Epidemiologic Transitions: Migration and Development of Obesity and Cardiometabolic Disease in the Developing World

Terrence Forrester
Tropical Metabolism Research Unit, Tropical Medicine Research Institute, The University of the West Indies, Kingston, Jamaica

Abstract
For centuries, the challenge has been the maintenance of bodyweight in the face of marginal food availability. Since the industrial revolution, energy expenditure related to economic activity and domestic life has fallen progressively as technological innovation has replaced muscular power with labor-saving devices. This fall in activity energy expenditure however has not been associated over this entire period with population weight gain. In the 1970s and the 1980s, there was an abrupt uptick in the rate of rise of relative weight in industrialized countries followed rapidly by developing countries. This has led to high and increasing rates of overweight and obesity in high-income countries worldwide, but also an alarming inclusion of low- and middle-income populations in this obesity epidemic. The precise drivers of these concurrent epidemics are not agreed, but probably include on the one hand an increase in dietary energy intake resulting from the impact of industrialization and globalization on food availability and price. On the other, there is the facilitating underlying status of a steadily falling activity energy expenditure as muscle power as an input into economic production as well as household and leisure activities has been supplanted. The rise in population weight without accompanying linear growth manifests as obesity. The accretion of fat as well as the response to other environmental exposures during progressive industrialization and modernization has evoked an accompanying epidemic of cardiometabolic pathology that has significant impact on health as well as macroeconomics. Given the power and presumed irreversibility of industrialization and globalization, our ability to reverse these obesity epidemics is heavily dependent on new knowledge being developed which gives insight with prevention and therapeutic implications on the proximal and distal drivers of this progressive positive energy balance.
Introduction

Since the 1970s and 1980s, there has been a simultaneous rise in population weight gain, beginning first in richer industrialized countries, but rapidly also becoming evident in low- and middle-income countries [1, 2]. When quantified in 2004, almost 1.8 billion people globally were overweight (body mass index, BMI >25) and approximately 500 million, obese (BMI >30) [1]. Indications are that the epidemic will continue to grow in size insofar as about 170 million children (<18 years) are either overweight or obese [3]. Overweight tracks, so that on average, fat children become fat adults, and herein lies a glimpse of the future, absent successful intervention.

Obesity is associated causally in some instances, comorbidly in others, with a group of non-communicable diseases (NCDs) including hypertension, diabetes, stroke, coronary artery disease and cancers of breast, colon, and prostate [4–6]. Obesity and its comorbidities were calculated in 2004 to account for 36 million disability-adjusted life-years and the health service costs attributable to obesity amounted to up to 6% of total spend [5, 7]. The NCDs are also the leading causes of mortality, globally, and the projections are for a continued rise pari passu with the continued evolution of the obesity epidemics.

The specific determinants of sustained weight gain leading to obesity in most populations worldwide remain unknown despite a clear understanding of the theoretical basis of energy balance [8]. Sustained population weight gain appears to take place when populations adopt a capitalist or industrial style economy, and this is presumed to change the environments in myriad dimensions including wealth accumulation, the contingent allocation of disposable income on goods and services that affect energy balance both proximally and distally, alteration in the quality, quantity, price and availability of foods, and uptake of energy-saving technologies into the economy as well as into the domestic space [8]. The final common pathway is a positive energy balance, an accumulation of the excess calories when intake exceeds expenditure, whether due to an increase in energy intake, reduction in physical activity energy expenditure, or both, to varying degrees [9–11].

Poorer countries bear the brunt of the epidemic of NCDs, a counterfactual but unfortunately accurate assessment, for while prevalence rates might be higher in many high-income countries, the rate-population product places the major burden squarely in the low- and middle-income countries [1, 5, 12]. There is also a fear that as these previously undernourished or marginally nourished poor populations accumulate wealth, the magnitude of their response to the obesogenic influences that accompany that increase in per capita GDP will exceed those experienced in richer populations. Thus, it has been proposed that exposure to undernutrition in early life, and intergenerational undernutrition modifies the susceptibility to become obese and develop a comorbid NCD. This is because undernutrition in pregnancy is now recognized to affect
the metabolic and physiological equilibrium set points and adaptive capacities of the child, and later, adult offspring through epigenetic mechanisms which increase susceptibility to obesity and the cardiometabolic diseases [13–15]. Such mechanisms give rise to phenotypes that are characterized by capacities that are constrained metabolically and physiologically and set points that are inappropriate in relation to demands that exist in our current globalized economies. Thus, when such individuals and populations come to operate in environments where the chronic obesogenic exposures place demands on systems that exceed their programmed capacities, they are metabolically and physiologically overwhelmed resulting in dysfunction that manifests as obesity and its comorbidities. This scenario in which pregnancy undernutrition developmentally entrains individuals to fit a nutritionally deprived environment, but such individuals come to live in obesogenic environments is most obviously evident in countries with a history of intergenerational undernutrition such as India, China, Latin America and sub-Saharan Africa. Here, for convenience we will emphasize the experience among African origin peoples with obesity and cardiometabolic comorbidities as an example, but the principles are equally applicable to all previously poor and marginally nourished populations.

**Obesity and Comorbidities**

There is evidence that worldwide the rate of population weight gain is high. For some populations where there are longitudinal data available on weight and height, it has been shown that the trajectory of adiposity has become steeper in the last two decades of the 20th century [2]. Thus, in the United States, an abrupt inflexion in the annual rate of rise of BMI was noted in 1985 [16]. The cause remains to be explained as there were no recorded similarly abrupt associated changes in dietary intake patterns or physical activity. Norway demonstrated a similar phenomenon in 1995 [17]. Among African populations, there is also evidence of an uneven secular trend in population weight gain, but overall trend data are scarce [11, 18]. Thus, the phenomenon of increasing obesity is global, but there is evidence that the trajectory of increase is higher in countries that have previously been poor. However, population trend data are not widely available across the different stages of economic development, and thus the validity and generalizability of this observation and the underlying explanations are unknown.

Between the 16th and 19th centuries, some 15 million people from Western Sub-Saharan Africa experienced forced migration to the Caribbean and the United States. Today, the contemporaneous existence of genetically related populations living in three settings that are at distinctly different stages on the economic development scale (low income, West Africa; low middle income, Jamaica and high income, United States) creates a context where ecologic comparisons of the impact of socioeconomic environment on health are possible
These contrasts are made possible by the natural migration experiment; there is compression in the time dimension, allowing the evolution of the socioeconomic impact on a single population to be imputed by cross-sectional comparisons of populations captured at different stages of the process. These migrant populations suffered significant social and economic disparities as slaves then; social inequalities still persist centuries later, and within country, it is unsurprising, given knowledge of the drivers of inequalities in health, that these populations experience higher rates of obesity and comorbid hypertension, diabetes and atherosclerotic vascular disease [20]. At the ecologic level, what is observed is a gradient of obesity and related disorders, rural to urban in country, as well as a gradient by country trans-Atlantic, West Africa through the Caribbean and into the United States [20–23]. Thus, populations living in sub-Saharan Africa today like in many other low- and low- to middle-income countries, show higher rates of obesity in urban populations compared to those rural (fig. 1) [24]. Compared to derived populations in the Caribbean, these ancestral populations from West Africa have lower relative weight (BMI) and percent fat, and African-Americans have the highest rates of overweight and obesity of the three groups of populations. Hypertension rates are also lowest in West-African populations, and there is a positive gradient of prevalence rates as populations traverse the nutrition and epidemiologic transitions with African-Americans displaying the highest rates (fig. 2, 3) [19]. This is also true for diabetes (fig. 4) [21]. Although stroke and myocardial infarction have not been evaluated in this manner in these populations, the risk markers for these atherosclerotic sequelae are also present in all these populations and also demonstrate a gradient in level, east to west. Lastly, the relevance of economic advancement and its coincident social and cultural changes are seen in the strong positive association of obesity and its comorbid cardiometabolic diseases with the per capita GDP (fig. 5).
Fig. 2. Prevalence of hypertension in populations of African origin living contemporaneously in West Africa, the Caribbean and the United States (International Collaborative Study of Hypertension in Blacks, 1995). Hypertension defined as systolic blood pressure $\geq 140$ or diastolic blood pressure $\geq 80$ or taking hypertension medication.

Fig. 3. Population prevalence of hypertension and mean BMI in African-origin populations in West Africa, the Caribbean and the United States (International Collaborative Study of Hypertension in Blacks, 1995).
Energy Balance: Activity Energy Expenditure and Population Weight Gain

There are a few simple and appealingly straightforward questions regarding the relationship of activity energy expenditure to population weight, and rate of weight gain, that are unfortunately not accompanied by readily available answers. These include: (a) Is population weight gain associated with a reduction in activity energy expenditure? (b) Will small increases in activity energy expenditure to match the calculated average daily energy surplus prevent weight gain? (c) What is the role of dietary energy intake as well as pattern of dietary...
intake (fats and sugars consumption) on energy balance and weight gain? A wealth of theoretical propositions does exist, but unfortunately very scant and inconsistent data relevant to all these questions are available and therein lies the imperative for scaled up research in these areas [8]. Most of the information available globally is questionnaire based. Questionnaire estimates of activity energy expenditure are notoriously unreliable, and thus it is probably unsurprising that the findings relating activity energy expenditure to baseline weight and weight change on follow-up are not consistent. However, the use of doubly labeled water (DLW) to estimate energy expenditure provides much greater precision; indeed DLW is the gold standard against which other methods are used in free-living populations. Nevertheless, the data available with this precise instrument remain conflicting [9, 10, 25, 26]. Thus, the relationship of baseline activity energy expenditure with body composition is itself inconsistent. Additionally, the relationship of activity energy expenditure with weight gain on follow-up is even more inconsistent. For example, in Jamaica and Nigeria, DLW-measured activity energy expenditure has been shown in a cross-sectional analysis to be inversely associated with body fat. However, this intuitively correct association is not consistently demonstrated even when precise tools like DLW are used in other samples from the same and related populations. So too are the data relating activity energy expenditure at baseline and weight change on follow-up. This is so in circumstances for example, in which the rate of weight gain in Jamaica was large (1.4 kg/year), some three times that observed in both Nigeria and among US Blacks [11]. Longitudinal studies performed on samples drawn from these populations did not reveal a strong relationship of weight gain to baseline energy expenditure [11, 25, 26]. Perhaps this observation of inconsistent relationships between activity energy expenditure and cross-sectional and longitudinal weight change is explainable by a long-standing hypothesis that there is a minimum energy expenditure threshold of TDEE 1.7•REE, below which weight gain is predictable and inexorable; above this point, weight gain is arrested [27]. It is thus important to more fully characterize, using appropriately precise measures, the energy expenditure of populations that are at different points in the nutrition and epidemiologic transitions.

However, if we are honest, an equally obvious interpretation of these data is that in our environments, despite the existence of a historical decline in physical activity related to the removal of muscular power from industrial and agricultural production as well as domestic life, it is the dietary intake that more consistently and powerfully determines energy excess and therefore weight gain [2]. Thus, in addition to possessing tools to accurately measure energy expenditure, it is now vital to acquire similar tools for the assessment of dietary pattern and energy intake. It is equally important to measure dietary energy intake as well as aspects of the diet such as the energy density related to oil/fats and simple sugars in order to arrive at a full understanding of the contributions to energy excess in populations that are gaining weight.
Energy Balance: Dietary Energy Intake and Population Weight Gain

The evidence that dietary energy intake has increased over decades and that this increase was coincident with the take-off of population weight gain comes from national food consumption data [1, 2, 7]. Such studies show that the increase in food energy supply beginning around the 1970s in the United States provided the appropriate level of exposure to have resulted in the rise in obesity in that country beginning in that decade. Similar conclusions have been drawn about the food energy supply and the timing of the obesity epidemic in the UK. The increase in food energy supply was related to increased consumption of some foods such as cereals as well as an increase in the intake of fats, oils and simple sugars in the form of sweetened drinks. In an economic model, consumption is driven both by availability and price, but in addition also by successful market-ing, and all three variables have apparently altered dramatically within the time frame of the epidemic. Industrialization and globalization as they have affected economic and social life in African-origin populations have had identical effects on food and implicitly dietary energy intake [28].

Conclusions

After centuries wherein the challenge has been the maintenance of body-weight in the face of marginal food security, there has been an abrupt uptick in the rate of rise of relative weight globally which began insofar as we can ascertain in the 1970s and the 1980s. This has led to high and increasing rates of overweight and obesity in high-income countries worldwide, but also an alarming inclusion of low- and middle-income populations in this obesity epidemic. The precise drivers of these concurrent epidemics are not agreed, but probably include, on the one hand an increase in dietary energy intake resulting from the impact of industrialization and globalization on food availability and price. On the other, there is the documented steady reduction in activity energy expenditure derived from muscular power as an input into economic production as well as on household and leisure activities. This fall in physical activity energy expenditure has been underway since at least the start of the industrial revolution three centuries ago without an apparent rise in population weight gain. Dietary energy intake is thus highlighted as the probable major determinant of this weight gain. The rise in population weight without accompanying linear growth manifests as obesity. The accretion of fat as well as the response to other environmental exposures during progressive industrialization and modernization is associated with cardiometabolic pathology that has huge impact on health as well as macroeconomics. Given the power and presumed irreversibility of industrialization and globalization, our ability to reverse these obesity epidemics is heavily dependent on new
knowledge being developed which gives insight with prevention and therapeutic implications on the proximal and distal drivers of this progressive positive energy balance.

References


6 Australian Institute of Health and Welfare (AIHW) and National Heart Foundation of Australia: The Relationship between Overweight, Obesity and Cardiovascular Disease. Canberra, AIHW (Cardiovascular Disease Series No 23), 2004.


