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Can Breastfeeding Reduce the Risk of Childhood Obesity?

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1. Introduction

Over the last two decades, rates of childhood obesity have increased to a point where the World Health Organization (WHO) has described it as "globesity" to indicate the "escalating global epidemic of overweight and obesity" (WHO, 2011). A number of factors have been identified that increase the risk of childhood obesity, including: high birth weight (Apfelbacher et al., 2008), mother's pre-pregnancy Body Mass Index (BMI) (Catalano et al., 2009; Gibson et al., 2007; Vos & Welsh, 2010), weight gain through pregnancy and diabetes in mother (Boney et al., 2005), mother's/father's BMI (Moens et al., 2009; Whitaker et al., 1997), early introduction of solid foods (Huh et al., 2011) and screen time (Fulton et al., 2009; Sisson et al., 2009), lower educational level, smoking in the home, and breastfeeding for less than three months (Apfelbacher et al., 2008). In some studies, low socioeconomic status (SES) and household income levels are seen as predictors (Veugelers & Fitzgerald, 2005; Vieweg et al., 2007); conversely, other studies have reported high socioeconomic status as a predictor (Cui et al., 2010). In addition, living in a single-parent family has been cited as a predictor of childhood obesity (Gibson et al., 2007), while maternal psychopathology has been shown to be an inconsistent predictor of childhood obesity (Gibson et al., 2007; McConley et al., 2011).

Childhood obesity can have very serious short and long term adverse health consequences on quality of life, performance achieved and long term health and life expectancy. It is estimated that the obesity epidemic will increase both direct and indirect costs to society and the health care and social insurance systems. One major concern is that obesity has been shown to track through the lifecycle. Obese children most often become obese adolescents (Deshmukh-Taskar et al., 2006) and obese adolescents have a 33% increase in the probability of having a life threatening event before the age of 60 (Baker et al., 2007).

There are few proven treatment options for either obese children or obese adults and those that do exist tend to be costly (Flodgren et al., 2010; Summerbell et al., 2004). Many feel the emphasis must be on the primary prevention of childhood obesity. It has been suggested that early infant nutrition may be one important factor in this pursuit. There are many well-evidenced reasons to support breastfeeding as it relates to the improved health of the mother and baby, but research that exists on breastfeedng as a public health obesity intervention is equivocal. The objectives of this chapter are to 1) describe the epidemiology of childhood obesity and its related comorbid conditions 2) discuss the biology of breastmilk and to explain

how it differs in constituents and method of delivery from infant formula and 3) to summarize the high level research (i.e., systematic reviews and meta analysis) that examines the relationship between breastfeeding and childhood obesity.

1.1 Definition of obesity/childhood obesity

Obesity is defined as excessive accumulation of fatty tissue that can hinder the effective functioning of the human body (WHO, 2000). As a result, health problems may ensue, leading to a poor quality of life and decreased lifespan. When seen in childhood, the issues of obesity can be far more extensive, as health problems occur earlier in life and place a burden on the child, family, community and health care system.

The calculations that determine obesity in children are not as clear-cut as one might think. It has been suggested that the standard BMI (weight (kg)/height (m^2)) should not be used as the sole measurement, since children's height and weight vary greatly during major growth spurts (Logue & Sattar, 2011). In addition, waist circumference should be included in the calculation, as central obesity is considered a major risk factor for Type 2 Diabetes Mellitus (DM) and Coronary Artery Disease (CAD) (Kovacs et al., 2010; Steene-Johannessen et al., 2010; Stevens et al., 2010).

Currently, the classification of overweight and obesity varies among major agencies (Table 1). The US Centre for Disease Control (CDC), International Obesity Task Force (IOTF) and WHO base their determinations on different populations during different time periods. The CDC surveyed American children from 1963-1994 (National Centre for Health Statistics, 2002). In 2000, the IOTF based their criteria on children from US, Brazil, Great Britain, Hong Kong, Netherlands and Singapore (Cole et al., 2000). In comparison, in 2006, the WHO collected data on children from US, Brazil, Ghana, India, Oman and Norway (WHO Multicentre Growth Reference Study Group, 2006) who lived in optimal health conditions, and were exclusively/primarily breastfed for up to 4 months, with some breastfeeding extending until 12 months of age. Using information and extrapolating for children, the cut-off points are given below. These guidelines should be considered when reviewing data on obesity rates in various reports, as different definitions provide varying prevalence estimates. For example the CDC cut-offs tend to report a much higher prevalence of obesity compared to the other growth references (Twells & Newhook, 2011).

Another point to consider is whether or not growth charts for infants (based on age) should be modified based on infant feeding method. Formula fed children grow faster than those who are breastfed (Dewey, 1998; Kramer et al., 2004; Victora et al., 1998), possibly due to the higher protein content of formula versus breast milk, which should be considered when referring to patterns of infant growth (Alexy et al., 1999; Koletzko et al., 2005). Even with guidelines, such as those from CDC, IOTF, and WHO, it is suggested that childhood overweight and obesity are often underdiagnosed. The development of a better diagnostic tool may be beneficial, since it is within childhood that interventions might prove to be more effective (Benson, 2009).

	US CDC	IOTF	WHO
Overweight	>85th	≥91st	>84th
Obese	≥95th	≥99th	>97.7th

Table 1. Childhood obesity rankings (percentile)

1.2 Global prevalence of childhood obesity

The prevalence of obesity is increasing throughout the world, particularly among children. In 2008, 1.5 billion adults (20 and older) were overweight; comparatively in 2010, 43 million children under the age of 5 were overweight (WHO, 2011a).

Human obesity develops as a result of interactions between genes, environmental factors, and behaviour. It has been found that genetic disorders such as Bardet-Biedl and Prader-Willi syndromes can directly cause obesity and, as a result of the study of twins and adopted children, heritability estimates of 40–70% have been reported (Farooqi & O'Rahilly, 2006). The relatively recent rise in global obesity rates over the last 25 years however suggest that mainly environmental factors are involved. Current environmental and lifestyle issues (social, behavioural, cultural and community) which promote an imbalance between energy intake and energy expenditure are purported to be the main causes of increasing obesity rates. The sedentary lifestyle and high-caloric diet seen in many populations have led to alarming rates of childhood obesity. As shown in Figure 1, since 1976 the United States has seen a surge in obesity rates among all age groups of children; particularly, obesity rates among preschool children aged 2-5 increased from 5.0% to 10.4% over the 1976/1980 to the 2007/2008 time span (Ogden & Carroll, 2010). Similar increases have been reported in regional populations in Canada (Canning et al., 2007).

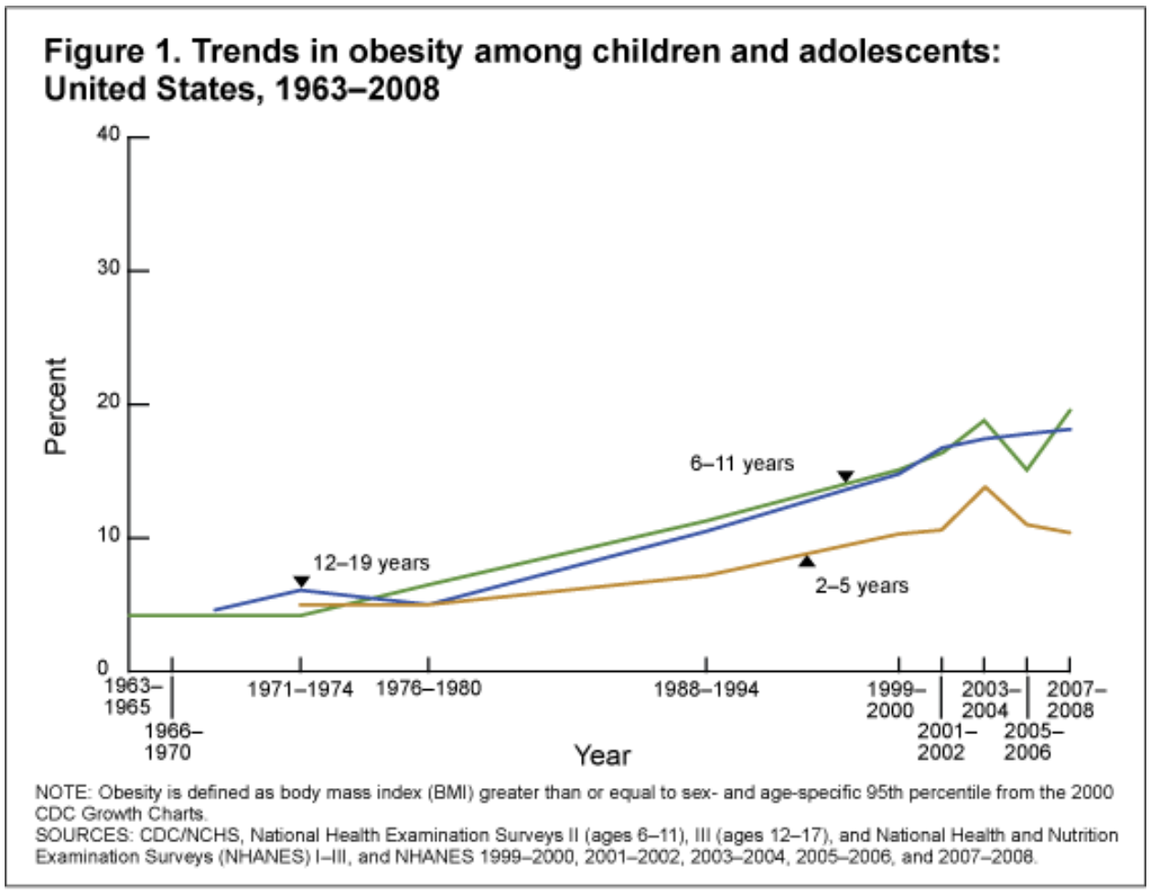

Fig. 1. Prevalence of obesity among children and adolescents: United States, Trends 1963-1965 through 2007-2008 (Ogden & Carroll, 2010).

Geographical differences in childhood obesity are also being observed. As can be seen in Figures 2 and 3, the 2010 worldwide prevalence varies among countries. Obesity rates are rising in low and middle income countries, a trend which had been previously seen in high income countries (WHO, 2011b). Although in the early 2000s Scandinavian countries had lower childhood obesity rates than Mediterranean, their rates (along with those of the US, Japan, United Kingdom, Spain, France and Greece) showed major increases throughout (Dehghan et al., 2005). Interestingly, more recent studies indicate that in France, Switzerland, Sweden, the US and UK, the childhood obesity trends appear to be stabilizing (Stamatakis et al., 2010; The Health and Social Care Information Centre, 2010). It has been suggested that in developed countries, the childhood obesity epidemic may be slowing as a result of increased awareness and policy programs that have been put in place in very recent years (Stamatakis et al., 2010).

Additionally, there is discussion regarding rural versus urban childhood obesity rates. Several authors (Bruner et al., 2008; Ismailov & Leatherdale, 2010; Simen-Kapeu et al., 2010) found that rural Canadian children were more likley to be overweight/obese; however, the WHO stated in 2011 that obesity is rising in urban settings (WHO, 2011a).

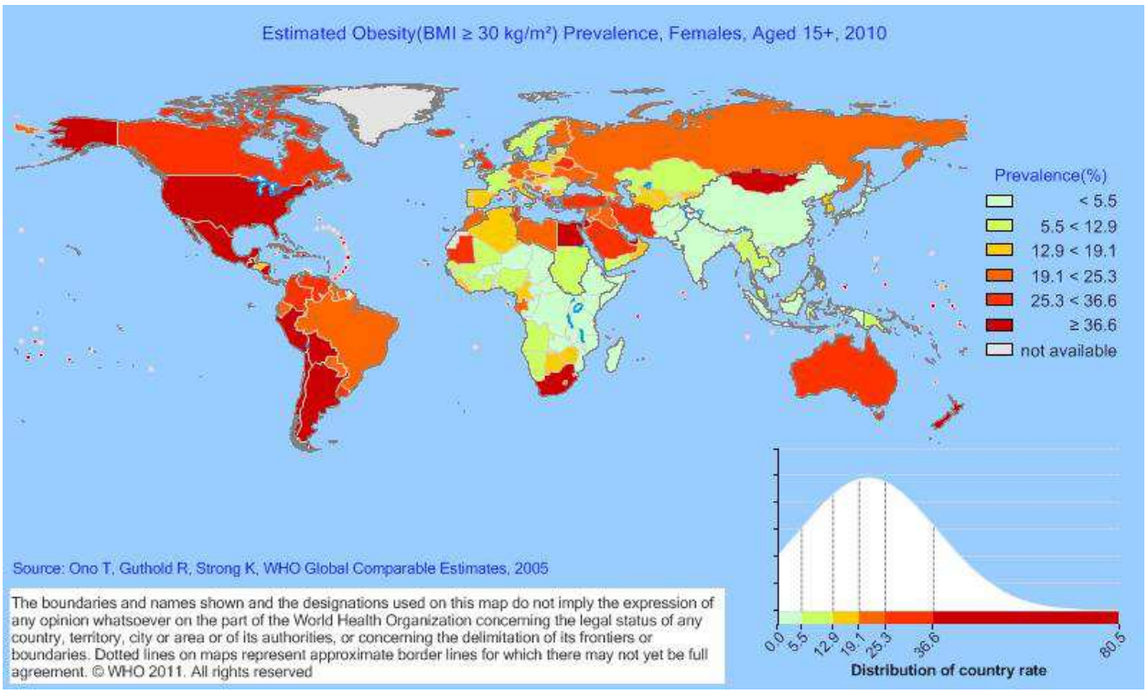

Fig. 2. Estimated Obesity Prevalence in Females 15 years and over, 2010 (WHO, 2011b).

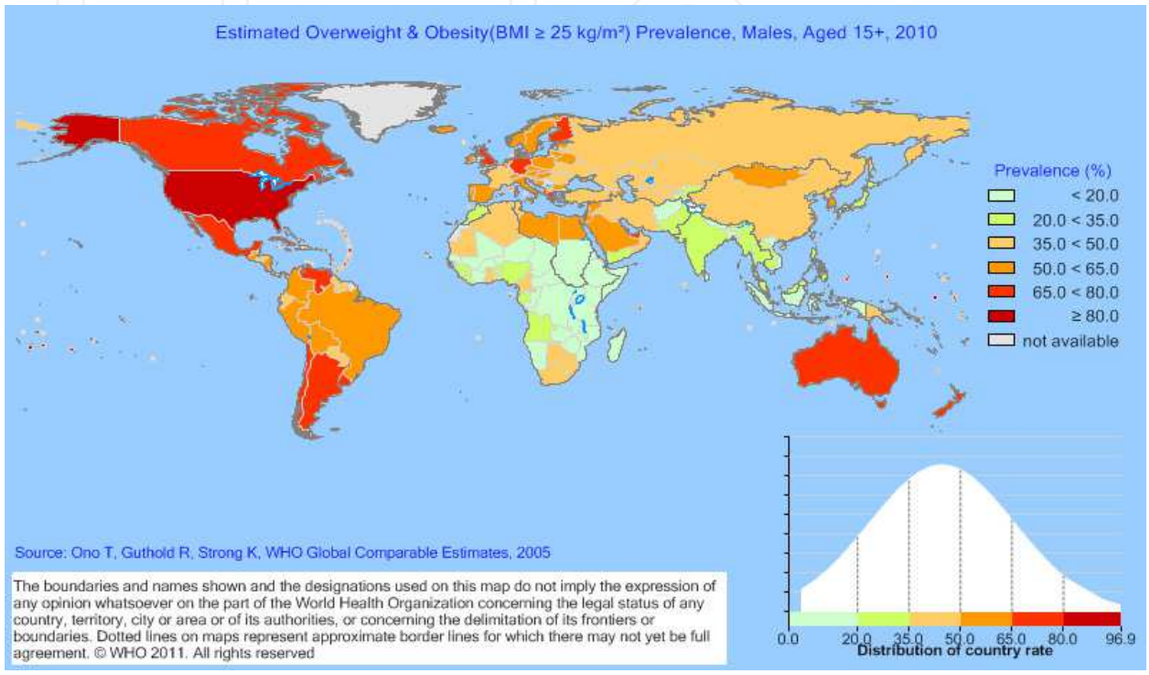

Fig. 3. Estimated Obesity Prevalence in Males, 15 years and over, 2010 (WHO, 2011b).

1.3 Health consequences of obesity

Overweight and obesity are responsible for 2.8 million adult deaths per year and add to the burden on the healthcare system, in particular, 44% of Diabetes Mellitus, 23% of Ischemic Heart Disease, and 7-41% of certain types of cancers (WHO, 2011a). Even more alarming is that childhood obesity puts the child at risk for disease in most of the body systems (see Figure 4) even before it predisposes the person to future adult obesity/ disability and premature death. As adiposity and age increase, if the child's body mass does not normalize, the obesity will track into adolescence and then adulthood (Biro, 2010). This may result in cardiovascular effects (raised systolic blood pressure and structural cardiac enlargement) (Logue & Sattar, 2011) and a potentially life threatening event. Interestingly, it was found that being an obese adolescent increased one's risk for adult morbidities, even if the person achieved normal weight during adulthood (Biro, 2010).

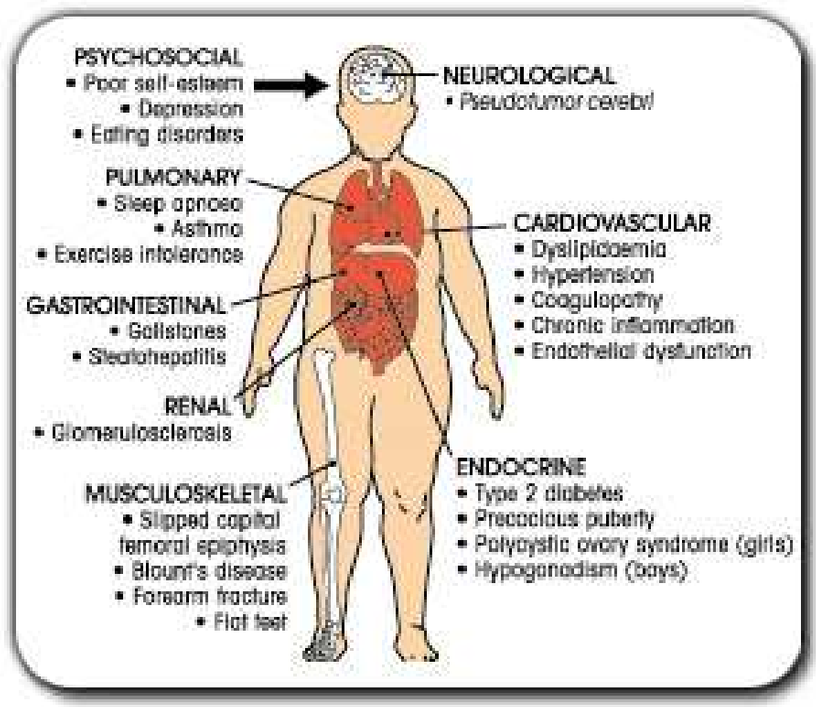

Fig. 4. Complications of childhood obesity (Ebbeling et al., 2002).

There is extensive evidence to suggest that obesity in children adversely affects most body systems, as outlined in the following sections.

1.3.1 Endocrine

Even though the definition of **Type 2 Diabetes Mellitus (DM**) has recently been updated to include an elevated glycated haemoglobin of at least 6.5 (Abrams & Levitt Katz, 2011), obese children are being treated for ketoacidosis as a result of an inadequate insulin response and before receiving a diagnosis of DM (Abrams & Levitt Katz, 2011; Stamatakis et al., 2010). When you add central abdominal obesity, hypertension, and dyslipidemia (lowered High Density Lipoproteins and raised triglycerides) to DM, a **metabolic syndrome** is identified in the child (Nyberg et al., 2011; Philippi-Hohn, 2010). This has been recognized as a predictor for higher cardiovascular morbidity/mortality in adults (Abrams & Levitt Katz, 2011).

Leukocyte telomere lengths (LTL) are DNA-protein complexes, found at the end of linear chromosomes, that protect those chromosome ends from degradation. In adults, shortened LTLs are associated with cardiovascular disease, Type 2 DM, insulin resistance, impaired glucose tolerance, and hypertension. In a large case controlled study of LTLs in obese children, prepubescent and pubescent males and females (< 9 years of age) were found to have significantly shorter lengths, putting them at greater risk to develop these health issues during adulthood (Buxton et al., 2011).

Androgen (male) hormones are present in adipose tissue. In the presence of obesity, these levels increase leading to hyperandrogenism, a medical condition characterized by excessive production and/or secretion of androgens. In women, these high levels of the male hormone, along with irregular menstruation and obesity, can lead to polycystic ovarian syndrome, which can subsquently develop into female subfertility (Abrams & Levitt Katz, 2011).

1.3.2 Renal

Obesity is linked to **end-stage renal disease (ESRD)** and **chronic kidney disease (CKD)** (Savino et al., 2011). Obese children have shown a significant positive correlation between microalbuminemia and BMI (Savino et al., 2011), waist circumference, systolic and diastolic blood pressure (Savino et al., 2011), insulin resistance and fasting glucose level (Sanad & Gharib, 2011). Subjects who had microalbuminemia were found to have a lower High Density Lipoprotein (HDL) cholesterol, and higher Low Density Lipoprotein (LDL) cholesterol and triglycerides (Sanad & Gharib, 2011). This is significantly associated with **metabolic syndrome**. Metabolic syndrome, alone, is an independent risk factor for **ESRD** and **CKD** (Sanad & Gharib, 2011; Savino et al., 2011) in adults.

Nitrous oxide (NO) is an important modulator of renal function and structure and, when impaired, renal complications may ensue. An inverse relationship was seen between NO levels and obese children (Savino et al., 2011).

1.3.3 Cardiovascular

Excessive inflammation and oxidation, which are possible biochemical links between obesity and cardiovascular events in adults, have been found in young obese children. Additionally, left ventricular (LV) mass and hypertrophy, LV dysfunction, and a three-fold higher risk of early development of arterial hypertension have been found in obese children (Philippi-Hohne, 2010). These disorders predispose obese children to future cardiovascular disease (Oliver et al., 2010).

1.3.4 Gastrointestinal

Increased Gastrointestinal (GI) disturbances such as constipation, gastroesophageal reflux (Philippi-Hohne, 2010), Irritable Bowel Syndrome, and abdominal pain (Abrams & Levitt Katz, 2011) have been found in obese children. Also, in pediatric autopsies of overweight and obese children between the ages of 2-19 years, Schwimmer et al. (Abrams & Levitt Katz, 2011) found **non–alcoholic fatty livers** in the majority of cases. Twenty-three percent of those cases had progressed to steatohepatitis, which can develop into fibrosis and cirrhosis of the liver.

1.3.5 Pulmonary

With the exception of **adeno-tonsillar** involvement**,** significantly more obstructive apnea/hypopnea events per hour of sleep have been found in obese children (Abrams & Levitt Katz, 2011). This chronic nocturnal hypoxemia can lead to pulmonary hypertension (Philippi-Hohne, 2010), which is a precursor to lung damage. Researchers have found a significant correlation between obesity and current asthma among children and adolescents. This relationship was stronger in nonatopic (non-allergic) children as compared to atopic (allergic – positive response to at least 1 allergen) (Stamatakis et al., 2010; Visness et al., 2010).

1.3.6 Musculoskeletal

Orthopedic complications such as poor balance, pain (especially in knee joints), impaired mobility, lower extremity malignancy, and fractures requiring surgical interventions (Abrams & Levitt Katz, 2011) have all been documented in obese children.

1.3.7 Psychosocial

Interestingly, in countries considered the 'happiest' in the world, the rates of adult and childhood obesity are low (Goran & Ventura, 2011). People in Denmark, Sweden, Norway, Finland, and the Netherlands ranked their country high in meeting their psychological, social and basic needs. Childhood overweight and obesity were 17% (combined) in these countries. In Griffith et al.'s (2010) review of 42 studies, self-esteem and quality of life were significantly lower in obese youth. Physical competence, appearance, and social functioning were also compromised (Goran & Ventura, 2011). Lower self-esteem was also found in overweight and obese children (Cornette, 2008). Females who were very overweight reported lower self-esteem than females classified as moderately overweight (Cornette, 2008). Overweight/obese children were more likely to be victims and perpetrators of bullying than 'normal' weight children. Bullying experienced by overweight/obese children was generally name-calling related to their size; however, when the overweight/obese child was the bully, racial, color or religious slurs were used against the other person (Janssen et al., 2004). As well, a lower quality of life was experienced by obese children. Schwimmer et al. (2003) found that obese children rated their quality of life to be comparable with that of young cancer patients on chemotherapy. In particular, teasing at school, difficulties playing sports, fatigue, and sleep apnea greatly decreased the quality of life in these children.

1.3.8 Cancers

Although cancers do not develop until later in life, several are more prevalent in obese individuals when compared to their normal weight counterparts. Eesophageal, thyroid, colon, and renal cancers are more prevalent in obese men while endometrial, gall bladder, esophageal, adenocarcinoma, and renal cancers are more common in obese women (Biro, 2010).

2. Biology of breastmilk

In humans, early infant nutrition appears to be critically important in determining long-term positive or negative health effects. A complex fluid, breastmilk is the optimal source of nutrition for infants and appears to be a crucial factor in determining health and preventing chronic disease. The following section reviews the process of breastmilk production, the protective components of breastmilk purported to be linked with the prevention of childhood obesity, and the relationship with metabolic programming. It also contrasts breastmilk and breastfeeding with infant formula and associated methods of feeding.

2.1 Process of production

During pregnancy, the breast prepares anatomically and physiologically for infant feeding. Lactogenesis is the the production of human milk and occurs due to a complex interplay of pituitary, ovarian, thyroid, adrenal, and pancreatic hormones. Lactogenesis occurs in stages. The initiation of lactogenesis results from a fall in plasma progesterone and high prolactin levels during the post-partum period. Lactogenesis stage 1 occurs in the latter stage of pregnancy. The breast produces thick yellow colostrum and at this stage high progesterone levels inhibit milk production. Lactogenesis 2 occurs at or soon after birth progesterone

levels drop and prolactin levels remain high. This results in milk production. Upon stimulation of the breast, prolactin levels in the blood rise, peak in about 45 minutes, and return to the pre-breastfeeding state about three hours later. The release of prolactin triggers the cells in the alveoli to make milk. During lactogenesis stage 3, the more that milk is removed from the breasts, the more the breast will produce milk. Milk supply depends strongly on how often the baby feeds and how well it is able to transfer milk from the breast. At two-three days post-partum, when mothers usually feel their milk "coming in", secretion of milk rapidly increases and major changes in milk composition occur over the next ten days. The changes over this period subsequently result in the establishment of mature milk (Lawrence, 1999). The volume of milk increases gradually, starting at about 100ml/day increasing to about 600 ml/day by day four (Lawrence, 1999) adapting to the new infant's energy needs.

2.2 Nutritional components and properties

Human milk is not a uniform body fluid but a heterogeneous secretion of the mammary gland. Its composition is frequently changing throughout the day and throughout feedings. Breastmilk contains many bioactive factors (lactoferrin, oligosaccharides, long-chain polyunsaturated fatty acids, glycoproteins and antibodies) that do not function primarily as nutrients but may control nutrient use or play a role in regulating metabolic pathways(Labbok et al., 2004). Breastmilk also contains other biologically active factors, which include hormones, growth factors and cytokines. These are all involved in energy balance regulation and seem to be important for infant nutrition and growth(Savino et al., 2009).

Milk composition changes during a feeding period, as well as throughout the day. Concentrations of protein, fat, carbohydrates, and minerals change, in addition to osmolarity and pH. There are more than 200 components of human milk which include an assortment of several molecules, many of which have yet to be specifically determined. There are differences in foremilk and hindmilk, and colostrum differs from transitional and mature milks.

Colostrum is the first mammary secretion of lactation, and consists of a thick, yellowish fluid that is small in volume and is high in protein . Secretory immunoglobulin A (sIgA) is particularly plentiful and prominent. Transitional milk occurs next from approximately seven to ten days to two weeks postpartum. During this time the concentration of immunoglobulins and total protein decreases whereas lactose, fat, and total caloric content increases. In mature milk, water is the largest constituent. The main fats in human milk are triacylglycerols, phospholipids, and their component fatty acids. Fat concentration changes depending on many different factors and conditions. Fat provides about 50 percent of the infant's calories. Human milk also consists of casein and lactalbumin (whey) proteins. The principal carbohydrate in human milk is lactose. Human milk also contains minerals, electrolytes, trace elements, vitamins, enzymes, hormones, bile salts, and growth factors(Lawrence, 1999).

Other important compounds in breast milk are leptin, ghrelin, adiponectin, resistin. Synthesis of leptin occurs in adipose tissue (Zhang et al., 1994). Leptin production occurs in mammary glands, and epithelial cells secrete leptin in milk fat globules (Smith-Kirwin, 1998). Milk-borne leptin has been implicated not only in growth, but also in short-term appetite regulation in infancy, especially during lactation(Savino et al., 2009). Leptin in breastmilk could exert a long-term effect on energy balance and body weight composition(Savino et al., 2009).

Ghrelin is a protein produced primarily by the stomach, involved in both short-term regulation of feeding and the long-term regulation of weight and energy metabolism(Savino et al., 2009). It has many endocrine and non-endocrine functions, including energy balance regulation (Hellstrom, 2009). As a component of breastmilk, ghrelin plays a role in both short-term regulation of food intake (by stimulating appetite) and in long-term body-weight reduction (by inducing adiposity). Considering these effects, ghrelin could be one of the factors through which breastfeeding may influence infant behavior and body composition(Savino et al., 2009).

Adiponectin is the most abundant adipose-specific protein. It has multiple identified functions(Scherer et al., 1997) and a reduction in its expression is associated with insulin resistance(Savino et al., 1997). It has been shown that adiponectin levels correlate negatively with the degree of adiposity in children between five and ten years of age(Nader et al., 2002). It is present in human milk(Martin et al., 2006) and may influence infant growth and development.

Resistin is a cytokine that is secreted by adipocytes(Steppan, 2001). It is expressed in the human placenta and may play a role in the regulation of energy metabolism during pregnancy. It has also been identified in human milk(Ilcol et al., 2008) and may be important for the metabolic development of infants(Savino et al., 1997).

2.3 Learned self-regulation

One hypothesis regarding the association of breastfeeding and obesity prevention relates to the ability of breastfed infants to self-regulate their energy intake to match their energy needs (Owen et al., 2005). Infants fed directly at the breast must actively suckle to draw milk out, whereas infants are passive when being fed from a bottle. The control of caregivers in bottle-feeding could lead to infants' poor self-regulation on the basis of internal cues of hunger and satiety. Infants who are bottle-fed in early infancy are more likely to empty the bottle or cup in late infancy than those who are fed directly at the breast. Bottle-feeding, regardless of the type of milk, is distinct from feeding at the breast in its effect on infants' self-regulation of milk intake (Fein & Grummer-Strawn, 2010).

Infants who are fed from the breast can control milk intake, because they decide when to start and when to stop sucking. Mothers who breastfeed often develop a feeding style that is less controlling, thereby allowing their infants to maintain their natural ability to regulate their energy intake (Taveras et al., 2006). Also, infants fed at the breast need to suck nonnutritively until the milk-ejection reflex occurs, and it is known that as sucking pressure decreases, the duration of each suck lengthens, and suckling frequency decreases from nonnutritive sucking (NNS) to nutritive sucking (NS) (Richard & Alade, 1992). This transition from NNS to NS may play an important role in establishing infants' self-regulation of milk intake (Mizuno & Ueda, 2006).

The taste and smell of breastmilk changes from meal to meal, depending on what the mother has consumed, which exposes the breastfed infant to a wider array of sensory experiences than the formula- fed infant. It is possible that the breastfed infant may be programmed to accept a different food selection which may affect dietary habits later in life(Koletzko, 2009). These factors regarding taste and smell remain constant during bottle-feeding. For example, breast milk fat content toward the end of the feeding episode is much higher than that at the start of the feeding, which might signal to the infant that the feeding episode is coming to an end. In contrast, bottle-fed infants are not exposed to such

"physiologic signaling" during the feeding episode. Research on regulation of food intake by chemosensory receptors suggests that bottle-fed infants who had no exposure to the varied flavors of breast milk may miss the important oropharyngeal sensory experience that is needed for the development of physiologic regulation of food intake later in life (Poothullil, 1995).

2.4 Differences from formula

Infant formula tends to have a higher average caloric density compared to breast milk with higher energy supplies per kilogram of bodyweight (Heinig et al., 1993). It has been suggested that a higher protein intake in formula-fed infants could increase the risk of obesity later in life by causing an earlier adiposity rebound(Taylor et al., 2005). Adiposity rebound refers to the period of increasing body mass index after the early childhood nadir, usually at about 6 years old (Medlexicon International Limited, 2011).

Also, a higher protein intake during infancy may promote the stimulation of insulin release and the programming of higher long-term insulin concentrations. Consistent with this occurence, formula-fed infants have been found to have higher plasma insulin levels and insulin-like growth factor 1, than those who were breastfed(Lucas et al., 1981). The growth acceleration hypothesis suggests that the protective benefits of breastfeeding on the development of long-term obesity may be due to a slower pattern of growth in breastfed infants when compared with formula-fed infants(Singhal & Lucas, 2004).

3. Breastfeeding as a prevention strategy for childhood obesity

It has been suggested that the choice of early infant feeding method may impact the growth trajectory of an infant and how that infant develops through childhood, adolescence and into adulthood. A large number of primary studies and several systematic reviews and meta-analyses have compared breastfeeding to formula feeding and the subsequent development of overweight and/or obesity overtime. Many of these studies have reported an inverse relationship between breastfeeding and childhood obesity. The prevalence of childhood obesity continues to increase in most developed countries and there is evidence it tracks through the life cycle. As there are a very limited number of effective treatment options available for either obese children or adults, the emphasis must be put on identifying, evaluating and implementing effective methods of primary prevention for obesity.

Due to the nature of the relationship and the inability to feasibly or ethically assign infant feeding choice via a randomization process, researchers have had to rely on observational studies to provide empirical evidence for what may be a causal protective relationship. The previous section described the plausible mechanisms by which breastfeeding can influence early growth and development. The final section in this chapter will summarize the systematic reviews and meta-analyses on breastfeeding and obesity conducted over the last 10 years and provide some thoughts on future research.

The protective effect of breastfeeding against childhood obesity was initially proposed by Kramer in 1981 (Kramer, 1981) and since then numerous studies have been conducted in order to examine the relationship between breastfeeding and childhood overweight and obesity (Twells & Newhook, 2010; Bergmann et al., 2003; Hediger et al., 2001, Buyken et al., 2008; Dewey, 1998; Kramer et al., 2007; Metzger & McDade, 2010; Savino et al., 2009; Singhal

& Lanigan, 2007; Victora et al., 1998; Araujo et al., 2006; Burdette et al., 2006; Gummer-Strawn & Mei, 2004). More recently, a number of systematic reviews and meta-analyses on this topic have been conducted (Arenz et al., 2004; Harder et al., 2005; Owen et al., 2005; Owen et al., 2005, Horta et al., 2007). Four of these reviews suggest that breastfeeding provides a small but significant protective effect (Arnez et al., 2004; Harder et al., 2005, Owen et al., 2005, Horta et al., 2007) against the development of overweight and/or obesity while one study suggests there is no effect of breastfeeding on differences in mean BMI between groups that were breastfed compared to those formula- fed (Owen et al., 2005).

3.1 Evidence from observational studies

The first systematic review and meta-analysis on the association of breastfeeding and childhood obesity was conducted by Arenz et al. in 2004. This review included studies published in English, French, Italian, Spanish and German from 1966 to December 2003. The researchers examined the relationship between breastfeeding and childhood obesity in children at least one year of age. Obesity was defined as a BMI greater than the 90th, 95th or 97th percentile(s). Only studies that adjusted for at least three potential confounding or interacting factors (e.g., birth weight, parental overweight, parental smoking, dietary factors, physical activity and socioeconomic status (SES) or parental education) and reported an odds ratio (OR) or relative risk (RR) with the last follow-up between 5 and 18 years of age were included in the meta-analysis. Nine of 28 eligible studies met the inclusion criteria including two prospective cohort studies and seven cross-sectional studies totalling more than 69,000 children from developed countries.

The meta-analysis used both fixed and random effects models and pooled crude and adjusted odds ratios (AOR) from the individual studies. Definitions of breastfeeding and other infant feeding methods were not always consistent across studies, especially the definition of exclusive breastfeeding. Sensitivity analyses were carried out to identify potential sources of heterogeneity by testing the strength of the findings stratified by study design, exposure ascertainment and the selection of study participants.

The combined crude odds ratio comparing any breastfeeding to no breastfeeding was calculated for six of the nine studies and was 0.67 (95%CI 0.62-0.73). The AOR calculated for the nine studies was 0.78 (95% CI 0.71-0.85) for both fixed and random effects models. The protective effect of breastfeeding was more pronounced in studies with adjustment for less than seven potential confounding factors (AOR 0.69 95%CI 0.59-0.81) compared to adjustment for seven or more potential confounding factors (AOR 0.78 95%CI 0.70-0.87). The evidence for duration of breastfeeding was inconsistent. Four studies demonstrated an inverse association between breastfeeding duration and the prevalence of obesity in the crude and adjusted analysis. One study reported a dose-response relationship in the crude analysis that lost significance after adjustment. Three studies reported no significant effect of duration of breastfeeding.

These results suggest a significant protective effect of breastfeeding against obesity. Although there was no indication of heterogeneity between the individual studies and several confounders were adjusted for in the analysis, there was some evidence of publication bias with smaller studies tending to report higher protection against obesity. Residual confounding could not be ruled out.

The second systematic review and meta-analysis was published by Owen et al. in 2005 who also published another meta-analysis in the same year. The first of Owen et al.'s two meta-

analyses included studies from the same time period as Arenz et al., (1966-2003) but involved a broader search strategy. The objective was to examine the relationship between breastfeeding and obesity assessed at any age. The definition of obesity was flexible, although most studies used a cut-off of >95th or 97th percentiles. Only those studies that reported crude OR's were included. Of 61 studies reviewed, 28 provided 29 AOR's including a total of 298,900 subjects aged 0.5-33 years of age. A fixed effects model was used and meta regression and sensitivity analysis examined the influence of a number of confounding factors that included parental body size, SES, and maternal smoking.

Twenty-eight of the 29 studies reported that breastfeeding was associated with a lower risk of obesity including four estimates for infants, 23 for children and two for adults. The fixed effects model indicated that breastfed infants were less likely to be obese than those formula-fed: OR 0.87 (95% CI 0.85-0.89). Six studies allowed for the adjustment of the following confounders: SES, parental BMI, and current maternal smoking or maternal smoking in early life. The pooled OR in these studies was reduced but breastfeeding remained a significant protective factor. Adjusting for birth weight in ten studies had no real effect on the OR and there was no clear evidence that the protective effect of breastfeeding changed with increasing age of outcome assessment.

In 14 studies that provided data on breastfeeding duration, the protective effect of breastfeeding over formula-feeding was greater among subjects breastfed for at least 2 months OR 0.81 (95% CI 0.77-0.84), compared with those never breastfed. The smaller studies reported a greater protective effect (OR 0.43 95% CI 0.33–0.55) and the larger studies reported a less protective effect (OR 0.88 95%CI 0.86–0.90) providing some evidence for publication bias and selective reporting. There was evidence of heterogeneity among the studies ($p<.001$) and residual confounding could not be ruled out.

Although the results demonstrated a protective effect of breastfeeding against the development of later obesity, the authors suggested that a protective effect may be due to confounding by SES and parental body composition and suggested that a further review was required that included large unpublished studies (the subject of the second Meta-analysis by Owen et al).

Harder et al. (2005), published a third systematic review and meta-analysis in the same year. Unlike the previous two reviews, Harder et al., attempted to assess the effect of *duration* of breastfeeding on the risk of overweight in order to examine whether a dose-response relationship existed. Study eligibility included any original report comparing breastfed subjects with exclusively formula-fed subjects at any age. Studies were included if the findings used OR's and/or contained data for the calculation of an OR for the risk of overweight with reference to feeding history. In addition information on the *duration* of breastfeeding had to be reported. All definitions of overweight and obesity were included. At risk of overweight was defined most often as BMI > than the 85th and >90th percentiles and obesity was defined most often as BMI > 90th, 95th and 97th percentiles.

The final analysis included 17 studies published between 1966 and December 2003 including 120,831 subjects from Great Britain, United States, Canada, Germany, Australia, New Zealand and Czechoslovakia. Fourteen studies provided results on more than one category of breastfeeding duration.

Using a random effects model, the duration of breastfeeding was inversely associated with the risk of overweight: OR 0.94 (95% CI 0.89-0.98). The odds of overweight were reduced by 6% per month of breastfeeding. In addition, a pooled OR for overweight calculated for five time

periods (<1, 1-3, 4-6, 7-9 and >9months) indicated that the risk of overweight continued to decrease by breastfeeding duration reaching a plateau at 9 months. For nine months or more of breastfeeding the OR was 0.68 (95%CI 0.50-0.91). A supplementary trend analysis of 11 studies demonstrated that a dose-response relationship existed between breastfeeding duration and the risk of overweight with an OR of 0.96(95%CI 0.94-0.98) per month of breastfeeding.

The age at examination had little influence on the effect of breastfeeding duration on the risk of overweight. The pooled OR from five studies that investigated subjects up to five years of age was 0.97 (95% CI 0.94-0.99) while for six studies on subjects six years of age or older, was 0.96 (95% CI 0.93-0.99). A sub-group analysis demonstrated that varying definitions of overweight and obesity influenced the estimate of the OR only marginally.

The authors reported that the duration of breastfeeding was inversely and linearly associated with the risk of overweight. In addiition the risk of overweight was reduced by 4% for each month of breastfeeding up to a duration of breastfeeding of 9 months. This finding was independent of the definition of overweight and obese and age at follow-up, suggesting a dose-response relationship. The authors concluded that although the result was relatively small in size, the association, if causal, may be an important factor from a population health perspective. There was no evidence of publication bias.

A critique of this meta-analysis was published as a letter (Quigley, 2006) suggesting a number of methodological concerns existed that included: non-consideration of ethnic background or SES as confounders, inconsistent definitions of both breastfeeding and overweight/obesity, and the reliance on crude OR's. A response was published by the authors (Harder & Plagemann, 2006) suggesting that irrespective of how the subgroups were defined as overweight, the pooled adjusted OR's did not differ from the unadjusted OR inferring a high stability of effect size despite heterogeneity in exposure and/or outcome definition, thus strengthening even more the validity and conclusions of the study.

In 2007, the USA Department of Health and Human Services Office on Women's Health requested that the Agency for Healthcare Research and Quality (AHRQ) summarize the literature and report on the relationship of breastfeeding and various infant and maternal health outcomes including childhood obesity (Ip et al., 2007). Due to restrictions of resources and time, the AHRQ relied primairly on a review of the exiting systematic reviews and meta-analyses, and based on a scoring system (discussed below), provided a grade for each review that assessed the strength of methodological rigor. A, B or C grades were given. Studies given an A grade (good) were described as those having the least bias and consequently results that were considered valid. A grade A study was considered high in quality in that it included; a rigorous systematic review and/or meta-analysis; a clear description of the population, setting, interventions and comparison groups; a clear description of the content of the comparison groups; appropriate measurement of outcomes; appropriate statistical assumptions and analytic methods. In addition the study authors demonstrated; appropriate consideration and adjustment for potential confounders; rigorous assessment of individual study quality; no reporting errors; and well-reasoned conclusions based on the data reported.

Studies given a B grade (fair/moderate**)** were considered susceptible to some bias, but not sufficient to invalidate the results. B grade studies did not meet all the criteria in category A because of some deficiencies. Grade B studies for example may have demonstrated suboptimal adjustment for potential confounders and be missing information, making it difficult to assess limitations and potential problems.

Studies given a C grade (poor) had significant biases that could invalidate the results. The study either did not consider potential confounders or adjust for them properly. Grade C studies had serious errors in design and analysis and had large amounts of missing information and discrepancies in reporting.

The AHRQ scheme for grading reviews was supplemented with the MOOSE guideline (Meta-analysis of Observational Studies in Epidemiology) and an additional checklist of items that were used to evaluate the quality of the systematic reviews specific to observational studies (Stroup et al., 2000; Ip et al., 2007).

Based on the search criteria by the AHRQ, the meta-analyses by Arenz, Harder and the first of two by Owen were reviewed. After careful appraisal and evaluation Arenz et al., was given an A grade and both the meta-analyses by Harder et al. and Owen et al. were given B grades, primarily due to the suboptimal consideration for potential confounding.

The fourth and second meta-analysis by Owen et al. was also published in 2005 and included both published studies from the previous meta-analysis and unpublished data. In contrast to the previous analyses, studies were included that examined the influence of infant feeding on obesity measured as *mean BMI* throughout life from 6 weeks after birth. Observational studies of cross-sectional and longitudinal cohort design were included and case-control studies that could not provide reliable data for comparisons based on mean BMI were excluded. Seventy studies were reviewed providing a total of 414,750 subjects in the age range from 1 to 70 years of age. Mean differences in BMI were required for analysis.

Using a fixed effects model, the mean difference in BMI between those initially breastfed and those formula-fed was examined. Meta-regression was used to examine: influence of study size, quintiles of age at outcome measurement, year of birth and the collection method of infant feeding status on the study outcome. Meta-regression and sensitivity analyses were also used to examine the influence of exclusive feeding, or duration of breastfeeding adjusting for study size.

In this meta-analysis, breastfeeding was associated with a slightly lower BMI compared with formula- feeding; mean difference -0.04 (95%CI -0.05, -0.02). The mean difference was larger for small studies: -0.12 (95%CI -0.29, 0.04) and smaller for larger studies: mean difference -0.03 (95%CI -0.04,-0.01) suggesting some publication bias. If the meta-analysis was restricted to 11 studies that adjusted for age, SES, maternal smoking and maternal BMI, the mean difference was reduced to non-significance between the groups from a mean difference of -0.12 (95% CI: -0.16, -0.08) to a mean difference of -0.01 (95%CI -0.05,0.03) demonstrating both the impact and the need to adjust for relevant confounders.

The authors concluded that the potential effect of breastfeeding on mean BMI was small and non-significant after controlling for confounding factors. Differences in BMI may be strongly influenced by publication bias. Therefore in contrast to the three previous meta-analyses, Owen et al. concluded that the promotion of breastfeeding although important for other reasons was not likely to reduce mean BMI.

In 2007, the WHO published a report summarizing the literature on the "Evidence of the Long-Term Effects of Breastfeeding: Systematic Reviews and Meta-Analysis" (http://www.ahrq.gov/downloads/pub/evidence/pdf/brfout/brfout.pdf) conducted by Horta et al. This report included a section on the long-term effects of breastfeeding on obesity. Previous published meta-analyses were briefly summarized and a new meta-analysis was conducted that considered all the papers included in previously published meta-analyses, and those papers newly identified by two independent literature searches at

the WHO. The analysis included 33 studies providing 39 estimates on the effect of breastfeeding on the prevalence of overweight/obesity with follow-up from 1 to 66 years of age. In a random effects model, breastfed individuals were less likely to be considered overweight/obese OR 0.78 (95% CI 0.72-0.84). In spite of the evidence of publication bias, a protective effect of breastfeeding was observed among the larger studies (>1500 participants). Studies that controlled for SES and parental weight reported that breastfeeding was associated with a lower prevalence of obesity. Therefore, according to Horta et al., the evidence suggested that breastfeeding may have a *small protective effect* on the prevalence of obesity.

In 2008, Cope and Allison published a critical review of the WHO report (Cope & Allison, 2008). The authors argued that due to the limitations of the empirical evidence currently available and the major shortcomings of these studies (e.g., publication bias, confounding, residual confounding, self-selection) there was insufficient evidence at this time to suggest that breastfeeding protects against either obesity in childhood or adulthood. The authors questioned the overall conclusion of the WHO report and concluded that "while breastfeeding may have benefits beyond any putative protection against obesity, and benefits of breastfeeding most likely outweigh any harms, any statement that a strong, clear or consistent body of evidence shows that breastfeeding causally reduces the risk of overweight or obesity is unwarranted at this time" (pp.594).

In summary, four of the five meta-analyses published in the last 10 years demonstrate a modest protective effect of breastfeeding against the risk of overweight and obesity. These analyses conducted by different groups of investigators using different inclusion criteria and varying methodologies demonstrated independently a significant association. The findings have remained consistent despite the different settings and populations studied, the use of crude and adjusted data and the varying definitions of infant feeding and overweight. In addition the evidence of a dose-response relationship was observed in some analyses. As a result, there is evidence to support a causal protective relationship between breastfeeding and later risk of overweight; however these conclusions must be placed in the context of the limitations of the study designs currently being used to examine this relationship. There are several weaknesses that limit the validity of meta-analyses of observational studies on the association between breastfeeding and risk of overweight and obesity. Tthese include: publication bias, heterogeneity between studies, the lack of adjustment of confounders and issues of residual confounding. Breastfeeding is associated with many other factors that can influence a child's weight status (e.g., maternal BMI, education, and smoking during pregnancy) and other family lifestyle habits that may be difficult to assess and adjust for in non-experimental study designs. It must be acknowledged that failing to limit the impact of these factors may result in spurious conclusions. (Berylein & Von Kries, 2011).

3.2 Randomized controlled trials

The doubled-blinded randomized controlled trail (RCT) is the gold standard in study design for minimizing biases and increasing the internal validity of the research (Sackett et al., 1991). Although interventional or experimental studies based on random assignment of the mother–infant pair to infant meeting method (breastfeeding or formula-feeding) would provide strong evidence for the association between breastfeeding and risk of overweight/obesity, they are for obvious reasons not feasible or ethical. However,

one large RCT conducted in Belarus in 1996-1997 randomized mother-infant pairs to a breastfeeding promotion intervention(Kramer et al., 2001). The Promotion of Breastfeeding Intervention Trial (PROBIT) was a cluster-randomized trial in which a total of 16,491 healthy mothers of full-term singletons with birth weights ≥ 2500g were randomly assigned to a breastfeeding promotion intervention and followed for 12 months. A total of 13,889 children were followed up at 6.5 years. The intervention based on the Baby Friendly Hospital Initiative (BFHI) and developed by the World Health Organization and the United Nations Children's fund was to promote and support breastfeeding, especially in mothers who have chosen to initiate breastfeeding. The control maternity hospitals and polyclinics continued the practices and policies in effect at the time of randomization (Kramer et al., 2001).

The intervention was successful in increasing the breastfeeding rates in the intervention group compared to the control group (43.3% versus 6.4%, $P < 0.001$), duration and exclusivity of breastfeeding; however evidence from the PROBIT study failed to demonstrate an effect of a breastfeeding promotion intervention on children's BMI. For children followed up at 6.5 years, there were no significant differences in mean BMI (i.e., 15.6kg/m^2 in both arms of the trial) or in the prevalence of overweight; OR 1.1(95%CI 0.8-1.4) or obesity; OR 1.2 (95%CI 0.8-1.6) between the two groups. (Kramer et al., 2007). The authors concluded that the PROBIT trial did not provide conclusive evidence for or against a potential effect of breastfeeding on body composition.

There have been a number of limitations of the PROBIT trial discussed regarding its ability to draw conclusions on the relationship between breastfeeding and childhood overweight and obesity. First, the study was not designed to study childhood overweight/obesity directly but rather to study the effects of the BFHI on breastfeeding exclusivity and duration. It may be that the study had insufficient statistical power to assess the outcome of childhood obesity. Second, only women who initiated breastfeeding were enrolled in the study and therefore there was no comparison group of formula-feeding mothers, therefore a self-selection bias existed. Third, the authors examined mean BMI and differences in mean BMI between the intervention and control groups but did not examine any changes in percentage overweight or obese (Berylein, 2011). If breastfeeding is affecting mainly the tail end of the distribution and not the mean BMI as has been suggested it might have been useful to examine the percentages of overweight and obese in both groups(Grummer-Strawn, 2004).

3.3 Future research

Observational studies will remain the primary study design used to collect information on the relationship between breastfeeding and obesity. As has been discussed, challenges arise in that observational or non-experimental studies (i.e., cohort, case-control and cross--sectional) are inherently biased with issues of misclassification of exposure (i.e., self-reported breastfeeding duration), confounding (i.e., mothers/family lifestyle, SES, maternal smoking), residual confounding and self–selection that are difficult to fully adjust for in analysis. However, the validity and reliability of these studies can be improved significantly by: defining careful subject selection criteria; using common and consistent definitions of infant feeding especially that of "exclusive" breastfeeding; ensuring the reliable collection of duration of infant feeding data by trained research personnel; guaranteeing the use of standard cut-off criteria for measuring childhood overweight and obesity such as the WHO criteria (WHO Multicentre Growth Reference Study Group, 2006) as well as blinding

assessment of the outcome. A well-designed observational study should provide high quality evidence upon which to draw valid conclusions.

In the context of these limiations, future prospective cohort studies may attempt to include, if data is available, a sibling analysis that provides an opportunity to control for hereditary and confounding factors (e.g., household/family/environmental factors) that are often difficult to adjust for in other studies (Metzger & McDade, 2010). In addition although RCT's are unlikely to be conducted in this area, there is the opportunity to randomize and evaluate the effectiveness of breastfeeding promotion interventions such as the one conducted in Belarus. A well-designed and performed RCT that includes randomization, allocation concealment, clear definitions of breastfeeding exposure compared with non-breastfeeding, and blinded assessment of outcomes could provide the best evidence in supporting the causality of breast milk in affecting the risk overweight and obesity. Analysing the differences in the degree of breastfeeding between the two groups as a result of the intervention will provide researchers with the opportunity to investigate differences in health outcomes such as the development of childhood overweight or obesity between the two groups. This may be the strongest study design available assuming the study is sufficiently powered to evaluate this secondary outcome.

3.4 Conclusion

In conclusion, the prevalence of childhood obesity has increased dramatically in many developed countries over the last few decades and its association with increased morbidity and mortality is of great public health concern. The tracking of childhood obesity and the lack of available effective treatment options suggest that areas of primary prevention must be the focus if we want to curb and reduce this epidemic. Although there are many limitations of the type of study design currently used to explore this relationship, the research evidence that does exist supports a modest but significant protective effect of breastfeeding against the development of obesity with some evidence of a dose-response relationship. It is recognized that breastfeeding is only one of many factors that impact body composition, but as the many benefits of breastfeeding are well-evidenced, continuing to promote and encourage breastfeeding may have the additional benefit of helping to protect against the obesity epidemic.

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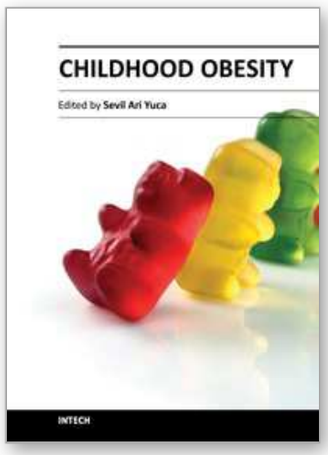

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This book aims to provide readers with a general as well as an advanced overview of the key trends in childhood obesity. Obesity is an illness that occurs due to a combination of genetic, environmental, psychosocial, metabolic and hormonal factors. The prevalence of obesity has shown a great rise both in adults and children in the last 30 years. It is known that one third of children who are obese in childhood and 80% of adolescents who are obese in their adolescent years continue to be obese later in life. Obesity is an important risk factor in serious illnesses such as heart disease, hyperlipidemia, hyperinsulinemia, hypertension and early atherosclerosis.

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