

Getting Fat: "What" is Eaten is as Important as "How much" is Eaten

Shrimpton $\textbf{R}^{\star},$ Bazzano A and Mason J

Department of Global Community Health and Behavioral Sciences, School of Public Health and Tropical Medicine, Tulane University, New Orleans, USA

*Corresponding author: Roger Shrimpton, Department of Global Community Health and Behavioural Sciences, Tulane School of Public Health and Tropical Medicine, 1440 Canal Street, New Orleans, LA 70112, USA, Tel: +1 504-988-5388; E-mail: rshrimpt@tulane.edu

Received date: July 05, 2017; Accepted date: July 12, 2017; Published date: July 19, 2017

Copyright: © 2017 Shrimpton R, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Obesity is becoming an enormous global problem and urgent measures are needed to contain it. Traditional thinking that it is just a problem of energy balance has led to educational approaches to get people to eat less and exercise more becoming the standard interventions. However, new evidence suggests that it is not just how much you eat, but what is being eaten that is driving the problem. Evidence for the various causalities, especially sugar intake, and the sort of approaches needed to stop the problem getting worse are presented. Policy actions to promote healthy diets cannot just focus on information based approaches that will help the public make better informed choices (e.g. media campaigns, dietary guidelines). In addition, more structural approaches are needed such as fiscal measures, and restrictions on advertising. Policy assessments should be carefully designed based on a theory of change, using indicators of progress along the various pathways towards the long-term goal of reducing obesity rates. The most important research needed is to strengthen the monitoring and evaluation of such programme approaches using a plausibility approach. No country has demonstrated success in controlling obesity yet.

Introduction

Traditional thinking that "eating too much" and "moving too little" are the causes of the rising global obesity problem is coming under increasing scrutiny. While recently commenting on the lack progress in obesity prevention Taubes resurrected the hypothesis that obesity is a hormonal, regulatory fat accumulation disorder, triggered not by energy imbalance but by the quality and quantity of the carbohydrates in the diet [1]. Ludwig and Freidman have also suggested that overeating may be secondary to a diet induced metabolic dysfunction, caused by eating too many refined carbohydrates [2]. A recent dynamic time series analysis of US and global population data suggests that while increases in carbohydrate consumption are associated with increasing weight, increases in fat consumption are associated with decreasing weight [3]. The importance of these findings, suggesting that "what" is eaten is as important as "how much" is eaten as a cause of obesity, has enormous implications for adoption of the right policies and strategies to tackle the problem. Achieving the WHO Global Nutrition Target 4, of no increase in childhood overweight [4], as well as Global Non-Communicable Disease

(NCD) Target 7, to halt the rise in diabetes and obesity [5] will surely depend on programs and interventions doing much better than they have done so far.

The problem

The global prevalence of overweight and obesity combined was 38 percent in adults (over 20 years) and 24 percent in children and adolescents (under 20 years) in 2013 [6]. Between 1980 and 2013 the worldwide prevalence of overweight and obese combined rose by 27.5 percent in adults and 47.5 percent in children and adolescents. Furthermore, the increase in prevalence is accelerating, with over half of the rise occurring during the eight-year period from 2000 and 2008. The increase in prevalence of overweight and obesity in low and

middle income countries (LMICs) was greater than in higher income countries (HICs) over the last three decades [7].

Globally, an estimated 422 million adults were living with diabetes in 2014, compared to 108 million in 1980 [8]. Diabetes occurs either when the pancreas does not produce enough insulin (type 1), or when the body cannot effectively use the insulin it produces (type 2). Most diabetic patients (over 90%) suffer from type 2 diabetes (T2D), which is by nature a progressive disorder with a slow and insidious onset, and therefore frequently under reported. The global prevalence (agestandardized) of diabetes has nearly doubled since 1980, rising from 4.7% to 8.5% in the adult population. This reflects an increase in associated risk factors such as being overweight or obese. Diabetes caused 1.5 million deaths in 2012. Higher-than-optimal blood glucose caused an additional 2.2 million deaths, by increasing the risks of cardiovascular and other diseases. Forty-three percent of these 3.7 million deaths occur before the age of 70 years, and this percentage is higher in LMICS than in HICs.

The global economic impact of obesity has been estimated at roughly US\$2.0 trillion, or 2.8 percent of global GDP [9]. Over the next 20 years, the cost of treating NCDs and cumulative output losses globally are likely to be at least US\$30 trillion and \$47 trillion, respectively, representing 48 percent and 75 percent, respectively, of global GDP in 2010 [10].

The causes

The lack of consumption of vegetables, fruits and whole grains, as well as overconsumption of processed foods, especially those high in fat and sugar were strongly associated with the increase in adult body weight in the USA [11]. Systematic reviews find that processed meats and sugar sweetened beverages are among the foods that increase the risk of cardiovascular disease and diabetes [12]. But we must be clear that is not carbohydrate intake that is the problem, it is sugar intake. The new WHO guideline confirms that adults and children should maintain a reduced intake of free sugars over the life course and recommends that intake are reduced to less than 10% of total energy intake [13]. Global assessments of sugar intake as a percentage of energy range from 20.0% to 38.4% in young children and 13.5-24.6% in adults [14]. Per capita sugar consumption is strongly associated with the prevalence of diabetes in a 165-country analysis, with the strongest association found in the Asia region [15]. Various systemic reviews and meta-analysis provide evidence that consumption of free sugars or sugar sweetened beverages (SSBs) promotes weight gain in adults and children [16, 17].

All over the world ultra-processed foods are displacing unprocessed foods in the diet, and in Brazil for example already contributed about a third of dietary energy in 2003 [18]. The major problem with ultra-processed foods is that they have a glycaemic index (GI) score of over 70, which is high, and means that blood sugar levels remain high for a long period after the food is eaten. Examples of high GI foods include instant noodles, doughnuts, white bread and soda for example. Low GI diets can help to reduce body weight and improve blood lipid profiles [19] and higher dietary GI is associated with increased risk of T2D [20] and CHD [21].

Diet-induced obesity is associated with chronic low grade systemic inflammatory state. The visceral fat deposits in the abdomen, in addition to secreting hormones such as leptin that help regulate feeding behavior and energy expenditure, are involved in producing this inflammatory response [22]. The metabolic abnormalities of obesity, including insulin resistance and T2D, are related to this inflammatory state [23, 24]. The exact causes of the low grade systemic inflammation of obesity are uncertain, with some considering it related to changes in gut bacteria caused by a western diet high in fat and/or refined carbohydrates. [25, 26, 27].

The solutions

All too often the only policy response to problems of overweight and obesity is to rely on nutrition education to encourage people to make healthy choices. A review of actions taken by international organizations, governments, civil society and the private sector to promote healthy diets found they were largely inconclusive in their effectiveness [28]. One of the problems this review found was that the nature and extent of these actions vary considerably across the globe, but it was still concluded that consistent, concerted and comprehensive nutrition education action is not yet in evidence anywhere, and on its own nutrition education is unlikely to succeed. In reality, a nutrition education approach is unlikely to be successful considering the cacophony of the dietary advice already out there in the public domain. Much of this "noise" in the messaging being created by research findings funded by the sugar industry to exonerate sugar as a major risk factor and to emphasize saturated fat as the main driver of CHD [29].

Labels which describe the nutrition content of foods can in theory permit the discerning consumer to make more informed purchases. WHO provides guidance in developing or adapting nutrient profile models, and based on these nutrient profiles, claims such as "low fat" or "healthy choice" on food labels and in marketing can be regulated by national food standards authorities [30]. Labels are most used by educated and wealthier segments of the population however, and evidence for the effectiveness of "front of pack" labeling is still weak [31].

Interest in using taxation to control the consumption of unhealthy foods has increased since 2011 when the UN General Assembly recommended "fiscal measures" to improve diets in order to address NCDs as a matter of priority in national development plans [32]. There is reasonable and increasing evidence that appropriately designed taxes on sugar sweetened beverages would result in proportional reductions in consumption, especially if aimed at raising the retail price by 20% or more [33]. Furthermore, there is similar strong evidence that subsidies for fresh fruits and vegetables that reduce prices by 10-30% are effective in increasing fruit and vegetable consumption [33]. Greater effects on the net energy intake and weight may be accomplished by combining subsidies on fruit and vegetables and taxation of target foods and beverages. Emerging evidence from Mexico shows that the introduction of a soda tax has resulted in decreased purchases of taxed sugar-sweetened beverages by 12% and increased purchases of untaxed alternatives (mainly water) by 4% [34].

Public health efforts need to be redoubled to protect children from marketing of energy-dense, nutrient-poor foods and beverages [35]. The World Health Assembly (WHA) recently endorsed a resolution calling for national and international action to reduce the impact on children of marketing of foods high in saturated fats, trans-fatty acids, free sugars, or salt [36], yet little is known about country compliance. If the experience gained with Code of Marketing of breast milk substitute is anything to go by it will not be an easy task. This code was adopted by the WHA in May 1981 and as of 2016 an estimated 135 countries had some form of Code-related legal measures in place, an increase from 103 in 2011 [37]. However, only 39 countries have legislation incorporating all or most Code provisions. Furthermore, thirty-four years after the adoption of the Code, global sales of breast-milk substitutes total US\$ 44.8 billion, and this number is expected to rise to US\$ 70.6 billion by 2019 [38].

Taking a life-course perspective is essential for preventing T2D, as it is for many health conditions including other NCDs [39]. Early in life, when eating and physical activity habits are formed and when the long-term regulation of energy balance may be programmed; there is a critical window for intervention to mitigate the risk of obesity and T2D later in life. There is a growing body of evidence which suggests that breastfeeding has protective roles against obesity, hypertension, dyslipidemia, and T2D during adulthood [40].

Scientific advances provide a wealth of new evidence to identify several key dietary priorities for cardio metabolic health [41]. These include food-based priorities for more fruits, non-starchy vegetables, nuts, legumes, fish, vegetable oils, yogurt, and whole grains; and fewer processed (sodium-preserved) meats and foods higher in refined carbohydrates and salt. Moving forward, priorities should include comprehensive policy actions that create an enabling environment for infants and children to learn healthy food preferences and targeted actions that enable disadvantaged populations to overcome barriers to meeting healthy preferences [42]. Policy assessments should be carefully designed on the basis of a theory of change, using indicators of progress along the various pathways towards the long-term goal of reducing obesity rates [43]. The most important research needed is to strengthen the monitoring and evaluation of such programmes using a plausibility approach [44].

Conclusions

The problems of obesity and diabetes the world is now facing are not only massive, but also largely underestimated. The economic

Page 2 of 4

consequences of carrying on as we are will be catastrophic. Effective new approaches are available to prevent obesity and type 2 diabetes as well as to prevent the complications and premature death that can result from all types of diabetes. These include policies and practices across whole populations and within specific settings (school, home and workplace) that contribute to good health for everyone, such as exercising regularly and eating healthily. However, policy actions to promote healthy diets cannot just focus on information based approaches that will help the public make better informed choices. (e.g. media campaigns, dietary guidelines). In addition to these, more structural approaches are needed such as fiscal measures, and restrictions on advertising. The public cannot be left to make sensible choices all on their own.

References

- 1. Taubes G (2013) The science of obesity: what do we really know about what makes us fat? An Essay by Gary Taubes BMJ 346: 1-5.
- 2. Ludwig DS, Friedman MI (2014) Increasing adiposity: Consequence or cause of obesity. JAMA 311: 2167-2168.
- 3. Riera-Crichton D, Tefft N (2014) Macronutrients and obesity: Revisiting the calories in, calories out framework. Econ Hum Biol 14: 33–49.
- McGuire S (2015) Comprehensive implementation plan on maternal, infant and young child nutrition Geneva, Switzerland, 2014. Adv Nutr 6: 134-135.
- 5. Global action plan for the prevention and control of NCDs 2013-2020. Geneva: World Health Organization.
- Ng M, Fleming T, Robinson M, Thomson B, Graetz N, et al. (2014) Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet 384: 766-781.
- 7. Finnucane MM, Stevens GA, Cowan MJ, Danaei G, Lin JK, et al. (2011) Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group (Body Mass Index). National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet 377: 557-567.
- 8. WHO (2016) Global report on diabetes. Geneva: World Health Organization.
- Dobbs R, Sawers C, Thompson F, Manyika J, Woetzel J, et al. (2014) "Overcoming Obesity: An Initial Economic Analysis." London: McKinsey Global Institute.
- Bloom DE, Cafiero ET, Jane-Llopis E, Abrahams-Gessel S, Bloom LR, et al (2011) The Global Economic Burden of Non communicable Diseases. Geneva: World Economic Forum.
- Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB (2011) Changes in Diet and Lifestyle and Long-Term Weight Gain in Women and Men. N Engl J Med 364: 2392-2404.
- 12. Micha R, Shulkin ML, Penalvo JL, Khatibzadeh S, Singh GM, et al. (2017) Etiologic effects and optimal intakes of foods and nutrients for risk of cardiovascular diseases and diabetes: Systematic reviews and metaanalyses from the Nutrition and Chronic Diseases Expert Group (NutriCoDE). PLoS One 12: e0175149.
- WHO (2015) Guideline: sugars intake for adults and children. Geneva: World Health Organization.
- 14. Newens KJ, Walton J (2016) A review of sugar consumption from nationally representative dietary surveys across the world. J Hum Nutr Diet 29: 225-240.
- 15. Weeratunga P, Jayasinghe S, Perera Y, Jayasena G, Jayasinghe S (2014) Per capita sugar consumption and prevalence of diabetes mellitus global and regional asociations. BMC Public Health 14: 186.
- Malik VS, Schulze B, Hu FB (2006) Intake of sugar sweetened beverages and weight gain: a systematic review. American Journal of Clinical Nutrition 84: 274-288.

- 17. Te Morenga L, Mallard S, Mann J (2012) Dietary sugars and body weight: systematic review and meta-analyses of randomized controlled trials and cohort studies. BMJ 345: e7492.
- Monteiro CA, Levy RB, Claro RM, de Castro IR, Cannon G (2011) Increasing consumption of ultra-processed foods and likely impact on human health: evidence from Brazil. Public Health Nutr 14: 5-13.
- Thomas D, Elliott EJ, Baur L (2007) Low glycaemic index or low glycaemic load diets for overweight and obesity. Cochrane Database of Syst Rev 18: CD005105.
- 20. Bhupathiraju SN, Tobias DK, Malik VS, Pan A, Hruby A, et al. (2014) Glycemic index, glycemic load, and risk of type 2 diabetes: results from 3 large US cohorts and an updated meta-analysis. Am J Clin Nutr 100: 218-232.
- 21. Fan J, Song Y, Wang Y, Hui R, Zhang W (2012) Dietary glycaemic index, glycaemic load, and risk of coronary heart disease, stroke, and stroke mortality: a systematic review with meta-analysis. PLos ONE 7: e52182.
- 22. Kwon H, Pessin JE (2014) Adipokines mediate inflammation and insulin resistance. Front Endocrinol Lausanne 71: 1-13.
- 23. Gregor MF, Hotamisligil GS (2011) Inflammatory mechanisms in obesity. Ann Rev Immunol 29: 415-445.
- Canale MP, de Villahermosa SM, Martinho G, Rovella V, Noce A, et al. (2013) Obesity-Related Metabolic Syndrome: Mechanisms of sympathetic over activity. Int J Endocrinol 31: 1-12.
- 25. Cox AJ, West NP, Cripps AW (2014) Obesity, inflammation, and the gut microbiota. Lancet Diab Endocrinol 3: 207-215.
- Nieuwdorp M, Gilijamse PW, Pai N, Kaplan LM (2014) Role of the micro biome in energy regulation and metabolism. Gastroenterology 146: 1525-1533.
- Kotzampassi K, Giamarellos-Bourboulis EJ, Stavrou G (2014) Obesity as a consequence of gut bacteria and diet interactions. ISRN Obes 651895: 1-8.
- 28. Hawkes C (2013) Promoting healthy diets through nutrition education and changes in the food environment: an international review of actions and their effectiveness. Rome: Nutrition Education and Consumer Awareness Group, Food and Agriculture Organization of the United Nations.
- Kearns CE, Schmidt LA, Glantz SA (2016) Sugar Industry and Coronary Heart Disease Research: A Historical Analysis of Internal Industry Documents. JAMA Intern Med 176: 1680-1685.
- 30. WHO (2010) Nutrient profiling: Report of a WHO/IASO technical meeting, Geneva: World Health Organization.
- Campos S, Doxey J, Hammond D (2011) Nutrition labels on prepackaged foods: a systematic review. Public Health Nutrition 14: 1496-1506.
- 32. United Nations General Assembly (2011) Political Declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-Communicable Diseases. New York: United Nations.
- 33. WHO (2016) Fiscal policies for diet and prevention of noncommunicable diseases: technical meeting report. Geneva, Switzerland.
- Colchero MA, Popkin BM, Rivera JA, Ng SW (2016) Beverage purchases from stores in Mexico under the excise tax on sugar sweetened beverages: observational study. BMJ 352: h6704.
- Lobstein T, Jackson-Leach R, Moodie ML, Hall KD, Gortmaker SL, et al. (2015) Child and adolescent obesity: part of a bigger picture. Lancet 385: 2510-2520.
- WHO (2010) Set of recommendations on the marketing of foods and non-alcoholic beverages to children. Geneva: World Health Organization.
- 37. WHO, UNICEF, IBFAN. Marketing of breast-milk substitutes: National implementation of the international code. Status Report 2016.
- Rollins NC, Bhandari N, Hajeebhoy N, Horton S, Lutter CK, et al. (2016) Why invest, and what it will take to improve breastfeeding practices? Lancet 387: 491-504.
- **39.** Baird J, Jacob C, Barker M, Fall CHD, Hanson M, et al. (2017) Developmental Origins of Health and Disease: A Lifecourse Approach to the Prevention of Non-Communicable Diseases. Healthcare 5: 1-12.

Page 3 of 4

Page 4 of 4

- Kelishadi R, Farajian S (2014) The protective effects of breastfeeding on chronic non-communicable diseases in adulthood: A review of evidence. Adv Biomed Res 3: 3.
- Mozaffarian D (2016) Dietary and Policy Priorities for Cardiovascular Disease, Diabetes, and Obesity: A Comprehensive Review. Circulation 133: 187-225.
- 42. Hawkes C, Smith TG, Jewell J, Wardle J, Hammond RA, et al. (2015) Smart food policies for obesity prevention. Lancet 385: 2410-2421.
- 43. Vandevijvere S, Swinburn B (2014) International Network for Food and Obesity/non-communicable diseases (NCDs) Research, Monitoring and Action Support (INFORMAS). Towards global benchmarking of food environments and policies to reduce obesity and diet-related noncommunicable diseases: design and methods for nation-wide surveys. BMJ Open 4: e005339.
- 44. Victora CG, Habicht JP, Bryce J (2004) Evidence-based public health: moving beyond randomized trials. Am J Public Health 94: 400-405.