REVIEW

Mini-review: Obesity in Caribbean Youth

EA Traboulay[1], OPA Hoyte[2]

ABSTRACT

Objective: *Our focus was on the determination of the growing number of youths of every race and ethnicity, diagnosed with obesity and its co-morbidities in the Caribbean. We reviewed the causes and strategies to combat obesity, and the implications of the fast food industry in enabling the escalation of obesity.*

Methods: *We consulted several databases such as PubMed, MEDLINE, the Obesity Gene Map Database, and the USEPA Toxicity Reference Database. Organizations such as the World Health Organization (WHO), Centres for Disease Control and Prevention (CDC), Organization for Economic Co-operation and Development (OECD), and the Pan American Health Organization (PAHO) were used as information sources.*

Results: *Transgenerational effects and triggers like obesogens, pathogens, environmental stress, antibiotics and gut microbiota are some of the causes of obesity, and some of these triggers are imprinted epigenetically early in embryonic development, leading to lifelong obesity. With an estimated population of 42 million in the Caribbean, the economic cost of obesity, including medical, absenteeism, presenteeism, insurance, disability, direct and indirect cost, was estimated at 68.5 billion USD with 88.2 million quality-adjusted life years lost.*

Conclusion: *Genome-wide association studies have established that genetics play a role in the aetiology of this "non-communicable" disease. While the development of personalized interventions according to genotype is futuristic, we must focus on effective nutrition and physical education (PE) classes in schools and establishing monitoring programmes using simple tools such as scales and tape measures as suggested intervention. A Pigovian tax to control the fast food industry is mandatory. Nevertheless, lifestyle adjustment, including alterations in diet and increased physical activity, continues to be a sound recommendation.*

Keywords: Epigenicity, gut microbiota, obesity, obesogens, transgenerational effects

Mini-revisión: Obesidad en la Juventud del Caribe

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RESUMEN

Objetivo: *Nuestra mira estuvo en determinar el número creciente de jóvenes de cada raza y grupo étnico, diagnosticados con obesidad y sus comorbilidades en el Caribe. Se revisaron las causas y las estrategias para combatir la obesidad, y las implicaciones de la industria de comida rápida en relación con el aumento de la obesidad.*

Métodos: *Consultamos varias bases de datos, tales como PubMed, MEDLINE, Mapa Genético de la Obesidad y USEPA Toxicity Reference Database. Organizaciones tales como la Organización Mundial de la Salud (OMS), Centros para el Control y Prevención de Enfermedades (CCPE), la Organización*

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para la Cooperación y el Desarrollo Económicos (OCDE), y la Organización Panamericana de la Salud (OPS) fueron utilizadas como fuentes de información.

Resultados: *Los efectos transgeneracionales y los factores desencadenantes como los obesógenos, los agentes patógenos, el estrés ambiental, los antibióticos y la microbiota intestinal, son algunas de las causas de la obesidad y algunos de estos factores desencadenantes se imprimen epigenéticamente temprano en el desarrollo embrionario, conduciendo a la obesidad de por vida. Con una población estimada de 42 millones en el Caribe, el costo económico de la obesidad – incluyendo médicos, ausentismo, presentismo, seguro, discapacidad, costos directos e indirectos – fue estimado en 68.5 billones de USD con 88.2 millones de años de vida ajustados por calidad perdidos.*

Conclusión: *Los Estudios de Asociación del Genoma Completo han establecido que la genética juega un papel en la etiología de esta enfermedad "no transmisible". Si bien por una parte el desarrollo de las intervenciones personalizadas según el genotipo es futurística, por otra parte debemos centrarnos en una nutrición eficaz y clases de educación física en las escuelas, a la par de establecer programas de control utilizando herramientas simples tales como balanzas y cintas métricas, como sugerencia de intervención. Un impuesto pigouviano para controlar la industria de comida rápida tiene que ser obligatorio. Sin embargo, el ajuste del estilo de vida – incluyendo las alteraciones en la dieta y el aumento de la actividad física – sigue siendo una recomendación acertada.*

Palabras claves: Epigenicidad, microbiota intestinal, obesidad, obesógenos, efectos transgeneracionales

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INTRODUCTION

"Young people are in a condition like permanent intoxication,
Because youth is sweet and they are growing."
Aristotle – Nicomachean Ethics

We have chosen to focus our study on the first 24 years of life of our Caribbean youth. A reason for targeting this age range in our study is the fact that there are currently 1.8 billion young people in the world, aged 10 to 24 years, and they comprise more than a quarter of the world's population (1). Every one of these young people is susceptible to obesity and its co-morbidities and presents an important window for intervention with regard to this disease.

The World Health Organization (WHO), the Centers for Disease Control and Prevention (CDC) and every medical and obesity association around the world have declared obesity a global epidemic. We are finding that newborns, children, adolescents and young people are exhibiting obesity at alarming rates. Global projections indicate that the number of obese adults in 2015 will increase to over 700 million, up from 500 million in 2008, and the number of overweight or pre-obese adults will rise to 2.3 billion or 33% of the global population in 2015 (2). In fact, a WHO document (3) indicated that 78% of all deaths in Trinidad and Tobago "could be attributed" to non-communicable diseases, which include obesity and its co-morbidities (4).

This mini-review is informative, but raises enough questions to pique curiosity and arouse the awareness of the reader regarding the health threat that obesity poses, and lead to lifestyle changes, which enhance physical well-being and longevity. We believe that many of the strategies to combat obesity, based on reducing caloric intake, are marginally effective due to an incomplete understanding of the multiple aetiologies of the disease, including genetics, epigenetics, environmental obesogens, bacteriological and viral pathogens, neurobiology, and molecular cell biology. Recognizing obesity to be as much a psychological issue as a physical condition, we recommend the integration of psychological techniques and therapies into weight management protocols focussing on treating the disease and not the symptoms.

METHOD

The methodological procedure used in this paper was a narrative approach with analysis from various sources. We consulted several databases such as PubMed, MEDLINE, the Obesity Gene Map Database, and the USEPA Toxicity Reference Database. Also used were leading organizations such as the WHO, CDC, the Organization for Economic Co-operation and Development (OECD) and the Pan American Health Organization (PAHO) as information sources. Because of the paucity of current Caribbean data on obesity, we have, therefore, resorted to using data from the United States of America (USA) and Latin America to demonstrate the nature and magnitude of the obesity problem, which we believe to exist worldwide.

LITERATURE REVIEW

Global obesity

World Health Statistics (5) revealed that the world is becoming "heavier", resulting in 2.8 million deaths per year from obesity. In a snapshot of global health, the WHO reported that, in the 28-year period 1980–2008, the global incidence of obesity almost doubled to half a billion people; if left unchecked, we believe it will double again before 2020. The 2012 report goes on to signify that overweight and obese individuals 20

years old and over were greatest in the WHO Region of the Americas, indicating that 62% were overweight in both genders and 26% were obese. The figures were comparatively lower in the South-East Asia Region where only 14% were overweight in both genders with a small margin of only 3% exhibiting obesity. In the European Region and the Eastern Mediterranean Region, over 50% of women were overweight and about half of overweight women were obese. The 2011 OECD report (6) shows that obesity varies almost ten-fold among 34 OECD countries, from a low of 4% in China to 35% or more in the USA and Greece (Fig. 1).

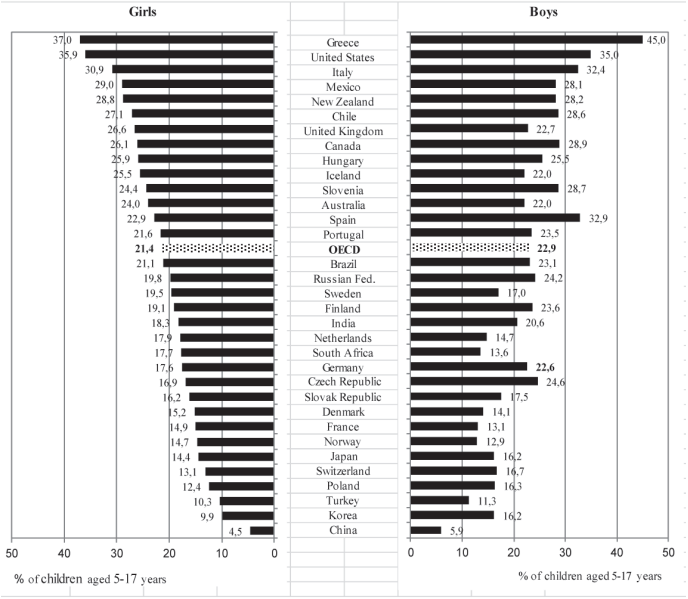
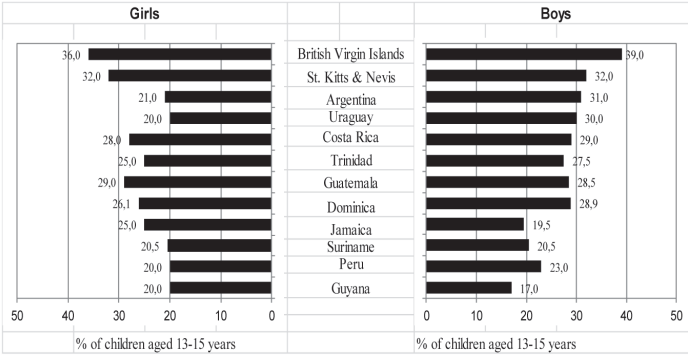


Fig. 1: Children (5–17 years) who are overweight including obese. Source: (5)

Caribbean and Latin America

Childhood obesity is a growing concern in the Caribbean. The 2011 Health Report Card for Trinidad and Tobago (4) and a recent report prepared by the Caribbean Food and Nutrition Institute (CFNI) over the period 2009–2010 (7), found that "23% of primary school children in Trinidad and Tobago were overweight/obese; a further 25% of secondary school children were overweight or obese with 14% being underweight." The Food and Agriculture Organization of the United Nations (FAO) nutrition country profile for Barbados (8) reflected the paucity of data by referring to a 1981 study which indicated that a body mass index (BMI) > 25 in school children between the ages of 10 and 19 years was fairly high, especially among girls; 20% among 10–14 year olds and 19% among 15–19 year olds.

A similar study conducted in 2003 for The Bahamas (9) indicated that female adolescents appeared to be at slightly greater risk for overweight and obesity than males, with approximately 20% of the females 15–16 years being overweight *versus* 16.2% of the males. A 2009 survey of students from Latin America and the Caribbean [LAC], conducted by PAHO (10), showed that the Virgin Islands recorded the highest obesity rate in 15–16-year student age groups, with boys recording 39% and girls 36%. We have compiled the data and plotted them in Fig. 2 to illustrate the magnitude of the problem. Schwiebbe and colleagues (11) investigated the 4–16-year age groups of children in Bonaire and found that 24.3% of the boys and 31.9% of the girls were overweight or obese. Further investigations revealed that 50% of the children had unhealthy food patterns, consuming less than two pieces of fruit per day and no vegetables.



Source: Author's construct compiled from (5, 10)

Fig. 2: Percentage of overweight students including obese 13–15 year olds in Latin America and the Caribbean.

DIAGNOSING OBESITY

Historically, BMI, developed in the early to the mid-nineteenth century, was the principal indicator of obesity, and remains the gold standard today. The BMI equation is expressed as body mass [kg] divided by the square of the height [m^2] (12, 13) and for children under five years of age, WHO Child Growth Standards (14) are used. In this paper, we use BMI measurements of ≥ 25 and ≥ 30 kg/m^2 at age 18 years to describe pre-obesity and obesity, respectively.

We have constructed Table 1 to show the relationship between BMI and the potential for diagnosing disease risk in our youth. Furthermore, we provide some basic non-pharmacological suggestions to improve health such as exercise for at least 150 minutes each week, eating nutrient-dense low-calorie foods such as fruits and vegetables high in fibre, together with olive oil as a fat source, broiled or grilled meat, and limiting salt and sugar.

What are the causes of obesity in our youth?

The central theme of this question lies within a historical and basic concept of nutrition and metabolism. We observe changes in our body mass when there is a disparity between the energy provided by the nourishment that we consume and the energy expended in our daily activities. This simple statement essentially conforms to the law of conservation of energy, adapted for thermodynamic systems, and we can articulate this equation in a straightforward manner:

Energy intake = energy burned + energy stored [Eq. 1]

Table 1: Body mass index (BMI) and associated disease risk in our youth

BMI (kg/m^2)	Obesity class	Potential risk of 20th century diseases	Non-pharmacological protocols
< 18.5	Underweight	Diseases are anorexia, chemical imbalances in the body, anaemia, osteoporosis, depression and reduced longevity.	Psychological and medical consultation required. Follow nutritious diet as shown in pre-obesity with light physical activity (PE).
18.5–24.9	Normal	OK	Light PE (1–3x/wk); follow a nutritious diet as shown in pre-obesity. Limit salt and sugar.
25.0–29.9	Pre-obesity	Diseases are hypertension, fatty liver, gout, elevated triglycerides, pulmonary disease, cataract, retina neuropathy, various cancers, stroke and gallbladder disease.	Moderate PE (3–5x/wk) with calorie restriction diet and **24-hr fasting (1x/wk)**. Focus on a variety of fruits; limit salt and sugar; reduce refined carbohydrate; increase fibre in diet; use vinegar in salads; eat more vegetables; use natural fats (olive oil *etc*), spices and herbs. Use lean protein broiled, baked or grilled. Psychological and medical consult.
30.0–34.9	Obesity I	Type 2 diabetes mellitus, gout, phlebitis, hypertension, fatty liver, pulmonary disease, various cancers, retina neuropathy, cataracts, inflammatory diseases, stroke, gallbladder disease, cancers and reduced longevity.	Active PE (3–5x/wk.). Intermittent **24-hr fasting (2x/wk)**. Diet and nutrition guidelines are the same as in pre-obesity. Need mandatory psychological and medical consult.
35.0–39.9	Obesity II	Associated diseases are the same as Obesity 1.	Active PE (3–5x/wk). Intermittent **24-hr fasting (2x/wk)**. Diet and nutrition guidelines are the same as in pre-obesity. Need mandatory psychological and medical consult.
≥ 40.0	Morbid obesity	Associated diseases are the same as Obesity 1.	Active PE (3–5x/wk). Intermittent **24-hr fasting (2x/wk)**. Diet and nutrition guidelines are the same as those listed in pre-obesity above. Need mandatory psychological and medical consult.

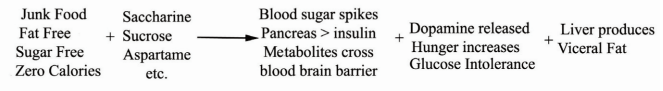
Source: Author's construct (2014)

Although fundamentally true, we run into problems when we look at homeostasis *in vivo*. Measuring energy intake is relatively simple, but determining calorie assimilation in the body is more problematic. To complicate our analysis, we must be aware that our body will neutralize any controlled decreases in food intake by an involuntary reduction in energy expenditure, making the weight loss conundrum even more challenging than a simple elucidation of equation 1.

Since junk food is highly processed and carefully engineered, it is easy to manipulate the contents to trick the neural circuitry of the brain. Take a moment, and recall the pleasurable sensation we feel after tasting our favourite chocolate cake or ice cream and wonder why. The reason is that our brain is being flooded with dopamine. An obese person instinctively chooses high-calorie and nutritionally deficient foods that produce a dopamine response. Do you think your brain will release dopamine at the sight or smell of a low-calorie vegetable such as broccoli or cauliflower? The pleasure reward response is just not there! A solution to the obesity epidemic lies in re-programming our neural circuits to reject high-calorie and nutritionally poor foods in favour of low-calorie and nutrient-dense foods.

The fast food industry employs a seemingly inefficient business model where 10 calories of energy are required to produce 1 calorie of junk food. Although this junk food is nutritionally deficient, it is tasty, and millions of obese people consume these products, making the fast food industry very profitable. We show the metabolism of junk food in equation 2.

[Eq. 2]



Suez and his team (15) found that non-caloric sweeten-

ers could actually hasten the development of glucose intolerance and metabolic disease by altering the make-up of the gut microbiota. Because these artificial sweeteners contain no fibre, they go directly to the liver, which produces visceral fat.

What is probably easier to explain than equations 1 and 2 is what we observe in the marketplace. The strategy of the fast food industry is to market the mass production of cheap, desirable, and readily available food. These conditions create a tipping point within our energy equation, resulting in a decrease in physical activity and the subsequent "eat more" gain weight phase. Researchers have quantified this energy trend in US children, which we have summarized in Table 2 from various sources (16–19). Rideout and other researchers (20–22) have also documented the trends that influence reduced physical activity in US children, which are summarized in Table 3.

The pathogen effect

Advances in DNA sequencing techniques have revealed a new level of complexity in the study of the microbiota and environment of the human gut, which we call the second genome. Bäckhed and colleagues (23) observed that gnotobiotic mice tend to be slimmer than normal mice. When they transplanted normal microbiota from the distal intestine of conventionally raised mice to the gnotobiotic mice, those rodents gained 60% in their body fat and exhibited insulin resistance within 14 days. By extrapolating to humans, we may speculate whether the trillions of microbes that outnumber the cells of our body and colonize our intestines could function as a metabolic organ that communicates with our own human metabolic apparatus. Can we now say that we have identified a possible obesogen target – gut microbiota?

Adenoviruses constitute another class of pathogens that infect humans (24, 25). Of the 52 adenoviruses that do, human adenovirus 36 [AdV-36] is the only one linked to obesity in humans (26) and this may be another target for identifying the function of adipogenic viruses in the human obesity story. Understanding the role of adenoviruses in the human body may illuminate and allow us to develop cause-specific treatments for obesity and, ultimately, its management and/or cure. Historically, we considered obesity a chronic "non-communicable disease"; however, we have reached the point of questioning whether obesity is contagious or whether it is an infectious disease such as a cold transmitted by a bacterial infection or like the flu as a viral infection.

Table 2: Trends in food supply and consumption in the United States of America relevant to obesity prevention over time

Variable	Changes in food consumption over time
Increase in portion size	Increased from 1977–1978 to 2003–2006 in youth 2–18 years of age. • Soft drinks increased by ~100 mL in size • Fruit drinks increased by ~43 mL in size • Hamburgers increased by 31 g/portion (+90 kcal/day) • Cheeseburgers increased by 22 g/portion (+90 kcal/day) • Pizza increased by 41 g/portion (+131 kcal/day) • Mexican fast food increased by 48 g/portion (+149 kcal/day)
Daily calorie intake	Increased in youth 2–18 years of age. • 1977–1978 = 1842 kcal/day • 1989–1991 = 1802 kcal/day • 1994–1998 = 1947 kcal/day • 2003–2006 = 2022 kcal/day
Consumption of sugar-sweetened beverage	Increased in youth 2–19 years of age. • 1988–1994 = 79% • 1999–2004 = 81%
Percentage of daily food eaten away from home	Increased in youth 2–18 years of age. • 1994–1998 = 32.7% • 2003–2006 = 33.9%
Percentage of fast food consumed at home *versus* in restaurants	Increased in youth 2–18 years of age. • 1994–1998 = 38% • 2003–2006 = 49%

Source: (16–19)

Table 3: Trends that influence sedentary behaviour in youths in the United States of America

Variable	Changes in youth behaviour over time
Hours of TV viewing	Increased in youth 8–18 years of age. • 1999 = 3:47 hr/day • 2004 = 3:51 hr/day • 2009 = 4:29 hr/day
Internet usage	Increased in youth 12–17 years of age. • 2000 = 73% use among US teens • 2009 = 93% use among US teens
Home Internet access	Increased in youth 8–18 years of age. • 1999 = 47% • 2004 = 74% • 2009 = 84%
Hours of playing video games	Increased in youth 8–18 years of age. • 1999 = 0:26 hr/day • 2004 = 0:49 hr/day • 2009 = 1:13 hr/day
Social networking use	Increased in youth 12–17 years of age. • 2006 = 55% use among teens • 2009 = 73% use among teens

Source: (20–22, 44)

Environmental stress and epigenetics

In studying how non-genetic transgenerational effects of adverse environmental exposures might contribute to population obesity trends, researchers have made significant advances over the years in their understanding of the basic epigenetic mechanisms involved in modifying the genome without changing the nucleotide sequence. These modifications, such as histone acetylation and DNA methylation, to name a few, occur during mitosis, can be inherited, and control the ability of the genome region to be transcribed into a specific phenotype. Non-genetic inheritance is all the more remarkable when transmission is down the male-line where paternal grandfathers transmit phenotypes to grandsons.

A landmark paper by Heijmans and colleagues (27) demonstrated that adult disease risk is associated with unhealthy environmental stress, which can cause epigenetic changes early in embryonic growth that persist throughout life. They studied pregnant individuals during the Dutch Hunger Winter, when the Germans imposed a food embargo on Holland during the winter of 1944/45. The team found that prenatal exposure to famine is associated with constant hypomethylation of the insulin growth factor 2 [IGF2], which they observed decades later in test subjects. Ravelli and colleagues (28) reported in a study of 741 subjects that men and women conceived during the same Dutch winter famine had higher rates of obesity at 19 years of age than those who were conceived before or after the famine.

Seminal papers by Hult and colleagues (29) at the Karolinska Institute in Sweden, and Fung (30) at Wheaton College in Illinois have shown that malnutrition in early gestation causes greater susceptibility to obesity. Regardless of ethnicity or geographic location, the researchers always observed the effects of famine and the resultant obesity. Hult *et al* tested 1339 adults born during the Biafra famine in Nigeria some 40 years ago, while Fung studied subjects in the Chinese Famine of 1959–1961. Their results showed that the adults studied attained a BMI of five points over the control group, and developed high blood pressure and glucose intolerance.

In layman's terms, what these groundbreaking papers suggest is that even today, any man or woman who is subject to environmental stress (malnutrition, abuse, drugs, alcohol, tobacco *etc*), will experience epigenetic programming effects. In the case of a pregnant woman, the results will affect her body, her unborn child, and her grandchildren. This is because the developing fetus within the mother already contains all the ova required for a lifetime in her ovaries and which will exhibit the same epigenetic programming caused by the mother's stress. When the oocyte of this F_1 offspring (daughter) is eventually fertilized by sperm, it will develop into a newborn individual (F_2 granddaughter) exhibiting an obesity phenotype. Other investigators corroborated these findings when studying rhesus monkeys treated with bisphenol A (BPA), a known obesogen, and found that hypomethylation occurred in the DNA, resulting in chromosomal impairment in the F_1 generation (31, 32).

Genetic factors

The search for the "Jack Spratt gene" was fraught with failure, but Neel (33) tackled the problem and proposed the "thrifty gene" hypothesis in the early 1960s. This hypothesis acknowledges that the "thrifty" genotype would have been invaluable for hunter-gatherer populations, particularly childbearing women. The rationale being that it would have allowed these women to fatten up quicker during times of plenty, and fatter individuals carrying the thrifty genes would live longer during times of food shortages. However, today we have a constant supply of food available in supermarkets and fast-food restaurants, and this "gene" efficiently prepares individuals for a famine that never comes. The result is extensive and protracted obesity with its health-related problems.

By tracking participants from birth through 38 years of age, Belsky and colleagues (34) showed that the genetic predisposition to obesity becomes detectable at about three years of age and stays on a developmental trajectory that has a cumulative effect on adult obesity. Numerous papers on families, adoptees, twins, and adopted twins have all established that genetic factors are likely to be accountable for 45–75% of the inter-individual difference in BMI (35, 36). These genetic components [monogenic and polygenic obesity] are many, and function through the full range of possible processes, including energy consumption, energy outflow, and the apportioning of nutrients between fat and sinewy muscle (37, 38).

Microdeletion of genes located at 16p11.2 appears to be a predisposing factor contributing to the obese condition. Walters and an international group of 55 researchers (39) have

Table 4: A selective list of genes associated with obesity phenotypes

Gene	Gene name	Location	Phenotypes measured
ACDC	Adipocyte, C1Q and collagen domain containing, adiponectin	3q27	Body mass index (BMI) and waist-to-hip ratio (WHR)
ADRA2A	Adrenergic receptor α-2A	10q24–q26	Skinfold ratio and abdominal fat
ADRA2B	Adrenergic receptor α-2B	2p13–q13	Basal metabolic rate and weight gain
ADRβ1	Adrenergic receptor β-1	10q24–q26	Weight, fat mass and BMI
ADRβ2	Adrenergic receptor β-2 surface	5q31–q32	WHR, obesity and BMI, subcutaneous fat and adipocyte lipolysis
ADRβ3	Adrenergic receptor β-3	8p12–p11.2	WHR, BMI and weight-gain capacity
LEP	Leptin (obesity homologue, mouse)	7q31.3	Obesity and BMI
LEPR	Leptin receptor	1p31	BMI, fat mass, overweight
NR3C1	Nuclear receptor subfamily 3, group C, member 1 (glucocorticoid receptor)	5q31	Obesity and overweight
PPARG	Peroxisome proliferative activated receptor, γ (gamma)	3p25	BMI, weight and fat mass
UCP1	Uncoupling protein 1 (mitochondrial, proton carrier)	4q28–q31	Weight, BMI and WHR
UCP2	Uncoupling protein 2 (mitochondrial, proton carrier)	11q13	Include BMI, obesity and skinfold thickness
UCP3	Uncoupling protein 3 (mitochondrial, proton carrier)	11q13	Include caloric intake, fat intake, fat mass, WHR, BMI and skinfold thickness

Source: (43)

shown that hemizygosity of a 600-kilobase (kb) region on the short arm (p) of chromosome 16 at position 11.2 from the centromere, containing about 25 genes, causes an extremely penetrant form of obesity that is often accompanying hyperphagia and rational infirmities. Furthermore, they show that a duplication of the gene results in a BMI of 18.5 kg/m^2 – borderline anorexia nervosa.

Bochukova investigated the copy-number variations (CNVs), which are sizeable DNA fragments erased or repeated in the genome. In 300 patients with severe early-onset obesity, plus 7366 healthy controls for CNVs, Bochukova found several deletions shared in patients with early onset obesity when compared to the controls. Furthermore, those individuals with deletions in the SH2B1 gene had hyperphagia and exhibited a disproportionate degree of insulin resistance for their level of obesity (40). These pivotal studies by Walters and Bochukova support the "genetic predisposing factor" in individuals with obesity.

Genes associated with obesity can be found in the Obesity Gene Map Database. We found numerous biomarkers connected to human obesity, directly or indirectly, and Snyder *et al* (41) have determined that they now number more than 425. There are some genes that are explicitly involved in controlling food intake, while others influence different metabolic and signalling pathways such as adipogenesis, which affects the energy balance equation. Therefore, body weights of those persons who are carriers of specific malfunctioning gene alterations, or polymorphisms experience fat accumulation (42). Table 4, compiled by Bell and colleagues (43), is provided here to illustrate the complexity of the problem and the numerous genes involved in searching for the "Jack Spratt gene".

ENVIRONMENTAL AND LIFESTYLE FACTORS

Studies and surveys conducted by Martinez *et al* using indirect indicators of physical activity such as TV viewing, numbers of cars by households or leisure time show that a decrease in energy outflow might be a major factor in the present-day pandemic of obesity (44).

Obesogens

We consulted the USEPA's Toxicity Reference Database and found several groups of chemicals that are the source of obesity in animals and humans. These obesogens, a term coined by Grün and Blumberg (45), are foreign chemicals that disrupt our hormones and normal body functions. This category of toxins comprises polychlorinated biphenyls (PCBs), dichlorodiphenyltrichloroethane (DDT) – a chlorinated organic insecticide, dioxin, some pesticides, herbicides, and many plasticizers (phthalate esters), like bisphenol A, which have been implicated in obesity. Rather than cite a litany of chemicals, we will look at one group of obesogens – plasticizers.

In 1976, Traboulay (46) found traces of mono-benzyl and monoethylhexyl phthalates in the Potomac River finished drinking water, which partially serves the greater Washington Metropolitan area. These phthalates constitute a family of synthetic compounds used in the manufacture of plastics. In this

experiment, conducted at Howard University, the phthalate esters were leaching out of PVC pipes in the chlorination tank just before the finished water entered the main water delivery system on its way to pumping stations. Subsequently, other investigators in 2007 and again in 2011 have shown a direct link between the increased presence of these synthetic compounds in urine and fat from the waist circumference of obese men (47, 48).

How can we avoid obesogens?

While we may say that obesogens are ubiquitous, we cite a few examples to show the enormity of the problem. Most obesogens are in our foods *eg* PCBs are in most non-organic fruits and kitchen vegetables. These would include peaches, apples, sweet bell peppers, celery, nectarines, strawberries, cherries, lettuce, imported grapes, carrots and pears. We must avoid beef and chicken raised with genetically modified (GM) corn or soy as well as farm-raised salmon. Without labelling information, we should consume organic fresh vegetables, wild fish and free-range chicken or chickens not fed with GM corn/soy products. Fruits that are safe for conventional diets include oranges, grapefruit, kiwi, bananas, pineapple, mango and watermelon.

We suggest removing insecticides, pesticides, and other chlorinated hydrocarbons from non-organic fresh vegetables by washing and soaking in a mixture of water and vinegar in a ratio of 3:1 for a few minutes or by washing in warm soapy water. We suggest that youth stay away from plasticizers like BPA, found in the coating of tin cans used to package foods such as baby formula, tuna, soup beans and tomatoes; and canned beverages such as energy drinks, and sports drink bottles made from plastic.

Genetically modified foods

As researchers, we have followed the genetically modified food debate for many years, and while the US has approved GM foods, the European Union (EU) has rejected its use and importation in the face of mounting pressure from US lobbyists. Without getting into the specifics of the GM debate, it is safe to say that the EU has decided to err on the side of safety, especially when the long-term effects of our health remains questionable.

As an example, one variety of GM corn incorporates a combination of four genetically engineered events (DP1507, MON89034, MON88017 and DP59112) that make this corn plant resistant to insect pests and noxious weeds. We see that currently there are six pesticides (Cry) and two herbicides (PAT) enzymes that were genetically engineered into the plant (Fig. 3). We may ask why four engineered events? The answer lies in the fact that insects and weeds soon build up a resistance to the toxins secreted by the corn plant and over time new toxins are needed. Another good question would be what are the effects of the insecticides and herbicides on the human body? We suggest that these toxins may be acting as obesogens, potential carcinogens, and/or possibly epigenetic modifiers.

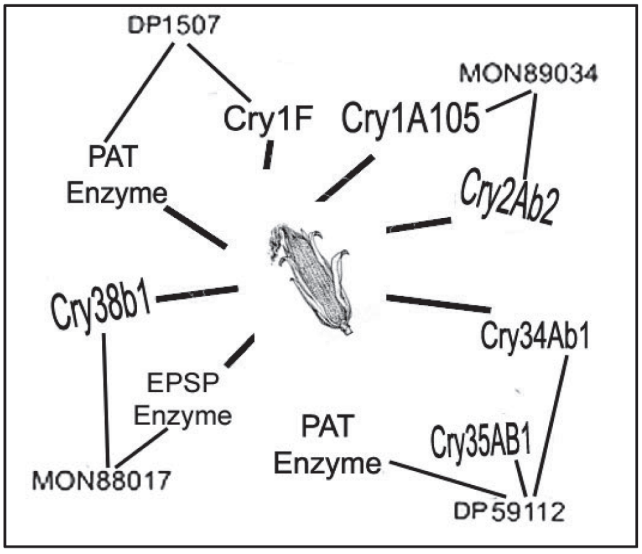


Fig. 3: This corn produces six insecticides producing toxins (Cry) and two herbicide toxins (PAT).
Source: Author's construct compiled from the GM Crop Database (2014).

The role of antibiotics in obesity

Ozawa (49) has documented that farmers have been administering antibiotics in animal feed for over 50 years to effect weight gain in cattle, swine, chickens and turkeys, which translated to a financial gain to the farmer. Cho and colleagues evaluated changes in gut microbiome in mice after antibiotic therapy and observed the following: 1) the consumption of antibiotics altered the bacteria in the guts of mice and 2) modified how the bacteria broke down nutrients, 3) activated more genes that converted carbohydrates into fatty acids and 4) turned on genes related to lipid conversion in the liver. Presumably, these shifts in molecular pathways enable fat build-up as observed in farm animals, and the treated mice became obese (50).

Transande and colleagues (51) have indicated that while antibiotics are important drugs, they come at a cost, which physicians and parents do not appreciate when provided at an early age to children. To confirm this theory, the researchers examined 11 552 children in the United Kingdom who had taken antibiotics in infancy (six months old) and found that they became overweight by the time they were three years old. Administration of antibiotics later in infancy (6–14 months and 15–23 months) was not always associated with increased body weight. Furthermore, the administration of non-antibiotic medications did not result in an increased body mass.

Lifestyle modification

The existence of a genetic propensity to obesity does not mean that obesity is preordained, since environmental factors are crucial for the appearance of genetic potential. Leibel and colleagues have shown that the static weight loss rule overlooks dynamic physiological adaptations to the altered body weight that leads to changes in both the resting metabolic rate as well

as the energy cost of physical activity (52). Therefore, we need to assist youth in making sound decisions in the way changes in diet or physical activity will transform their body shape over time.

Consider looking at a mathematical modelling methodology to human metabolism proposed by Thomas, as well as Song and colleagues (53–55). For a practical exercise, Hall and colleagues have developed a web-based body weight simulator, which illustrates the benefits over the static method, and can calculate and show graphically an individual's weight loss over time (56). See the programme at http://bwsimulator.niddk.nih.gov/.

THE ECONOMIC COST OF OBESITY

We could find no definitive quantitative data regarding the current economic burden that obesity and its co-morbidities place on the Caribbean, as most of the existing published base data are circa 2000 and older. Various researchers in the USA have used numerical models to evaluate the impact of obesity on a country's economy. Of interest are the various parameters used in determining direct medical costs, indirect costs and productivity costs (57–61), shown in Table 5. We have extrapolated and modified the data in Table 5 and adjusted the numbers in previous work by Caribbean researchers (62, 63) estimating the economic cost of diabetes and hypertension as a function of the gross domestic product (GDP).

We make the following assumptions: a) population in the Caribbean at 42 million; b) observed overweight and obesity at 30% of the population; c) the medical cost per capita at $700 US; d) absenteeism at $75 US per capita; e) presenteeism at $75 US per capita; f) indirect costs at $355 US per capita; g) direct costs at $250 US per capita and h) disability costs at $75 US per capita. Therefore, we estimate that a realistic cost of treating obesity and its co-morbidities today, in the Caribbean, is $68.5 billion US per year. When we look at years of life lost to obesity, we estimate an average of seven years/obese person, resulting in 88.2 million years of quality-adjusted life years lost in the Caribbean.

CAN OBESITY BE REVERSED OR CURED?

This is an uphill battle but the resounding answer is YES to both questions. Consider that from breakfast to dinner, mil-

Table 5: Key obesity parameters used in modelling obesity in the United States of America

Parameter	Remarks
Direct medical costs	The estimated annual direct cost of childhood obesity in the US is $14.3 billion with total obesity costs at $147 billion.
Productivity costs	
Absenteeism	Annual productivity losses due to obesity-related absenteeism of between $3.38 billion ($79 per obese individual) and $6.38 billion ($132 per obese individual).
Presenteeism	Obesity contributes to productivity loss if obese individuals are less productive while present at the workplace. The estimated monetary value of this loss among obese workers is $11.7 billion per year.
Disability	In addition to absenteeism and presenteeism, obesity may lead to an increase in disability payments and disability insurance premiums.
Premature mortality	Several studies have found a connection between obesity and premature mortality.
Health insurance	The size of the welfare loss due to the obesity externality in the US is at $150 per capita.
Transportation costs	Increases in body weight among Americans (*vis-à-vis* Japanese) mean that more fuel and, potentially, larger vehicles are required to transport the same number of commuters and travellers each year.
Human capital accumulation	Effects of obesity and overweight on educational attainment also represent a potential economic impact.

Source: (52–56)

lions of people globally devour "fast foods" from their favourite outlets. The battle begins with corporate philosophy whose top priority is to have a "fast food" outlet no more than four minutes from their clients and, if necessary, open all night long. Although by no means comprehensive because of obvious limitations, Fig. 4 is a graphical representation of our proposed *a priori* obesity syndrome hypothesis. Our purpose here is to illustrate the complexity of the syndrome and the challenges we face in finding a cure.

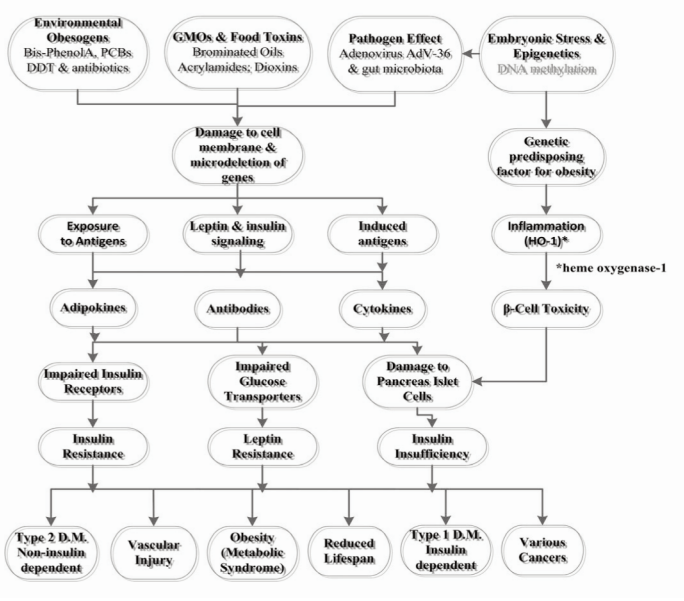


Fig. 4: Obesity syndrome hypothesis.
Source: Author's construct (2014)

There appears to be a positive aspect appearing on the horizon – synthetic biology. Rossger and colleagues (64) used biotechnology techniques to design a lipid-sensing regulator (LSR) encapsulated within an autologous designer cell which is implanted *in vivo* to control diet-induced obesity. The LSR monitors disease-relevant metabolites in the peripheral circulation and coordinates the secretion of therapeutic proteins in an evolving pathologic condition. This works because the LSR device contains a pramlintide molecule, which is a Food and Drug Administration (FDA) approved anorectic peptide hormone. This hormone controls hyperphagia, stimulates satiety and reduces gastric emptying. The anorectic hormone also restricts high-caloric food intake, diminishing hyperlipidaemic blood levels, decreasing body weight, and re-establishing the energy homeostasis of the organism. We hope that this procedure will soon lead to clinical trials in humans.

Can we emulate Europe's strategy to reduce obesity?

In 2003, the WHO (65) proposed a Pigovian tax, suggesting nations contemplate levying a tax on junk foods to persuade people to make healthy food choices. This tax gained traction in Europe as part of their attempts to counteract obesity. For example, Denmark instituted a tax on foods having more than 2.3% saturated fats, including meat, cheese, butter, edible oils, margarine, spreads, snacks, chocolate, ice cream, sugary drinks, and sweets. Consumers pay up to 30% more for a pack of butter and sweets. Economists have determined that tax earnings will exceed €200 million per year, and expect saturated fat consumption to decrease by 4%. However, lately, authorities are considering abolishing the "fat tax" because it said the tax had inflated food prices and put Danish jobs at risk. This political decision to sacrifice health and longevity in lieu of obesity for jobs needs further consideration by all parties.

The European Public Health Alliance (66) reports that the UK, Hungary, Italy and Finland have followed suit and they are expecting to receive an extra €70 million annually from this tax. France has instituted a similar tax policy on highly sugared beverages and drinks with artificial sweeteners and this levy will produce returns of €280 million annually. Although some Caribbean islands have legislated controls on trans fats in foods, they are not actively enforced (67). Caribbean politicians and legislators may want to consider this Pigovian tax strategy as a potential solution to their country's obesity problem.

SUGGESTED PROGRAMMES FOR GRADES K–FORM 5

Schools need a relatively simple and inexpensive programme to monitor their students. To be cost-effective, this programme need not involve additional staff, but it can use existing teachers, staff nurses or other staff to track the students' BMI and hip-to-weight ratio from primary through high school. We suggest that the main focal point to cure obesity is the school, from which emanates the other factors such as the roles played by communities, families, and organized physical activity (PE) activities, all acting in a concerted effort to reduce obesity. We have constructed Table 6 to provide the phenotypes of interest as well as the measurement methods that are easy to follow.

Primary prevention

A method of "primary prevention" should complement the monitoring programme outlined in Table 6. The definitive teaching tool is the food pyramid developed by Haddad and colleagues (68). Having discussed the link between childhood obesity and its adverse outcome on health in later years, we strongly believe that teaching our children about the food pyramid provides them with an important lesson in healthy eating. It is ideal for children because it gives them a visual reference to remember, but it also provides parents with additional information that they can appreciate. The challenge for parents is to prepare healthy foods in a way to interest their children, knowing that they are providing their children with one of the best gifts possible – the prospect of good eating habits and good health as adults.

Table 7 emphasizes activity strategies made into interesting and fun exercises that teach students the skills and the

Table 6: Common phenotypes used in monitoring obesity

Phenotypes	Measurement methods	Comments
Weight	Scales	Target body composition; quick and easy
Waist-to-hip ratio	Tape measure	Target body composition; quick and easy
Body mass index	Scales and tape measure	Target body composition; quick and easy
Skinfold thickness	Skin callipers	Target body composition; quick and easy

Source: Author's construct (2014)

Table 7: Suggested primary preventative programme in schools

School-based activity	Activity-related	Diet-related
Teach how to plant a kitchen garden	Introduce body mass index as well as the 50-yard dash for boys and girls for speed, agility, and body composition.	Eat more fruits, vegetables, legumes, whole grains, and nuts.
Teach about food systems	Chin-ups for boys and flex arm hanging for girls for upper body strength.	Remove sugars, solid fats and sugar-sweetened beverages from diet.
Teach value of nutrition	Require long jump, sit-ups, and push-ups for both boys and girls.	Balance the energy equation.
Teach preference for vegetables.	Decrease video games and television viewing.	For pregnant youth, suggest breastfeeding exclusively.
Teach skills necessary to make healthy food choices	Teach colours of healthy and unhealthy foods.	Ensure micronutrient intake to promote optimal linear growth.

Source: Author's construct (2014)

benefits of nutrition, diet, and physical activity. This will ensure the probability of shifting physical activity, eating, and weight management toward energy balance in the school population, including those individuals at high risk of becoming obese.

CONCLUSION

We conclude that reducing the incidence of obesity in any country, developed or developing, is feasible, but not easy. Research has uncovered many hitherto overlooked components of the health puzzle and has prompted a re-evaluation of old beliefs. We must recognize that some obesity determinants may have been operating in previous generations initiated by genetics or previous non-genetic epigenetic reprogramming of germ line cells.

RECOMMENDATIONS

We suggest the following simple rules:

- Avoid consuming foods with enticing labelling such as 'fat free' and 'sugar free' or 'zero calories' because they are still "junk food", nutritionally deficient and result in adverse health effects.
- Avoid eating something your great-grandmother would not acknowledge as food.
- Avoid food products with labelling information that a third-grader cannot pronounce.

We understand there are factors over which an individual has no control, such as genetics and, to a lesser extent, environmental toxins, and the presence of infectious agents in the environment. However, we cannot absolve ourselves of all responsibility for our health, since we are able to exercise control over lifestyle and reduce the likelihood of falling victim to obesity. For maximum effect and optimal results, the healthy lifestyle needs to start in early childhood with parent involvement. Furthermore, we must develop effective and meaningful educational programmes in our primary and high schools to bend the curve of youth obesity from a high of ~30% to a goal of 5%, which we suggest as a "good number" for the Caribbean. We believe that political leaders, school administrators, doctors, lawyers and teachers in the Caribbean can play a major role in improving obesity of our youth.

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