

The GraceKennedy Foundation Lecture 2016

**Overfed and Undernourished:
Dietary Choices in Modern Jamaica**
Marvin Reid

GraceKennedy Foundation

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The GraceKennedy Foundation and GraceKennedy Foundation Lectures

The GraceKennedy Foundation was established in 1982, in celebration of the company's 60th anniversary. The Foundation provides assistance in two main areas: education and the environment. This is accomplished primarily through the provision of grants to schools and charitable organizations; its scholarship and bursary programme; the GraceKennedy Jamaican Birthright Programme; the funding of two Professorial Chairs at The University of the West Indies and the Annual Lecture. Over the years the GraceKennedy Foundation has successfully established and fostered strong ties with a number of local and international organizations in order to have a significant impact on the groups and individuals that they assist. The lecture series serves as a good example of how the Foundation partners with esteemed academics and experts in the field to present topics that are critical to the development of the country.

Over the past 94 years, Grace Foods has placed great emphasis on providing wholesome, nutritious and delicious food products. The name Grace is synonymous with quality. With this in mind, we felt it was important to lead the discussion on the critical issue of nutrition. Nutrition plays a key role in the health of the nation and our hope is that the Lecture will be a springboard for change, not only in Jamaica but also across the Caribbean region.

We are confident that this Lecture will continue in the tradition of previous lectures and will become an invaluable resource for all who seek a deeper understanding of national issues.

The Foundation distributes copies of the lecture book to schools and public libraries across the island, and provides an e-book online at www.gracekennedy.com in the hope that the Lecture's reach will extend beyond those present at its delivery. The Foundation, as always, welcomes and looks forward to your comments.

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The GraceKennedy Foundation Lecture, 2016

OVERFED AND UNDERNOURISHED: DIETARY CHOICES IN MODERN JAMAICA

One of the first GraceKennedy Scholars was a tall teenage athlete from Ardenne High School who had won the bronze medal in the Long Jump at Boys' Championships in 1982 and the silver in 1983. He had also been selected to receive the Nellie Olsen Award for being the Most Outstanding student at Ardenne High. That scholar was Marvin Reid, who received the GraceKennedy Scholarship to study medicine in 1983.

In 2016, we welcome as our GraceKennedy lecturer, Professor Marvin Reid, who has more than fulfilled that early promise of excellence and who this afternoon, shares with us his thoughts on nutrition in a lecture entitled "Overfed and Undernourished: Dietary Choices in Modern Jamaica."

His lecture forces us to face and consider the choices we make daily about what we eat and the implications of those choices. The basic requirement of eating to satisfy metabolic demand is affected by many factors and influences; among these are our unique genetic makeup, our beliefs about food, our appetite, state of health and age or stage of life. Important also in our dietary choices are environmental factors such as availability, cost, cultural norms and government policies.

Understanding this is vital because of the increasing health risks posed by making bad nutritional choices. The significant research evidence detailing the links between nutritional status and disease has exposed these risks, many of which are associated with lifestyle choices. Obesity in children and adults is a major factor in diagnoses of diabetes and hypertension, while undernourishment, particularly in children, resulting in wasting, stunting and the repercussions of these conditions, is also a major concern. Although he does not provide us with prescriptions, Professor Reid offers a number of recommendations for consideration when we make choices about our daily diets.

Eminently qualified to guide us in our thoughts on these vitally important issues, Professor Reid qualified with the MBBS from The University of the West Indies (UWI) in 1988, then undertook postgraduate studies in clinical nutrition at Southampton University in the United Kingdom. He earned the PhD degree from The UWI in 1996, did postdoctoral studies at Baylor University in the USA, and has also pursued

a number of study programmes, at The UWI and at universities abroad, designed to enhance his clinical and research expertise. He has had a private family practice since 1996.

His association with the Tropical Medicine Research Institute (TMRI) began in 1990, when he worked as a Medical Officer. He was promoted to the rank of Professor in 2009 and, since 2013, he has been the Director of the Tropical Metabolism Research Unit (TMRU), an arm of TMRI and the premier nutrition research unit within The UWI. The major function of the TMRU is research but the Unit, under Professor Reid's guidance and management, also provides clinical and laboratory services as well as instruction for medical students and students of nursing – at undergraduate and postgraduate levels.

In addition to the TMRU's clinical service for children with severe undernutrition, and for volunteers of various clinical trials and experimental studies, care is also provided through the TMRU ambulatory clinic for 2 cohorts in ongoing research studies, the Vulnerable Windows Cohort (VWS) and the Jamaica Survivors of Malnutrition Cohort (JAMAKAS), as well as for subjects with various nutritional problems.

Professor Reid has authored and/or co-authored some 121 peer-reviewed journal articles and 1 book chapter as well as numerous other articles, abstracts, papers and technical reports; he has supervised students at the Master's and Doctoral levels and has also served as reviewer for a number of prestigious medical journals including the *West Indian Medical Journal*. He has received several awards for excellence in research including: the Mona Campus Principal's Award for research attracting the most research funds (2009); Award for best research publication, Faculty of Medical Sciences (2008, 2011, 2013 and 2014); and Award for best researcher, Faculty of Medical Sciences (2012 and 2014). In 2014, he received the Vice Chancellor's Award for Excellence in Research.

In addition to his considerable work in nutrition, Professor Reid is a key player in the activities of the Caribbean Network of Researchers on Sickle Cell Disease and Thalassemia as well as the Global Network on Sickle Cell Disease.

He has been a member of the Executive of the Medical Association of Jamaica (MAJ) since 2009 and served as Vice President and Chair of the Education Committee of the MAJ from 2013–2015. He functions in a

similar capacity with the Caribbean College of Family Physicians, and is the Chairman Elect of this body for 2017–2019.

His non-medical activities have included being Board Member at Ardenne High School and President of the Ardenne Alumni Association for 2007–2008. He is the current President of the Rotary Club of New Kingston.

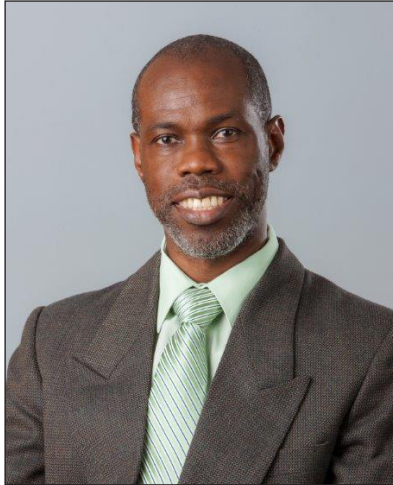
His talent and interest in sport continued into university, where he won gold medals in long jump in every Interhall and Intercollegiate competition for the period 1984–1988. He established Intercollegiate and Mutual Life Games records in 1984 and an Inter-University campus record in 1987. In that year, he was named Sportsman of the Year for The UWI, and he represented Jamaica in Track and Field at the World University Games in long jump. He also represented Jamaica in Volleyball 1985–1995 at the Pan-American Games and the Caribbean Volleyball Championships, and was a member of the 1992 silver medal team that competed at the Caribbean Championship.

Professor Reid has two children.

Elsa Leo-Rhynie

Chair, GraceKennedy Foundation

March 2016



Marvin Reid

THE LECTURE

CHAPTER 1

The Principles of Nutrition

About the Lecture

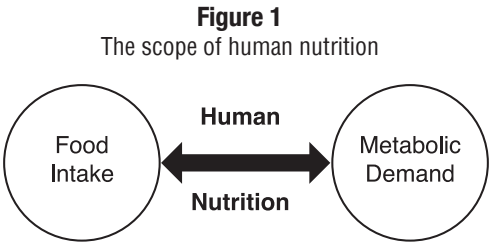
How do our dietary lifestyle choices affect our body functions? What are the medical consequences of the dietary choices we make? This lecture is intended to provide you with a framework for understanding how the body works when it is interacting with foods. I will share the state of our understanding of the impact of our dietary lifestyle choices on body function, as well as the medical consequences of those choices, by drawing on research findings that have been pioneered here in Jamaica. Since there are many different sources and types of foods as well as many different individual situations, I will not seek to offer a prescription for the many possible nutritional ills that can befall an individual or, for that matter, a population. I will, instead, focus on the extremes of nutritional maladaptation, obesity and undernutrition.

What Nutrition Is

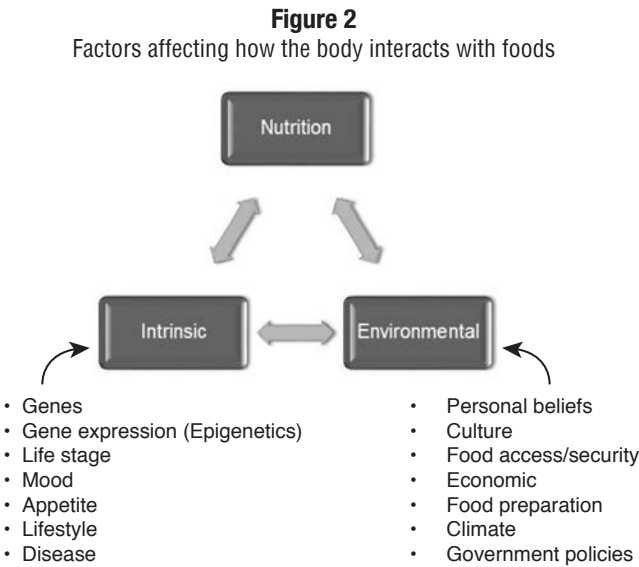
Nutrition is the branch of science that deals with what we eat and how our body utilizes what we eat to survive. As a science discipline, nutrition makes use of many different types of experiment or observations and measurements to assess the relationship of what we eat to how it is utilized in the body and the subsequent outcomes. In Jamaica, the Tropical Metabolism Research Unit (TMRU), a division of the Tropical Medicine Research Institute (TMRI) at The University of the West Indies, was established some 60 years ago to study the causes and consequences of poor nutrition. The findings from the work of this Unit have been used to better treat people with malnutrition both internationally and locally, as well as to inform national, regional and international nutritional policies. In this Lecture I will draw upon the lessons that we have learnt in understanding how poor nutrition affects body function and contributes to illness.

Every living person interacts with the science of nutrition every day and this is probably the reason for the many varied interpretations and applications of nutritional principles. In fact, I often think of our approach to nutrition in the same way that I think of our approach to religion, where many people hold on to beliefs and practices because of sheer faith and not

necessarily facts. In short, nutrition encompasses the interaction between food intake and the body’s metabolic demands (Figure 1), where the body’s metabolic demand is a measure of the body’s tissue activity at any instance in time.



The science and practice of nutrition, or the interaction between food intake and metabolic demand, is affected by many factors both at the individual level and at the environmental or societal level (see Figure 2). We will consider these factors sequentially.



What Affects Nutrition?

The Individual Level

At the individual level, genes are the full complement of genetic material that a person inherits from his or her parents. The genes are composed

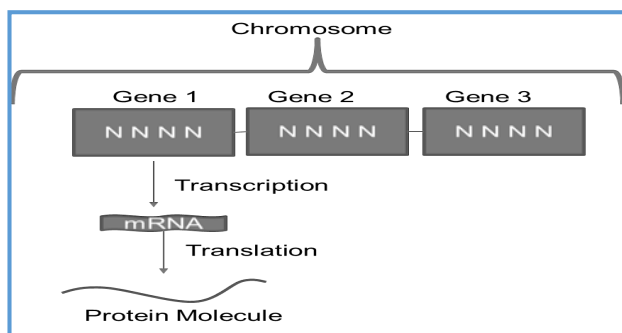
structurally of chemicals called nucleotides, arranged in specific sequences (Figure 3). The expression of genes determines the characteristics of a person. The processes involved in the expression of genes are complex and Box 1 illustrates some of the complexities. Of importance, though, is the observation that changes in gene expression can occur either by structural changes in the gene itself or through changes in the outcome of processes involved in gene expression, called epigenetics. Some epigenetic changes are influenced by the availability of specific nutrients.

BOX 1: More About Genes

Genes are organized to form chromosomes. When genes are expressed, the information contained in the sequence of nucleotides is first transcribed onto a molecule called Messenger Ribonucleic Acid (mRNA). The information contained in mRNA molecules is, in turn, translated into proteins. The outcome of genes being expressed is the phenotype. Not all genes are expressed at the same time nor do genes remain structurally inert during the life of a cell. The regulation or control of gene expression is complex but in general, genes have associated with them promotor and repressor regions. The promoter region is that section that initiates transcription of a particular gene while the repressor region is that section that prevents the transcription of a particular gene. Thus, there are several opportunities for gene expression in a cell to be altered without changing the sequence of nucleotides.

Figure 3

The functional organization of genetic material in a cell. Chromosomes are composed of genes. Genes in turn are made of nucleotides (N). The process of transferring information from genes to Messenger Ribonucleic Acid (mRNA) is Transcription. The process of transferring information from mRNA to protein is called Translation.



Alteration may take place by turning on or off promotor/repressor regions, or by the chemical modification of the nucleotides themselves, the mRNA or the protein product resulting from the translation of mRNA. These modifications are called epigenetic changes and represent the second individual factor of our discussion. Some of these epigenetic changes are inheritable.

It is believed that these epigenetic responses allow the body to adapt to environmental stimuli such as the availability of nutrients (1). For example, in The Gambia there are two seasons, a rainy season (August–September) and a dry season (March–May). The rainy season is characterized by the consumption of a surfeit of folate-containing foods when compared to the dry season. Folate foods are rich in methyl groups (a molecule composed of one carbon atom bonded to three hydrogen atoms and abbreviated -CH_3) and act as methyl donors in biochemical reactions in the body. Scientists found that Gambian children who were conceived during the rainy season had significantly greater methylation at specific gene sites than those conceived in the dry season (2). Methylation refers to the chemical modification of a compound whereby a methyl group is added to it. This important observation provides the mechanistic framework to explain the Development Origin of Chronic Disease hypothesis. In summary, nutrient availability during the early development and growth of an embryo or infant may result in epigenetic changes. These epigenetic changes may alter the organism's response to the environment, resulting in increased susceptibility to certain diseases.

The third individual factor under consideration is life stage. In human biology, we often speak of the life course, a sequence of biological growth phases that an individual undergoes from conception through to adulthood. These phases are all unique within individuals and impose specific demands on the body if they are to be successfully navigated without ill effect. In brief, we may describe these as an intra-uterine period (from conception to delivery); infancy (0–2 years) childhood (2 years to onset of puberty); adolescence (pubertal period); and adulthood which may be further subdivided into young, middle age and elderly. There is now a wide body of evidence that poor nutrition at any phase but especially at the intra-uterine and infancy phases, can have persistent effects on later phases. For example, scientists have reported that poor growth during the intra-uterine phase may predispose individuals to chronic diseases like hypertension and diabetes mellitus in adulthood (3, 4).

The fourth individual factor is mood. Many of us have the experience that when we are happy or sad our dietary consumption changes. Thus, it is not difficult for us to appreciate that one's mood can modulate one's intake of food. Indeed, there is experimental data to support the belief that there is a relationship between our mood and our food intake (5). For example, there is evidence from experimental studies that nerve signals from the stomach to the brain during food ingestion influence mood. Additionally, there is data from animal studies that suggests that stomach dysfunction may be a risk factor for mood disorders such as anxiety and depression. Further, there is a view that eating disorders such as anorexia nervosa and bulimia represent the extreme spectrum of the relationship between mood and food intake whereby people with these disorders use binge eating to cope with a preponderance of negative emotions (6).

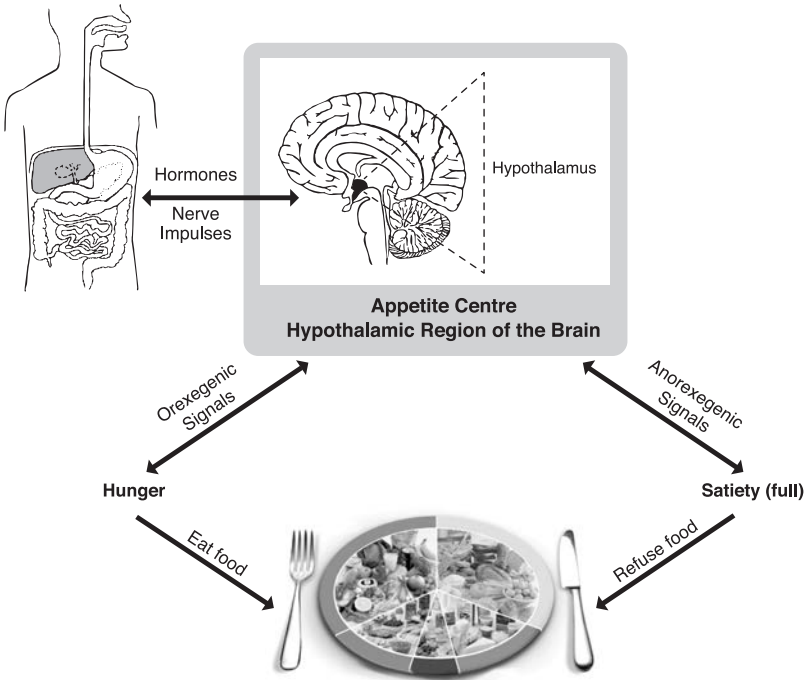
The fifth individual factor is appetite. Our desire to eat is what is referred to as our appetite. In humans there are many signals or cues to eat. From a physiological standpoint, appetite plays an important role in maintaining energy balance. In fact, the control of appetite is quite complex. Conceptually, we think of the regulation of appetite as occurring over two phases: a short-term phase and a long-term phase. Briefly, we believe that there are specific areas in the brain that are responsible for responding to and coordinating diverse signals including electrical impulses, chemical and hormonal signals from the gut and from other areas of the brain, to initiate and terminate feeding (Figure 4). Signals that stimulate appetite are described as orexigenic while those that inhibit appetite or induce satiety are described as anorexigenic. Dysfunction in the regulation of appetite has been implicated in many diseases. I have posited, for example, that a dysfunction in appetite plays an important role in explaining why many people with sickle cell disease are thin and have low weight for their age (7). We refer to this as "wasting".

The sixth individual factor is lifestyle. For the purpose of this lecture we are referring to the balance of activity between work and leisure as well as the consumption of socially accepted drugs such as alcohol and tobacco. The level of physical activity that an individual participates in will affect his or her health and nutritional status. There is energy expended to participate in physical activity. This energy expenditure may be broadly classified as Non-Exercise Activity Thermogenesis (NEAT) or Exercise Activity Thermogenesis (EAT) (8). So what kinds of activity result in

the generation of NEAT and EAT? In general, EAT refers to the energy expended in purposeful exercise activity such as when we go to the gym, play sport or train for a sporting discipline. NEAT, on the other hand, is expended on both voluntary activities such as ambulation and posture, or involuntary activity such as fidgeting. In other words, leisure time activity, sitting, standing, walking, gardening and household chores result in the generation of NEAT. Non-exercise activity thermogenesis as a proportion of an individual's total energy expenditure is about 6–10% in individuals with a mainly sedentary lifestyle but accounts for up to 50% of total energy expenditure or more in highly active subjects. In fact, the inter-individual variation in NEAT between individuals of similar age, gender and body mass can be as great as 2000 kilocalories (kc) per day (8). Thus, it is thought that differences in NEAT between individuals contribute to the development of obesity.

Figure 4

Control of appetite. The appetite centre in the hypothalamic region of the brain receives inputs or messages from the brain and the gut. The inputs are a combination of chemicals called hormones, e.g., leptin, or nerve impulses. Signals that cause the sensation of hunger are orexogenic in contrast to anorexigenic signals that inhibit food intake and cause the feeling of satiety.



The last individual factor that affects the relationship between dietary intake and metabolic demand is disease. There are many diseases that affect nutritional status and, correspondingly, inappropriate nutrition can cause disease or exacerbate existing disease. Some diseases, for example, sickle cell disease, and injuries such as burns, increase the metabolic rate and the demand for nutrients while inappropriate feeding in sick patients can cause death from the inability to properly process the food intake, a condition referred to as “refeeding syndrome”.

Environmental Factors

Personal beliefs are intricately linked to culture. Most times, eating takes place in the presence of other people and it is often perceived as an enjoyable part of our cultural experience. An important influence on personal belief is faith, which can affect feeding practices, food and lifestyle choices. Examples of how faith influences our eating practices may be seen in various religions, such as Hinduism and Islam and among Seventh Day Adventists.

Norms are our understanding of what is expected of us as acceptable behaviour. We notice what others do and base our behaviour on those observations. Norms are important determinants of behaviour because adherence or non-adherence to norms will lead to societal judgements. In general, people conduct themselves to obtain social approval and avoid disapproval and other negative social sanctions. Thus, norm adherence is more likely when the risk associated with non-adherence is high, when there is uncertainty about what constitutes correct behaviour, and when there is greater shared identity with the norm-referent group. Social norms, therefore, are the implicit codes of conduct that guide our actions. As they relate to food and eating, social norms modulate our food choices and the amount of food we consume. Eating norms are followed because they provide information about safe foods and facilitate food sharing. These norms may also affect food choice and intake by altering how we perceive ourselves and/or by altering the pleasure we obtain from seeing, smelling, touching and eating food (9).

Changes in the weather pattern of a country or region will affect food production and thus can influence the nutritional status of a population (10). For example, following natural disasters there may be food scarcity and, if prolonged, famine may result, leading to an increased prevalence

of malnutrition and undernutrition. Additionally, the yield of staple crops grown in a region and the survival of livestock may be affected by climate change.

A country's public health policy is a vehicle through which governments seek to modulate health risks to the population (11). Typically, policies are effected through a combination of laws, taxation and incentives. A topical public health policy controversy is the taxation of sugar-sweetened beverages (SSB) (12). Increased consumption of sugar-sweetened beverages has been linked to increases in obesity in both high-income and low- and middle-income countries. In New York City, they have reported a 35% decrease in the number of New York City adults consuming one or more sugary drinks a day and a 27% decrease in public high school students doing so from 2007 to 2013 because of the imposition of a sugar tax in 2006 (13). In the Caribbean, Barbados recently introduced a tax on SSB (14).

Metabolic Demand

An implicit assumption of human nutrition is that homeostasis or balance occurs whenever dietary intake matches metabolic demand. It is when there is no balance that disease may occur. But what constitutes this metabolic demand? Metabolic demand is the summation of metabolic activities of all tissues in a human body. For example, all cells in the body are continually building and breaking down molecules to supply their needs and dispose of waste. There is also the perpetual movement of molecules across cell borders as well the physical motion of cells. All these processes require energy. From an energetics point of view, we classify energy expenditure from metabolic activities in two broad categories: a variable component and a basal component. We can view this basal component as the minimum energy that the organism expends to live. We determine this basal component by measuring the amount of energy expended after resting for 12 hours in a thermoneutral (neither cold nor hot, typically 25°C) environment with the person fasting but having no mental or pathological stress. In the language of nutrition, we say that this is the basal metabolic rate. The basal metabolic rate can be measured using either direct calorimetry or indirect calorimetry. In direct calorimetry, the heat generated from metabolic activities is measured as change in body temperature. Indirect calorimetry is based on the principle that when tissue oxidizes food it utilizes oxygen and that

carbon dioxide and urea are produced. We therefore measure the oxygen consumed, the carbon dioxide produced and urea lost from the body and use these to calculate the metabolic rate.

The variable component of metabolic demand includes energy expended to process food intake (thermogenic effect of food), energy for maintaining body temperature, physical activity and growth. The latter two components, physical activity and growth, contribute most to the difference in the magnitude of the metabolic demand between individuals of similar age. A living organism will always have a metabolic demand. However, we eat intermittently. Hence, to satisfy the metabolic demand, the immediate source of nutrients will be our body stores. Thus, when a person eats he or she is, in essence, replenishing body stores that have been used. This principle, that in the normal state it is the metabolic demand that determines what humans consume – the demand-led principle – is a fundamental tenet of human nutrition. An implication of this concept is that if intake fails to satisfy the metabolic demand at any instant in time there will be a consequence whereby there will be an alteration in form (body composition) and function. When the functional and structural changes are within the physiological or normal limits of the organism we say that the organism has adapted to the mismatch. When the functional and structural changes are outside the normal limits, then disease supervenes.

In this chapter, I have introduced you to the basic principles of nutrition. I have shown that nutrition is more than the process of ingesting food. In much broader terms, nutrition encompasses the factors that may impact and modulate dietary intake both at the individual and the environmental level. I have also introduced the concept of metabolic demand, which is the summation of all the metabolic needs of the body at an instance in time, and I have suggested that it is metabolic demand that drives our need for food intake. Finally, I have restated a basic nutritional tenet which is that if metabolic demand is not instantaneously met there will be functional and structural consequences which, at the extremes, will lead to disease.

CHAPTER 2

Obesity and Chronic Non-Communicable Disease

What the Body Mass Index Tells Us

Epidemiology is that area of science that assesses or measures the occurrence, distribution and factors related to the causes of disease in a population. Good quality data on the chronic disease profile of Jamaica has been provided through the national surveys performed by the Epidemiological Research Unit of the Tropical Medicine Research Institute at The University of the West Indies. Their latest national survey was done between 2007 and 2008 and there are plans to perform another in 2016. Epidemiological studies in Jamaica have reported that non-communicable diseases, including cancers, are the major health problems in Jamaica. For example, diseases of the heart and blood vessels (cardiovascular diseases or CVD) such as strokes, heart attack, heart failure, atherosclerosis and hypertension are common, with about 36% of Jamaicans between 15 and 74 years having at least one cardiovascular disease. The prevalence of hypertension was approximately 25% in both men and women. The estimated prevalence for diabetes mellitus was 7.9% with a higher prevalence in women compared to men, 9.3% and 6.4%, respectively.

The level of obesity at the population level and somewhat at the individual level can be assessed by computing body mass index (BMI). This measure is computed by dividing a person's weight by the person's height in metres squared. In other words, the body mass index measurement is found by solving the formula:

$$BMI (kg/m^2) = \frac{Weight (kg)}{Height (m)^2}$$

The BMI is a practical measure of the amount of fat that an individual has because there is a reasonable correlation between BMI numbers and estimates of the body fatness assessed with direct measurements. In the Jamaican context, scientists from the Tropical Medicine Research Institute performed experiments to determine the relationship between BMI and the amount of fat in males and females. They did this experiment in volunteers from Nigeria, in African-Americans from the United States of America

(USA), and in people in Jamaica. They found that in these populations, BMI was strongly related to fatness with BMI accounting for about 61% percent to 85% of the variance in percentage of body fat (15). In other words, if the BMI of two Jamaicans differed by 1 unit then the average difference in amount of fat between them is about 0.61 units. Additionally, the amount of body fat in the volunteers varied among the countries with the Nigerians having the lowest, Jamaica being intermediate and the USA the highest. The amount of fat in the body also varied between the sexes at all three sites with women having more body fat at each level of the BMI. Thus, if a Jamaican male and female had the same BMI number, the woman had more fat tissue in her body. The World Health Organization (WHO) has recommended the following cut-points to classify nutritional status by BMI for adults greater than 18 years (see Table 1).

Figure 5
 Prevalence of malnutrition types in Jamaica
 (Jamaica Health & Lifestyle Survey 2008)

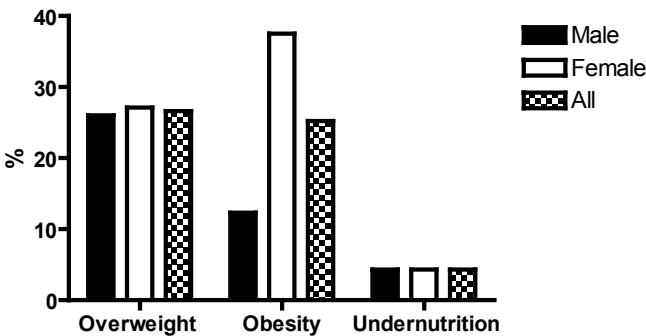


Table 1
 Nutritional status according to BMI values in adults

BMI	Nutritional Status
Below 18.5	Underweight
18.5–24.9	Normal or Healthy Weight
25.0–29.9	Overweight
30.0 and above	Obese

Using the BMI cut-points, the 2008 Jamaica Health and Lifestyle Survey (JHLS) reported that 25% of the Jamaican population was obese with a female to male ratio of 3:1 (16). Approximately 70% of women were classified as having central obesity by both waist circumference and waist-to-hip ratio criteria; 20% of men had elevated waist circumference and 9% had increased waist-to-hip ratio. Central or truncal obesity (commonly known as the “spare tyre” or belly fat) is significant as it is believed that the types of fat cells that are found in the central or truncal region are different structurally and functionally from fat found at other sites in the body (17). For example, it is believed that the excessive accumulation of fat in the truncal region (visceral adiposopathy) results in the excessive production of chemical substances, many of which are inflammatory and can affect blood vessel functions, leading to cardiovascular disease (17). In short, truncal obesity is an additional risk factor for cardiovascular disease. There were no significant differences in the prevalence of CVD risk factors studied for participants living in rural communities when compared to participants living in urban communities.

The Costs of Disease

Expenditure on diabetes and hypertension alone during the period 2000 to 2010 accounted for 5.9% of Jamaica’s GDP (18). The data reports on prevalence estimates but do these prevalence estimates translate into death? The Global Burden of Disease Study 2010 (GBD 2010) is a collaborative project of nearly 500 researchers in 50 countries led by the Institute for Health Metrics and Evaluation (IHME) at the University of Washington. This study sought to quantify levels and trends of health loss due to disease, injury, and risk factors. Their data indicates that the top six causes of premature death in Jamaica were the human immunodeficiency virus and acquired immunodeficiency syndrome (HIV and AIDS), stroke, interpersonal violence, diabetes, ischemic heart disease and hypertensive heart disease. In other words, cardiovascular disease accounted for four out of the top six for a cumulative percentage of 26.1%. As such, chronic cardiovascular non-communicable diseases exert a tremendous cost burden on the health system and on the Jamaican population. An interesting feature of these diseases, though, is that they are often preventable through modification of health risk behaviours. Health risk

behaviours are unhealthy behaviours that are amenable to change. They include a sedentary lifestyle, poor dietary practices, risky sexual behaviour, and alcohol and tobacco consumption. In fact, the Global Burden of Disease 2010 study ranks dietary risk as the leading factor contributing to Jamaica's disability-adjusted life years (DALY). DALY is a measure of disease burden determined by estimating the number of years lost due to ill-health, disability or early death.

The finding of a significant burden of non-communicable diseases and disproportionate burden of obesity in women in Jamaica is mirrored by data from other countries. Global survey data indicates that the prevalence of both male and female overweight and obesity varies by region and has rapidly increased between 1980 and 2008 (19). The high global prevalence of female overweight and obesity is correlated to a country's economic status as assessed by gross national product or gross domestic product. Improvements in economic growth in many developing countries have resulted in changes in habitual diets and lifestyle. These changes in dietary pattern have been termed the nutrition transition. As nutrition transitions emerge, diets tend to include greater amounts of fat, sugar and refined carbohydrates, and lifestyles become increasingly sedentary (20).

The data in the preceding paragraphs was focussed on adults but many Caribbean countries, including Jamaica, are also observing increased prevalence of obesity in children (21). For example, the prevalence of overweight children in the age range 0 to 5 years in Jamaica was estimated at 6% (21) in 1993.

From our exploration of nutritional principles in Chapter 1, you will recall that a person who is overweight or obese has a mismatch between intake and metabolic demand. In short, this individual has a chronic excess of intake of energy relative to the energy expended in daily life resulting in alteration in form; that is, a change in body composition due to an increase in the fat content of the body. There is also a change in body function such as low exercise capacity and physical disability (22) as well as disease outcomes such as premature death. We would therefore expect that in many westernized societies there would be a good correlation between energy intake and the prevalence of overweight and obesity (23). Indeed, this is the situation reported in many studies but it is not a universal finding and in studies that have failed to find an association between energy intake and levels of obesity, scientists have challenged the notion that body weight is

simply a function of “calories in” and “calories out” (24). As a consequence, there emerged the view that the source of the calories may be more important. The major sources of energy in diets are carbohydrates, fats and proteins. Collectively, these are called macronutrients. Cross-sectional and clinical trials studied suggest that increased carbohydrate intake and/or protein intake are associated with lower levels of obesity as measured by BMI, while an increased level of fat intake is associated with increased level of fatness (25–27).

Dietary Intake of Jamaicans

In Jamaica, Jackson and colleagues used a food frequency questionnaire to assess the diets of adult Jamaicans predominantly living in the Spanish Town area to determine if there were differences in the dietary consumption among those who were overweight and obese when compared to those who were not (28). This was part of a larger study comparing the diets of people of African ancestry living in Africa, the United Kingdom and the Caribbean. The researchers reported that there was no difference in the dietary intake of energy between subjects who were obese and those who were lean. Further, only protein consumption in females was related to being overweight, with women who consumed 87 grams or more of protein daily at increased risk of being overweight when compared to those who consumed 75 grams or less daily. To put it in perspective, a typical slice of hard-dough bread has 4 grams of protein, so the women who consumed about 3 slices of bread more per day were at increased risk of being overweight. In the Jamaica Health and Lifestyle Survey which sampled people across Jamaica, there was no difference in the pattern of food consumption in those individuals living in urban communities when compared to those living in rural communities (Figure 6). Of interest is the finding that more than 90% of Jamaicans eat less than the recommended three servings of either fruit or vegetables in a day.

Dr. Joanne Smith of the TMRI conducted a more extensive assessment of the macronutrient composition of people living in a rural setting in St. Mary as compared to those living in the urban setting of Hope Pastures and Mona Heights and found no difference in the energy and macronutrient consumption of the people sampled.

Figure 6
Fast food and sugar-sweetened beverage (SSB)
consumption in Jamaica (JHLS 2008)

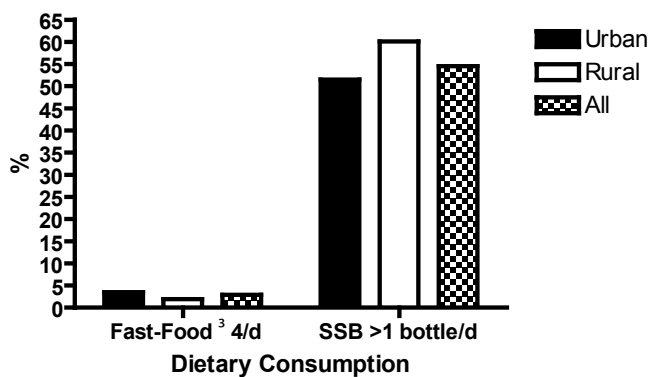


Figure 7
Energy Intake in urban and rural Jamaicans by sex
Joanne Smith (2014)

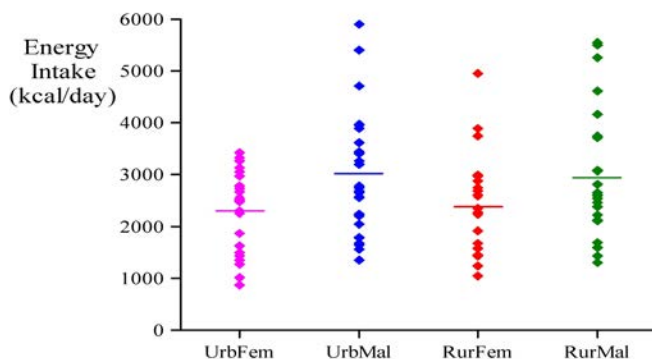
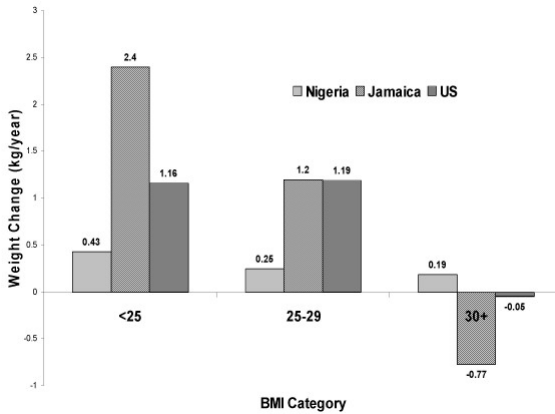


Figure 8
Weight change per year by BMI categories



Source: Durazio-Arvizu et al BMC Public Health. 2008;
8: 133. doi: 10.1186/1471-2458-8-133.

Durazo-Arvizu et al (29) analyzed weight data collected during the period 1995–1999 in adults over the age of 19 in Nigeria, Jamaica and the USA and reported that the rate of weight gain in Jamaicans was the highest in those who were lean at the initial assessment and averaged 2.4 kg/year. In subjects who had repeated weight measurement in Dr. Smith’s study, the average rate of weight gain was 0.12 kg/year and she found that energy-adjusted carbohydrate and protein intakes were not significant predictors of change in fat mass.

In the studies discussed so far the diets of Jamaicans have been assessed using tools called food frequency questionnaires or 24-hour recall. A food frequency questionnaire asks participants to report the frequency of consumption and approximate portion size of food and beverage items over a defined period of time. These questionnaires are frequently tested prior to being used in the studies to ensure that the foods that participants usually eat are represented. However, questionnaire-acquired data frequently suffers from mis-reporting, with those who are overweight and obese under-estimating their energy consumption. The best method of assessing energy intake is to measure energy expenditure over a 7-day period by using doubly labelled water studies. Water is composed of two types of atoms,

hydrogen and oxygen. In a double water label study, participants are given special water in which the hydrogen is substituted with its stable isotope version called deuterium and the oxygen atom of water is substituted by its stable isotope version called oxygen-18 (^{18}O) hence the term “double water”. Stable isotopes are non-radioactive atoms which have different masses compared to their natural variants. Upon drinking the “double” water, the “double” water is distributed throughout the body and is involved in body metabolism; it is excreted or lost from the body in breath and urine. Therefore, periodic collection of urine and breath samples are done over a 7-day period and measurements on these samples allow the energy expenditure to be determined. We can calculate the level of under-reporting by using the formula

level of underreporting%

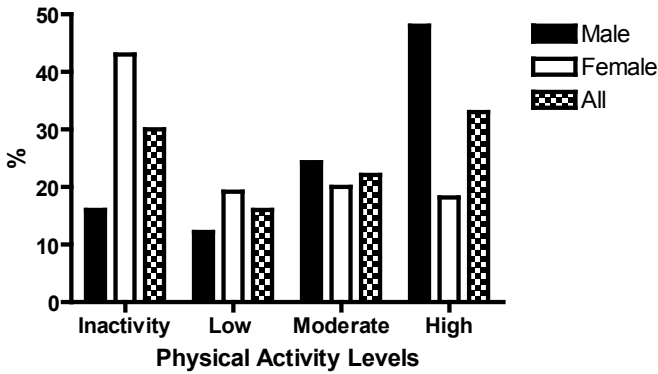
$$= \frac{\text{Calculated energy on questionnaire} - \text{true intake from double water labelled measurement}}{\text{true intake from double water labelled measurement}} \times 100$$

Using this technique it was found that the level of under-reporting of total energy intake was, on average, 17.9% (30). In other words, when scientists calculated the amount of energy reported on the questionnaire completed by participants and then measured what it should be using doubly labelled water, they found that Jamaicans under-reported their intake on the form as a proportion of their true intake by approximately 18%. Under-reporting was greatest in obese individuals, ranging from 44% in obese males to 25% in obese females.

The findings from nutritional research in Jamaica, such as those cited above, reveal to us that the dietary pattern is becoming increasingly westernized. This is so in both rural and urban settings. This has resulted in an increased rate of weight gain especially in those who are in the appropriate weight group. Despite these observations there does not appear to be any correlation between the total energy intake and macronutrient composition of the diet and obesity. However, this lack of a relationship may have arisen because of the tendency for Jamaicans, especially obese Jamaicans, to under-report their consumption patterns.

Physical Activity among Jamaicans

Figure 9
Physical activity levels in Jamaicans (JHLS 2008)



The other side of the energy balance equation is physical activity. The Jamaica Healthy Lifestyle Survey reported that about 30% of Jamaicans surveyed were classified as being physically inactive (Figure 9). This represented an increase of 76% over the proportion classified as physically inactive when the survey was performed in 2000. The assessment of physical activity was done using questionnaires. However, it is the view of some scientists that conclusions based on questionnaire data may be flawed (31). As discussed earlier, the gold-standard method to measure energy expenditure is using the doubly labelled water isotope technique. In a recent review of studies employing this technique, total energy expenditure adjusted for weight and age was not different between developing and industrialized countries (32). This is contrary to a commonly held perception that the reason for the lower rates of obesity in rural Africa, India and China, when compared to developed westernized countries is, in part, the more strenuous daily work routines in the lesser developed countries (32).

Dr. Cargill of the TMRI measured energy expenditure in a sample of urban participants and rural participants living in St. Mary using the doubly labelled water technique and found that after adjusting for differences in age, sex, weight and educational status, rural participants had higher total energy expenditure than urban participants. Despite this, there was no difference in the level of adiposity or fat between urban and rural dwellers, calling into question the role that energy expenditure plays in the obesity

epidemic. Indeed, if we recall, total energy expenditure is composed of non-exercise activity thermogenesis (NEAT) and exercise activity thermogenesis (EAT). At the individual level, it is supervised exercise that is efficacious in weight reduction and loss of body fat from the abdomen – the so-called visceral fat (33). Thus, the lack of difference in adiposity observed between rural and urban dwellers in Jamaica may be related to the distribution of energy expenditure between non-exercise activity thermogenesis and exercise activity thermogenesis. In short, the intensity of the exercise may be insufficient to induce differences in obesity.

Causes of Obesity

The epidemiological data begs the question, Why do people become overweight and obese on these nutritional transition diets; that is, diets which are low in fruit/vegetables, high in sugars and fats?

Many observers have noted a clustering of obesity in families. While it could be true that families share a common environment there have been studies that indicate that mutations of single genes, otherwise called monogenetic defects, are associated with extreme obesity. Mutations are alterations in genetic structure caused by errors in making copies of the gene during cell division or by environmental factors which directly damage the gene. It was not until the advent of new techniques such as genome wide association studies (GWAS) that there has been significant progress in understanding the genetic contribution to obesity. In GWAS studies many common genetic variants in different individuals are assessed to see if any variant is associated with a trait. These studies have found that variations in genes that regulate appetite or hormonal control of metabolism are associated with obesity. For example, the fat mass and obesity-associated (FTO) gene is connected to obesity by regulating appetite, food choice and hormone regulators such as growth hormone (34–36).

The Drifty Gene Theory seeks to integrate genetic, evolutionary and metabolic mechanisms to explain why people become obese (37). In this theory it is believed that the genes that control the appetite and regulate how much we eat have become less sensitive in each succeeding generation, probably through genetic mutations. This results in people consuming more food before they feel full or experience satiety.

BOX 2 : The Drifty Gene Theory

The starting point of the “drifty gene” theory is the observation that many animals store energy as fat. The stored fat acts as an energy back-up or reservoir for periods in which food supply and, therefore, energy supply, will be short. This storage of fat limits the mobility of the animal which increases the risk that the animal may be killed by predators. In order to improve the chances of survival it is in the best interest of the animal, from an evolutionary perspective, to have two regulatory set points: a high set point which prevents the animal from eating too much and storing too much fat thus becoming obese, and a lower regulatory set point which prevents the animal from eating too little, having little storage fat and then dying quickly if there is food shortage. In this theory, human obesity probably occurs because the evolutionary forces to maintain tight control of the upper set point have decreased over time because the risk of humans being killed by predators has been reduced. In other words, the control mechanisms to limit over-eating of food and subsequent fat storage have become impaired over time. How might this impairment of the upper set point have occurred? It is believed that permanent alterations (mutations) of the genes that control the upper set point occurred over the period of our existence, resulting in diminished functioning of this upper set point and this, in turn, predisposes affected individuals to become obese when there is a surfeit of energy supply. In other words, the ability to control one’s intake and not become engaged in binge eating or over-eating has decreased over time.

The Drifty Gene Theory is often combined with the Nutrient Specific Hunger Hypothesis where it is believed that we eat to satisfy a particular macronutrient and thus over-consume calories to meet that need. The nutrient with most compelling data is protein and this is encapsulated in the protein leverage hypothesis (38). The hypothesis suggests that people need to eat certain amounts of protein and this target is an intrinsic characteristic of an individual. When exposed to an environment in which the protein-energy ratios of foods are low, the individual will over-consume energy to satisfy the protein demands and thus become obese.

BOX 3 : John’s Protein Target

To illustrate this let’s take the case of John, a student at The University of the West Indies, Mona. We have measured John’s protein target and it is 100 grams per day. John will eat a variety of food such that at the end of the day,

the amount of protein consumed is 100 grams. If John is fed diet A which contains 10 grams of protein with a total energy content of 100 kcal, John will consume $100/10 \times 100 \text{ kcal} = 1000 \text{ kcal}$ per day to satisfy his protein target. If John is fed diet B which contains 20 grams of protein and total energy content of 100 kcal, then John will need to eat only $100/20 \times 100 = 500 \text{ kcal}$ per day to satisfy his protein target. We say that diet A has a lower protein-energy ratio than diet B. Therefore to satisfy his protein target, if John only had access to diet A, this would result in John consuming much more energy than if he consumed diet B and this extra energy would be stored as fat and lead to obesity. The typical westernized diet has lower protein-energy ratios and thus, over-consumption of these diets may lead to obesity.

So far, I have shown that Jamaica has a high level of cardiovascular diseases and obesity. I have also discussed the theories about why individuals become obese. Specifically, I have highlighted that while there may be a genetic component, there is likely to be important metabolic and behavioural factors that contribute to obesity. A better understanding of these factors is becoming increasingly relevant because at the population level, it has increasingly become apparent that differences in gross energy intake and physical activity pattern do not fully explain the pandemic of the obesity problem. Thus, understanding why people become obese is important as it helps health care and other professionals to design better solutions to the problem. Alongside the obesity problem, Jamaica continues to grapple with the problem of undernutrition. I will now discuss some of the issues related to undernutrition in Jamaica.

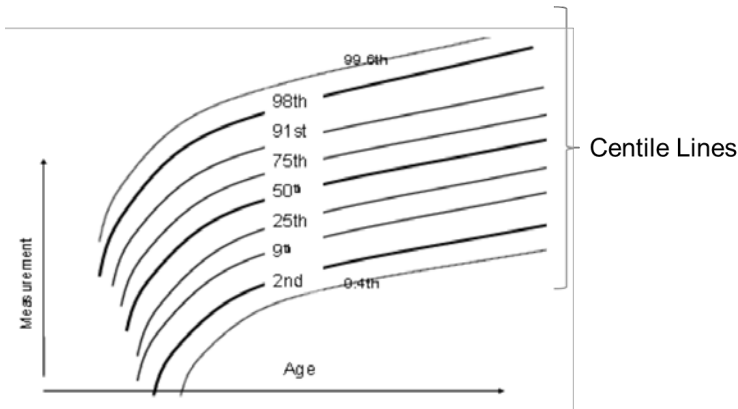
Undernutrition in Jamaica

Coexisting with obesity is undernutrition. Data from the Jamaica Healthy Lifestyle Survey indicates that approximately 4.3% of people between 15 years and 74 years were undernourished (Figure 5). This assessment was based on the number of people with BMI lower than 18.5 kg/m^2 . If we use the prevalence number estimated from the Survey (4.3%), based on population census figures from the Statistical Institute of Jamaica (STATIN), we could estimate that there are about 84,000 undernourished adults between 15 and 74 years in Jamaica. While BMI is a reasonable measure to assess undernutrition in adults it is more useful to supplement this measure with other measures to assess undernutrition in children. Similar to BMI, the measures used for children are based on measurement of the body contours or dimensions.

Measuring Undernutrition in Children

Figure 10

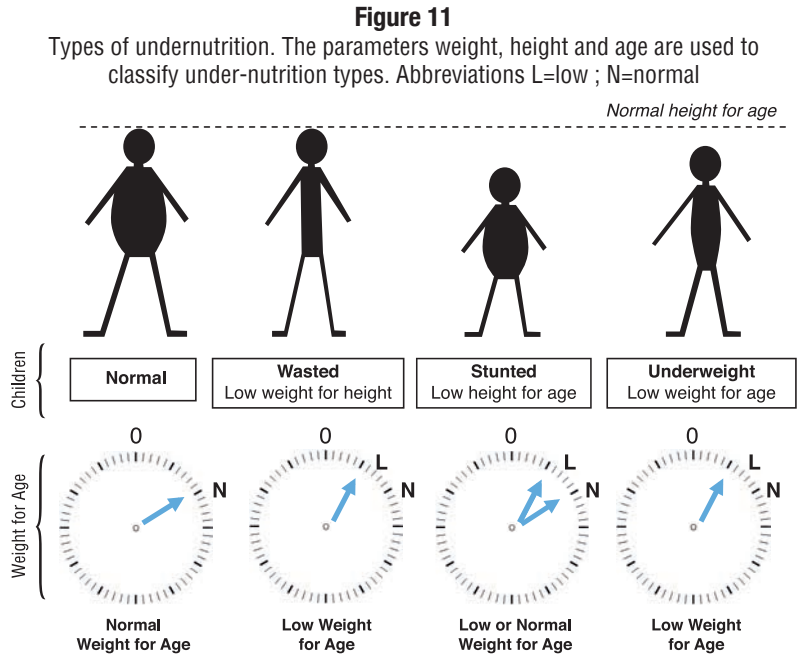
A typical growth chart. The 50th centile represents the average growth pattern of a normal child. Growth patterns at the 2nd centile line or below are classified as abnormal.



Typically, the body dimensions that we measure include the diameter of the head (head circumference), the upper arm diameter (mid upper arm circumference), height and weight. Collectively these measurements of body dimensions are called anthropometry. The head circumference gives an idea of whether the brain is growing normally while the weight, height and arm circumference represent markers of nutritional status. Children grow at varying rates at different ages and along different tracks, and this will produce varying anthropometric values at each age for each sex. In order to capture normal ranges for the anthropometric measurements, the WHO has created growth charts that reflect the normal range for these measurement at varying ages, specifically between 0–18 years, for each sex. The WHO growth charts are based on the growth of healthy breast-fed children in optimal conditions from six different countries. Growth data was collected from around 8,500 children who were exclusively breast-fed for the first 4 months of their lives, and were living in a well-supported health environment. The growth chart therefore tells us how big a child is expected to be at a particular age. We assess whether a specific measurement obtained on a child is normal by comparing it with the values on the WHO growth chart (39). Growth charts have lines drawn on them which are called centile lines. A centile line represents the proportion of the normal population of children that will have values at or below the line. The middle

line or median line represents the average growth pattern for a normal child. Fifty percent (50%) of children will grow at rates below this line and 50% will grow at rates above this line. Lines below the median line represent poorer growth compared to the median centile line. The WHO describes growth measurements that are below the 2nd centile as abnormal growth (39) (Figure 10).

Using weight, height and age of children, the WHO recognizes three broad categories of undernutrition. These are wasting, stunting and undernutrition. This is illustrated in Figure 11.



Stunting

Stunting has been defined as attained length or height at a particular age that is lower than expected. Technically, it is defined as more than 2 standard deviations (z score <-2) below the median value (50th centile line) on the growth reference curve. This corresponds to the 2nd centile line on the WHO growth chart. Worldwide, linear growth retardation or stunting occurs predominantly in the first 2–3 years of life. In Jamaica, the prevalence of stunting, in children under 5, has been estimated at 4.8 % (40) although there is wide geographic variability. In older children (11–12

year olds), the prevalence is about 3% (41). Additionally, we performed an anthropometric survey of school-aged children between 6 and 16 years old as part of a study assessing the cardiovascular risks of infants and adolescents in Jamaica, and in that study the prevalence of stunting was 2.1% (42). Within the Caribbean in general, stunting in children under 5 years has been estimated at 8.2 % and in developing countries, the pool estimate is 32% (43).

Stunting classically is thought to reflect long-term exposure to nutritional inadequacy (44) and is associated with increased rates of illness, death, reduced cognitive outcomes and school achievement, adult work capacity, and increased cardiovascular risk and disease in adulthood (43, 45–51). Thus, there is great interest in identifying interventions that reduce the burden of stunting on a country (52). There is a considerable amount of data from studies conducted in developing countries to address stunting. The data suggests that a package of interventions that includes the promotion of breast-feeding, education on complementary feeding (the introduction of foods other than breast milk or infant formula into the infant's diet), food and micronutrient supplementations is effective in reducing death associated with stunting as well as the frequency of stunting (52). For example, in Jamaica it has been reported that providing energy and protein supplementation in the form of a milk-based supplement between the ages of 9 months and 2 years resulted in an average gain of 1 cm more in height in the supplemented group when compared with the non-supplemented group after 2 years (50). In further follow-up on these study participants, those who were given psychosocial stimulation benefitted from improved cognition and exhibited less violent behaviour as adults (53). An interesting finding from further observations on this group of participants, who are now all adults, is that children born to a stunted parent had lower cognition compared to children born to a non-stunted parent (54). This suggests that the effects of stunting in childhood, especially on cognition, seem to be able to be transmitted to offspring; this further compels us to find interventions that can mitigate stunting.

The Effects of Breast-feeding

The pattern of weight gain and the rate of post-natal growth in the first year of life is influenced by breast milk and the frequency and timing of

complementary foods (55–58). For example, Dewey et al (57) reported that the average weight gain of infants being breast-fed for 12 months is lower than that of formula-fed infants, even after complementary foods were introduced. In contrast, Butte et al (56) reported that at one year old there was no significant difference in nutrient intake and growth pattern among infants who were breast-fed for 4 months and formula-fed infants. However, in that study, infants who were breast-fed had lower amounts of body fat at 3, 6 and 9 months of age.

The WHO has recommended exclusive breast-feeding up to six months of life to achieve optimal growth, development and health of infants (58). Infants who are exclusively breast-fed for six months experience less illness from pneumonia than those who are partially breast-fed or not breast-fed (43). Despite this recommendation, however, the prevalence of exclusive breast-feeding for the first 6 months of an infant's life in Jamaica and the Caribbean remains low (21). Prevalence data was obtained by questionnaire methods where mothers reported on their practices. This introduces considerable bias especially in an environment where mothers feel compelled not to disappoint their care providers. Consequently, we have used a new measurement technique to assess whether women are exclusively breastfeeding their babies. This method involves giving lactating mothers a special water called deuterium to drink. This water will mix with the mother's milk and the child will ingest this deuterium during breast-feeding. We then take salivary samples from the baby and the amount of deuterium present in the saliva is an indication of the amount of breast milk the baby has ingested. If the baby is being fed other liquids apart from breast milk, the concentration of deuterium in the saliva of the tested infant will be lower than expected. Using this technique, called the mother–child dose method, we have investigated the prevalence of exclusive breast-feeding of lactating mothers at 6 weeks after birth. We found that exclusive breast-feeding occurred in 37% of women. This proportion is much lower than that reported by data collected by the Ministry of Health. Their data, using self-reporting of mothers attending 6-weeks clinic, is that the exclusive breast-feeding rate is approximately 50% (Figure 12). In other words, half of the mothers attending the 6-weeks clinic reported that they were exclusively breast-feeding their baby.

Figure 12

Self-reported exclusive breast-feeding rates in Jamaican mothers attending 6-weeks clinic between 2001 and 2011. (59)

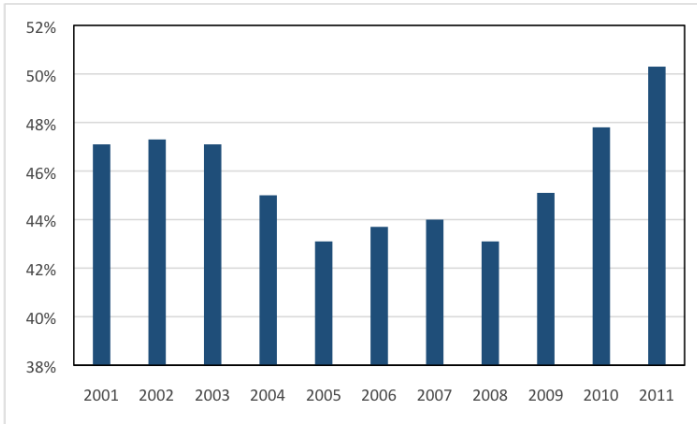
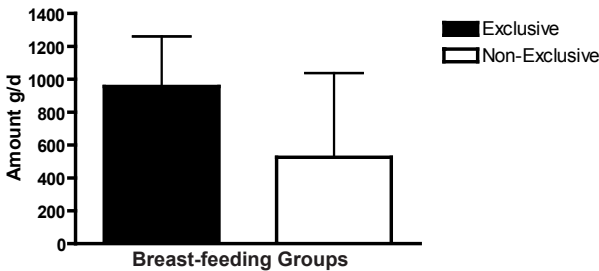


Figure 13

Breast milk intake in Jamaican infants

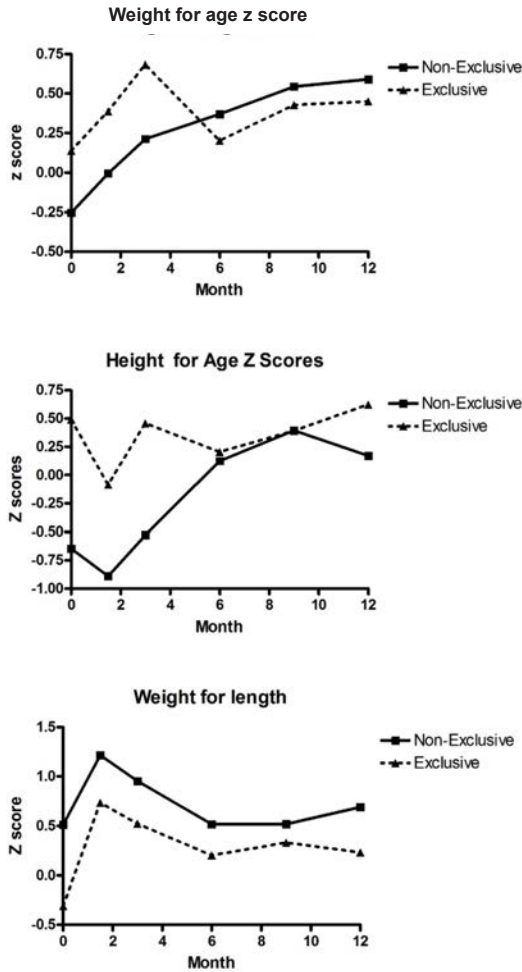


Although it is commonly agreed that breast milk is nutritionally uniquely suited to the human infant and contains hundreds of unique components and living cells to protect infants from infection and to aid development, the non-exclusively breast-fed infants received on average about 74% of the amount of breast milk compared to the exclusively breast-fed infants. In fact, the mean breast milk consumption in the non-exclusive breast-feeding group was mean±sd 704±349 g/d which was significantly less than the exclusive breast-fed group of mean±sd, 956±304 g/d (Figure 13).

Additionally, we found that the growth pattern of the exclusively breast-fed infants was different from non-exclusive breast-fed infants (Figure 14).

At 1 year, the exclusively breast-fed infants had less weight standardized for both their height and age, suggesting that they were leaner and will perhaps have less risk of cardiovascular illness as they age.

Figure 14
Growth pattern in the first year of life
according to breast-feeding mode



In addition to breast milk, an important nutrient required for growth and cognitive development in infants is iron. Iron concentration in human milk is low (0.2–0.4 mg/L) but the form it is in will allow good uptake in the breast-fed child. The good uptake, therefore, partly compensates for its low concentrations (60). This is the basis of the WHO recommendation of exclusive breast-feeding for 6 months to prevent the development of iron deficiency anemia in term, healthy infants. Notwithstanding, in Jamaica, iron deficiency is common with prevalence in children <5 years estimated to be at about 30% (61).

Wasting, Underweight and Other Forms of Undernutrition

The second broad category of undernutrition is wasting; that is, being thinner than expected for one's height. Wasting is more reflective of acute undernutrition. We use the weight for length growth chart to assess wasting and, technically, the WHO defines wasting as below minus two standard deviations from the median centile on the weight for height growth reference chart. The third broad category of undernutrition is being underweight. We define being underweight as having a lower weight than expected based on one's age. Thus, the reference growth chart used to assess underweight is the weight for age growth chart.

A special form of severe undernutrition is kwashiorkor (Figure 15). This syndrome, as a distinct entity, was recognized in 1908 in Latin America and was known as “multideficiency syndrome”; in Europe it was called “flour dystrophy” (62, 63). Later, it was described from Africa by Cicely Williams, a Jamaican doctor, in the English literature, and given the name kwashiorkor which has persisted to today (64). The Tropical Metabolism Research Unit uses the Wellcome classification to define kwashiorkor. This classification uses two principal criteria: the presence of oedema and being underweight. Oedema refers to swelling of the body tissue due to accumulation of water in the subcutaneous tissues. Typical body sites where one can find oedema are the feet, back and around the eye orbit. Specifically, the underweight criteria is set at a weight for age of between 60% to 80% of the weight for age growth reference (65). In addition to these criteria, children with kwashiorkor syndrome have many typical features such as an enlarged liver which is often filled with fat, peeling skin rashes, changes in the colour of the hair, and ulcers in the mouth. Such children are often difficult to treat and the death rate tends to be high (63).

In contrast to kwashiorkor, another severe form of undernutrition is marasmus. We define a child as having marasmus if he or she is less than 60% of weight for age reference and there is no oedema. The child displaying marasmus is, thus, very thin and markedly underweight (Figure 15).

Figure 15

Picture of marasmic child (left); hair change in kwashiorkor (centre);
oedema and skin changes in kwashiorkor (right)

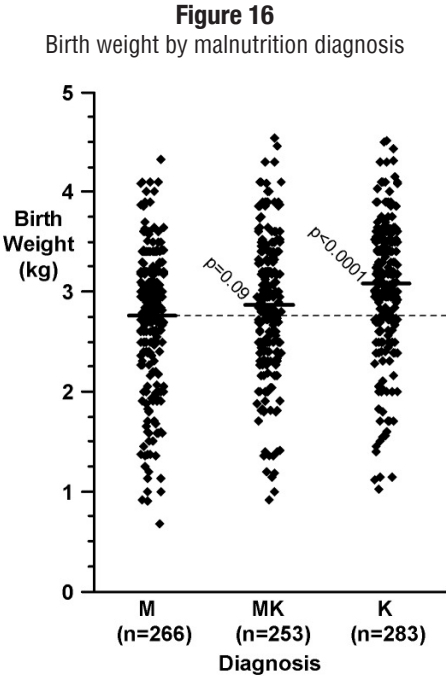


For years, many textbooks have attributed the cause of kwashiorkor to a protein deficiency and marasmus to both protein and energy deficiency. Collectively, these two disorders or syndromes are sometimes referred to as protein-energy malnutrition. However, we have studied these diseases on our Tropical Metabolism Research Unit ward for close to 60 years, even though our admission numbers have declined from 50 or more in the 1990s to about 20 infants with severe undernutrition annually. We can state that these diseases are not due to protein deficiency per se. Dietary assessment has failed to show consistent dietary differences between the children who develop marasmus when compared with those that suffer from kwashiorkor. Quite to the contrary, our data suggests that the problem is likely to be how these children metabolize food, in particular protein. Specifically, our data indicates that there is an inappropriate protein metabolic response to poor diet (66–69) as well as cell membrane damage from free radicals (70). A by-product of many reactions within cells is highly reactive molecules called free radicals. These molecules have the ability to react with many compounds in a process called oxidation. The generation of too many free radicals can harm the cell so, as a consequence,

cells have developed protective systems called antioxidants to neutralize the effects of free radicals. Additionally, some antioxidants are nutrients such as vitamins E and C. Within cells a major antioxidant is glutathione. What is the nature of inappropriate protein metabolic response? In brief, the body will need to mount an immune response to assist in recovering from illness. We found that children with kwashiorkor were not able to maintain their protein breakdown rates when they were ill. Consequently, the slower protein breakdown rate of children with kwashiorkor reduced the supply of most amino acids, resulting in decreased availability for the synthesis of plasma proteins involved in nutrient transport and the immune response (69). Another consistent finding in children with kwashiorkor was that they had low concentrations of glutathione in cells and when we replenished this with amino acid supplementation they lost oedema and recovered faster (70).

But why do some children develop kwashiorkor and some develop marasmus when exposed to inappropriate nutrition? We know that the pre-illness diet is not significantly different between those children that progress to kwashiorkor when compared with those who progress to marasmus. This raises the issue of whether there may be a genetic component. We have investigated this, looking at differences in the genes that code for enzymes that are involved in glutathione metabolism. Recall that we said that glutathione is the major antioxidant system found in cells and that children with kwashiorkor had low levels. We found that genetic variation within the Glutathione-S-Transferase gene may increase the risk that an infant exposed to inappropriate diet may develop kwashiorkor (71). This gene is an important gene that participates in regulating the amount of glutathione in cells. Another interesting thought related to the observed differences that we found in the metabolic profile of children who developed kwashiorkor as compared with marasmic children is that the metabolic differences might arise from exposures in the womb that induce changes in metabolic capacity, and which persisted after the birth of the infant. In this scenario, children who were exposed to adverse experiences within the womb would develop the capacity to strive on “less” and would have lower birth weight. On the other hand, infants exposed to an unrestricted intrauterine environment would grow well in the womb, manifested by normal birth weight but lack the ability when exposed to adverse postnatal experience to adapt

appropriately. If this thought is true, then we would expect children with kwashiorkor to have higher birth weights than children who developed marasmus. This is, indeed, what we found when we examined the birth weight of children who were admitted to the TMRU for treatment of kwashiorkor and marasmus (Figure 16) (72).



Source: Reproduced from Forrester et al (2012)
PLoS ONE 7(4): e35907. doi:10.1371/journal.pone.0035907

Undernutrition Resulting from Poor Quality Diet

We have looked at types of undernutrition especially in children and spent some time examining the consequences as well as the possible mechanisms that contribute to the development of one type of undernutrition as compared to another. We also made the observation that the pre-illness diets across the broad categories of undernutrition are probably not much different. While there are many factors contributing ultimately to what a child consumes, including parenting skills and practice, household composition (whether the father is present or not), socioeconomic status

of the household, sanitation, food availability and the child's activity and clinical state, there is general agreement that poor quality diets contribute to the undernutrition of children. For example, Green et al (73), using data from 124 countries, have found that an overall increase of 100 kcal/day/person was associated with a 0.84% reduction (95% CI -0.97% to -0.72%) in child stunting from 1985 to 2009 across countries. Further, greater intakes of energy from vegetable sources and milk were associated with 3.1% and 2.1% lower stunting rate respectively.

Most dietary surveys have identified snacking as a major contributor to poor quality diet. Typically, most snacks are high in sugars and salt but provide little critical nutrients such as vitamins, micronutrients or protein. In Jamaica, we are familiar with snacks such as cheese puffs, fried banana and plantain chips. It is not uncommon for snacks to provide more than a third ($> 1/3$) of total daily energy intake but less than 1/6 of essential nutrients (74) in children. Additionally, porridge is a common breakfast staple and weaning food for infants. However, unless it is prepared thickened and fed with a spoon it is unlikely to provide the energy that weaning infants need to grow satisfactorily (75).

This chapter reported data which indicated that Jamaica has the twin burden of obesity and undernutrition and the medical consequences from infancy through to adulthood. Additionally, we explored some of the possible mechanisms that give rise to these public health problems and highlighted the work that scientists in Jamaica, especially those at the Tropical Medicine Research Institute, are doing to gain greater understanding and, ultimately, to design better solutions to these problems.

CHAPTER 3

What Can We Do?

The Key Points – A Recap

We have spent some time discussing the fact that Jamaica is in the midst of a nutrition transition and that this has resulted in the twin threats of undernutrition and obesity. Key points and findings which you should bear in mind are that:

- A person who is undernourished or obese has a mismatch between his or her intake and metabolic demand.
- Overweight is common in Jamaican children with a prevalence of 6%; obesity has a prevalence in adults of 25%.
- Obesity is a major contributor to the high levels of diabetes and cardiovascular disease observed in Jamaica.
- Truncal obesity or belly fat is an additional risk factor for cardiovascular disease.
- Wasting, stunting, and other forms of undernutrition are common in both children and adults.
- There are no major differences in dietary pattern between people living in rural and urban communities and, in both communities, vegetable and fruit consumption are low.

Addressing the Problems

How can we reduce these problems? Clearly, the recommendations I make will need to operate at the policy level as well as at the individual level. However, the focus of the recommendations I make in this Lecture will be at the individual level and will be more of a general nature. This is because addressing specific nutritional needs will require an individualistic approach.

Understanding Food Groups

The first competency we need is to know foods. Nutritionists divide foods into groups or categories:

- Cereals or staples
- Legumes, nuts and oilseeds

- Vegetables
- Fruits
- Food from animals
- Fats, oils

From a nutrient perspective, these categories overlap but they make communication about diets easier. Let's briefly discuss the nutritional properties of each group.

Cereals and Staples

This group includes starchy fruits, roots, tubers and ground provisions. They are a source of energy and are rich in carbohydrates and fibre. They will also provide some protein and fat. A special property of carbohydrates is their glycaemic index (GI), which is the propensity of carbohydrate-enriched foods to increase blood glucose concentration relative to glucose. Many studies have shown that the regular consumption of diets containing high-GI foods is associated with an increased risk of Type 2 diabetes and cardiovascular disease. In contrast, the inclusion of low-GI foods in the diet, with no change in the total amount of carbohydrate consumed, may improve blood glucose control, reduce serum fats, prolong endurance during physical activity, promote longevity and improve vascular and insulin sensitivity (76–80). Among staples, dasheen and cassava are high GI foods while boiled green banana, white yam and Irish potato are classified as intermediate GI foods.

Legumes and Nuts

Legumes or peas are good sources of protein but do not contain all amino acids. They are also good sources of fibre and minerals.

Vegetables

This consists of dark-green leafy vegetables, yellow and other vegetables. Vegetables can be eaten either raw or cooked and play an important role in human nutrition, being mostly low in fat and carbohydrates but high in vitamins, minerals and fibre. The nutritional value of vegetables is affected by heat.

Fruits

Fruits are good sources of vitamins and minerals such as potassium. They are especially good source of vitamin C. For example:

- Citrus (50 mg)
- Mango (53 mg)
- Guava (218 mg)
- Cherries (1790 mg)

Fruits tend to be low in sodium, calcium, iron, zinc, protein and fat.

Food from Animals (Meat and Dairy)

This includes poultry, fish, milk, cheese, yoghurt and egg. As a category, animal meats are good sources of:

- Protein
- Vitamin B12
- Calcium
- Cholesterol

A source of confusion is the classification of meat as red or white. A practical definition is that meat from mammals is red while meat from poultry and fish is white. Using this definition, pork is red meat; it is primarily because of lobbying from industry sources that pork is often classified as white, especially in cooking circles. In an effort to clarify, the United States Department of Agriculture uses the myoglobin content – the major molecule responsible for the colour of red meat – to classify a meat as red or white.

Fats and Oils

Sources of fats and oils include meat fat, avocado (pear) and ackee. In general, fats can be saturated or unsaturated. Saturated fats are fat molecules that are composed of carbon and hydrogen atoms that are linked together by chemical bonds; they have no double bonds between carbon atoms. Saturated fats are typically solid at room temperature. Saturated fats are found in the fatty portions of meats and butter. Unsaturated fats have at least a single double bond in their structure (monosaturated) and are polyunsaturated if they have more than one double bond. Unsaturated fats are typically found in plant-based oils such as corn, palm, or olive oil, and fish such as mackerel.

How Much Is a Serving?

Now that we have some insights about foods we need to apply a tool to communicate amounts. Nutritionists often use the term serving. So how much is a serving? Well, what is described as a serving is dependent on the food group. A “serving” is the amount of food recommended by health professionals and is usually food-category specific. For example:

Table 2
Identifying Serving Size

Food category	Examples of One Serving
Staples	<ul style="list-style-type: none">• ½ cup sliced yam• 1 medium green banana• 1 slice bread• 1/2 cup cooked rice, pasta, or cereal
Vegetables	<ul style="list-style-type: none">• 1 cup raw leafy vegetables (about the size of a small fist)• 1/2 cup cut-up raw or cooked vegetables• 1/2 cup vegetable juice
Fruits	<ul style="list-style-type: none">• 1 medium fruit (about the size of a cricket ball)• 1/4 cup dried fruit• 1/2 cup fruit juice
Foods from Animals (Dairy Products)	<ul style="list-style-type: none">• 1/2 cup whole milk• 1 and 1/2 oz fat-free or low-fat cheese
Foods from Animals	<ul style="list-style-type: none">• 2–3 oz cooked lean meat, poultry or fish
Fats and Oils	<ul style="list-style-type: none">• 1 tsp soft margarine• 1 tsp vegetable oil
Legumes and Nuts	<ul style="list-style-type: none">• 1/3 cup or 1 and 1/2 oz nuts• 2 tbsp peanut butter
Sweets and Added Sugars	<ul style="list-style-type: none">• 1 tbsp sugar• 1 tbsp jelly or jam• 1 cup lemonade

Because of this confusion many professional nutritional groups are tending to use weight-based measurements.

Measuring Calories

The other skill that we need to master is to understand the concept of calories and be able to measure calories. All the macronutrients that we consume, whether carbohydrates, fats or protein, can be oxidized to produce energy. The unit of measurement of this energy is the calorie. If we use the metric

units then this would be the joule. The conversion between these two units is: 1 calorie = 4.186 joule. The amount of energy that a person needs will vary according to age, sex and activity level. A useful Jamaican resource that assists in estimating the amount of calories in foods is the *Caribbean Calorie Counter* by Patricia Thompson. Also, the National Heart, Lung, and Blood Institute of the National Institute of Health has published some tables of expected caloric needs at different ages, which I am reproducing here as a guide.

Table 3
Calories Needed each Day for Boys and Men

Age /years	Not Active	Somewhat active	Very Active
2–3	1000–1200	1000–1400	1000–1400
4–8	1200–1400	1400–1600	1600–2000
9–13	1600–2000	1800–2200	2000–2600
14–18	2000–2400	2400–2800	2800–3200
19–30	2400–2600	2600–2800	3000
31–50	2200–2400	2400–2600	2800–3000
>51 years	2000–2200	2200–2400	2400–2800

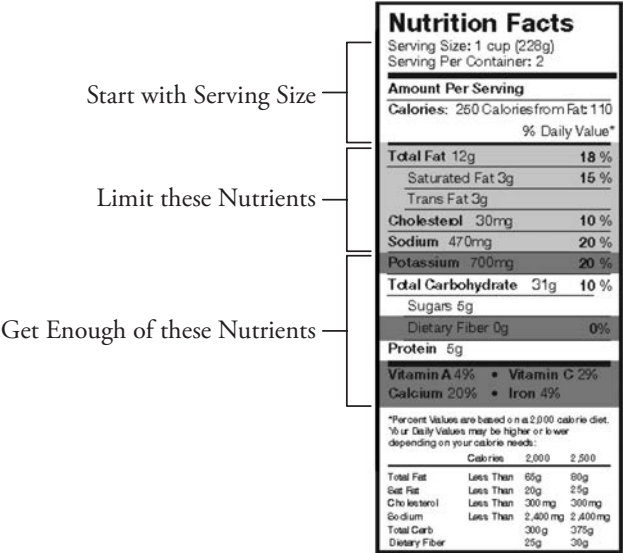
Table 4
Calories Needed each Day for Girls and Women

Age /years	Not Active	Somewhat active	Very Active
2–3	1000	1000–1200	1000–1400
4–8	1200–1400	1400–1600	1400–1800
9–13	1400–1600	1600–2000	1800–2200
14–18	1800	2000	2400
19–30	1800–2000	2000–2200	2400
31–50	1800	2000	2200
>51 years	1600	1800	2000–2200

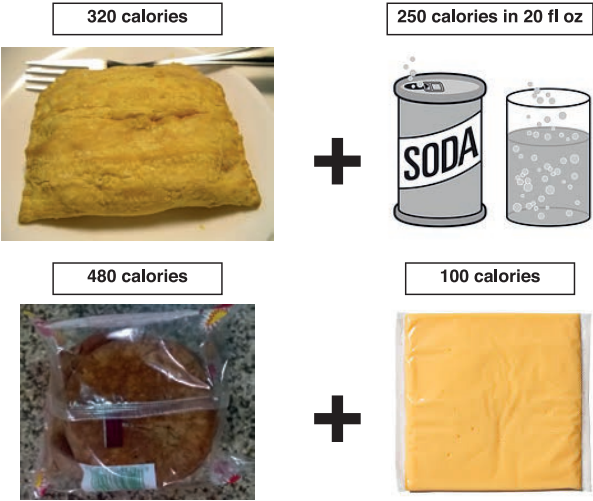
The final competency we need is to know how to read food labels (see Figure 17).

Reading Food Labels

Figure 17
A sample of a food label



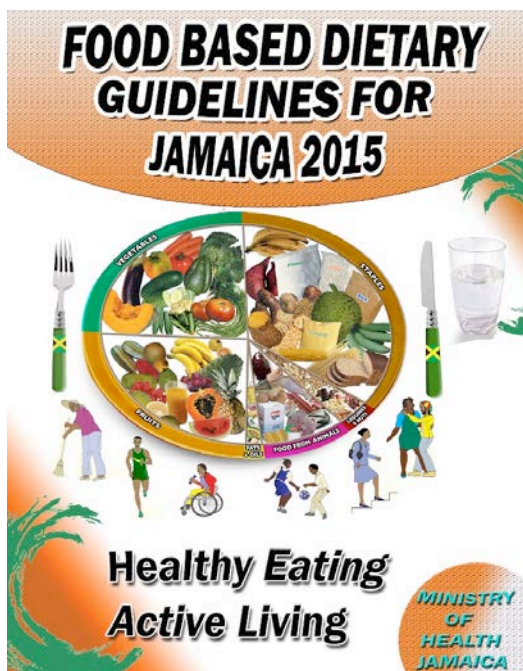
It is important to determine what constitutes a serving and how many servings are in a package.



In the example of the patty and soda, consuming all of it would mean taking in $228 \times 2 = 456$ calories since it has 2 servings per container. Using

data from Thompson's calorie counter book, a typical lunch-time snack for many Jamaicans, a beef patty, and a 20 fl oz can of soda, would provide $320 + 250 = 570$ calories. This represents almost 20% of the total daily needs of a young adult male. A bun and cheese sandwich is 580 calories.

The Ministry of Health has published a food-based guideline to assist Jamaicans in making healthier food choices. The food guide is a plate depicting the recommended proportions of six food groups for healthy eating. It seeks to promote variety, adequacy, balance and control. The size of each food group segment reflects the volume the group should contribute to the total daily diet. In addition, the food guide promotes the drinking of water and participation in physical activity. Items such as salty and sugary food and highly-processed products, the intake of which is recommended to be reduced, were not depicted in the food guide.



General Tips Through the Life Course

Infancy

The World Health Organization has recommended that children should exclusively be breast-fed for the first 6 months of life. This will result in

optimal growth of the infant. The period of weaning can be challenging and the exact timing and types of food are controversial. However, we do have evidence that energy-dense infant cereal which is frequently used at this stage is of importance (75). Additionally, high intake of cow's milk protein in formula-fed infants is associated with higher weight gain and increased adiposity, which have led to recommendations to limit protein intake in later infancy. However, recent data has suggested that giving the protein as meat-based puree may not be associated with a similar adverse risk as in milk feeds (71).

The Elderly

As we age there is gradual loss of muscle mass and strength. At its extreme, this contributes to the frailty observed in the elderly. This loss of muscle mass and strength is called sarcopenia. Sarcopenia occurs because ultimately, the rate of muscle breakdown exceeds muscle build up. Protein intake and physical activity are the main stimuli for muscle protein synthesis and hence building muscle. A dietary protein intake of 1.0–1.2 g/kg (body weight)/day divided between each daily meal of 25–30 grams of high quality protein per meal is recommended to prevent sarcopenia. Additionally, resistance training and perhaps the use of amino acid-based supplements like beta-hydroxy-beta-methylbutyrate (HBMB) may be helpful (81).

The Obese

There is no magic bullet to yield sustained weight loss in the obese individual who wants to lose weight. As a consequence, there are many dietary products, plans and programmes marketed for use. The overarching principle is that sustained weight loss requires an appropriate dose of physical activity combined with some dietary programme. In general, clinical trials have indicated that low-carbohydrate (<20% of total energy) interventions led to significantly greater weight loss than did low-fat interventions. For those who are morbidly obese (BMI > 35), especially if they have another cardiovascular disease, there is now the option to supplement dietary and exercise regimes with a surgical intervention.

In summary, in this chapter we sought to establish the key tools that are required to make healthy food choices: knowing your food groups, appreciating the concept of calories and the relevance of nutritional

labelling. We also discussed a few broad principles in the nutritional care of select vulnerable groups – infants, the elderly and the obese.

Our Food Choices Are Our Responsibility

In this Lecture, I have attempted to provide the framework for you to understand nutritional recommendations. This is relevant because the pattern of illnesses that we face in Jamaica is now, more than ever, being driven by what we consume and what we feed our children. Of special note is that it is clear from research performed at the TMRU and elsewhere that how a pregnant mother is fed and the feeding of her child in the first 2 years of life are critical factors that may determine the long-term response of the child to changes in his or her nutritional environment. As a developing country, we are faced with the twin burden of obesity and undernutrition, the direct consequences of being overfed and undernourished. Irrespective of this, the common thread through both problems is poor nutritional quality characterized by insufficient fruits and vegetables and high intake of refined carbohydrates and fat.

We have also explored our understanding of the metabolic changes which occur in obesity and undernutrition and highlighted the work that scientists at the TMRI have done to further our understanding of the problem so that better solutions can be created. As with any complex problem the solution will be multifactorial, requiring attention at the public policy level; public education, health promotion, legislation, social programmes, poverty alleviation, improved water and sanitation but more importantly, as individuals, we will have to take personal responsibility for our food choices. Within this context we should aim to:

1. Consume a balanced diet that provides your body with all its nutrient requirements, which will consist of a range of selections taken from all food groups. Typically this will consist of 5 or more servings of fruits and vegetables, 5 or more servings of starch, 1 or more serving from a protein source, and less than a serving of fats or oils daily. Where possible include peas, beans and nuts in your daily meals and reduce your intake of salty, processed foods, sugary foods and drinks.
2. Consume the right level of calories for you, depending on your age, physical activity level and whether you are afflicted with an illness.

3. Strive to make physical activity part of your daily routine. Intensity is also important and adults should try to include at least 150 minutes of moderate-intensity exercise per week. Exercise recommendations can be met through 30–60 minutes of moderate-intensity exercise such as brisk walking for five days per week or 20–60 minutes of vigorous-intensity exercise such as jogging for three days per week. As we get older, weight training should be included in our exercise programme.

References

1. Waterland RA, Michels KB. Epigenetic epidemiology of the developmental origins hypothesis. *Annu Rev Nutr.* 2007;27:363-88.
2. Waterland RA, Kellermayer R, Laritsky E, Rayco-Solon P, Harris RA, Travisano M, et al. Season of Conception in Rural Gambia Affects DNA Methylation at Putative Human Metastable Epialleles. *PLoS Genetics.* 2010;6(12).
3. Barker DJ, Martyn CN. The maternal and fetal origins of cardiovascular disease. *Journal of Epidemiology and Community Health.* 1992;46(1):8-11.
4. Lindblom R, Ververis K, Tortorella SM, Karagiannis TC. The early life origin theory in the development of cardiovascular disease and type 2 diabetes. *Mol Biol Rep.* 2015;42(4):791-7.
5. Holtmann G, Talley NJ. The stomach-brain axis. *Best Pract Res Clin Gastroenterol.* 2014;28(6):967-79.
6. Leehr EJ, Krohmer K, Schag K, Dresler T, Zipfel S, Giel KE. Emotion regulation model in binge eating disorder and obesity--a systematic review. *Neurosci Biobehav Rev.* 2015;49:125-34.
7. Reid M. Nutrition and sickle cell disease. *Comptes rendus biologies.* 2013;336(3):159-63.
8. Levine JA. Nonexercise activity thermogenesis--liberating the life-force. *J Intern Med.* 2007;262(3):273-87.
9. Higgs S. Social norms and their influence on eating behaviours. *Appetite.* 2015;86:38-44.
10. Machalaba C, Romanelli C, Stoett P, Baum SE, Bouley TA, Daszak P, et al. Climate Change and Health: Transcending Silos to Find Solutions. *Ann Glob Health.* 2015;81(3):445-58.
11. Samuels TA, Guell C, Legetic B, Unwin N. Policy initiatives, culture and the prevention and control of chronic non-communicable diseases (NCDs) in the Caribbean. *Ethnicity & health.* 2012;17(6):631-49.
12. Block JP, Willett WC. Taxing sugar-sweetened beverages: not a "holy grail" but a cup at least half comment on "food taxes: a new holy grail?". *International journal of health policy and management.* 2013;1(2):183-5.
13. Kansagra SM, Kennelly MO, Nonas CA, Curtis CJ, Van Wye G, Goodman A, et al. Reducing sugary drink consumption: New York City's approach. *American journal of public health.* 2015;105(4):e61-4.

14. Sandy D. Tax on sweet drinks [WWW]. Barbados Today; 2015 [cited 2016 2 January]. Available from: <http://www.babadosoday.bb/2015/06/15/tax-on-sweet-drinks/>.
15. Luke A, Durazo-Arvizu R, Rotimi C, Prewitt TE, Forrester T, Wilks R, et al. Relation between body mass index and body fat in black population samples from Nigeria, Jamaica, and the United States. *Am J Epidemiol*. 1997;145(7):620-8.
16. Ferguson TS, Francis DK, Tulloch-Reid MK, Younger NO, McFarlane SR, Wilks RJ. An update on the burden of cardiovascular disease risk factors in Jamaica: findings from the Jamaica Health and Lifestyle Survey 2007-2008. *The West Indian medical journal*. 2011;60(4):422-8.
17. Farb MG, Gokce N. Visceral adiposopathy: a vascular perspective. *Horm Mol Biol Clin Investig*. 2015;21(2):125-36.
18. Metivier C, Gittens-Baynes K. Confronting Chronic Non-Communicable Diseases: Response Programmes of Selected Caribbean Countries. *Caribbean Journal of Public Sector Management* 2014;XI(1):1-11.
19. Kanter R, Caballero B. Global gender disparities in obesity: a review. *Advances in nutrition (Bethesda, Md)*. 2012;3(4):491-8.
20. Popkin BM. Global nutrition dynamics: the world is shifting rapidly toward a diet linked with noncommunicable diseases. *The American journal of clinical nutrition*. 2006;84(2):289-98.
21. Gaskin PS, Nielsen AL, Willie D, Durant TC. Early childhood nutritional status in CARICOM countries: an overview with respect to five nutrition related millennium development goals. *J Environ Public Health*. 2014;2014:580928.
22. Miller CT, Fraser SF, Levinger I, Straznicky NE, Dixon JB, Reynolds J, et al. The effects of exercise training in addition to energy restriction on functional capacities and body composition in obese adults during weight loss: a systematic review. *PLoS One*. 2013;8(11):e81692.
23. Brown RE, Sharma AM, Ardern CI, Mirdamadi P, Mirdamadi P, Kuk JL. Secular differences in the association between caloric intake, macronutrient intake, and physical activity with obesity. *Obesity research & clinical practice*. 2015.
24. McAllister EJ, Dhurandhar NV, Keith SW, Aronne LJ, Barger J, Baskin M, et al. Ten putative contributors to the obesity epidemic. *Critical reviews in food science and nutrition*. 2009;49(10):868-913.

25. Austin GL, Ogden LG, Hill JO. Trends in carbohydrate, fat, and protein intakes and association with energy intake in normal-weight, overweight, and obese individuals: 1971-2006. *The American journal of clinical nutrition*. 2011;93(4):836-43.
26. Hooper L, Abdelhamid A, Moore HJ, Douthwaite W, Skeaff CM, Summerbell CD. Effect of reducing total fat intake on body weight: systematic review and meta-analysis of randomised controlled trials and cohort studies. *BMJ (Clinical research ed)*. 2012;345:e7666.
27. Naude CE, Schoonees A, Senekal M, Young T, Garner P, Volmink J. Low carbohydrate versus isoenergetic balanced diets for reducing weight and cardiovascular risk: a systematic review and meta-analysis. *PLoS One*. 2014;9(7):e100652.
28. Jackson M, Walker S, Cruickshank JK, Sharma S, Cade J, Mbanya JC, et al. Diet and overweight and obesity in populations of African origin: Cameroon, Jamaica and the UK. *Public health nutrition*. 2007;10(2):122-30.
29. Durazo-Arvizu RA, Luke A, Cooper RS, Cao G, Dugas L, Adeyemo A, et al. Rapid increases in obesity in Jamaica, compared to Nigeria and the United States. *BMC Public Health*. 2008;8:133.
30. Orcholski L, Luke A, Plange-Rhule J, Bovet P, Forrester TE, Lambert EV, et al. Under-reporting of dietary energy intake in five populations of the African diaspora. *Br J Nutr*. 2015;113(3):464-72.
31. Dhurandhar NV, Schoeller D, Brown AW, Heymsfield SB, Thomas D, Sorensen TI, et al. Energy balance measurement: when something is not better than nothing. *Int J Obes (Lond)*. 2015;39(7):1109-13.
32. Dugas LR, Harders R, Merrill S, Ebersole K, Shoham DA, Rush EC, et al. Energy expenditure in adults living in developing compared with industrialized countries: a meta-analysis of doubly labeled water studies. *The American journal of clinical nutrition*. 2011;93(2):427-41.
33. Fisher G, Hunter GR, Allison DB. Commentary: physical activity does influence obesity risk when it actually occurs in sufficient amount. *Int J Epidemiol*. 2013;42(6):1845-8.
34. Fall T, Ingelsson E. Genome-wide association studies of obesity and metabolic syndrome. *Mol Cell Endocrinol*. 2014;382(1):740-57.

35. Kuhn AB, Feis DL, Schilbach L, Kracht L, Hess ME, Mauer J, et al. FTO gene variant modulates the neural correlates of visual food perception. *Neuroimage*. 2016;128:21-31.
36. Lukasova P, Vankova M, Vcelak J, Vejrazkova D, Bradnova O, Stanicka S, et al. Fat mass and obesity associated gene variants are associated with increased growth hormone levels and affect glucose and lipid metabolism in lean women. *Physiol Res*. 2015;64 Suppl 2:S177-85.
37. Speakman JR. Evolutionary perspectives on the obesity epidemic: adaptive, maladaptive, and neutral viewpoints. *Annu Rev Nutr*. 2013;33:289-317.
38. Sorensen A, Mayntz D, Raubenheimer D, Simpson SJ. Protein-leverage in mice: the geometry of macronutrient balancing and consequences for fat deposition. *Obesity (Silver Spring)*. 2008;16(3):566-71.
39. World Health Organization. WHO Child Growth Standards: Length/Height-for-Age, Weight-for-Age, Weight-for-Length, Weight-for-Height and Body Mass Index-for-Age: Methods and Development. . Geneva, Switzerland: 2006.
40. The Planning Institute of Jamaica, The Statistical Institute of Jamaica. Jamaica Survey of Living Conditions. Kingston: Stephenson's Litho Press; 2002.
41. Jackson M, Samms-Vaughan M, Ashley D. Nutritional status of 11-12-year-old Jamaican children: coexistence of under- and overnutrition in early adolescence. *Public health nutrition*. 2002;5(2):281-8.
42. Wilks RJ, McFarlane-Anderson N, Bennett FI, Reid M, Forrester TE. Blood pressure in Jamaican children: relationship to body size and composition. *The West Indian medical journal*. 1999;48(2):61-8.
43. Black RE, Allen LH, Bhutta ZA, Caulfield LE, de Onis M, Ezzati M, et al. Maternal and child undernutrition: global and regional exposures and health consequences. *Lancet*. 2008;371(9608):243-60.
44. Duggan M, Golden B. Deficiency diseases. In: Geissler C, Powers H, editors. *Human Nutrition*. 11th ed. Edinburgh: Elsevier Churchill Livingstone; 2005. p. 517-35.
45. Drewett RF, Corbett SS, Wright CM. Cognitive and educational attainments at school age of children who failed to thrive in infancy: a population-based study. *J Child Psychol Psychiatry*. 1999;40(4):551-61.

46. Grantham-McGregor SM, Walker SP, Himes JH, Powell CA. The effect of nutritional supplementation and stunting on morbidity in young children: the Jamaican study. *Trans R Soc Trop Med Hyg.* 1993;87(1):109-13.
47. Sawaya AL, Sesso R, Florencio TM, Fernandes MT, Martins PA. Association between chronic undernutrition and hypertension. *Matern Child Nutr.* 2005;1(3):155-63.
48. Stein AD, Wang M, Ramirez-Zea M, Flores R, Grajeda R, Melgar P, et al. Exposure to a nutrition supplementation intervention in early childhood and risk factors for cardiovascular disease in adulthood: evidence from Guatemala. *Am J Epidemiol.* 2006;164(12):1160-70.
49. Walker SP, Gaskin P, Powell CA, Bennett FI, Forrester TE, Grantham-McGregor S. The effects of birth weight and postnatal linear growth retardation on blood pressure at age 11-12 years. *J Epidemiol Community Health.* 2001;55(6):394-8.
50. Walker SP, Grantham-McGregor SM, Himes JH, Powell CA, Chang SM. Early childhood supplementation does not benefit the long-term growth of stunted children in Jamaica. *The Journal of nutrition.* 1996;126(12):3017-24.
51. Walker SP, Wachs TD, Gardner JM, Lozoff B, Wasserman GA, Pollitt E, et al. Child development: risk factors for adverse outcomes in developing countries. *Lancet.* 2007;369(9556):145-57.
52. Bhutta ZA, Ahmed T, Black RE, Cousens S, Dewey K, Giugliani E, et al. What works? Interventions for maternal and child undernutrition and survival. *Lancet.* 2008;371(9610):417-40.
53. Walker SP, Chang SM, Vera-Hernandez M, Grantham-McGregor S. Early childhood stimulation benefits adult competence and reduces violent behavior. *Pediatrics.* 2011;127(5):849-57.
54. Walker SP, Chang SM, Wright A, Osmond C, Grantham-McGregor SM. Early childhood stunting is associated with lower developmental levels in the subsequent generation of children. *The Journal of nutrition.* 2015;145(4):823-8.
55. Butte NF, Garza C, Smith EO, Nichols BL. Human milk intake and growth in exclusively breast-fed infants. *J Pediatr.* 1984;104(2):187-95.
56. Butte NF, Wong WW, Hopkinson JM, Smith EO, Ellis KJ. Infant feeding mode affects early growth and body composition. *Pediatrics.* 2000;106(6):1355-66.

57. Dewey KG. Growth characteristics of breast-fed compared to formula-fed infants. *Biol Neonate*. 1998;74(2):94-105.
58. World Health Organization. Report of the expert consultation on the optimal duration of exclusive breastfeeding. Report of An Expert Consultation; 28-30 March; Geneva, Switzerland: Department of Nutrition for Health and Development. Department of Child and Adolescent Health and Development, World Health Organization; 2001.
59. Ministry of Health Jamaica. National Infant and Young Child Feeding Policy. Kingston Jamaica: 2014 June.
60. Domellof M, Lonnerdal B, Abrams SA, Hernell O. Iron absorption in breast-fed infants: effects of age, iron status, iron supplements, and complementary foods. *The American journal of clinical nutrition*. 2002;76(1):198-204.
61. Caribbean Food and Nutrition Institute (CFNI). The Pan American Health Organization (PAHO): Micronutrient study report - Jamaica. 1998.
62. Golden MH. Evolution of nutritional management of acute malnutrition. *Indian Pediatr*. 2010;47(8):667-78.
63. Waterlow JC. Protein-energy malnutrition: the nature and extent of the problem. *Clin Nutr*. 1997;16 Suppl 1:3-9.
64. Williams C. Kwashiorkor: a nutritional disease of children associated with a maize diet. *Lancet*. 1935(Nov. 16):1151-2.
65. Wellcome Trust Working Party. Classification of infantile malnutrition. *Lancet*. 1970;2(7667):302-3.
66. Badaloo AV, Forrester T, Reid M, Jahoor F. Lipid kinetic differences between children with kwashiorkor and those with marasmus. *The American journal of clinical nutrition*. 2006;83(6):1283-8.
67. Green CO, Badaloo AV, Hsu JW, Taylor-Bryan C, Reid M, Forrester T, et al. Effects of randomized supplementation of methionine or alanine on cysteine and glutathione production during the early phase of treatment of children with edematous malnutrition. *The American journal of clinical nutrition*. 2014;99(5):1052-8.
68. Hsu JW, Badaloo A, Wilson L, Taylor-Bryan C, Chambers B, Reid M, et al. Dietary supplementation with aromatic amino acids increases protein synthesis in children with severe acute malnutrition. *The Journal of nutrition*. 2014;144(5):660-6.

69. Jahoor F, Badaloo A, Reid M, Forrester T. Protein metabolism in severe childhood malnutrition. *Ann Trop Paediatr*. 2008;28(2):87-101.
70. Badaloo A, Reid M, Forrester T, Heird WC, Jahoor F. Cysteine supplementation improves the erythrocyte glutathione synthesis rate in children with severe edematous malnutrition. *The American journal of clinical nutrition*. 2002;76(3):646-52.
71. Marshall KG, Howell S, Reid M, Badaloo A, Farrall M, Forrester T, et al. Glutathione S-transferase polymorphisms may be associated with risk of oedematous severe childhood malnutrition. *Br J Nutr*. 2006;96(2):243-8.
72. Forrester TE, Badaloo AV, Boyne MS, Osmond C, Thompson D, Green C, et al. Prenatal factors contribute to the emergence of kwashiorkor or marasmus in severe undernutrition: evidence for the predictive adaptation model. *PLoS One*. 2012;7(4):e35907.
73. Green R, Sutherland J, Dangour AD, Shankar B, Webb P. Global dietary quality, undernutrition and non-communicable disease: a longitudinal modelling study. *BMJ Open*. 2016;6(1):e009331.
74. Hess J, Slavin J. Snacking for a cause: nutritional insufficiencies and excesses of U.S. children, a critical review of food consumption patterns and macronutrient and micronutrient intake of U.S. children. *Nutrients*. 2014;6(11):4750-9.
75. Stephenson DM, Gardner JM, Walker SP, Ashworth A. Weaning-food viscosity and energy density: their effects on ad libitum consumption and energy intakes in Jamaican children. *The American journal of clinical nutrition*. 1994;60(4):465-9.
76. Jovanovski E, Zurbau A, Vuksan V. Carbohydrates and endothelial function: is a low-carbohydrate diet or a low-glycemic index diet favourable for vascular health? *Clin Nutr Res*. 2015;4(2):69-75.
77. McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, Jacques PF. Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. *Diabetes care*. 2004;27(2):538-46.
78. Ramdath DD, Isaacs RL, Teelucksingh S, Wolever TM. Glycaemic index of selected staples commonly eaten in the Caribbean and the effects of boiling v. crushing. *Br J Nutr*. 2004;91(6):971-7.
79. Rizkalla SW. Glycemic index: is it a predictor of metabolic and vascular disorders? *Curr Opin Clin Nutr Metab Care*. 2014;17(4):373-8.

80. Wolever TM, Brand-Miller JC, Abernethy J, Astrup A, Atkinson F, Axelsen M, et al. Measuring the glycemic index of foods: interlaboratory study. *The American journal of clinical nutrition*. 2008;87(1):247S-57S.
81. Yanai H. Nutrition for Sarcopenia. *J Clin Med Res*. 2015;7(12):926-31.