The contribution of infant food marketing to the obesogenic environment in Australia

Julie Smith BA, BEd (Hons), PhD

ABSTRACT

Obesity has been growing rapidly among both children and adult Australians in recent decades, raising concern at the associated chronic disease burden, and generating debate over the extent of individual versus government responsibility. This paper briefly reviews recent scientific evidence on links between poor early life nutrition and obesity in later life, which suggests that artificial baby milk rather than breastfeeding in infancy is associated with a 30-50% higher likelihood of later life obesity. It then presents data on long-term trends in breastfeeding in Australia and on consumption of Infant milk products since 1939.

Evidence is also presented of increased marketing and promotion of breastmilk substitutes from the mid 1980s, including through the healthcare system, associated with the emergence of increased competition in the Australian infant food industry. This collaborative marketing effort by industry and health professionals in turn contributed importantly to the sharp decline in breastfeeding from the mid 1980s. As a consequence, most Australians born since 1985 were exposed to artificial baby milk in early infancy. A substantial proportion of Australian infants are still partially fed with artificial baby milk in the first 12 months of life.

The example of infant food highlights that the healthcare system and the food industry, and not just individual mothers’ choices, have contributed to poor infant nutrition and obesity trends in Australia. Redressing healthcare system and industry practices to restore a supportive environment for breastfeeding is thus argued to be a necessary element of the public health response to the current obesity problem.

INTRODUCTION

The proportion of adult Australians who are overweight or obese has doubled since 1980 (Australian Institute of Health and Welfare 2003a). The proportion of children who were overweight tripled between 1985 and 1995, and continues to rise (Australian Institute of Health and Welfare 2004). Rising rates of childhood and adult obesity have also become evident in other developed and developing countries since the early 1980s (World Health Organization 2000; Janssen, Katzmarzyk & Boyce 2005). Obesity will persist into adulthood for 50% of obese children or adolescents (Dietz 1998a) although adolescent obesity is more predictive of obesity in adult life than earlier childhood obesity (Whitaker et al 1997). The link between obesity and chronic illnesses such as diabetes and coronary heart disease also makes it of increasing public health concern (Dietz 1998b; Reilly et al 2003).

Responsibility for addressing the obesity problem remains contested. Commonwealth Parliamentary Secretary for Health and Aging, Christopher Pyne recently commented that “it is not the role of government to provide individuals with a healthy diet in a free society”, and cited breastfeeding as an area of ‘parental responsibility’, where individual women should be encouraged to play their part in the battle of the bulge (Pyne 2006). Likewise the Commonwealth Minister for Health and Aging Tony Abbott was reported as saying that “the only person responsible for what goes into my mouth is me, and the only people who are responsible for what goes into kids’ mouths are the parents” (Sydney Morning Herald 12 April 2006, p 4).

Others believe obesity is a reflection of the environment in which people live – a policy of exhorting citizens to adopt wiser lifestyles only makes sense if “people have the capacity and freedom to make wiser choices” (Leichter 2003). This view emphasizes an ‘ecological’ explanation for rising obesity – a perspective which argues that the social, physical and market environment in which we live makes healthy eating and adequate exercise into difficult choices, encouraging an unhealthy lifestyle (Fjeller & Swinburn 1997).

This paper aims to illustrate the likely links between the current increase in obesity and long-term trends in increased artificial milk feeding of infants during the past sixty years, and to show that environmental factors, including the healthcare system and commercial marketing practices, have been important in shaping major change in infant feeding practices since the mid 1980s. This change in infant feeding practices is likely to have contributed to the current obesity problem, emphasizing that a view of obesity as solely an
individual or parental responsibility cannot reasonably be sustained.

METHODS AND DATA SOURCES
This paper briefly reviews the epidemiological literature on the links between artificial feeding and later life obesity, and summarizes possible mechanisms for this association. Results for recent meta-analyses and systematic reviews identified from a PubMed search are reported. Keywords included breastfeeding, infant feeding and obesity. The public policy framework on obesity and breastfeeding is summarised to illustrate the growing consensus of public health experts on links between infant feeding practices and the growing problem of obesity.

Long-term trends in infant feeding in Australia have been compiled from historical data sets on breastfeeding and Australian Bureau of Statistics publications on apparent consumption of infant milk products. Victorian infant health clinic data on rates of "full" breastfeeding provide the main information on Australian breastfeeding trends for the period from 1927 to the present (Mein-Smith 1991; Mortensen 2001). Full breastfeeding is defined as excluding artificial baby milk but not excluding solid foods and drinks (Mortensen 2001). Breastfeeding initiation (at first clinic visit) is also presented for Victoria from the 1990s. Time series data on artificial baby milk consumption between 1939 and 1999 is compiled from annual Australian Bureau of Statistics publications on "apparent consumption of foodsuiffs" (Australian Bureau of Statistics 1939–1998, 2000). This data was published until 1999 for milk products: categories included those mainly used for artificial feeding of infants - 'infant and invalids food', 'condensed, concentrated, evaporated milk', and 'powdered milk'. 'Invalids' included mothers who were encouraged to drink milk based dietary supplements, including artificial baby milks, supposedly to increase their milk supply (Thorley 2002). These trends in infant feeding practices are also related to information obtained from historical studies of infant feeding and maternity care practices in Australia, and to evidence on how hospital and maternity care practices hinder or support breastfeeding mothers.

Data on long term trends in print media advertising of commercial infant milk products is presented to show how this corresponds to trends in breastfeeding and infant milk product consumption. Commercial infant milk product advertising for the period 1950–1985 was examined to identify the number of advertisements in various categories and trends over time. Data on the advertising of artificial baby milk was collected through systematic sampling of 203 issues of the Australian Women's Weekly (that is, all issues during the month of January for the period 1950–1980 plus all issues during May and September in every 5th year for the same period plus all issues during 1985) and 362 issues of the Medical Journal of Australia (that is, all issues in every 5th year for the period 1950–1980).

BREASTFEEDING AND OBESITY – WHAT ARE THE LINKS?
More than 25 years ago epidemiological research showed that artificially-fed infants had a higher incidence of becoming overweight and obese in adolescence (Kramer 1981). The precise magnitude of the relationship between infant nutrition and later life (childhood, adolescence and adulthood) obesity remains unclear because of weaknesses in research design and the possibility of residual confounding. It is difficult to demonstrate a relationship between breastfeeding and adult obesity because of the increasing number of variables that affect the risk of obesity with greater age (Dewey 2003). Dewey (2003) noted a tendency for breastfeeding in infancy to have stronger effects in adolescence than in early childhood, suggesting that early life feeding may have a programming effect that does not manifest itself until the preadolescent and adolescent growth spurt appears. Nevertheless, meta-analysis has shown that the association of obesity with artificial feeding in infancy remained regardless of whether obesity was measured in infancy, childhood or adulthood; the authors concluded that the protective effects of breastfeeding were independent of later-life diet and physical activity patterns (Owen et al 2005). Owen and co-workers (2005) concluded that only a substantial study showing what would be an impossibly large (that is, greater than 40%) protective effect of artificial feeding over breastfeeding could challenge current evidence that later life obesity is more significantly common in those who were artificially-fed as infants. Such effect sizes are not large compared to other clinically relevant factors such as parental overweight, but the effect could be of public health significance given the wide extent of childhood obesity and of artificial feeding (Dewey 2003).

Dewey (2003) showed that in studies showing significance, artificially-fed infants were 20–40% more likely to be obese (defined as body mass index (BMI) >95th percentile) than breastfed infants (adjusted odds ratio (OR) for breastfed infants during early childhood 0.70–0.84; OR for breastfed infants during late childhood adolescence 0.78–0.80). While there were plausible behavioral and biological mechanisms for the effect, this result might be explained by residual confounding variables such as child feeding practices and physical activity which are difficult to measure.

Arenz and co-workers (2004) performed a systematic review and meta-analysis of nine studies with almost 69 000 participants. This analysis found a small but consistent effect of breastfeeding on childhood obesity; there was a 28% higher risk among artificially-fed infants (pooled adjusted OR for breastfed infants 0.78) (Arenz et al 2004). Potential confounders of birth weight, maternal overweight, maternal smoking and socioeconomic status contributed to a difference between crude and adjusted odds ratios of up to 0.14 in individual studies. A subsequent review of 28 studies with 298 900 participants concluded that artificial feeding increased the risk of later life obesity by 15% compared to those who were initially breastfed (OR for breastfed infants 0.85) (Owen et al 2005). However, when this data was adjusted for birth weight and maternal factors such as BMI and socioeconomic status, the association between artificial feeding and increased risk of later obesity was considerably weakened (pooled adjusted OR for breastfed infants 0.93) (Owen et al 2005).
Causality is difficult to prove in observational studies and there is often uncertainty about whether associations are causal, or whether residual confounding explains some or all of the effect. However, a dose-responsive relationship suggests causation, making it less plausible that an association is due to confounding factors.

Dose response
The following studies have found a dose-response of obesity to artificial feeding in early life. A meta-analysis of 17 different studies measuring duration of breastfeeding, with almost 121 000 participants, found that the probability of overweight/obesity in later life was reduced by 4% for each month of breastfeeding. Breastfeeding for less than one month increased obesity risk by nearly 50% compared to breastfeeding for more than six months (Harder et al 2005) (OR 0.68 for breastfed infants). A significant protective effect of breastfeeding against obesity has been shown for infants who had been breastfed exclusively for approximately four months or for a substantial duration. For example, infants who were breastfed longer than six months or breastfed exclusively for more than four months, had a reduced risk of obesity in childhood or adolescence of approximately 50% (OR 0.70) (Bogen, Hanusa & Wearke 2004; Grummer-Strawn & Mei 2004). In other studies measuring duration, breastfeeding reduced later life obesity by between 20% (OR 0.78 for mainly or only breastfed for greater than six months vs only or mainly artificially-fed) (Gillman, Rifas-Shiman et al 2001) and 45% (OR 0.57 for 6–12 months of breastfeeding vs never breastfed) (von Kries et al 1999). The association between early feeding and later obesity was found to be stronger in studies where breastfeeding was sustained beyond two months, or where initial feeding was exclusive (pooled OR 0.76 for exclusive breastfeeding beyond two months, and 0.81 for breastfeeding duration greater than two months) (Owen et al 2005).

These greater observed effects in studies with more precise measurement of duration or intensity of exposure is important, because it suggests that pooled estimates from meta-analyses of studies with ambiguous measures of exposure may underestimate the magnitude of effects. Many studies have defined breastfeeding without minimal criteria for exclusivity or duration, and categorised ‘mixed fed’ infants as either ‘breastfed’ or ‘formula fed’. This definition is often necessary to obtain sufficiently large sample sizes of breastfed infants in populations where exclusive or sustained breastfeeding beyond the early weeks is rare. More precise categorisation of exposure, such as comparing predominantly or exclusively artificially-fed with predominantly or exclusively breastfed from birth to six months, may result in larger effect sizes than shown by pooled estimates from meta-analysis of numerous mixed quality studies.

Confounding
While imprecise exposure measurement may underestimate the role of breastfeeding in protecting against later life obesity, residual confounding on the other hand may result in overstated effects of infant feeding on later life obesity. Few of the studies of infant feeding and obesity risk control for all important confounders (Arenz et al 2004), and it is difficult to accurately assess parental attributes or family environments associated with dietary habits or physical activity levels (Dewey 2003).

A within-family study of 2709 families that included 2372 discordantly fed sibling pairs, has shown that those breastfed for a longer duration (four months longer, on average) had a 6–8% lower risk of overweight in adolescence (Gillman et al 2006). This finding suggests that apparently protective effects of breastfeeding are not due to unmeasured socio-cultural factors because the association of breastfeeding duration with adolescent obesity for siblings raised in the same family environment was similar to the results for the whole sample. Nelson and co-workers (2005) used a within-family approach to examine this issue and did not find an inverse association. However, their sample was limited to only 112 sibling pairs, and compared siblings who had ever been breastfed with those who had never been breastfed.

A recent study of 15 253 adolescents, from relatively homogeneous socioeconomic and parental occupational backgrounds, found that exclusive breastfeeding reduced obesity by around 25–35%. This figure was obtained after adjusting for important confounders including maternal overweight/obesity, maternal diabetes, household income, maternal smoking, and birth weight, as well as dietary intake and physical activity (adjusted OR 0.66–0.73) (Mayer-Davis et al 2006).

Behavioural and biological mechanisms
Despite inadequacies in existing epidemiological research, it is likely that current observational findings reflect causality rather than unmeasured socioeconomic or biological confounding variables since animal studies and a small number of experimental studies show consistent results. There are also biologically plausible mechanisms by which artificial feeding may increase obesity risk (von Kries et al 1999; Dietz 2001; Gillman et al 2001; Dewey 2003; Arenz et al 2004; Harder et al 2005; Singhal 2006). Suggested mechanisms involve the metabolic programming effects of human milk, as well as effects of early feeding practices on infant and maternal feeding behaviours.

Behavioural mechanisms
Breastfeeding facilitates development of self control, and may also shape later food preferences towards healthy eating since components of human milk and the sucking experience affect feeding behaviours of the mother and the child. Although infants can self-regulate energy intake, artificially-fed infants have less self-control over how much milk they will take (Birch & Fisher 1998), partly because the carer has more control over the artificially-fed infant's intake and may keep feeding even when the baby is full (Tomon et al 1975). It may also relate to more vigorous feeding that has been associated with bottle feeding due to larger feeds and higher sucking pressure (Lucas, Lucas & Bauman 1979; Agras et al 1987).

Parental interference, such as encouragement to eat or excessive food restrictions, can override the development of self control in children, and increase obesity risk (Ventura et al 2005).
Studies show that breastfeeding mothers cannot easily monitor and manipulate milk intake, their feeding style is more responsive to infant feeding cues of hunger and satiety (Taveras et al. 2004). Reinforcing this is the common view that a heavy infant is a sign of successful feeding and parenting (Baugham et al. 1998). As the taste of breastmilk varies with the mother’s diet, breastfed babies accept new foods more readily (Sullivan & Birch 1994). This point is significant because infants naturally resist eating new foods and prefer sweet or salty tastes (Birch & Fisher 1995). The consistently bland flavour of artificial baby milk may make the infant less willing to try new foods and artificial feeding may thereby reinforce innate preferences for salty and sugary foods and influence later life dietary choices.

Artificially fed infants also may have less control over food intake because artificial baby milk lacks components of human milk (see below) which inhibit appetite and produce satiety.

Biological mechanisms
There is growing evidence that diet in infancy has short- and long-term effects on how the body metabolizes food, as well as influencing food intake levels and composition. Research in the 1990s has shown that the normal energy intake of infants has been considerably overestimated (Dewey et al. 1995; Whitehead 1995). Lower weight gain by breastfed infants reflects their lower mean caloric intake (Heining et al. 1993). Hence, regulatory nutrition standards for artificial baby milks are based on excessive assumptions about normal intake and cause over-feeding. Excessive intake of energy and protein in infancy is now a recognized factor in causing rapid early weight gain and altered nutritional programming (Lucas 1991, 1998, 2000; Stettler et al. 2002) that increases the risk of obesity in adolescence and adulthood (Baird et al. 2005) and plays a key role in adversely programming related health outcomes such as heart disease and diabetes (Singhal & Lucas 2004).

Components in breastmilk (such as the complex and dynamic mix of nutrients, hormones, growth factors and fats) play a key role in developing body systems to appropriately regulate food intake, process fats and sugars, and influence fat formation and body weight (Petrouschke, Rohrig & Hauner 1994; Hauner, Rohrig & Petrouschke 1995; Hamod 2001). Human milk contains leptin which controls appetite and satiety, and energy expenditure, and regulates weight gain; it may perform a counter-regulatory role to insulin in the body (Casabrelli et al. 1997; Lyle et al. 2001; Singhal et al. 2002; Agostoni 2005; Miraletes et al. 2006). Artificially-fed infants not only have significantly higher energy intakes than breastfed infants, their feeding triggers different hormonal responses to feeding; higher insulin concentrations found in artificially-fed infants are likely to stimulate fat deposition and early development of adipocytes (Lucas et al. 1980; Lucas et al. 1981).

Importantly, the dietary fat composition of artificial baby milk also differs in important ways from breastmilk, and this promotes excessive and abnormal fat cell proliferation and development (Kolezko et al. 2001; Ailhood & Guesnet 2004; Ailhood et al. 2006). Notably, commercial infant milks also have a cholesterol and saturated fatty acid content more markedly different from mature breastmilk than the unmodified cows’ milk used for artificial feeding until the late 1960s (Martin et al. 2005). Most significantly, recent research on the fatty acid composition of the fats in mothers’ milk and artificial baby milk shows that commercial infant milks marketed between 1980 and 1995 have had extremely high (up to 120%) ratios of linoleic acid to α-linolenic acid, which are known from animal studies to strongly promote fat cell and adipose tissue formation and result in highly abnormal weight gain (Ailhood et al. 2006).

These altered hormone levels and fat concentrations in infancy disturb the normal functioning, growth and development of body organs and tissues, and hence increase the propensity for fat and glucose metabolism disorders (Singhal 2006). Studies have found artificial feeding in infancy is associated with increased chronic disease symptoms in later life, such as high cholesterol levels, blood pressure, insulin resistance, and atherosclerosis (Ravelli et al. 2000). For example, artificial feeding in full-term infants is associated with higher blood pressure in adolescence and adulthood (Lawlor 2005; Martin, Gunnell & Smith 2005), and artificially-fed infants have higher levels of cholesterol and low density lipoproteins, suggesting long-term effects on fatty acid metabolism (Owen et al. 2005). Breastfed infants therefore have fewer risk factors for cardiovascular disease and diabetes in later life. More recently, Owen and co-workers (2005) have shown artificial feeding in infancy increased the risk of type 2 diabetes in later life by around 40% (adjusted OR 0.59–0.61 for breastfed infants). Breastfeeding in infancy is likely to reduce the risk of obesity through similar processes to those by which it protects against these other conditions in the metabolic syndrome (Plagemann & Harder 2005).

Public policy, breastfeeding and obesity
Over the past decade, national and international frameworks for policy have acknowledged the public health significance of links between poor infant feeding practices and later life obesity and chronic disease.

In its report on Diet, Nutrition and the Prevention of Chronic Diseases the World Health Organization (WHO) (2003) cited inadequate breastfeeding as a possible risk factor for chronic disease, finding “increasingly strong evidence that a lower risk of developing obesity may be directly related to the length of exclusive breastfeeding although it may not become evident until later in childhood” (World Health Organization 2003, p. 32).

Expert guidelines and reviews have also identified artificial feeding in infancy as a significant risk factor for key components of the ‘metabolic syndrome’ - overweight, high blood pressure and cholesterol, heart disease and diabetes (American Academy of Pediatrics 2005). The recently revised breastfeeding policy of the Royal Australasian College of Physicians also notes that breastfed infants have improved obesity and diabetes outcomes compared to artificially-fed infants (Royal Australasian College of Physicians 2006).

In Australia, the promotion of breastfeeding and the improvement of infant nutrition was prioritised within a policy framework addressing diet-related disease and early death in 200
in Eat Well Australia (National Public Health Partnership 2001). Australia’s National Obesity Task Force Report noted in 2003 that ‘effective prevention needs responses from all parts of society to encourage more active living and healthy living, beginning from the very start of life with breastfeeding’ (Commonwealth of Australia 2003, p 3). It listed ‘breastfeeding’ as a ‘setting’ that was a key ‘best-buy’ government response to the problem of obesity. The 2003 Australian Dietary Guidelines for Children and Adolescents also noted the link between inadequate or inappropriate fetal and early infant nutrition and the consequent chronic disease in adulthood, and cited evidence from research in the 1990s that breastfeeding infants may have reduced risk of autoimmune diseases and adiposity later in childhood (National Health and Medical Research Council 2003). The National Chronic Disease Strategy adopted by Australian governments in November 2005 included the promotion of breastfeeding as a key direction for preventative action (National Health Priority Action Council 2005).

The European Charter on Countering Cerebral Obesity was adopted in November 2006 and included ‘promoting breastfeeding’ in a package of essential preventative actions. Suggested measures included breastfeeding friendly hospital practices, adequate paid maternity leave and regulation of infant food marketing, as well as health professional training, and support for mothers (World Health Organization 2006a).

**BREASTFEEDING AND ARTIFICIAL INFANT MILK FEEDING IN AUSTRALIA – HISTORY AND TRENDS**

The end of ‘easy breastfeeding’

McCalman (1984) observed that ‘the 1930s marked the beginning of easy breastfeeding in Australia’. In the decades after 1900, medical opinion had played an increasing role in determining infant feeding practices. Breastfeeding was strongly and forcefully promoted by the health profession in the early decades of the 19th century (Reiger 1985). However, the use of patent infant foods had become more widespread in the latter part of the 18th century (Siskind, Del-Mar & Schofield 1993). Tinned condensed milk and powdered milk became available in Australia from the 1850s (Lund-Adams & Heywood 1995), and improved transportation and refrigeration also increased dairy milk supply in the cities by the 1890s. These developments led to a debate within the Australian medical profession on artificial feeding, and ultimately to medical endorsement of certain proprietary brands of infant foods and milk as ‘safe’ for infants from six months. Similarly, in the United States (US), the paediatric profession had developed strong collaboration with the infant food industry in order to better control infant food quality and marketing practices, and to establish the discipline as an expert in the science of infant care (Greer & Apple 1991). In the US, the success of the safe milk campaigns convinced paediatricians that breastfeeding was largely irrelevant to infant health. Influential research in the late 1920s found that newborns fed evaporated milk gained more weight than other infants, contributing to medical acceptance of artificial feeding (Greer & Apple 1991). By the 1930s artificial baby milk was generally accepted by the medical profession as ‘safe’ (Greer & Apple 1991).

Meanwhile, hospital birth and medicated childbirth increased during the 1930s and was nearly universal by 1945 (Lewis 1976; Reiger 1985). Although there was pressure to breastfeed, the regime imposed by hospitals and clinics made breastfeeding difficult, and some medical observers noted that the move to hospital birth had reduced breastfeeding (Smibert 1988; Mein Smith 1997; Reiger 2001; Thorley 2003). The experience of mothers was that ‘the idea of breastfeeding was supported, but the art of breastfeeding was underminded’ (Thorley 2001a, p 26).

The infant welfare movement and its associated emphasis on clean milk and strict hygiene in the ‘scientific’ care and feeding of infants also strongly influenced infant feeding practices in Australia (Reiger 1985; Mein Smith 1997). A focus on measurement and science permeated the infant welfare movement. From the 1930s, the ‘scientific’ infant care practices promoted through the clinics also promoted the belief that a heavy infant is a healthy infant. Weight gain became the main focus of infant health from this time. Test weighing scales were the symbol of ‘scientific’ infant care. Perceived ‘insufficient’ weight gain led many mothers to introduce artificial baby milks on the advice of health professionals, or to abandon breastfeeding due to ‘inadequate milk’ (Thorley 2003).

Throughout the post-war era there was also widespread use of infant age-weight charts, developed in the US during the 1950s. These were based on artificially-fed populations, which have now been found to overestimate normal weight gain of infants (World Health Organization 2006b). These growth reference charts did not account for the normal decrease in growth velocity of infants at approximately three months of age, and ‘the perceived failure to grow at the normal rate led women to give up or supplement breastfeeding’ (National Public Health Partnership 2001, p 64).

**Breastfeeding trends in the 20th century**

Breastfeeding had been universal in Australia until the second half of the 19th century, although parr or panada (that is, semi-liquid or soft food for infants such as bread boiled to pulp and flavoured) was often introduced early (Wickes 1953). Until the late 1930s, virtually all mothers initiated breastfeeding, and the usual weaning age was approximately nine months (Reiger 1985; Siskind, Del-Mar & Schofield 1993; Lund-Adams & Heywood 1995; Mein Smith 1997). However, a decline in breastfeeding commenced during the early 1940s and accelerated from the mid 1950s. Those now reaching middle age in Australia – those born since the 1950s – are the first generation in history that have not been substantially breastfed in infancy, and those born since around 1970 were the first generation in which use of commercial baby milks was widespread as the alternative to breastfeeding.

Figure 1 shows the high breastfeeding initiation rates that were typical in Australian cities early in the century and the sharp fall in breastfeeding initiation and increased supplementation from 1942. There was a brief recovery after World War II, then an accelerating decline in breastfeeding initiation and duration from the mid-1950s. Breastfeeding initiation and duration rose again from the late 1960s, but this increase was not continued through
the 1980s. While breastfeeding initiation appears to have risen in recent years (Graham et al 2005), there has been no increase in breastfeeding duration since 1985, and recent state surveys suggest a possible decline in rates of exclusive breastfeeding at six months (Gabriel et al 2005; Hector, Webb & Lynner 2005).

Figure 1. Long term trends in breastfeeding in Australia – initiation and full breastfeeding at 3, 6, and 9 months

![Breastfeeding Trends](image)

Just as the earlier fall in breastfeeding had been led by middle-class, educated women (Lund-Adams & Heywood 1995), so the rise in breastfeeding from the 1970s mainly occurred among those of higher socioeconomic status. Babies from more deprived backgrounds typically breastfeed for only a short time, if at all, and are weaned onto artificial baby milk and solids in the first six months. In 2001, around 20% of children from the most disadvantaged families had never been breastfed (Australian Institute of Health and Welfare 2003b). This rate could partly explain why lower socioeconomic groups experience a particularly high prevalence of obesity and its related risk factors.

Consumption of commercial baby milk products, 1939-1999
The above evidence on breastfeeding trends is consistent with the evidence on sales of commercial infant milk products since 1939. Table 1 demonstrates that there was a substantial acceleration in sales and consumption of specialised infant milk products beginning in the 1950s. Peak consumption of condensed, evaporated and powdered milk products, commonly – although not exclusively – used in artificial infant feeding, occurred in the early 1970s, when breastfeeding rates were at their lowest point. At this time home-modified cows’ milk was increasing discouraged by medical advice. The apparent consumption of infant and invalid milk products for each baby born in Australia is presented in Figure 2 for the period 1939–1999. This figure shows that sales of artificial baby milk per infant became significant from the 1940s, with very rapid growth from the mid 1950s. Sales continued to rise steadily although from the 1970s, the composition changed in favour of commercial baby milks as medical advice discouraged the use of home-modified baby milks.

Table 1: Trends in consumption of milk products used for infant feeding, Australia 1939-1999*

<table>
<thead>
<tr>
<th>Year ended June</th>
<th>Infants and invalids food (kg per capita)</th>
<th>Condensed concentrated evaporated milk (kg per capita)</th>
<th>Powdered milk (kg per capita)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1939</td>
<td>0.5</td>
<td>2.0</td>
<td>1.2</td>
</tr>
<tr>
<td>1949</td>
<td>0.6</td>
<td>3.4</td>
<td>1.5</td>
</tr>
<tr>
<td>1959</td>
<td>1.0</td>
<td>4.1</td>
<td>1.1</td>
</tr>
<tr>
<td>1971</td>
<td>1.0</td>
<td>6.8</td>
<td>4.8</td>
</tr>
<tr>
<td>1979</td>
<td>1.2</td>
<td>2.9</td>
<td>2.2</td>
</tr>
<tr>
<td>1989</td>
<td>1.4</td>
<td>1.8</td>
<td>1.9</td>
</tr>
<tr>
<td>1999</td>
<td>NA</td>
<td>0.8</td>
<td>1.5</td>
</tr>
</tbody>
</table>


Figure 2. Annual consumption of commercial infant milk, per infant, Australia, 1939-1999*

![Commercial Infant Milk Consumption](image)


INCREASED COMPETITION AND MARKETING OF INFANT MILK PRODUCTS
The accelerated decline in breastfeeding and rise in artificial baby milk consumption from the 1950s was contemporaneous with increased competition between manufacturers of artificial baby milks and the associated more active promotion and marketing of these food products through the Australian healthcare system. A scramble for market share, including direct-to-consumer advertising and more vigorous promotion to hospitals and health professionals, drastically increased the size of the market for artificial infant milk products in the space of a decade. In the process, this reduced the ‘market’ for breastfeeding.

Until the 1950s, advertising of infant milk products was not common. Thorton (2003) attributes this mainly to the lack of competition between brands in the 1940s and 1950s. Nestle's Lactogen dominated the small Australian market in artificial baby milk from the 1920s, so advertising was unnecessary (Thorton 2001a, 2003). Glaxo and Kariain products were also available but most mothers who were artificially feeding used home-modified fresh or powdered cows' milk, evaporated milk or Lactogen through the early postwar years (Mein Smith 1997).
From around the mid 1950s, amidst worldwide dairy expansion, and with the lifting of wartime import controls in Australia, US companies Mead Johnson and Wyeth began to market their products in Australia. This change prompted the incumbents in the Australian market to promote their products directly to mothers (Thorley 2003).

Ironically, the main distribution and marketing outlet for these proprietary products was through the health system, and another reason for the lack of advertising had been the close relationship Nestlé (later the Carnation Company) had with the hospitals: promotion was mainly directed at health professionals through company representatives, as in the US (Thorley 2003). A marketing strategy aimed mainly at physicians had characterised marketing of artificial baby milk in the US from the 1930s after the medical profession and the industry formalised collaboration in development and advertising of artificial infant foods (Greer & Apple 1991). Similar links between medicine and commerce were forged in Australia (Minchin 1988). A similarly ‘beneficial, reciprocal relationship’ had also been established with Australian hospitals during the 1940s by Nestlé; the companies’ booklets were available in the hospitals and baby clinic staff recommended the product to mothers who could afford it (Thorley 2003). The use of US pharmaceutical companies were very well practiced in marketing to health professionals and the healthcare system. As a consequence of their experience and relationships, manufacturers were well placed to take advantage of new mothers’ uncertainties and their struggles with milk supply that were attendant on the restrictive breastfeeding practices of the era’ (Thorley 2003).

From the 1940s, virtually all mothers gave birth in hospital and the rigidity of hospital practice was increasing unhelpful to breastfeeding (Thorley 2001, 2003). Distribution of their promotional materials by maternity staff thus gave the companies access to virtually all mothers. From the late 1950s, hospitals and health professionals were also enlisted in free distribution of artificial baby milk to hospitals and to mothers at discharge and this represented valuable endorsement by the health service and medical staff (Thorley 2003). By the mid 1970s, hospitals were ‘display cases’ for the promotion of artificial feeding (Lund-Adams & Heywood 1995). Manufacturers of artificial baby milk produced booklets on parenting and feeding that were widely distributed through the hospitals and were slant towards product promotion, and against breastfeeding (Thorley 2003). Practices that have been shown to be harmful to breastfeeding success (such as labour analgesia, separation of mothers and their newborn, highly restrictive feeding regimes, use of complementary feeds, and commercial discharge packs) have been widespread in Australian hospitals and the health system from the 1950s and remained commonplace for several decades (Fankin et al 1995; World Health Organization 1998).

Over this same period, direct advertising to mothers also increased dramatically. For example, the number of advertisements for infant foods and milks in The Australian Woman’s Weekly multiplied seven-fold in the decade from 1955, with the number of these magazine advertisements peaking in the early 1970s (Figure 3).

**DISCUSSION: DID MARKETING MATTER?**

Health authorities such as the World Health Organization recognize that marketing is a powerful influence on health behaviors (Hastings et al 2004). Economic research shows advertising is an important form of competition in highly concentrated industries, and significantly affects consumer behaviour and sales (Roberts & Samuelson 1988; Saffer & Chaloupka 2000). It has also been shown in the pharmaceutical and tobacco industries that competition through strategies such as brand proliferation, market segmentation, and complementary direct-to-consumer advertising and medical detailing, can expand the size of the total market (Brekie & Kuhn 2006), and does not just shift consumers between commercial brands (Chaloupka et al 2005). In 1981, the World Health Assembly concluded that, ‘the marketing of breastmilk substitutes requires special treatment,
which makes usual marketing practices inappropriate for these products’ (World Health Organization 1981).

It is shown above that the increasingly competitive market in commercial infant feeding products from the 1950s in Australia led to more aggressive product promotion, more usage of artificial baby milk, and less breastfeeding.

There are many influences on infant feeding decisions, and advertising is not the only aspect of commercial marketing and promotion (Hastings et al 2004). Advertising of commercial infant feeding products is only indicative of marketing and promotion trends. Data is presented on advertising compiled from two publications, the Medical Journal of Australia and the Australian Women’s Weekly, which may not be fully representative of trends in other publications. Nevertheless, these had wide influence and readership among Australian health professionals and women. It is reasonable to expect that the trends reflected in data on advertising in these publications are a reasonable indication of trends in the marketing and promotion activities of companies selling infant milk products. Historical studies of maternal and child health care and feeding practices confirm that there were corresponding trends of increased promotional activity evident in the health care system. Data presented here does not include electronic advertising, and will thus under-represent marketing and promotion through electronic media, such as television and radio. It is likely that television advertising became more important during the 1960s and 1970s, thus data on print media advertising will increasingly understate the extent of advertising to mothers before the World Health Organization International Code on Marketing of Breastmilk Substitutes (WHO Code) was agreed in the early 1980s (World Health Organization 1981). The data series presented also excludes infant foods other than milk products. Advertising of such products during the period was extensive and also contributed to premature weaning from breastfeeding. Analysis of the changing extent of advertising of these products is needed to better reflect the extent of commercial pressures undermining exclusive and sustained breastfeeding. This paper presents data for the period 1950 to 1985 only. The introduction of the WHO Code and the growth of electronic media make compilation of valid data a more complex task for later years. Notably however, direct advertising of artificial baby milk to mothers, including both print and electronic media, appears to have reemerged in the past decade, associated with more vigorous marketing and promotion of specialised products including ‘follow on’, ‘toddler’ and ‘organic’, and supplemented products, and such trends have recently been documented in the US (Government Accounting Office 2006; Oliveira & Davis 2006).

It is difficult to relate historical trends in infant feeding practices directly to rising levels of obesity in the Australian population because of the absence of reliable and accurate national data on breastfeeding patterns and on pediatric and adult obesity. Available national, state and local data on infant feeding patterns suffers from inconsistent definitions over time, differences in collection methods, and potential for selection bias. Figure 1 is based on a data series compiled from several smaller surveys and from Victorian clinic data; it also uses early data from Sydney (Armstrong 1939), and central Melbourne (Mein Smith 1997). Queensland data for the post-war period confirms a similar pattern outside Victoria (Siskind, Del-Mar & Schofield 1993). Data from mothers attending clinics, such as the Victorian series, suffer from self-selection bias and may not show the true prevalence of breastfeeding. Collection methods and precise definitions of breastfeeding practices also change over time. Nevertheless, the long term trends in breastfeeding evident in Figure 1 are likely to accurately reflect the broad trends over the century, and are consistent with patterns and time trends in other developed countries such as the US, Japan, Norway, and Sweden. The data series compiled from Australian Bureau of Statistics publications for apparent consumption of milk products can be expected to be a reasonably accurate portrayal of national trends. However it is possible that long term changes in patterns of household usage of powdered milk and evaporated milk products may reduce the accuracy of this data series as an indicator of trends in artificial infant milk feeding. Likewise the category ‘infant and invalids food’ does not distinguish artificial baby milk from similar products marketed for ‘invalids’, but the latter represent only a small proportion of consumption in this category (Connaughton K 2005, pers. comm.). The trend towards the use of artificial baby milk rather than powdered milk and evaporated milk products from the late 1960s is consistent with medical recommendations to use artificial baby milk rather than modified cows’ milk from the early 1970s in Australia, and supports this series does provide a reasonable representation of trends in infant feeding practices over the 1960s and 1970s. The continuing rise in consumption of commercial infant milk from the early 1970s despite rising breastfeeding initiation is likely to reflect the replacement of home-prepared products by commercial infant milk since the 1970s, and the continued short duration of exclusive breastfeeding through to the present time. Since the mid 1980s it is also likely to reflect expanded marketing and sales of artificial infant milk targeting mothers of older infants (‘follow on’ or ‘toddler’ milks). These emerged in the mid-1980s as a strategic response to the WHO Code which constrains marketing of products for younger infants (Greer & Apple 1991; Minchin 1998).

National data on obesity is available only sporadically from the mid-1980s, and data from surveys in earlier periods is insufficient to show long term trends in obesity. It is clear however, that the increase in pediatric obesity since the mid 1980s has been greater than the increase over the previous 16 years (Flood et al 2003). Concern about obesity has been fueled by the dramatic increase in prevalence from 8–9% to 17–20% among adults and from approximately 1% to approximately 5% since the early/mid 1980s (Australian Institute of Health and Welfare 2003a, 2004).

Although the magnitude of the effect of artificial infant feeding on later obesity is moderate in clinical terms, it is likely to be of public health importance, as a high percent of the population is exposed to premature weaning from breastmilk in infancy. For example, Dietz (2001) estimated that the population attributable risk of overweight from formula feeding in the US is 15–20%; (40% of US infants are not breastfed in hospital and 70% in
weaned from breastfeeding by six months of age). In Australia the attributable proportion is lower because rates of artificial infant feeding are currently lower than in the US. Nevertheless, among Australian adults born between 1956 and 1976 (now aged 45–60), some 30–50% were artificially-fed at hospital discharge, and 80–90% by twelve weeks (see Figure 1). These rates are comparable with current US levels and suggest infant feeding practices during the 1960s are of public health significance in the current rise in adult obesity. Even during the 1980s and 1990s, 10–20% of Australian infants were artificially-fed at hospital discharge, and around 60% were artificially-fed at six months of age (National Health and Medical Research Council 2003). This implies some 10–20% of current obesity in adults or in some population subgroups may be attributable to exposure to artificial feeding in infancy (that is, assuming an OR of 0.75–0.85 and exposure rates of 10–20%).

As the recent rise in childhood obesity has occurred amidst an increase in breastfeeding, some commentators discount the role of infant feeding as an explanation of current obesity trends (Dewey 2003). However, while breastfeeding initiation increased from the early 1970s until the mid 1980s, there has been little change in either initiation or duration since then. Consumption of artificial baby milk on the other hand, has continued to increase, reflecting the continued short duration of exclusive breastfeeding, and the increased promotion and availability of ‘follow on’ and ‘toddler’ milks. Commercial products have also largely replaced home-prepared artificial baby milks since the early 1970s. There is also evidence that the composition of artificial baby milks were highly distorted during the 1980s and early 1990s, with a fatty acid composition that is likely to have been highly obesiogenic, and this may outweigh the effects of increased breastfeeding initiation at the population level.

Furthermore, the recent rise in childhood and adolescent obesity has been identified in cohorts that were born in earlier years. Recent studies in the US and Denmark suggest a birth cohort effect for childhood and adolescent obesity that is consistent with early life factors contributing to current child and adult obesity. For example, Ogden and co-workers (2002) used US data to show that obesity rates among children and adolescents born since the mid to late 1960s are 11–15% compared to rates of between 4–6% among children and adolescents born before that time. Similarly time series data for boys and young men in Denmark shows that rates of obesity increased among those born from the early 1940s to the mid-1950s, and from the late 1960s onward, suggesting that changes in the early life of these cohorts resulted in an increase in obesity at later ages (Olsen et al 2006). Analysis of data from Sweden shows that the risk of obesity in adolescent men more than doubled for those born in 1973 compared to those born in 1953, though the trend slowed somewhat during the 1970s (Rasmussen 1999). These identified periods of change in early life environment coincide with decreased breastfeeding and increased artificial feeding in Northern European populations during the 1960s and early 1970s. These trends are consistent with a ‘programming effect’ of commercial artificial feeding, which has amplified in recent years, and this is an important area for future research.

**CONCLUSION**

Those who view obesity as simply a matter of individuals eating too much and exercising too little might well consider the case of infant feeding. Can it really be supposed that mothers in the 1960s were ‘choosing’ en masse not to breastfeed? Or did the Australian healthcare system and commercial practices stand in the way of mothers ‘choosing’ to feed human milk to their young? Likewise, can we continue to argue that breastfeeding was simply a matter of individual choice, and that individuals control their own obese or overweight destiny in the face of evidence that nutrition in early life ‘programs’ later life metabolic disorders and obesity?

While there is room for debate on the magnitude of the effect, artificial infant feeding is well established as an important risk factor for pediatric obesity (Reilly 2006), and is likely to be of population health significance given Australia’s past and probably current exposure levels (Dietz 2001; Dewey 2003). It is therefore ironic, that for several decades, Australia’s maternal and child health care system and industry regulatory framework permitted and facilitated commercial promotion of unhealthy infant feeding practices. That is, the healthcare sector itself was complicit in the decline in breastfeeding and increased exposure to a risk factor for obesity. The continuing significance of poor maternity care practices as a barrier to breastfeeding is shown by the Commonwealth government’s call in 2006, to improve breastfeeding rates in Australia through, inter alia, ‘in-hospital practices such as rooming-in with the baby and not giving supplemental feeds or pacifiers’ (Pyne 2006).

This paper highlights the role of increased market competition in the commercial infant food industry in creating an ‘obesiogenic environment’ – one which undermined breastfeeding – since the 1950s. Competition for market share through advertising and promotion has expanded the size of the market. It also points to the possibility that the epidemic of artificial feeding in Australia since the 1950s could help explain the current epidemic of obesity among middle aged adults. The unbalanced fatty acid composition of artificial infant milks, especially during the 1980s and 1990s, has added fuel to the obesity problem in children and adolescents through exposure to food products during the critical early life period.

Finally, the experience of successful commercial marketing of artificial feeding of infants through the health system during the 1950s and 1960s emphasizes the need for governments and health professional organisations to introduce effective restraints on the promotion and marketing of artificial feeding for infants and young children, including of ‘follow on’ and ‘toddler’ milks, and other commercial substitutes for breastmilk, including infant foods and drinks.

**REFERENCES**


Lawlor DA 2005, Infant feeding and components of the metabolic syndrome, findings from the European Youth Heart Study, Arch Dis Child 90: 582-588.


Lester IH 1994, Australia's Food and Nutrition. AGPS, Canberra.


ABOUT THE AUTHOR:
Dr Julie Smith has authored three books and numerous articles in scientific journals, including on economic aspects of breastfeeding. Her published research includes pioneering estimates of the economic value of breastmilk, and the attributable hospitalisation costs of artificial infant feeding. Her other recent publications argue the absence of a ‘level playing field’ in the ‘market’ for mothers’ milk, and set out the case for measuring breastfeeding in Australia’s GDP.

Correspondence to:
Dr Julie Smith
Australian Centre for Economic Research on Health (ACERH)
Australian National University
Canberra ACT 0200, Australia
Julie.Smith@anu.edu.au

© ABA 2007