Early Onset Obesity in Infants and Children: Nature, Nurture or Both? Commentary on Cooke, Hinkley, Chaput & Tremblay, Oken, Paul, Savage, Anzman-Frasca, Birch, and de Silva-Sanigorski & Campbell

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Topic
Obesity

Commenting on:

- Appetitive traits and weight in children – Lucy Cooke
- Environmental/family/psychosocial influences on physical activity in young children – Trina Hinkley
- Obesity at an early age and its impact on child development – Jean-Philippe Chaput and Angelo Tremblay
- Prenatal origins of obesity: Evidence and opportunities for prevention – Emily Oken
- Obesity prevention during infancy: A change of focus – Ian Paul, Jennifer Savage, Stephanie Anzman-Frasca and Leann Birch
- Obesity prevention in the preschool years – Andrea de Silva-Sanigorski and Karen Campbell

Introduction

By the year 2030, obesity rates are predicted to rise from about 30% to nearly 42% of the population of the United States.[1] Childhood obesity rates alone have tripled in the last 30 years with 1 out of 3 U.S. children considered obese or overweight, with the latest prevalence of obesity in the nation being 16.9% with higher levels among some minority groups.[2] Over 10% of U.S. children under the age of two are already obese and 20% of children ages 2 to 5 are overweight.[2] From a public health perspective, this rise in obesity is associated with earlier onset of associated problems like type 2 diabetes, fatty liver disease, high blood pressure, and heart disease, with exacerbated complications in children including disruption of normal development, psychosocial distress, and long-term health care burdens. Beyond individual co-morbid conditions associated with

obesity, the Centre for Disease Control and Prevention (CDC) predicts healthcare costs will top $550 billion by 2030 if rates continue to grow.[1]

This series of six papers[3-8] addresses factors contributing to the development of obesity in early life. While each paper has a distinct approach to childhood obesity prevention, in the end, each gets at the age-old debate: nature versus nurture. This commentary organizes the discussion by examining nature versus nurture approaches to childhood obesity prevention and synthesizes conclusions into a series of policy implications.

Research and Conclusions

Nurture

Chaput and Tremblay[3] hypothesize that obesity may be socially determined, viewing the epidemic in children as a symptom of modern living (computer-dependent, sleep-deprived, physically-inactive, stressed), rather than a genetic or pathological process. Focusing on a systems approach to modifying the environment and lifestyle in order to make the "healthy choice the easy one," the authors focus on interventions targeting key areas of child development: active play and short sleep duration. Similarly, Hinkley[4] targeted interventions at increasing physical activity and reducing screen-time, and sought to determine the specific individual, social and environmental factors (or "correlates") that can be the target of interventions. Both studies limit their intervention scope to two specific activities, and recognize the need for more objective measures of success beyond weight and self-reports of activity, screen-time or sleep.

Researchers in Australia have taken system approaches to obesity prevention a step further by examining where specifically obesity prevention efforts will be most effective in a child's day. Examining preschool-age environments (home, school, healthcare, etc.), de Silva-Sanigorski and Campbell[5] found home-based or healthcare-based interventions to have a larger effect on *body mass index (BMI)* reductions compared to interventions in educational settings. However, the study lacked longitudinal-data, needed guidance from behavioural theories, and failed to control for parental involvement, which has been shown as a key contributor to excessive weight-gain in preschool years.[9] In contrast, Paul et al.[6] focused on parenting during infancy as a means to prevent early obesity by shifting dated clinician recommendations from the prevention of "failure to thrive" (i.e., when the rate of weight gain is significantly lower than that of other children of similar age and gender) to preventing early obesity.

The articles discussed above, not only target key activities, but seek to determine in which part of a child's day an intervention can be most impactful and which social correlates, particularly parents, can be most influential in obesity prevention. However, while researchers focusing on "nurture-based" or socio-environmental approaches agree that prevention of childhood obesity should be the first line of treatment and incorporate multi-factor environmental approaches, it is important not to discount "nature" and be open to using multi-factor interventions that also account for genetic or prenatal conditions.

Nature

While "nurture" certainly plays a role in obesity and offers more tangible potential solutions, "nature" also likely plays an influential role. In contrast to the previous papers, Cooke[7] hypothesizes that eating behaviours have a genetic component that may render certain individuals susceptible to environmental food cues. This paper reviews studies that suggest that responsiveness to food, lack of sensitivity to fullness, tendency to find food reinforcing, and higher speeds of eating all have a heritable components. However, the degree to which each eating behaviour is expressed is determined by environmental conditions, emphasizing the role of parents in the development of eating behaviour in young children. The author notes that much research blames parent feeding styles for a child's obesogenic eating behaviour; but new research suggests a bi-directional process in which parents respond to a child's genetic eating style and weight, which are in turn influenced by the environment.

Oken[8] also suggests a link among body weight, genes and the intrauterine environment but focuses on maternal behaviours during pregnancy. Specifically, this paper reviews data on maternal prenatal obesity, excess weight gain, gestational diabetes, and smoking as predictors of later obesity and adverse complications in infants. Previous research suggests a genetic component for obese mothers having obese children; however, new evidence suggests that the obese intrauterine environment also programs body weight. Further research is needed to clarify if it is the intrauterine experiences that program long-term weight gain or if they are just markers for shared genetic traits.

What's missing?

Several other key issues not mentioned in these papers should be highlighted. Childhood obesity in the U.S. is highest in lower-income populations, highlighting the economic contributions to obesity development. In addition, new studies show that early-life critical periods may be more impactful among certain sub-groups of the population, including low-income Hispanic populations where the emergence of childhood obesity is most rapid in the first few years of life.[2] For example, Hispanic three-year-old children are twice as likely to be overweight or obese compared to blacks or whites.[10] It is important to understand the nature and nurture factors at play in specific sub-groups of the population at greater risk.

In addition, emerging evidence suggests that beyond maternal obesity and smoking, other intra-uterine exposures to certain environmental pollutants or contaminants, termed obesogens or endocrine disruptors, also play a role in the programming of fat cells and life-long susceptibility to obesity.[11,12] In addition to this new theory, we need to understand more about how genetic variants influence early-onset obesity. While obesity genes have been identified, they only explain a small portion of the variance, although new genes are being identified that are specific to early development of obesity.[13]

Breastfeeding is another important developmental factor to consider. In developed countries, there is a robust association between breastfeeding, especially extended breastfeeding >12 months and a lower risk of becoming overweight during childhood and adolescence, even after accounting for maternal obesity and family lifestyle

behaviours.[14,15] Finally, there is likely to be important interactions between nature and nurture. For example, evidence shows that breastfeeding protects against the negative influence of the peroxisome proliferator-activated receptor gamma 2 (PPARg2) Pro 12Ala *polymorphism* on weight gain in the first 18 months of life and BMI in adolescents.[16,17] Other examples of gene-environment interactions are likely to play a critical role in the early development of obesity.

Another issue that needs to be considered is whether today's food environment of highly processed foods is applicable to infants and children. One particular aspect is the increase in dietary fructose due to the use of high fructose corn syrup as an added sweetener.[18] This high level of fructose consumption may be more problematic for infants and children. Studies show a strong link between high sugar consumption and obesity beginning in infancy.[19] Why? Because from an evolutionary perspective babies and infants are not programmed to handle fructose,[20] which is not present in breast milk. Lactose, the principal sugar in mother's milk, is made from glucose and galactose. The metabolic process required to handle fructose only emerges later in development, so this is another reason why promoting breastfeeding may be particularly important to prevent childhood obesity.

Development and Policy Implications

Childhood obesity prevention efforts have generated momentum in the last several years with initiatives spanning across academia, non-profits, local and state governments, and even the White House. The previously-mentioned papers target obesity prevention from diverse perspectives offering a set of policy implications that target systems change, focus on modifiable risk factors, change social norms, improve evaluation measures of success, and reexamine long-held public policies that shape the U.S. food supply.

A systems approach works at different levels of society – individual, interpersonal, organizational, community and public policy – and focuses on modifying environments and lifestyles in order to make the healthy choice, the easy choice. The policy implications of the majority of the papers targeted the interpersonal and organizational levels. More specifically, at an interpersonal level, researchers believe parents are key to preventing of childhood obesity. Policies are needed to educate parents and help limit sedentary activity, boost sleep and increase physical activity. In addition, at an organizational level, researchers emphasize policies that target healthcare providers and key institutions where children spend time (childcare settings, schools, etc.) and using them to disseminate health education to make parents aware of the increased health risks of prenatal obesity, smoking, gestational diabetes, etc. Utilizing existing infrastructure as a cost-effective means for delivering preventive interventions helps focus efforts on vulnerable age groups at various school, childcare, and home-based settings. In addition, policies targeting lifestyle and eating behaviour modification need to be grounded in evidence-based practice and sound behavioural theory models.

In addition to changing dated social norms like preventing "failure to thrive," policies need to further address laws for food marketing to children, portion control, and overall public policy towards food subsidies that have created an environment of excess and

cheap overeating. Several of the researchers point to the IOM's recently published "Early Childhood Obesity Prevention Policies" as a key resource for policy design and implementation.[21] But do these go far enough? The U.S. food supply needs a massive overhaul requiring a paradigm shift to re-align the balance between making healthy fresh foods more available and less expensive, and cheap processed foods more expensive. For example, Farm Bill subsidies and legislation should be reexamined to reduce political and economic incentives that promote items like cheap, nutrient-poor high fructose corn syrup (HFCS). Even a simple change to corn subsidies alone can potentially remove HFCS from the food supply, lessening the obesogenic environment of cheap, super-sized, sugar-sweetened beverages, which are the largest source of calories and added sugar in the American diet.[22] Individuals can force industry to make changes by rejecting foods made with high fructose corn syrup. This will also force food and beverage manufacturers to re-consider using HFCS and turn to using natural sugar instead. This might increase the cost of foods and beverages, which will in turn force the food industry to re-consider portion sizes and force consumers to purchase more fresh foods instead of highly processed items. Actions such as this are needed to share the burden of effort for obesity prevention between individuals, federal and state governments, and the ever-expanding food and beverage industry.

In conclusion, childhood obesity prevention requires a multi-faceted approach targeting "nature," "nurture" and nature-nurture aspects. Policy recommendations need to focus on systems change, modifiable risk factors, social norms, in addition to a much-needed reexamination of food and agricultural policies that shape our nation's food environment.

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