

Pediatric Obesity: It's Time for Prevention Before Conception Can Maternal Obesity Program Pediatric Obesity?

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Abstract: Global increases in obesity have led public health experts to declare this disease a pandemic. Although prevalent in all ages, the dire consequences associated with maternal obesity have a pronounced impact on the long-term health of their children as a result of the intergenerational effects of developmental programming. Previously, fetal under-nutrition has been linked to the predisposition to pediatric obesity explained by the adiposity rebound and 'catch-up' growth that occurs when a child born to a nutrient deprived mother is exposed to the obesogenic environment of present day. Given the recent increase in maternal overweight/obesity (OW/OB) our attention has shifted from nutrient restriction to overabundance and excess during pregnancy. Consideration must now be given to interventions that could mitigate pregravid body mass index (BMI), attenuate gestational weight gain (GWG) and reduce postpartum weight retention (PPWR) in an attempt to prevent the downstream signaling of pediatric obesity and halt the intergenerational cycle of weight related disease currently plaguing our world. Thus, this paper will briefly review current research that best highlights the proposed mechanisms responsible for the development of child OW/OB and related sequelae (e.g. type II diabetes (T2D) and cardiovascular disease (CVD)) resulting from maternal obesity.

Keywords: pediatric, maternal, obesity, developmental programming, disease, gestational weight gain, intervention

Introduction

The overwhelming prevalence of obesity in developed, developing and first world nations has lead public health experts to call this increase in adiposity the world's largest and fastest growing epidemic. Aside from the millions of dollars of health care funding lost on account of obesity, the economic costs fall second to the devastating decline in health and thus quality of life. Obesity is relevant to all age-groups, but has a significant impact on the health, well-being and longevity of two specific populations; women of childbearing age and children. In Canada 23.1% (5.5 million Canadians) are obese and 36.1% (8.6 million) are overweight according to directly measured body mass index scores [(BMI) weight (kg)/height (m²)] (Tjepkema, 2008). Even more startling is the dramatic and progressive rise in pediatric overweight/obesity accounting for 26% of 2–17 year old Canadian children and youth (Shields, 2008). These are alarming statistics as overweight children have a tendency to remain overweight as adults or progress to an obese state and carry with them an array of obesity-related health problems. Of particular concern, from 1999 to 2002, 54.5% of women of childbearing age (20–39 years) were overweight (BMI > 25), 29.1% were classified as obese (BMI > 30) and 5.6% considered morbidly obese (BMI > 40) (Hedley et al. 2004). The parallel increase in obesity rates in the pediatric and maternal population, supports the relationship that having an obese parent will increase the likelihood that an overweight/obese child will remain obese in adulthood; thus exacerbating the current epidemic (Whitaker et al. 1997) and suggesting that the parental environment can have a significant impact on the long-term health of their child.

As the prevalence of overweight/obese (OW/OB) women and children continues to rise so to does their risk of disease and death. Overweight and obese adults have a 50%–100% increased risk of all-cause mortality, primarily attributed to cardiometabolic disease when compared to normal-weight individuals (National Institutes of Health 1998). Severe obesity may also reduce life-expectancy in today's youth, causing them to live shorter lives than their parents (Olshansky et al. 2005). Irrespective of life-expectancy, obese kids may be forced to cope with increased susceptibility to the many cardiovascular, metabolic,

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pulmonary, musculoskeletal, gastrointestinal and psychosocial disorders that accompany increased adiposity such as hypertension, diabetes, sleep apnea, osteoarthritis, fatty liver disease and depression (Princeton University and The Brookings Institution 2008).

The pathophysiological consequences of obesity have a profound impact on the health of OW/OB pregnant women as they progress to term. Similar to the obese child, OW/OB women are also at increased risk of diabetes (i.e. gestational diabetes mellitus (GDM)), hypertension, osteoarthritis and certain cancers, including breast, endometrial and colon (Hall and Neubert, 2005; Krishnamoorthy et al. 2006), as well as a decline in psychological well-being and quality of life (Janicke et al. 2007). However, it may be of even greater importance to note that obesity during pregnancy not only directly affects the mother, but has been implicated in many adverse pregnancy outcomes that can influence the child's short and long-term health. Maternal obesity is strongly associated with increased susceptibility to fetal macrosomia, neural tube defects, preterm birth, increased cesarean section, postpartum infection, pre-eclampsia, gestational hypertension (Smith et al. 2008) and gestational diabetes (GDM) (Arendas et al. 2008); demonstrating that the intrauterine milieu and its regulation throughout gestation must maintain homeostatic balance in order to promote optimal child health.

Whether early life obesity is onset by an imbalance between energy intake and expenditure, possession of a more susceptible genotype or interactive epigenetic mechanisms activated by one's daily environmental exposure, the adaptive responses that occur *in utero* and developmentally program the child's subsequent predisposition to cardiovascular disease and obesity are a potential target for strategic preventive interventions. With the Institute of Medicine making childhood obesity prevention a national priority item in the United States (Office of the Surgeon General 2008), Obesity Canada and the Canadian Medical Association revising clinical practice guidelines calling for innovative preventive measures (Lau et al. 2007), the recently demonstrated poor achievement in the Active Healthy Kids Canada Report Card (Active Healthy Kids Canada 2008) and the Foresight program's vision to develop a sustainable response to obesity (Foresight, 2007) there is no better time than the present to start to design

and implement effective intervention strategies. Targeting women's health during the pregravid, gestational and postpartum periods, crucial times of growth, development and physiological change in mother and child, may prove to be a worthy pursuit in our attempt to halt the intergenerational cycle of obesity currently plaguing our world.

Thus, the purpose of this paper is to provide an audience of health care providers current information about the intergenerational effects of obesity in support of the fetal origins hypothesis. We highlight how maternal lifestyle, particularly a poor intrauterine milieu (via. excess energy consumption and decreased expenditure), can increase offspring susceptibility to obesity and/or its related precursors presumably through developmental programming. By identifying gaps in the literature and stressing the importance of prevention before conception, we hope to demonstrate the need for multidisciplinary interventions in and around gestation as a means to attenuate the prevalence of pediatric obesity, maternal obesity and their associated complications and comorbidities in the mother, her child and thus future generations.

Methodology

The OVID Medline database was queried using the keywords obesity and pregnancy. From 1950–2008 over 2,000 articles were initially identified from which the authors manually screened the sources to include only those that directly pertained to developmental programming of pediatric obesity, its related co-morbidities and the effects of maternal obesity during pregnancy. After careful examination the remaining 189 sources were assessed for content that was relevant to and could provide the best evidence in support of maternal programming of disease *in utero*. Finally, articles included in this review were selected based on scientific merit by demonstrating a relationship between maternal obesity and negative offspring health outcomes that may result in clinical manifestation of down-stream diseases such as T2D, CVD and/or their precursors. Further articles were included if they supported the purpose of the analysis and were related to an originally queried source, but may not have been initially identified through the OVID Medline search. The following is a brief review that attempts to highlight a chronological continuum from maternal obesity to

disease progression in their offspring, with a focus on metabolic and cardiovascular sequelae.

Fetal Origins of Obesity—The Role of Undernutrition

Although this paper will focus on the role of fetal ‘overnutrition’ given the impact of the obesity epidemic on women and children, the concept of developmental programming will be introduced from an ‘undernourished’ perspective as this was the original condition from which the relationship between the maternal intrauterine environment and long-term health of the offspring was first recognized. The intrauterine milieu’s impact on adult health outcome was first described by Barker and colleagues almost two decades ago. By retrospectively studying the effects of intrauterine growth and maternal physique on adult blood pressure, a strong relationship was established between the mother’s internal environment, child’s birth weight and the development of hypertension in adulthood (Barker et al. 1990). Furthermore, it was proposed that improving maternal health, particularly nutrition, may have a role in preventing such outcomes later in life. Arising from this original work, ‘The fetal origins of adult disease hypothesis’ has since attributed early environmental factors, predominantly maternal nutritional status and its effect on the fetus *in utero*, to the premature programming of risk for cardiovascular and metabolic disease in adulthood (Barker et al. 2002; Barker, 2005; Barker et al. 2005; McMillen and Robinson, 2005). Developed as a causal explanation related to how low birth weight leads to disease progression, the ‘thrifty phenotype hypothesis’ was proposed. Briefly, it states that having a suboptimal intrauterine milieu, as a result of fetal nutrient deprivation, will facilitate an adaptive growth and development response of vital tissues and organs at the expense of others. This is proposed to lead to altered metabolic mechanisms that enhance postnatal survival in a time of nutrient restriction. However, this relationship becomes problematic when the postnatal nutritional environment is one of abundance, not deprivation as experienced *in utero*, and a metabolic mismatch is established developmentally programming the child for ‘catch up’ growth and susceptibility to cardiometabolic disease later in life (Hales and Barker, 1992; Hales and Barker, 2001; Hales and Ozanne, 2003). As a result of maternal undernutrition, metabolic adaptive

responses that occur in the fetus initially designed to enhance survival may in fact be undesirable for postnatal growth and development if the child’s environmental exposure pre and post natal are dissimilar.

Maternal Obesity Programs Pediatric Obesity and Metabolic Function

Despite the abundance of research that links ‘catch up growth’, ‘adiposity rebound’ and early onset obesity to fetal nutrient deprivation *in utero* (Ong et al. 2000), the developmental programming of pediatric obesity, its precursors (i.e. impaired glucose tolerance (IGT), insulin resistance, etc) and resulting co-morbidities (e.g. T2D, CVD, cancer) will be discussed from here, with a focus shifted towards maternal overnutrition and the corresponding habitus of obesity.

Viewing pregnancy as a critical period of pediatric obesity prevention is a relatively novel area of study given the significant rise in maternal obesity in recent years. As such, the acute effects on fetal growth and development *in utero* and subsequent predisposition to obesity in response to maternal over nutrition and obesity are just beginning to attract attention in the literature. Maternal obesity is a well-recognized risk factor for *fetal macrosomia* and several epidemiological studies have highlighted a U or J-shaped curve/relationship between birth weight and adolescent weight as well as adult fat mass with babies large or small for gestational age being at increased risk (Curhan et al. 1996; Pettitt and Jovanovic, 2001; Rogers, 2003; Wei et al. 2003; Ozanne et al. 2004; Ong, 2006; Druet and Ong, 2008) as well as increased maternal BMI leading to greater susceptibility of fetal death due to placental dysfunction (Nohr et al. 2005). Thus, as weight increases above and beyond that considered healthy so to does the risk of chronic disease, pregnancy complications and all-cause mortality.

Furthermore, pregravid OW/OB and surpassing gestational weight gain (GWG) requirements are strongly associated with an increased risk of fetal overgrowth, having a child born large for gestational age, infant fatness (Ehrenberg et al. 2004) and post-partum weight retention (PPWR) (Amorim et al. 2007; Huang et al. 2008). These women are also at higher risk of maternal hyperglycemia and GDM (Hillier et al. 2007), conditions that have also been independently linked to having

an overweight/obese child (Pettitt et al. 1983; Pettitt et al. 1985; Pettitt et al. 1987; Pettitt et al. 1993; Noussitou et al. 2005; Rosenberg et al. 2005; Schaefer-Graf et al. 2005; Malcolm et al. 2006; Allen et al. 2007; Chu et al. 2007; de Campos et al. 2007). The resulting implications of increased maternal adiposity and the corresponding risk this has on the child's future health must be addressed.

During pregnancy maternal insulin sensitivity is naturally decreased as a protective mechanism of survival that attempts to direct a portion of nutrients consumed by the mother to the developing fetus to ensure adequate energy partitioning for optimal growth (King, 2000). However, when exposed to an obesogenic environment, we presume that maternal overnutrition results in a positive energy balance. The surplus of energy would then need to be stored by the mother with additional fuel redirected to the child and stored, increasing their risk for metabolic disease; a situation that is exacerbated in children of GDM pregnancies (Dabelea et al. 2008).

This association between maternal OW/OB and childhood overweight was first demonstrated in rodent models (Vickers et al. 2000; Vickers et al. 2003; Bayol et al. 2007; Ferezou-Viala et al. 2007; Harvey et al. 2007; Samuelsson et al. 2008; Shankar et al. 2008). The resultant sequelae (i.e. obesity) present in offspring of OW/OB mothers has been most strongly attributed to altered neuroendocrine regulation of appetite signaling pathways (Muhlhausler et al. 2006). However, since the appetite regulatory system of the rat develops postnatally and the same system develops before birth in sheep and humans, investigations utilizing lamb models are more relevant to human physiology and reproduction, and thus warrant discussion.

Appetite regulation and energy homeostasis are regulated through a variety of intricate neuroendocrine pathways that act in response to systemic glucose, insulin and leptin concentrations at the level of the hypothalamus to control the expression of the orexigenic neuropeptides, neuropeptide Y (NPY) and agouti-related peptide (AGRP) and their anorexigenic counterparts, pro-opiomelanocortin (POMC) and cocaine-and amphetamine-regulated transcript (CART) (Kalra et al. 1999). The novel work of Muhlhausler and colleagues, demonstrated for the first time, an alteration in offspring appetite regulatory system function in response to increased maternal nutrition during late gestation (Muhlhausler et al. 2006). This group demonstrated that at 30 days of age, lambs born to ewes that consumed 40% more

than their daily energy requirements in late gestation displayed increased milk intake, plasma glucose concentrations and subcutaneous adiposity when compared to controls. The relative increase of adipose tissue was directly related to circulating glucose concentrations in early life, which is consistent with children born to mothers with GDM or glucose intolerance (Dabelea et al. 2008). Furthermore, a decrease in leptin sensitivity with increased adiposity was seen in lambs of over-nourished ewes and this was attributed to down-regulation of the leptin receptor in the arcuate nucleus of the brain resulting in leptin resistance. Increased maternal nutrition resulted in increased, appetite inhibiting, POMC expression as plasma glucose concentration increased. Although NPY and AGRP expression was not different when compared to controls, CART expression in response to increased nutrient intake and fat mass was also reduced when lambs were exposed to excess nutrient supply before birth which could have implications for regulation of energy balance. Overall, the authors attributed the inability of over-nourished mothers to up-regulate hypothalamic anorexigenic pathways in response to increased adiposity to a central resistance to the actions of leptin and consequently may lead to increased childhood obesity in OW/OB mothers consuming excess calories in gestation (Muhlhausler et al. 2006). These findings, which accredit the programming of pediatric obesity to leptin resistance, are in agreement with others rodent models (Franke et al. 2005; Ferezou-Viala et al. 2007) including those which demonstrated reversal of glucose intolerance and diet induced obesity through maternal leptin administration (Stocker et al. 2007). It has also been cited that untreated GDM and hyperglycemia in pregnancy can result in inappropriate programming of appetite regulating networks in the brain contributing to development of later OW/OB (Franke et al. 2005). Increased caloric consumption above that needed for optimal growth and development of the fetus can result in excessive GWG and subsequent PPWR. This demonstrates that increased maternal adiposity may enhance the susceptibility to glucose intolerance, T2D and metabolic dysfunction in both mother and child.

Maternal Obesity Programs Pediatric Obesity and Cardiovascular Disease

Overfeeding practices leading to excessive energy intake, coupled with decreased expenditure through

reductions in daily physical activity have contributed to global increases in maternal obesity. In addition to the concept that *obesity begets obesity*, increased maternal adiposity has also been identified as an important determinant of the metabolic syndrome in children (Boney et al. 2005). A poor intrauterine milieu and the resultant presentation of pediatric obesity leads to a plethora of metabolic abnormalities, particularly a proinflammatory response as a result of increased fat mass and release of adipokines in the blood, that predispose the child of an OW/OB mother to cardiovascular disease in adulthood (Weiss and Caprio, 2005). As the rates of pediatric and maternal obesity continue to rise, it is important to understand the underlying mechanisms that increase one's susceptibility to comorbidities (e.g. hypertension) directly related to pediatric obesity in an attempt to improve the quality of life of this population and prevent disease progression into adulthood.

Initially epidemiological evidence showed a non-significant relationship between maternal obesity and offspring blood pressure, yet demonstrated that underweight resulted in increased blood pressure, supporting the Barker hypothesis (Godfrey et al. 1994; Clark et al. 1998;). However, in light of the current obesity epidemic, more recent research draws parallels between increased maternal adiposity and offspring hypertension (Phillips et al. 2005) while making associations with GDM (Lee et al. 2007). In fact, it is now accepted that maternal obesity is associated with a poor maternal metabolic profile which is partially attributed to increased blood pressure, insulin resistance, hyperglycemia, and elevated blood lipids (Wilson and Grundy, 2003). The resulting metabolic platform, if not transformed prior to conception, may result in a suboptimal 'obesogenic' intrauterine environment that has great potential to developmentally program pediatric obesity and cardiovascular disease. Recent work in animal models, mimicking the human obese condition, has provided impressive evidence linking the obesogenic gestational environment to negative health consequences in offspring (i.e. hyperphagia, insulin resistance, obesity, and hypertension) (Samuelsson et al. 2008). Obesity, induced in female mice by feeding an obesogenic diet (16% calories from saturated fat, 30% simple sugar) 6 weeks before mating and maintained during pregnancy and lactation was examined and compared to a control to assess effects on offspring metabolic and cardiovascular health.

Offspring of both groups were weaned onto a standard diet and studied at 3 and 6 months postpartum. The obesogenic diet was designed to resemble that which is consumed by many of us in today's society and was compared to a control diet which consisted of 3% fat and 7% sugar. The mothers consuming the obesogenic diet, comprised of highly palatable fats and refined sugar, had offspring who displayed hyperphagia, increased adiposity, adult symptoms of the metabolic syndrome, including hypertension, as well as decreased physical activity patterns (Samuelsson et al. 2008). Both maternal caloric intake and weight gain were significantly greater in the mice fed the obesogenic diet resulting in 4-fold increase in abdominal fat mass (4.00 ± 0.42 g) when compared to control fed a standard diet (1.02 ± 0.08 g). Offspring of the obese mothers were hyperphagic from 4 to 6 weeks, with increased adiposity and reduced activity levels at 3 months when compared to controls (Samuelsson et al. 2008). Furthermore, at 6 months offspring of obese mothers were heavier, displayed increased abdominal obesity associated with adipocyte hypertrophy and arterial endothelial dysfunction. Of greatest concern was the presentation of hypertension in the offspring of the obese mothers at 6 months postpartum (134 mmHg in obese offspring vs. 124 mmHg in controls), as measured by systolic radiotelemetry (Samuelsson et al. 2008). The phenotypic characteristics presented in the offspring of obese mothers demonstrate, for the first time, the drastic metabolic and cardiovascular effects induced by excessively feeding on high-fat diets during pregnancy and throughout lactation, which promoted maintenance of their obese state. In an attempt to quantify the results and understand the ramifications of maternal obesity, Samuelsson and colleagues, suggested that in addition to genetic predisposition, a complex interaction between dietary and obesity related metabolic sequelae initiate a cascade of internal processes with the final outcome being increased susceptibility to pediatric obesity in their offspring. However, increased adiposity (Rahmouni et al. 2005) and endothelial dysfunction (Armitage et al. 2004) have both been cited as possible independent mechanisms contributing to the hypertensive condition of obese offspring.

Presently, the literature has demonstrated that not only feeding preferences for a palatable high fat diet can be programmed *in utero* as a result of maternal behavior (Bayol et al. 2007), but physical

activity habits as demonstrated through a reduction in energy expenditure and excessive food consumption (i.e. hyperphagia) can be programmed as well (Samuelsson et al. 2008). Misinformed maternal lifestyle choices, especially poor feeding preferences during pregnancy, may predispose the developing child to traits that result in perpetual weight gain and early onset of overweight, premature metabolic and cardiovascular disease and significant decline in quality of life.

Although the molecular mechanisms of action are likely related, the interaction between these complex systems have not, as of yet, been completely identified or studied sufficiently to establish concrete therapeutic targets due to the relative novelty of the research addressing maternal obesity and its related sequelae on mother and child. This ‘gap’ in the literature suggests that prevention before conception might be an optimal, albeit potentially unrealistic, clinical strategy for reducing pediatric obesity prevalence. Preliminary research has identified the role of excess glucocorticoids and circulating proinflammatory cytokines as a result of increased fat mass in females of childbearing age and their corresponding effects on the hypothalamo-adrenal-pituitary axis (MohanKumar et al. 2007), leptin resistance (Rahmouni et al. 2005) and dysfunction of the

appetite regulating neuropeptides POMC, AGRP, NPY and CART (Muhlhausler et al. 2006) as the most promising areas of future research proposed to identify therapeutic targets that would offer clinical benefits by altering the appetite regulatory center.

The literature associated with the fetal programming of pediatric obesity is derived from multiple levels of research (i.e. epidemiological, animal model, and human studies) which presents evidence suggesting that a highly palatable diet, rich in refined sugar and saturated fat and consumed beyond the nutritional requirements of pregnant women, combined with inactivity, can be detrimental to fetal development *in utero*. As a result, this exposure may have drastic ramifications on the long-term health of the child. In essence, the child will then carry with them these risk factors and/or conditions into adulthood, perpetuating the intergenerational effects of obesity exacerbated by maternal adiposity (Fig. 1). By modifying the behavior of women during pregnancy and preventing abnormal/excessive GWG through nutritional and activity interventions, the burden of increased fat mass can be minimized and a healthy, optimal lifestyle and future for the child could potentially be ‘programmed’. Animal model results from female offspring born to high-fat diet-induced

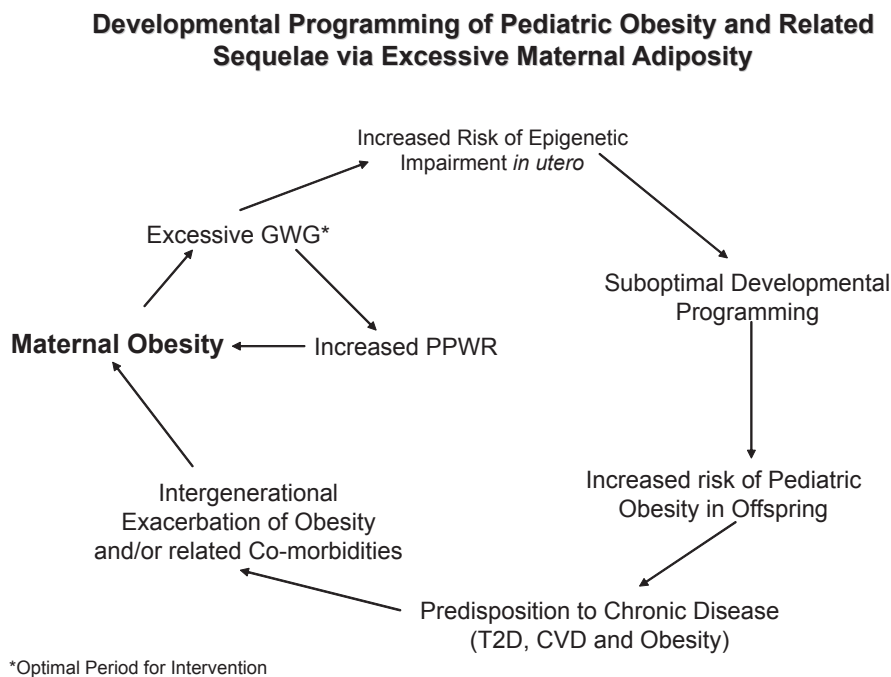


Figure 1. The role of maternal obesity in development of childhood OW/OB, T2D and CVD (GWG, gestational weight gain; PPWR, postpartum weight retention; T2D, type 2 diabetes; CVD, cardiovascular disease; OW/OB, overweight/obesity).

obese mothers are encouraging. In an investigation in which obese mothers who switched from a high-fat diet to one of balance nutrient composition during the periconceptual, gestation and lactation periods birthed female offspring that were 'resistant' to the deleterious effects of consuming a high fat diet at weaning (Gallou-Kabani et al. 2007). The ability of female offspring to 'resist' impaired glycemia, increased body fatness and a hyperphagic state postpartum with *ad libitum* access to fatty foods suggests that interventions during pregnancy can have important health implications. These findings stress the need to test the effects of lifestyle interventions (i.e. diet and exercise) aimed at preventing excessive GWG in a controlled, clinical setting as a means to prevent abnormal developmental programming or attenuate the mechanisms that promote such drastic alterations. To prevent the onset of pediatric obesity and the associated problems associated with deleterious downstream signaling as a result of poor maternal lifestyle choices, innovative intervention strategies are needed that cater to the lifestyle of the mother and encourage support from the entire family.

Where Should we Go from Here?

Poor maternal lifestyle behavior leading to *in utero* exposure to an obesogenic fuel supply, rich in saturated fat and sugar, may alter hypothalamic regulatory centers that control appetite, food consumption and activity patterns thereby programming pediatric obesity. Of great concern are those behaviors that lead to excessive GWG. An increase in adiposity beyond the Institute of Medicine recommendations, in addition to having OW/OB parents, has been linked to early childhood overweight (Dubois and Girard, 2006; Wrotniak et al. 2008). Thus, strategies that reduce 'risky' lifestyle behaviors such as poor dietary choices leading to overconsumption and energy storage, lack of physical activity resulting in suboptimal energy expenditure or a combination thereof, warrant clinical intervention that educate those involved so they can reap the benefits of prevention before conception or at least during gestation. Recently, one controlled trial has had success in preventing excessive GWG and deterioration of glucose metabolism in obese women using dietary counseling sessions (Wolff et al. 2008). A few have successfully limited PPWR

(Leermakers et al. 1998; O'Toole et al. 2003), whereas others displayed an inability to limit GWG in obese women (Gray-Donald et al. 2000; Polley et al. 2002; Olson et al. 2004; Kinnunen et al. 2007). To date, a small number of trials have assessed the combined effects of nutrition and exercise on limiting GWG in a controlled fashion. Artal and colleagues were able to attenuate GWG in obese women with GDM (Artal et al. 2007) whereas others were not as successful (Hui, 2006). The need for interactive, educational and activity-based interventions during pregnancy highlighting individualized ways in which nutrition and physical activity can be incorporated into daily life to improve child health outcomes are desperately needed. Promoting a balanced lifestyle during pregnancy and making changes to one's habits during this time are not only ideal, but are supported by the recent call in the literature for well-designed, randomized controlled trials evaluating the effectiveness of lifestyle interventions during this period (Dodd et al. 2008; Gavard and Artal, 2008). Achieving this objective through an intensive family-centered approach that is individualized to meet the needs of each person while adhering to an overall group treatment plan has potential for success as long as it is sustainable. Exercise participation has been deemed safe during pregnancy (Davies et al. 2003; Gavard and Artal, 2008), and resistance training has been shown to limit the need for insulin therapy in overweight women with GDM (Brankston et al. 2004), demonstrating clear clinical advantages. The benefits of this type of exercise delivered in a pregnancy-specific modality may prove beneficial in limiting GWG. Furthermore, greater nutrition knowledge has been linked to lower PPWR one year postpartum in a low income population (Nuss et al. 2007). Thus, the possibility exists that pregnancy-specific, individualized, family-centered lifestyle interventions focusing on nutrition and physical activity may have a beneficial effect on limiting GWG and PPWR, and in turn, limit the future presentation of pediatric obesity and its related sequelae.

By quantifying the needs and wants of pregnant women through prenatal lifestyle questionnaires and discussions in a clinical setting focused on nutrition and exercise, healthcare teams will be able to design and implement intervention strategies that aim to prevent the programming of pediatric obesity adding more weight to the statement

that ‘an ounce of prevention is worth much more than a pound of cure’.

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Disclosure

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