

Obesity and type 2 diabetes: preventing associated complications

Editorial

Type 2 Diabetes (T2D) and obesity go hand in hand. Many of the complications in both diseases are due to underlying primary drivers, insulin resistance, and metabolic syndrome. In this millennium, two diseases have reached *A State of global epidemics*.¹ In addition, obesity and T2D have become major public health issues, threatening the well-being of millions of people in both industrialized countries and emerging economies. In those who are genetically susceptible, taking in more energy than is necessary favors the development of glucose intolerance and subsequent development of T2D and associated complications.²

Like other diseases, obesity has

- i. A cause (caloric imbalance, availability and abundance of food, and consumption of low-nutritious, high-caloric food)
- ii. Pathology (adipocyte-mediated excessive production of inflammatory cytokines and hormones)
- iii. Pathophysiology (an environmentally and psychologically inducible dysregulation of appetite, low activity levels, body fat distribution, psychological issues, and deranged body-weight-controlling mechanisms),³
- iv. A disease that can be treated with anti-obesity medication.

However, not all obese patients experience complications such as T2D, Cardio Vascular Disease (CVD), and strokes.^{4,5} However, there are no specific, sensitive, and cost-effective markers or tests available for separating those who are vulnerable to developing complications from those who have low risks. Obesity is much more complicated than being just a lifestyle issue. It involves abnormalities of the mitochondria, which play central roles in energy expenditure and energy balance, and also has genetic predispositions.⁶ Mean while, obesity is becoming the leading preventable cause of cancer.⁷⁻⁹

Although there are common causes, each individual has a different set of risk factors that lead to the development of overweight, obesity, and T2D; if the issue of excess weight is unattended and untreated, these can lead to serious complications.¹⁰⁻¹² Excess visceral adiposity is positively correlated with the insulin resistance, T2D, certain cancers, and premature deaths.¹³ An effective strategy to control this necessitates both, preventing individuals from becoming overweight and early identification of the cause(s) of obesity and interventions. With such knowledge, clinicians should then could individualize care and treatment plans to target weight loss, particularly to lose intra-abdominal fat (using waist size is a surrogate marker), which would improve patient quality of life and generate cost savings in the long run.

Different people have different causes and underlying genetic susceptibilities for accumulating fat and experiencing of T2D and obesity.¹⁴⁻¹⁵ Thus, one management plan will not work for everyone.¹⁶ The management approaches need to be cause-oriented (elimination of root causes in a given person),⁶ systematic, and based on individualized

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methods to identify those who are prone to complications to be able to treat it cost-effectively. In addition, treatment strategies must be timely, appropriate, affordable, acceptable, and cost-effective, and patients must be able to adhere to the interventions.

By the law of conservation of energy, obesity is the result of an imbalance between energy intake and energy expenditure; fat content increases when energy intake is consistently greater than energy expenditure. Because of inherent difficulties with energy expenditure, older people accumulate greater amounts of visceral fat more easily than do younger individuals.¹⁷ Similarly, a greater amount of visceral fat accumulates in women during the perimenopausal period,¹⁸ in part because of relative estrogen deficiency.¹⁹

The increase in body weight [fat] associated with overconsumption of calories and engaging in less-than-optimal physical activity,^{20,21} which is influenced by socio-economic status, beliefs and social, cultural, and behavioral factors. However, the stigma associated with the obesity hindering the successful outcome of many.²² The health risks associated with obesity also depend on the distribution of body fat; accumulation of intra-abdominal fat being the worst. For example, people with less visceral fat but higher amounts of peripheral subcutaneous fat, even though may have higher BMI, can be healthy²³ and may experience less obesity-associated complications.¹⁴

In white Caucasians, the use of the Body Mass Index (BMI) is a sensitive tool for categorizing those who are overweight versus those who are obese.²⁴ Although BMI alone is useful for white Caucasians, because of its lack of sensitivity, by itself, is not useful for most other ethnic groups, such as Asians, blacks, Hispanics, and Pacific Islanders, for the identification of obesity or assessment of future CVD risks.²⁵ However, the sensitivity of BMI improves when it use in combination with waist circumference measurement. The latter can be easily done in each biannual clinic visit and noted in medical records for decision-making and follow-up.

In most obese patients, the metabolic damage is due to excess visceral (abdominal) fat. Visceral obesity is strongly linked with insulin resistance and the metabolic syndrome.^{26,27} Metabolic studies conducted in several laboratories have confirmed that among equally obese patients, subjects with excess visceral adipose tissue have the metabolic profile with the highest risks,^{28,29} including insulin resistance and compensatory hyperinsulinemia. Furthermore, atherogenic

dyslipidaemia and insulin resistance frequently present in patients with abdominal obesity.^{10,15,30}

However, there are no direct, practical, and cost-effective methods of measuring the severity of metabolic syndrome or determining who is at high risk for experiencing complications.^{14,31} The currently available biochemical methodologies are not specific enough to determine who is at risk versus who is not. Nevertheless, expensive high-tech testing is available for quantifying visceral fat to identify those who are at risk of CVD. Because of the lack of cost-effectiveness, it is hard to justify the use of these imaging techniques and non-invasive methods for routine investigation and assessment of obesity and for cardiovascular risks.

Therefore, a simple anthropometric measurements, such as the waist circumference and waist-to-hip ratio, together with basic blood lipid profile scan be cost-effectively use in identifying those who are at a higher risk for complications and monitoring the progress.^{16,32} In obese patients, measurement of abdominal girth is the most cost-effective (but underutilized) and the easiest way to identify and monitor patients who are at a high risk for CVD.

Despite medical advances and the availability of a series of new pharmaceutical agents, lifestyle changes, healthy eating, and increased physical activity (if adhered to advice and treatments by patients) are still the most cost-effective approach and the fundamental basis of preventing and managing obesity and T2D. Even for those obese persons who would benefit from pharmacotherapy or bariatric surgery, positive lifestyle changes are essential for the longer-term success in weight maintenance and the disease control.^{6,33}

The combination of sustainable lifestyle changes and pharmacotherapy (or bariatric surgery in highly selected patients) not only would maintain a patient's weight at a lower physiological set point but also would minimize the long-term complications of T2D and obesity.³⁴ However, because of the poor compliance with instructions and medications, the overall effectiveness of lifestyle changes and therapies for weight loss is limited. However, encouraging and close monitoring in a motivated person, set goals can be achieve successfully.

Evidence-based research on lifestyle interventions have demonstrated that effectiveness of reducing the body weight by 5 to 10% in improving insulin resistance,³⁵ and decreasing the incidence of CVD, heart failure, stroke, cancer, diabetes, and all-cause mortality.³⁶⁻³⁸ These alone should encourage overweight and obese patients to consider losing weight. In addition, such patients should be offer advice and guidance not only on healthy life styles and education, but also about the causes leading to excess weight, assisting them to adhere to a weight-reducing diet, a reasonable physical activity regimen, and periodically monitoring the patient's progress. Medications and bariatric surgery are effective but not the first set of options. Even when such treatments offered, lifestyle and behavioral changes need to be complementary.³⁹

It is easier to prevent obesity than to lose weight; one needs to identify risk factors in individual patients at the earliest possible time. Those who are overweight or obese with excess visceral fat, and engaged in smoking, drinking excessive amounts of alcohol, partaking of the least amount of physical activities, and having an adverse CVD family history are at the highest risk for insulin resistance, T2D, CVD, and premature death.⁴⁰⁻⁴²

In these high-risk populations, timely and effective interventions (goal of achieving approximately, 10% weight loss) would significantly reduce the human and financial costs, as well as productivity lost

from complications associated with metabolic syndrome, T2D and obesity.^{26,43} Since these diseases and associated complications predict to triple during the next two decades, policy makers must pay attention to this progressive, insidious epidemic, and proactively determine the right path. However, tackling T2D, obesity, and metabolic syndrome, requires prioritization of resources, combined approaches, and paradigm shift in thinking.

Obesity is more than simply a thermodynamic, genetic, or a metabolic problem of handling calories; it is also an inflammatory disease, impairment of the neuro hormonal system, and a behavioral disorder leading to dysregulation of metabolism and energy balance that results in accumulation of visceral fat leading to serious multi-system complications. In addition to reasonable weight reduction programs, controlling the obesity-associated complications requires coordinated, individualized, cause-driven, and complication-centric approaches. Understanding the cause of obesity in each patient would greatly facilitate the development of an individualized, sustainable treatment plan that would lead to successful outcomes.

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Conflict of interest

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References

- Arnold M, Pandeya N, Byrnes G, et al. Global burden of cancer attributable to high body-mass index in 2012: a population-based study. *Lancet Oncol.* 2015;16(1):36-46.
- Prentki M, Nolan CJ. Islet beta cell failure in type 2 diabetes. *J Clin Invest.* 2006;116(7):1802-1812.
- Aronne, LJ. Therapeutic options for modifying cardiometabolic risk factors. *Am J Med.* 2007;120(3 Suppl 1):S26-S34.
- Must A, Spadano J, Coakley EH, et al. The disease burden associated with overweight and obesity. *JAMA.* 1999;282(16):1523-1529.
- Hinnouho GM, Czernichow S, Dugravot A, et al. Metabolically Healthy Obesity and Risk of Mortality: Does the definition of metabolic health matter? *Diabetes Care.* 2013;36(8):2294-2300.
- Sunil Wimalawansa. Pathophysiology of obesity: Focused, cause-driven approach to control the epidemic. *Global Advanced Research Journal of Pharmacy and Pharmacology.* 2013;2(1):1-13.
- Meyerhardt JA, Catalano PJ, Haller DG, et al. Influence of body mass index on outcomes and treatment-related toxicity in patients with colon carcinoma. *Cancer.* 2003;98(3):484-95.
- Chan DS, Vieira AR, Aune D, et al. Body mass index and survival in women with breast cancer-systematic literature review and meta-analysis of 82 follow-up studies. *Ann Oncol.* 2015;26(10):1901-1914.
- Efstathiou JA, Bae K, Shipley WU, et al. Obesity and mortality in men with locally advanced prostate cancer: analysis of RTOG 85-31. *Cancer.* 2007;110(12):2691-2699.
- Neeland JJ, Ayers CR, Rohatgi AK, et al. Associations of visceral and abdominal subcutaneous adipose tissue with markers of cardiac and metabolic risk in obese adults. *Obesity (Silver Spring).* 2013;21(9):439-447.

11. Ross R, Després JP. Abdominal obesity, insulin resistance, and the metabolic syndrome: contribution of physical activity/exercise. *Obesity (Silver Spring)*. 2009;17(Suppl 3):1–2.
12. Després JP. The insulin resistance-dyslipidemic syndrome of visceral obesity: effect on patients' risk. *Obes Res* 1998;6(Suppl 1):8–17.
13. Renzaho AM, Halliday JA, Nowson C. Vitamin D, obesity, and obesity-related chronic disease among ethnic minorities: A systematic review. *Nutrition*. 2011;27(9):868–879.
14. Spinler SA. Challenges associated with metabolic syndrome. *Pharmacotherapy*. 2006;26(12 Pt 2):209–217.
15. Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. *Am J Cardiol*. 1999;83(9B):25–29.
16. Wimalawansa, SJ. Controlling obesity and its complications by elimination of causes and adopting healthy habits. *Advances in Medical Sciences*. 2014;3(1):1–15.
17. Pascot A, Lemieux S, Lemieux I, et al. Age-related increase in visceral adipose tissue and body fat and the metabolic risk profile of premenopausal women. *Diabetes Care*. 1999;22(9):1471–1480.
18. Zamboni M, Armellini F, Milani MP, et al. Body fat distribution in pre- and post-menopausal women: metabolic and anthropometric variables and their inter-relationships. *Int J Obes Relat Metab Disord*. 1992;16(7):495–504.
19. Tchernof A, Després JP. Sex steroid hormones, sex hormone-binding globulin, and obesity in men and women. *Horm Metab Res*. 2000;32(11–12):526–536.
20. Misra A, Khurana L. The metabolic syndrome in South Asians: epidemiology, determinants, and prevention. *Metab Syndr Relat Disord*. 2009;7(6):497–514.
21. Zamboni A, Marchiori M, Manzato E. Dyslipidemia in visceral obesity: pathophysiological mechanisms, clinical implications and therapy. *G Ital Cardiol (Rome)*. 2008;9(4 Suppl 1):29–39.
22. Sunil J, Wimalawansa. Stigma of obesity: A major barrier to overcome. *J Clin. & Translational Endocrinology*. 2014;1(3):73–76.
23. Brothers J, McBride M, Paridon A, et al. Fatness is not a factor of fitness: analysis of cardio respiratory data from healthy children over an 8-year period. *Cardiol Young*. 2012;23(1):47–53.
24. Da Costa LA, Arora P, García-Bailo B, et al. The association between obesity, cardiometabolic disease biomarkers, and innate immunity-related inflammation in Canadian adults. *Diabetes Metab Syndr Obes*. 2012;5:347–355.
25. Misra A, Shrivastava U. Obesity and dyslipidemia in South Asians. *Nutrients*. 2013;5(7):2708–2733.
26. Wimalawansa SJ. Visceral adiposity and cardio-metabolic risks: Epidemic of Abdominal Obesity in North America. *Research and Reports in Endocrine Disorders*. 2013;3:17–30.
27. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in women. *JAMA*. 1998;280(21):1843–1848.
28. Ross R, Freeman J, Hudson R, et al. Abdominal obesity, muscle composition, and insulin resistance in premenopausal women. *J Clin Endocrinol Metab*. 2002;87(11):5044–5051.
29. Bacha F, Saad R, Gungor N, et al. Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: race differential in diabetogenic and atherogenic risk factors. *J Clin Endocrinol Metab*. 2003;88(6):2534–2540.
30. Vinik, AI. The metabolic basis of atherogenic dyslipidemia. *Clin Cornerstone*. 2005;7(2–3):27–35.
31. Vega GL. Obesity and the metabolic syndrome. *Minerva Endocrinol*. 2004;29(2):47–54.
32. Minocci A, Guzzaloni G, Marzullo P, et al. Abdominal fat index by ultrasound does not estimate the metabolic risk factors of cardiovascular disease better than waist circumference in severe obesity. *Diabetes Metab*. 2005;31(5):471–477.
33. Mechanick JI, Garber AJ, Handelsman Y, et al. American Association of Clinical Endocrinologists' position statement on obesity and obesity medicine. *Endocr Pract*. 2012;18(5):642–648.
34. Wimalawansa SJ. Thermogenesis based interventions for treatment for obesity and type 2 diabetes mellitus. *Expert Reviews of Endocrinology & Metabolism*. 2013;8(3):275–288.
35. John M Jakicic, Sarah A Jaramillo, Ashok Balasubramanyam, et al. Effect of a lifestyle intervention on change in cardio respiratory fitness in adults with type 2 diabetes: results from the Look AHEAD Study. *Int J Obes (Lond)*. 2009;33(3):305–136.
36. Kelly GS. Insulin resistance: lifestyle and nutritional interventions. *Altern Med Rev*. 2000;5(2):109–132.
37. Cordain L, Eaton SB, Sebastian A, et al. Origins and evolution of the Western diet: health implications for the 21st century. *Am J Clin Nutr*. 2005;81(2):341–354.
38. Eaton SB, Konner M. Paleolithic nutrition. A consideration of its nature and current implications. *N Engl J Med*. 1985;312(5):283–289.
39. Kramer H, Reboussin D, Bertoni AG, et al. Obesity and albuminuria among adults with type 2 diabetes: the Look AHEAD (Action for Health in Diabetes) Study. *Diabetes Care*. 2009;32(5):851–853.
40. Nicklas BJ, Penninx BW, Cesari M, et al. Association of visceral adipose tissue with incident myocardial infarction in older men and women: the Health, Aging and Body Composition Study. *Am J Epidemiol*. 2004;160(8):741–749.
41. Kuk JL, Katzmarzyk PT, Nichaman MZ, et al. Visceral fat is an independent predictor of all-cause mortality in men. *Obesity (Silver Spring)*. 2006;14(2):336–341.
42. Boyko EJ, Fujimoto WY, Leonetti DL, et al. Visceral adiposity and risk of type 2 diabetes: a prospective study among Japanese Americans. *Diabetes Care*. 2000;23(4):465–471.
43. Garvey WT, Garber AJ, Mechanick JI, et al. American association of clinical endocrinologists and american college of endocrinology position statement on the 2014 advanced framework for a new diagnosis of obesity as a chronic disease. *Endocr Pract*. 2014;20(9):977–989.