
[6] Colditz GA. Economic costs of obesity and inactivity. Med Sci Sports Exerc 1999;31(suppl 11):S663–S667.
[7] Wolf AM, Colditz GA. Current estimates of the economic cost of obesity in the United States. Obes Res 1998;6:97–106.
[8] Mokdad AH, Bowman BA, Ford ES, et al. The continuing epidemics of obesity and diabetes in the United States. JAMA 2001;286:1195–1200.
[9] Leibson CL, Williamson DF, Melton LJ III, et al. Temporal trends in BMI among adults with diabetes. Diabetes Care 2001;24:1584–1589.
[10] Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med 1995;122:481–486.
[11] Colditz GA, Willett W, Stampfer MJ, et al. Weight as a risk factor for clinical diabetes in women. Am J Epidemiol 1990;132:501–513.
[12] Reaven GM. Role of insulin resistance in human disease (syndrome X): an expanded definition. Annu Rev Med 1993;44:121–131.
[13] Lamarche B, Tchernof A, Moorjani S, et al. Small, dense low-density lipoprotein particles as a predictor of the risk of ischemic heart disease in men: prospective results from the Quebec Cardiovascular Study. Circulation 1997;95:69–75.

[14] Despres JP. Health consequences of visceral obesity. Ann Med 2001; 33:534–541.
[15] Hopkinson ZE, Sattar N, Fleming R, Greer IA. Polycystic ovarian syndrome: the metabolic syndrome comes to gynaecology. BMJ 1998;317:329–332.
[16] Luyckx FH, Desaive C, Thiry A, et al. Liver abnormalities in severely obese subjects: effect of drastic weight loss after gastroplasty. Int J Obes Relat Metab Disord 1998;22:222–226.
[17] Clain DJ, Lefkowitch JH. Fatty liver disease in morbid obesity. Gastroenterol Clin North Am 1987;16:239–252.
[18] Clark JM, Brancati FL, Diehl AM. Nonalcoholic fatty liver disease. Gastroenterology 2002;122:1649–1657.
[19] Kyzer S, Charuzi I. Obstructive sleep apnea in the obese. World J Surg 1998;22:998–1001.
[20] Haraldsson PO, Akerstedt T. Drowsiness—greater traffic hazard than alcohol: causes, risks and treatment. Lakartidningen 2001;98:3018–3023.
[21] Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. Am J Med Sci 2001;321:225–236.
[22] Grunstein RR, Wilcox I. Sleep-disordered breathing and obesity. Baillieres Clin Endocrinol Metab 1994;8:601–628.
[23] Unger R, Kreeger L, Christoffel KK. Childhood obesity: medical and familial correlates and age of onset. Clin Pediatr 1990;29:368–373.
[24] Dixon JB, Chapman L, O'Brien P. Marked improvement in asthma after LAP-BAND surgery for morbid obesity. Obes Surg 1999;9:385–389.
[25] Kahrilas PJ. Gastroesophageal reflux disease. JAMA 1996;276: 983–988.
[26] Lundell L, Ruth M, Sandberg N, Bove-Nielsen M. Does massive obesity promote abnormal gastroesophageal reflux? Dig Dis Sci 1995;40:1632–1635.
[27] Rigaud D, Merrouche M, Le Moel G, et al. Factors of gastroesophageal acid reflux in severe obesity. Gastroenterol Clin Biol 1995;19: 818–825.
[28] Eckel RH, Krauss RM. American Heart Association call to action: obesity as a major risk factor for coronary heart disease. AHA Nutrition Committee. Circulation 1998;97:2099–2100.
[29] Bray GA. Health hazards of obesity. Endocrinol Metab Clin North Am 1996;25:907–919.
[30] Kannel WB, Cupples LA, Ramaswami R, et al. Regional obesity and risk of cardiovascular disease: the Framingham Study. J Clin Epidemiol 1991;44:183–190.
[31] Rexrode KM, Hennekens CH, Willett WC, et al. A prospective study of body mass index, weight change, and risk of stroke in women. JAMA 1997;277:1539–1545.
[32] Sebire NJ, Jolly M, Harris JP, et al. Maternal obesity and pregnancy outcome: a study of 287,213 pregnancies in London. Int J Obes Relat Metab Disord 2001;25:1175–1182.
[33] Perlow JH, Morgan MA, Montgomery D, et al. Perinatal outcome in pregnancy complicated by massive obesity. Am J Obstet Gynecol 1992;167:958–962.
[34] Galtier-Dereure F, Montpeyroux F, Boulot P, et al. Weight excess before pregnancy: complications and cost. Int J Obes Relat Metab Disord 1995;19:443–448.
[35] Allison DB, Fontaine KR, Manson JE, et al. Annual deaths attributable to obesity in the United States. JAMA 1999;282:1530–1538.
[36] National Institutes of Health. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Bethesda, MD: National Heart, Lung, and Blood Institute in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases, 1998. NIH Publication No. 98-40831-262.
[37] Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. N Engl J Med 1995;333:677–685.