Obesity and Hyperobesity: The Setting of an Ongoing Epidemic Landscape in North American Children

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Global Asymmetric Distribution of Childhood Obesity

Beyond the number of increased weekly amputations of limbs performed, heart diseases, cancer related causes and treatment of type 2 diabetes in adult populations, obesity has yet taken another unprecedented toll: the incidence of childhood obesity continues to surge in Canada, Mexico and the United States [Ebbeling 2009].

Regardless of the potential multifactorial nature of increased incidence of obesity, the fairly modern term of obesogenic environments has been coined and singled out as a modern driving force of this epidemic [Swinburn 1999]. In addition, during the last three years, epidemiological data taken into account body mass index (BMI) and waist circumference (WC) distribution against environmental effects had raised the possibility of a genetic influence since the occurrence of paediatric obesity [Wardle 2008].

However, beyond the indisputable impact of a genetic constituent, childhood obesity has become an asymmetric epidemic primarily affecting children in suburban areas of developing and developed countries based on poor diet habits and limited access to organized and routine physical activity [James 1997; Gordon-Larsen 2000].

While obesity rates have increased two to three fold during the last quarter of a century in England and the United States, during this same period a four fold increase in children obesity has taken place in Egypt [Wang 2002].

Figure 1 provides few examples of such an asymmetric distribution of global childhood obesity. It is striking that India attained comparable levels of childhood obesity (18.3% girls; 20.6% boys) equivalent to those found in countries such as Australia, England, Spain, Canada, Mexico and the United States.

Equally remarkable are such low levels of obesity in children in China (4.5% girls; 5.9% boys) by comparison to those found in Chile (27.1% girls; 28.6 boys) Brazil (21.1% girls; 23.1 boys) South Africa (17.7% girls; 13.6% boys) Canada (26.1% girls; 28.9% boys) the United States (35.9% girls; 35% boys) and to a lesser extent those detected in France (14.9% girls; 13.1% boys) [International Association for the Study of Obesity; iaso.org].

Although data compiled by the International Association for the Study of Obesity (IASO) expands from 1982 to 2008, it is interesting to underscore the contrasting differences between France and the rest of western European countries.

While France is affected by childhood obesity, the proportionate rates found in this country are significantly lesser than those found in surrounding western European nations. More conspicuous however, are the significantly lower rates of childhood obesity in China by comparison to those found in India, South Africa and Algeria (see Figure 1).

Although it is possible to argue that the differences seen amongst theses countries might be due to differences in reporting and accuracy of the data collected, this contrasting data emphasizes the multifactorial nature of obesogenic environments whereby ethnicity and genetic predisposition is only one of the many parameters impacting the onset of obesity in children globally.

This article provides our most current insight of the complex interdependency of key driving vectorial factors underscoring childhood obesity postulating a model concerning primary prevention as well as the potential long lasting evolutionary consequences of inaction towards promptly and sternly structuring a concerted global action plan to curve the current epidemic affecting children in all latitudes.

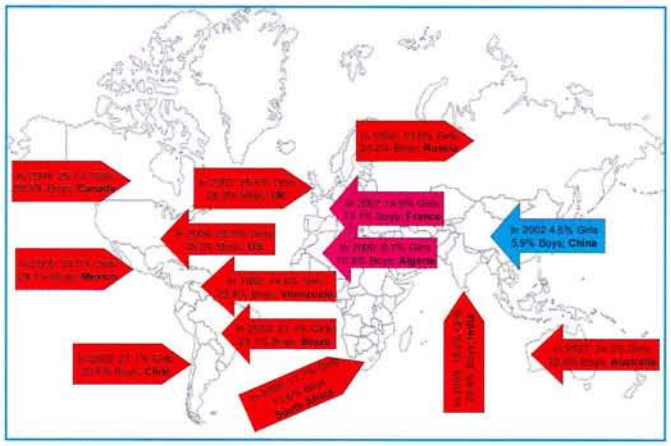

Figure 1. Commencing in the early 80's, obesity in children has become an asymmetrical global epidemic which has almost equally affected girls and boys in North America, Latin America, Europe, Asia and the Middle East. Further details on global obesity in children and adults can be accessed at the archives of the International Association for the Study on Obesity [IASO; www.iaso.org].

Obesogenic Environments: Key Driving Vectors of the Onset of Obesity in Children

Prime driving vectors of overweight and the onset of obesity have multiple origins and interdependent dynamics some of which have been related to environment, lifestyle and cultural factors [Dehghan 2005].

Regardless of individual cultural factors, increased caloric and fat intake seem to act as one of the most common pivotal factors, whereby increase intake of excessive sugar prevailing in soft drinks, traditional candy, frequent consumption of poor quality food portions and reduced physical activity are leading causes of overweight and obesity in children [Dehghan 2005].

Use of food fillers, higher sugar and sodium content in foods targeting children has been an important contributor to childhood obesity since the early 80's. According to a Federal Trade Commission Report entitled Sizing Up Food Marketing and Childhood Obesity [Federal Trade Commission 2009], obesity in children ages six to eleven has doubled since 1980 and the rates amongst teenagers twelve to nineteen has actually tripled during the same time period. One in five young adolescent suffers from obesity putting them at risk of type 2 diabetes, heart disease, osteoarthritis and cancer.

According to the Rudd Centre at Yale University, cereals marketed for youngsters contain almost double the amount of sugar, 65% less fibre and up to 60% more sodium than those for adults [Harris *et al.* 2010].

On the basis of an extensive and thorough analysis of current practices in the fast food industry targeted to children and young adolescents, the Rudd Centre prepared a series of recommendations to limit caloric intake in children. Table 1 summarizes the maximum acceptable calories and sodium content in food meals targeted to children and adolescent.

Acceptable Caloric Content in Meals Designed for Children

Meals targeted to Children	Maximum Calories	Maximum Sodium (mg)
Elementary school-age children (per meal)	650	636
Pre-school age children (per meal)	410	544
Regular Menu Items		
Lunch or Dinner main dishes (per individual item or meal)	700	720
Breakfast main dishes (per individual item or meal)	500	480
Sides, snacks and beverages (per individual item)	350	340

Table 1. Maximum Total Sodium and Caloric Content Acceptable in Meals Targeted to Children and Adolescent.

Current meals marketed to children and adolescent surpass the maximum acceptable level of calories and sodium content outlined within Table 1. Values reported within the Table represent the maximum content of calories and sodium acceptable for each meal indicated on the Table. [Harris et al. 2010].

Although limiting caloric and sodium intake is a straightforward and important determinant to slow down and perhaps decrease the incidence of obesity in children and adolescent, regulation on the industry has proven to be limited and inefficient.

Product labelling has improved, however, in several instances the amount of calories and sodium content in meals and snacks are often higher than those reported by manufacturers. Full description and disclosure of caloric and sodium intake in fast food restaurants is somewhat elusive and self regulated.

According to Harris et al. [2010] more than half a billion dollars (654 million USD) are spent on advertising by fast food restaurants directed to children and adolescent, a sizeable amount of resources that could be much better used to prevent and to reduce childhood obesity and the diversity of severe health problems associated to this condition.

The urgency to efficiently address and to prevent childhood obesity calls for the establishment of a voluntary and collaborative Global Task Force composed of top decision makers of the largest manufacturers and fast food retailers, public health officials, dieticians, food scientists, clinicians, public and private school officials and community members within most affected countries to strike an integrated Programme for the Primary Prevention and Early Treatment of Childhood Obesity under the general guidance and approval of the Health World Organization (WHO).

Incentives to participating manufacturers and food retailers within a collaborative Global Task Force to curve children obesity could involve research and development tax incentives to revamp their raw materials, supply chain of produce and of key ingredients of their most popular menu products.

Community spaces to set green houses to grow local produce dedicated to low income families could respond to the claim of several communities that families opt to eat in fast food restaurants due to the high price of meat, fruit and vegetables. Obesity has been quietly setting in, moving from adult population to children with an alarming swift pace. The consequences and fate of obese children and young adults will most likely mirror that already experienced within adult population.

By the year 2000, approximately 31% of adults in the United States were obese leading to an estimated 365,000 deaths per annum due to overweight, poor diet and physical stillness [Mokdad et al. 2004; Mokdad et. al 2005]. Nonetheless, the occurrence and interdependence between low income, poor diet, exposure and consumption of high caloric fast foods and related products, physical stillness, overweight and death rates induced by obesity is not limited to the United States.

Poor countries have been equally affected since the late 70's early 80's (refer to Figure 1). Before late 70's early 80's the occurrence of such high levels of obesity in both adult and children in countries with a tangible level of poverty was simply unimaginable. Although it is difficult to elucidate the exact external mechanistic factors influencing metabolomics underpinning obesity in such a diverse ethnicity, cultural eating habits and financial capability, global fast food chain industries have indeed permeated not only different cultures but financial status of targeted populations.

In relation to external factors influencing children eating habits and demands, Figure 2 summarizes the resources spent by fast food retailers towards marketing their products to children and adolescent as compiled by Harris *et al.* [2010]

The intensification of current alarming global frequency rates of childhood obesity affected by both external and internal risk factors call for scientists and clinicians not only to work together but to seek functional contributions by developing integrated strategic experimentation and planning of immediate viable interventions. As of to date, there are two surprising undertakings in the scientific and clinical arena. Some these findings and undertakings are described in the Sections that follow.

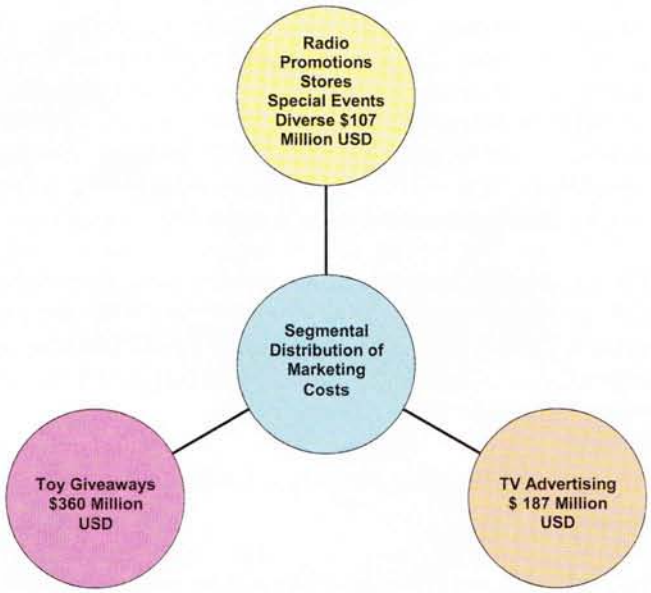

Figure 2. Segmental Distribution on Marketing Costs Associated to Branding Fast Food Consumables.

Cis & Trans Gene Activators Involved in Obesity Metabolomics

From a sheer scientific perspective, the genomic analysis of cis and trans activators of complex metabolomics such as those impinging obesity led to the recent discovery of a master gene controlling adipose gene expression [Small *et al.* 2011].

Briefly, the GIANT Consortium, the MAGIC Investigators, the DIAGRAM Consortium, and the MuTHER Consortium at Oxford University found that the CIS-acting expression quantitative trait locus (eQTL) of the maternally expressed transcription factor KLF14 associated to type 2 diabetes and high-density lipoprotein cholesterol, functions as an effective master gene TRANS-regulator of adipose gene expression.

This truly constitutes a major discovery towards understanding and effectively dissecting the impact of multifactorial regulators acting in response to diverse internal and external risk factors in complex diseases such as childhood obesity.

In addition, the characterization of CIS and TRANS activators acting in tandem during the onset or interrelated disorders such as type 2 diabetes, high-density lipoprotein cholesterol and obesity, most likely responding to external discrete risk factors, provides a unique opportunity to assess upstream internal factors that could trigger pleitropic metabolic peptide signalling cascades and metabolic disruptors.

Although further characterization of CIS/TRANS tandem activators cascades involved in the onset of obesity interrelated disorders is required, the discovery of the KLF14 master gene and its direct correlation with type 2 diabetes and high density lipoprotein cholesterol could be used as predictors of the onset of childhood obesity and hence of proactive screening and control of external risks factors such as exposure to foods with a high caloric content.

Metabolomics, genomics and proteomics must be at the centre of primary and secondary prevention of childhood obesity whereby metabolic and protein signatures should be standardized to better tailor early professional care of children at risk of becoming overweight and ultimately developing obesity.

Weight-Loss Bariatric Surgery in Children: The Gastric Bypass

Dyspnea, heart and liver enlargement and fat engorgement, high blood pressure, high-density lipoprotein cholesterol, arthritis, cyst-filled ovaries and other obesity-related disorders detected in teenagers, has forced hospitals in North America to commence surgical weight loss programmes tailored for children.

The Children's Hospital Boston, the Children's Hospital Medical Centre in Cincinnati and the Hospital for Sick Children (SickKids) in Toronto among other major North American hospitals, have set programmes to perform gastric bypass operations in children 12-17, a radical weight-loss procedure that until recent years had otherwise been performed exclusively in adult populations.

While the formal establishment of such drastic surgical interventions tailored for children and adolescents within reputable institutions is a reflection of the urgency required to curve morbid obesity and mortality within this young population, more robust and stern efforts need to be articulated to effectively address primary prevention of childhood obesity. Some considerations to harness a robust primary prevention of childhood obesity are put forward in the following Section.

Conclusions

Modelling an Operational Platform for the Primary Prevention of Childhood Obesity: Extrinsic Driving Factors

Low income, poor diet, exposure and consumption of high caloric fast foods and related products as well as physical stillness have all been

Extrinsic Factors Influencing the Onset of Childhood Obesity

Extrinsic Factor	Actions
Low Income	Adopt and or increase the existence of community gardens for locally grown produce Provide free to low cost high quality meals with acceptable caloric and sodium content in Public Schools within unfavoured communities. Increase family subsidies geared towards acquisition of basic foods in accordance to total family income and number of family dependents.
Poor Diet	Increase public regular access to information concerning daily recommended consumption of affordable healthful foods and of minimum recommended portions.
Exposure & Consumption of High Caloric Foods & Related Products	Increase dialogue and propose changes to food menus with major fast food chains and retailers. Proactively increase awareness of the consequences of daily or frequent consumption of fast foods within the most vulnerable population.
Physical Stillness	Increase access to free and or affordable organized physical activities within public schools and community centres particularly within poor suburban to rural areas.

Table 2. Summary of key driving extrinsic factors impacting the onset of childhood obesity and proposed actions

identified as pivotal extrinsic factors conducing to childhood obesity. Table 2 summarizes pressing actions that most be adopted to mitigate the impact of extrinsic factors in the onset of childhood obesity. Although some of the proposed actions have somewhat been currently implemented in various North American communities a greater concerted effort is needed to curve the current epidemic of childhood obesity.

Intrinsic Factors Impacting Childhood Obesity

The recent discovery of CIS/TRANS tandem activators cascades involved in the onset of obesity interrelated disorders such as type 2 diabetes and high density lipoprotein cholesterol, in particular of the maternally expressed transcription factor KLF14 acting as an effective master gene TRANS-regulator of adipose gene expression has provided a unique opportunity to develop sensitive and fast pace screening assays that could be potentially used to predict geographical and ethnic clusters of vulnerable children populations at risk of developing childhood obesity. These geographical and ethnic mappings of at risk children population due to alterations in genomic, proteomic and metabolic changes influencing the expression of factors such as KLF14 could be used to selectively and more intensively apply concerted actions to diminish the impact of extrinsic factors as summarized in Table 2.

It is sought that in addition to the ongoing aggressive implementation of surgery interventions such as gastric bypasses performed in obese children, proactive and strategic programmes and initiatives geared towards a more efficient primary prevention of childhood obesity will prevail in the years to come.

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