

Intergenerational Obesity: Contemporary Research for Informed Nutritional Counselling

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COMMENTARY

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The stories, skills, morals, and ideas that are passed on to children will impact them for the rest of their lives. As one looks to the future to support the next generation, it is imperative to understand the issues and hurdles that they will have to overcome, particularly in health and wellness. One such issue is the rising trend in the prevalence of obesity in childhood, adolescence, and early adulthood. In a 2016 data brief, the CDC noted that the prevalence of obesity among 12-19 year-old Americans was 21%, with the average weight of children in this age range continuing to steadily increase [1]. Obesity also tends to be familial. According to the UCSF Benioff Children's Hospital, American children with one obese parent have a 50% likelihood of becoming obese, while this increases dramatically in children with two obese parents to an 80% likelihood of becoming obese [2]. This poses a health risk to the child, as prospective cohort studies have identified an association

between obesity and dyslipidemia, cancers of the colon, kidneys, endometrium, gallbladder, and pancreas, cardiovascular disease, type 2 diabetes mellitus, depression, chronic joint pain, and decreased life expectancy [3]. Fortunately, childhood obesity is preventable, and strategic steps can be taken by dietitians and other healthcare practitioners to ensure that clients are provided with the information and tools to promote and maintain a healthy weight. In order to gain a clearer picture of the multifactorial issue of intergenerational obesity and means to break this cycle, it is first necessary to understand the obesogenic influences that will affect the client's child. Previous research has addressed these influences individually, but healthcare practitioners may benefit from a more summative review of the factors contributing to intergenerational obesity. The factors explored in this review include genetics, upbringing, culture, and socioeconomics.

If a mutation or irregularity occurs in any of the parental genes, it may affect the development and metabolism of the child. This is relevant to cases of obesity. It is well established that obesity can originate from a rare irregularity in gene loci across a single chromosome, as is the case with Prader-Willi syndrome [4]. However, it is much more common for obesity risk to be polygenic; it is influenced by an array of genes spanning multiple chromosomes, though the genome-wide association studies (GWAS) that examine loci such as the FTO and near-MC4R have been able to account for less than 1% of BMI variation from genetic influences alone [3]. Even if the influence is generally slight, contemporary geneticists such as Kari E.



White, Cecilia M. Lindgren, and Jessica L. Saben have discovered that certain mutations in the gene loci that govern adiponectin synthesis, lipogenesis, and metabolic efficiency will affect body fat distribution and obesity risk [5, 6]. For example, mitochondrial DNA is maternally inherited and has an influence on basal metabolic rate. When an egg is fertilized, it tags the mitochondria in the sperm cell with ubiquitin, which signals the body to destroy paternal mitochondria [7]. By default, the mother's genetic contribution to the mitochondria of her child's cells predominates over the father's contribution. Consequently, the rate and efficiency of a child's mitochondria will be reminiscent of the mitochondria of the mother [7]. If the mother's genome contains heritable irregularities in her mitochondrial metabolism that resulted in a reduced basal metabolic rate, then these irregularities may be passed on to her children in a process called transgenerational inheritance. This phenomenon was recently observed in a 2019 study that examined the effect of maternal metabolic disorder on three subsequent generations of mice. Female mice were treated with a high-fat, high-sugar diet until metabolic disorder developed, and these mice were significantly more likely to give birth to multiple generations of offspring with mis-expressed mitochondrial dynamic proteins DRP-1 and OPA17. Paternal contributions to obesity risk, on the other hand, can be influenced by short-term mechanisms. While women are born with approximately one million eggs which will last their entire lifetime, men manufacture sperm cells consistently via gametogenesis. Thus, sperm cells are susceptible to malformation if produced during a period of poor health status. More specifically, paternal nutrient deficiencies, nutrient excesses, scrotal overheating, exposure to endocrine disruptors, and radiation exposure can all promote either epigenetic alterations or stable mutations in the underlying genome of the sperm cells [8]. The 2016 TIEGER Study indicated that these mutations may adversely affect the offspring's risk of developing obesity and other metabolic complications [9]. This means that from birth, a child can be susceptible to obesity simply by having the

same genetic profile as his or her obese parents, and this genetic profile can even be affected by the acute health status of the father prior to conception. In practice, it is extremely valuable to encourage clients to be honest regarding their family's history of genetic irregularities, enzyme deficiencies, and pre-conception health status. Collection of these data as part of a patient or client's health history may provide useful insight into the ideal modality of obesity prevention and management. As the discipline progresses and the genetic component of obesity become better understood, this type of risk factor assessment may become standard practice.

Beyond the parental contribution to metabolic outcomes in children, is the extent to which environment and family social dynamics affect obesity. A recent review by Loos and Janssens reviewed the predictive capacity of two different methods for assessing obesity risk: traditional and genetic. Genetic methods of assessing obesity risk involved collecting the child's genetic information and assessing 32 gene loci associated with obesity [10]. Traditional methods simply assessed the child's weight and whether one, both, or none of the child's parents were obese. When applied to the same cohort, traditional methods of predicting obesity were shown to be significantly more accurate than genetic methods, supporting the notion that familial and environmental influences play a stronger role in the development of obesity than genetics alone [10]. Perhaps this information can provide some solace to expectant parents; the factors that have the most influence on the child's risk of obesity are the same factors over which the parents have the greatest control.

In the timeline of fetal development, the first environmental factors affecting a child's obesity risk are the flavor profile of the amniotic fluid and the duration of breastfeeding. Each of these factors has a significant priming effect. Infants and toddlers repeatedly exposed to particular flavors will favor those flavors. Duration of breast milk consumption throughout the first year of life can improve the child's metabolic outcomes later in life. During



gestation, and particularly during the third trimester, amniotic fluid is routinely swallowed by the developing fetus carrying certain flavor profiles from the mother's diet to the fetus [11]. In 2001, Dr. Julie Mennella and her research team discovered that when mothers drank carrot juice during pregnancy or lactation, their infants had fewer negative reactions to a carrot-flavored cereal than the infants of mothers who did not drink carrot juice [11]. Extrapolated to the prevention of childhood and adolescent obesity, pregnant and lactating mothers who regularly consume nutrient-dense foods may be able to prime their children to be more accepting of these foods later in life. Increasing the duration of breastfeeding is another option for mothers interested in preventing childhood obesity. A meta-study pooling the results of 226,508 participants revealed an inverse relationship between number of months that a mother performs breastfeeding and the infant's relative risk of obesity [12]. More specifically, breastfeeding for 7 months or more showed the greatest protective effect, with a 25% reduction in the relative risk of childhood obesity [12]. This serves as a reminder that obesity prevention begins with a healthy diet, both for expectant mothers and their nursing new-borns. While consumption of nutrient-dense foods during pregnancy and the duration of breastfeeding are both the prerogative of the mother, there are other behavioral factors affecting a child's obesity risk that can be influenced by any parent or guardian.

The family unit is one of the primary domains inviting socialization for children, and biological learning—both deliberate and unintentional. Elements of the underlying family lifestyle in the household strongly influences a child's weight status in adolescence and into adulthood. Factors including diet, hours of screen time, common leisure activities, time spent outdoors, and structured exercise are all modelled by the parents or guardians and observed by the children. Improving any of these areas can have a lasting effect on the child's norms. A 2018 study published in BMC Public Health found that behavioral interventions for preventing childhood obesity

have the greatest impact when they focus on behaviors of the entire household rather than behaviors of the child alone [13]. These findings are substantiated by a cross-sectional study that examined the relationship between the weight status of school-age children and whether their parents had established rules regarding screen time, fast food consumption, soft drink consumption, and fruit/vegetable intake [14]. The results of the study showed that simply establishing these rules had no significant effect on the weight status of the children [14]. In short, the precept "Do as I say, not as I do" does not work to curb weight gain in children and adolescents. Rather, the desired behaviors must be adopted and modelled by the parents or guardians themselves. In practice, healthcare practitioners should emphasize the importance of the standards of practice and rules in the household instead of solely promoting them to the adult client.

This parental influence extends to the way that food and weight standards are discussed. The results of a study published in JAMA Pediatrics indicated that 10-year-old girls who were told by family members or peers that they were "too fat" were 66% more likely to be obese by the age of 19 than those who did not receive this negative feedback [15]. The researchers speculated that such labeling may promote inappropriate coping mechanisms, including disordered eating. Instead, it is recommended that parents and guardians provide a strong emotional and logistical support network, teach their child or adolescent about the role of nutrition and exercise in preventing obesity, and empower their child by fostering a strong sense of self-efficacy [16]. The most effective behavioral tools in any parent's toolkit seem to be empowering their children to succeed in their weight loss goals, and then modelling the necessary healthful behaviors themselves [16]. A reasonable hypothesis is that this method of intervention may come with the added benefit of improving or maintaining the parents' or parent's weight status as well. Unfortunately, children also have social influences outside of the home that affect their likelihood of becoming obese.



The proliferation of affordable fast-food restaurants in the United States has been mirrored by enthusiastic public demand for these eateries. People of both high and low socioeconomic status eat out with somewhat similar frequency. Yet, the health burden of this behavior is greater among people of lower socioeconomic status. Poorer populations tend to economize their food purchases in favor of the greatest caloric content for the lowest price, which limits their options of nutrient dense foods [16]. From this perspective, nutrient-dense foods appear to be a luxury, while cheap, high-calorie foods are often the default choice. If a child sees a lush, multi-colored array of vegetables on the restaurant menu but watches their parent opt for fries, a cheeseburger, and a soda, then the parent has tacitly communicated that the cost of selecting a nutrient-dense meal is not worth the price. They subtly messaged a cost-benefit analysis of a meal to their child. Parents and guardians of lower socioeconomic status are fighting an uphill battle in which modelling healthful behaviors requires active effort and deliberation. Whether in a supermarket or at a restaurant, purchasing nutrient-dense foods can begin a trend of investment in one's health and the health of the family which in turn has significant bearings on future generations.

Unfortunately, there is a paucity of healthy food choices in select regions of the United States. These food deserts are devoid of nearby supermarkets or farmers' markets, and their inhabitants instead opt to shop at local gas stations, mini-marts, and convenience stores. As part of the PHRESH Study, a total of 40 Pittsburgh groceries and markets located in two such food deserts were examined to determine if differences existed between price of foods consumed and obesity [17]. The stores were audited for the cost of their culinary staples, fruits/vegetables, and junk foods, as well as the advertising methods for items in these categories. Here, 1,214 participants, all of whom were local shoppers, completed anthropometric measurements and socioeconomic surveys, and their purchasing habits were recorded. The study revealed that low-price stores and high-price stores commonly employed different marketing

strategies; low-price stores placed a greater emphasis on advertising their junk foods and convenience items, while high-priced stores prominently advertised their fruits and vegetables [18]. The researchers found an inverse relationship between the price of food at any given store and the prevalence of obesity among that store's shoppers. Given the opportunity, people in both Pittsburgh food deserts preferred purchasing junk foods and convenience foods over fruits and vegetables, even when the fruits and vegetables were comparably inexpensive [18].

This purchasing behavior and its effect on regional obesity is exacerbated by the presence of food swamps. A food swamp is an area replete with businesses that sell predominantly high-calorie foods. A 2017 study spanning 3,141 US counties concluded that food swamps may be more predictive of obesity rates than food deserts, and the researchers suggested that a more favorable balance of fast food restaurants, grocery stores, and convenience stores would be necessary to reduce the obesogenic effect of food swamps [19]. The residents of food swamps and food deserts should be more attentive to their purchases than the average consumer if they're to reverse the cycle of intergenerational obesity. A limited selection of nutrient-dense foods and/or an overwhelming surplus of calorie-dense foods can skew the shopping lists and eating habits of these consumers. Raising a child in either of these environments presents a potential threat to the child's weight status and future health status, highlighting the importance of paying close attention to the food climate of clients.

As the focus of public health has shifted from acute health issues toward lifelong issues of preventable chronic disease, parents are the stewards of their children's long-term health in more ways than ever before. The benefit is that parents can play an active role in the prevention of obesity in children. This role is made more manageable with education and intervention at any level, especially with the assistance of qualified healthcare professionals. Awareness of a genetic predisposition to obesity can lead to discussions of early preventative efforts. These conversations might



include maintaining a healthy parental diet prior to conception, breastfeeding the infant for greater than 7 months, and a maternal diet during conception and lactation that incorporates a high proportion of nutrient-dense foods. Behavioral interventions in the family might include modelling appropriate levels of activity and healthy food choices, with an emphasis on health education and promoting self-efficacy in food selection. Systemic interventions are harder to implement, but parents can use their awareness of their food climate to actively seek out means to increase the regularity with which they purchase nutrient-dense foods from healthy groceries. Any of these considerations may help individually, but their combination and synthesis can be especially effective. In practice, healthcare professionals can employ a multifactorial, research-driven approach to obesity prevention for current and future generations. Possible avenues of future research include an improved framework for the effect of particular gene loci on obesity risk, the potential benefit of an expanded client/patient health history, and improved methodologies of resource referral for patients in food swamps or food deserts. With sensitivity, quality care, and informed interventions, it is possible to end the cycle of intergenerational obesity.

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