

The Secretary  
Standing Committee on Health and Aging  
House of Representatives  
PO Box 6021  
Parliament House  
Canberra ACT 2600

Dear Ms Brown,

**Submission to Inquiry into the Health Benefits of Breastfeeding**

Please find attached our submission to the above inquiry.

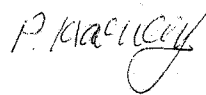
We mainly focus on chronic disease risk, which is our current area of research, but our submission also addresses the following three areas of the Committee's terms of reference:

- a) the extent of the health benefits of breastfeeding
- c) the potential short and long term impact on the health of Australians of increasing the rate of breastfeeding
- f) the impact of breastfeeding on the long term sustainability of Australia's health system

Yours Sincerely



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## Submission to the Standing Committee on Health and Aging - Inquiry into the Health Benefits of Breastfeeding

We have recently commenced a review of epidemiological studies which compare chronic disease incidence among those breastfed with those who were not breastfed.<sup>1</sup> This work is part of a wider study aiming to estimate the costs of chronic disease treatment incurred by artificial infant feeding and it extends previous research on the attributable hospitalization costs for infants and young children with common illnesses.<sup>1</sup>

We have looked at research findings for the following chronic diseases:

- obesity
- diabetes, types 1 and 2
- cardiovascular disease and disease risk
- asthma
- coeliac disease
- inflammatory bowel disease (including Crohns disease and ulcerative colitis)
- cancer

The following findings are preliminary. We expect to complete our research study later this year. In this paper, we present a summary of results from recent meta-analyses and provide a commentary on plausible causal mechanisms and methodological problems currently encountered in breastfeeding research.

Despite the benefits to the health and development of both infant and mother,<sup>2</sup> breastfeeding rates in Australia are poor. National<sup>3-5</sup> and state surveys<sup>6,7</sup> show that only around 5% of infants are exclusively breastfed at 6 months, and only around 10% receive any breastmilk at 12 months. In NSW, **up to a third of indigenous Australians are never breastfed.**<sup>6</sup> Nationally, individuals in the lowest two socioeconomic deciles are twice as likely as those in the top two deciles to have never breastfed.<sup>8</sup> Chronic disease is more prevalent among low socioeconomic groups and indigenous Australians.<sup>9</sup> Lack of breastfeeding in infancy thus helps perpetuate a cycle of educational, labour market and economic disadvantage. Around **30% of adults born during the 1960s and 1970s, and now in an age group experiencing a high incidence of chronic disease, were never breastfed.** With **only around 40% of Australian infants breastfed at six months of age**, less than half can be said to have a 'healthy start to life', and breastfeeding rates at six months may even be declining.<sup>6</sup>

### *Preliminary findings on chronic disease risk and infant feeding*

Our early findings suggest that 11–28% of chronic disease prevalence can be attributable to lack of breastfeeding in infancy. This figure is based on epidemiological research summarized in Table 1 below.

The effect estimates from the meta-analyses in Table 1 may appear small in size (take the RR of 1.06 for coronary heart disease for example) but they do have the potential to translate into the prevention

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Dr Smith is the recipient of an Australian Research Council Post Doctoral Fellowship (DP0451117) on 'the economics of mothers' milk and the market for infant food'. Dr Harvey is the editor of *Breastfeeding Review*, Australia's only peer reviewed professional journal on breastfeeding. Both researchers are volunteers with the Australian Breastfeeding Association (formerly Nursing Mothers').

of a substantial incidence of chronic disease.<sup>ii</sup> Martin and coworkers<sup>13</sup> theorised that reductions in mean blood pressure levels of the breastfed population would equate to the prevention of 3,000 coronary heart disease events and 2,000 strokes annually among those under the age of 75 years in the UK. Similarly, Akobeng et. al.<sup>18</sup> estimated that if 'no breastfeeding' was eliminated as a risk factor in babies born in England and Wales in 2002, then the number of cases of asthma, coeliac disease and obesity that could be prevented over 7-9 years would be 33,100, 2,655, and 13,639 respectively. In the US, researchers estimate that as much as 15-20% of childhood obesity could be avoided by reducing the country's 40% rate of exclusive artificial feeding.<sup>19</sup> Potential health system cost savings are large.

**Table 1: Results from epidemiological studies on infant feeding and later disease risk**

Chronic disease	Relative risk <sup>a</sup> for artificially-fed infants	Confidence limits <sup>b</sup>	Reference
Obesity	1.15	1.12-1.18	Owen 2005 <sup>10</sup>
	1.23	1.14-1.35	Harder 2005 <sup>11</sup>
Diabetes (type 1)	1.43	1.15-1.77	Gerstein 1994
Diabetes (type 2)	1.64	1.18-2.27	Owen 2006 <sup>12</sup>
Heart, stroke and vascular disease	Hypertension ~ 1.20 Coronary heart disease ~ 1.06 Strokes/ischemic attacks ~ 1.18	n.a.	Martin 2005 <sup>13</sup>
Asthma	1.37	1.23-1.66	Gdalevich, 2001 <sup>14</sup>
Coeliac disease	2.08	1.69-2.5	Akobeng 2006 <sup>15</sup>
Inflammatory bowel disease	Crohn's disease 1.49	1.16-1.92	Klement 2004 <sup>16</sup>
	Ulcerative Colitis 1.3	1.04-1.65	
Childhood cancer	All childhood cancers 1.28	1.01-1.64	Martin 2006 <sup>17</sup>

<sup>a</sup> Assuming relative risk (RR) approximates the inverse of the odds ratio (OR), where OR represents the protective effect of breastfeeding and RR represents the risk of artificial feeding.

<sup>b</sup> Confidence limits are based upon 95% confidence intervals of OR.

This is illustrated in Table 2, which calculates the population attributable risk proportion for various prevalences of exposure to artificial infant feeding.

For example, if around 60% of infants are exposed to premature weaning from breastfeeding (a definition corresponding approximately to the proportion of infants who were receiving no breastfeeding at 6 months of age), around 11-28% of chronic disease incidence could be avoided through increasing breastfeeding.

If breastfeeding rates were improved to National Health Target levels of around 80%, that is, an exposure of around 20% of the population, this would reduce the attributable proportion of chronic disease in the population to 4-11% (Table 2).

There are many uncertainties in this area of health research and some are quite important to consider. Appendix I discusses some methodological problems encountered in breastfeeding research and uses the recent research on obesity to illustrate these issues.

<sup>ii</sup> Although not summarised in Table 1, it is worth noting the work of Beral (2001) on breast cancer in the mother. This large meta-analysis found that breastfeeding could account for almost two-thirds of the estimated reduction in breast cancer incidence from 6.3 to 2.7 per 100 women by age 70, if women had the average number of births and lifetime duration of breastfeeding that had been prevalent in developing countries until recently.

**Table 2: Sensitivity analysis for different assumed 'breastfeeding' rates**

Assumed rate of population exposure (% 'not breastfed')	Proportion (%) of chronic disease in the population that is attributable to the exposure – range for chronic diseases in Table 1 <sup>iii</sup>							
	Obesity	Childhood cancer	Diabetes	CVD <sup>iv</sup>	Asthma	Coeliac <sup>v</sup>	Inflammatory bowel disease <sup>vi</sup>	Range for all conditions
100	19	22	39	17	30	26	29	17–39
90	17	20	37	15	28	24	26	15–37
80	16	18	34	14	26	23	24	14–34
70	14	16	31	12	23	21	22	12–31
60	12	14	28	11	21	18	19	11–28
50	10	12	24	9	18	17	17	9–24
40	8	10	20	7	15	15	14	7–20
30	6	8	16	6	11	12	11	6–16
20	4	5	11	4	8	9	7	4–11
10	2	3	6	2	4	5	4	2–6
0	0	0	0	0	0	0	0	0

*Possible mechanisms by which feeding in infancy affects later life chronic disease risk*

Recent neurobiological research provides new insights into how early brain functioning and development affects physical and mental health, learning and behaviour throughout the life cycle.<sup>20, 21</sup> The brain controls the main functions of all mammals including metabolism, reproduction, respiration, cardiovascular system, immune system, emotions, behaviour, response to stress and threats, and learning. Brain development is a complex interaction between nature and nurture. Early interactions with the main caregiver, including those via the sensory pathways as developed during breastfeeding, builds basic capabilities of the brain, and shapes later social and emotional functioning,<sup>22</sup> and intelligence, literacy, behaviour and physical and mental health.<sup>21</sup>

The higher risk of infectious illnesses and the immunological vulnerability of artificially fed infants is well known,<sup>23</sup> but more recently, evidence has been accumulating from systematic reviews and meta-

<sup>iii</sup> The population attributable fraction was computed using  $P_e(RR-1)/(1+P_e(RR-1))$  where  $P_e$  is the prevalence of exposure to artificial feeding, and RR is the relative risk calculated as the ratio of the incidence of morbidity in artificially fed infants to the incidence in breastfed infants.

<sup>iv</sup> Based on relative risk of 1.2.

<sup>v</sup> The relative risk ratio used of 1.3 refers to the risk associated with weaning from breastfeeding before introducing solids. As around 40-60% of Australian infants have been fully weaned from breastfeeding by 4-6 months when solids are commonly introduced, we have calculated this figure by assuming that 50% are not breastfeeding when solids are introduced.

<sup>vi</sup> The relative risk ratio used is 1.4, an average of the odds ratio for Crohn disease and ulcerative colitis.

analyses that artificial feeding of infants increases maternal and infant risk for a number of acute and chronic diseases. These include obesity<sup>10, 24</sup>, the 'metabolic syndrome' of diabetes,<sup>12</sup> cardiovascular disease risk including high blood pressure<sup>13</sup>, as well as some childhood cancers,<sup>25</sup> breast cancer in the mother,<sup>26</sup> and a range of chronic digestive (ulcerative colitis, Crohn's disease and coeliac)<sup>15, 16</sup> and allergic diseases.<sup>14, 27, 28</sup> Epidemiological studies also consistently show measurable cognitive development disadvantage in populations of children who were not breastfed as infants, notwithstanding methodological flaws leading to uncertainty about effect sizes.<sup>29, 30</sup> Meta-analyses<sup>31-33</sup> show breastfeeding confers permanent cognitive performance advantage of 2-5 IQ points, after adjustment for confounding variables such as maternal education or style, and family socioeconomic status. Studies on cognitive development also show effects which are longlasting<sup>34</sup> and greater for longer duration<sup>35</sup> and intensity/exclusivity<sup>36</sup> of breastfeeding, and for pre-term and small-for-gestational age infants.<sup>35-38</sup> Recent studies using diverse methodologies from both the natural and social sciences reinforce these findings.<sup>39-45</sup> Researchers are currently investigating possible links with later life mental health problems and vulnerability to stress among artificially-fed infants.<sup>46-48</sup>

Recent work by JS has focused on artificial baby milks and the obesogenic environment in Australia,<sup>49, 50</sup> and we summarise below the mechanisms by which infant feeding is suggested to influence later life obesity. Breastfeeding is likely to reduce the risk of obesity through similar processes to those by which it protects against other conditions in the metabolic syndrome.<sup>51</sup>

It is likely that the above observational findings reflect causality rather than unmeasured socioeconomic or biological confounding variables as 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<sup>11, 19, 24, 52-55</sup>

Although these mechanisms are still unclear, it is probably through the metabolic programming effects of human milk, as well as from effects of early feeding practices on infant and maternal feeding behaviours.

Firstly, breastfeeding facilitates development of self control, and may also shape later food preferences towards healthy eating, as components of human milk and the suckling 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<sup>56</sup>. Partly this is because the carer has more control over the artificially-fed infant's intake and may keep feeding even when the baby is full<sup>57</sup>. It may also relate to more vigorous feeding (larger feeds and higher sucking pressure) associated with bottle feeding<sup>58, 59</sup>.
- Parental interference, such as encouragement to eat or excessive food restrictions, can override the development of self control in children, and increase obesity risk<sup>60</sup>. Studies show that as breastfeeding mothers cannot easily monitor and manipulate milk intake, their feeding style is more responsive to infant feeding cues of hunger and satiety<sup>61</sup>. Reinforcing this is the common view that a heavy infant is a sign of successful feeding and parenting<sup>62</sup>.
- As the taste of breastmilk varies with the mother's diet, breastfed babies accept new foods more readily.<sup>63</sup> This point is significant because infants naturally resist eating new foods and prefer sweet or salty tastes<sup>56</sup> and the consistently bland flavour of artificial baby milk may make the infant less willing to try new foods. Artificial feeding may thereby reinforce innate preferences for salty and sugary foods and influence later life dietary choices.
- Artificially fed infants may have less control over food intake because artificial baby milk lacks components of human milk (see below) which inhibit appetite and produce satiety.

Secondly, there is also growing evidence that diet in infancy has short- and long-term effects on how the body metabolises food, as well as influencing food intake levels and composition.

- Research in the 1990s showed that the normal energy intake of infants has been considerably overestimated<sup>64,65</sup>. Lower weight gain by breastfed infants reflected their lower mean caloric intake<sup>66</sup>. Regulatory nutrition standards for artificial baby milks were based on excessive assumptions about normal intake and hence caused over-feeding. Excessive intake of energy and protein in infancy is now a recognised factor in causing rapid early weight gain and altered nutritional programming<sup>67-70</sup> that increases the risk of obesity in adolescence and adulthood<sup>71</sup> and plays a key role in adversely programming related health outcomes such as heart disease and diabetes<sup>72</sup>.
- Components in breastmilk (including a 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<sup>73-75</sup>. 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<sup>76-80</sup>. 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<sup>81,82</sup>.
- 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<sup>83-85</sup>. Notably, commercial infant milk also has 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<sup>86</sup>. 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 120:1) ratios of linoleic acid (LA) to  $\alpha$ -linolenic acid (LNA) Such a neonatal feeding regime which is known from animal studies to strongly promote fat cell and adipose tissue formation and result in highly abnormal weight gain. The LA content in formulas remains very variable, depending on the fat sources used. Furthermore, the fat content of commercial infant formulas continue to reflect standards and recommendations which are based on the very high LA/LNA ratios in milk of US, Dutch and UK women (13-14:1), rather than the more historically and biologically normal ratios of around 7-9:1 found in Northern European and Canadian women<sup>85</sup>.

These altered hormone levels and fat concentrations in infancy disturb the normal functioning, growth and development of body organs and tissues, increasing the propensity for fat and glucose metabolism disorders<sup>53</sup>.

Although the causal pathways for effects of breastfeeding are not fully understood, the effects of breastfeeding on mental development are also believed to result from maternal infant interactions during breastfeeding, alongside or interacting with the unique and highly complex composition of human milk.<sup>37, 40, 87, 88</sup>

### *Conclusion*

Our research is still at a preliminary stage. There are many uncertainties due to methodological flaws in existing research. These flaws work in the direction of understating the effect on chronic diseases risk from formula feeding in the case of how breastfeeding is defined, and overstate the risks where there is residual confounding. Overall, we would suggest that poor measurement of breastfeeding lowers the measured effects in these studies at least as much as does inadequate control for relevant confounding. Confidence in many of the studies is also weakened by the small sample sizes. High quality studies (such as those with large sample sizes, clear comparisons of substantially breastfed and substantially artificially-fed infants, and adjustment for confounding various) typically find an effect

between lack of substantial breastfeeding in infancy, and increases in incidence of later life chronic diseases.

Despite the uncertainties, we suggest there is enough evidence to show that breastfeeding affects chronic disease incidence at the population level and is therefore of significance to public health policy. Although the average effects are modest, the widespread population exposure to premature weaning means that relatively small effects from improving breastfeeding have a **potentially large impact on population health**. For example, researchers in the United States estimate that as much as **15-20% of childhood obesity could be avoidable by reducing the country's 40% rate of exclusive artificial feeding**.<sup>19</sup> Similar size effects have been found for several other chronic diseases. Likewise effects of artificial feeding during infancy on cognitive performance are only slightly less than the deficits from anaemia or lead burden,<sup>88</sup> and of the same order of magnitude as those from highly regarded early childhood education interventions, which raise IQ by 5 points.<sup>89</sup> However, population exposures to artificial feeding are much greater than for anaemia or lead. Hence relatively small effects can be highly important for public health.<sup>33</sup>

Breastfeeding is a one off 'intervention'<sup>vii</sup> that continues to reduce chronic disease risk throughout the life cycle. Unlike other interventions, such as exercise programs, or dietary changes, it does not have to be continued throughout the life cycle in order to maintain this protection, and so has no ongoing costs. This point means that it is likely to be very cost effective as a disease prevention measure.

There are few other **preventative health interventions** which have proven permanent effects in reducing risk factors for chronic disease or chronic disease in such a variety of settings.

**In summary, supporting breastfeeding as a policy priority is not a bad bet if you want to do something effective now, about an urgent problem which we still don't really know how to prevent.**

### *Recommended priorities*

Breastfeeding is already recognized to be a public health priority. Australian governments acknowledge its wide-ranging benefits in reducing ill health and chronic disease and facilitating normal child development. 'The encouragement and support of breastfeeding' is an Australian Dietary Guideline, acknowledging the nutritional, health, social and economic benefits it provides for the Australian community.<sup>9</sup> Encouragement of breastfeeding is a key component of recent public health and nutrition strategies targeting child health,<sup>94</sup> obesity<sup>95,96</sup> and chronic disease.<sup>54</sup> Breastfeeding also contributes to the national economic reform agenda - in 2006 the Council of Australian Governments prioritised chronic disease prevention and early childhood development on the national economic reform agenda because of the major implications for health system costs and labour productivity.<sup>97</sup> **More effective promotion of breastfeeding** would assist this agenda.

The Commonwealth and State governments need to 'back words with actions' in areas where government has primary responsibility. Many public health and child health and development strategies include the promotion of breastfeeding as a goal, but don't follow through with firm commitment to specific actions or measures to achieve this. Such measures should include the following:

#### **1. Collection of national, as well as state level breastfeeding data**

Good statistics are essential to design and evaluate programs and to target resources effectively. Australian collections on breastfeeding rates are *ad hoc* and full of gaps and inconsistencies, a

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<sup>vii</sup> Though strictly, it is premature weaning/artificial feeding that is the 'intervention' in these studies.

problem that dates back over a decade.<sup>90</sup> The lack of adequate statistics on breastfeeding reflects the low political priority given to it. For example, the 2001 ABS National Health Survey collected information on breastfeeding as one of a number of lifestyle behaviours and related characteristics which have been established as risks to health. In the next survey, the ABS will no longer include breastfeeding in its Survey, even though this is the only national source of information which links health risks and behaviours with obesity and chronic disease risk, and other data such as socioeconomic status, indigenous status, ethnicity etc. They do not see it as a priority, yet it affects many areas (such as diabetes) that they do collect data on.

## **2. Protection of breastfeeding from misleading and inaccurate commercial marketing and promotion**

This marketing and promotion relates to that targeted at health professionals and hospitals, as well as direct advertising to mothers, including media and shelf advertising, promotions, health professional education and sponsorship of continuing medical education etc.

## **3. Targeting of health system barriers to breastfeeding by low income and indigenous mothers**

Poor quality maternity care and outdated, inaccurate breastfeeding information is a particular problem for low income and indigenous mothers. Well-educated women in affluent communities are more likely to have access to a hospital meeting Baby Friendly Hospital standards for supporting breastfeeding, and to be cared for by well trained health professionals who are up-to-date and properly skilled to support breastfeeding. Strategically chosen geographic areas may be necessary as low income and indigenous mothers have a high rate of chronic disease and addressing this issue may require close targeting of well resourced programs.

## **REFERENCES**

- 1 Smith JP, Thompson JF, et al. Hospital system costs of artificial infant feeding: Estimates for the Australian Capital Territory. *Australian and New Zealand Journal of Public Health* 2002; 26:543-551.
- 2 Leung AK, Sauve RS. Breast is best for babies. *J Natl Med Assoc* 2005; 97:1010-9.
- 3 NHMRC Dietary Guidelines for Children and Adolescents in Australia incorporating the Infant Feeding Guidelines for Health Workers. Canberra: National Health and Medical Research Council, 2003.
- 4 Australian Bureau of Statistics. Breastfeeding in Australia, 2003.
- 5 Donath S, Amir LH. Rates of breastfeeding in Australia by State and socio-economic status: Evidence from the 1995 National Health Survey. *J Paediatr. Child Health* 2000; 36:164-168.
- 6 Hector D, Webb K, et al. Report on Breastfeeding in NSW 2004 (revised): NSW Centre for Public Health Nutrition, 2005.
- 7 Gabriel R, Pollard G, et al. Infant and child nutrition in Queensland 2003. Brisbane: Queensland Health, 2005.
- 8 Australian Institute of Health and Welfare (AIHW). Australia's Welfare 2003. AIHW. Canberra, 2003.
- 9 National Health Priority Action Council (NHPAC). National chronic disease strategy. In: Australian Department of Health and Aging, ed. Canberra, 2005.
- 10 Owen CG, Martin RM, et al. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics* 2005; 115:1367-77.
- 11 Harder T, Bergmann R, et al. Duration of breastfeeding and risk of overweight: a meta-analysis. *Am J Epidemiol* 2005; 162:397-403.
- 12 Owen CG, Martin RM, et al. Does breastfeeding influence risk of type 2 diabetes in later life? A quantitative analysis of published evidence. *Am J Clin Nutr* 2006; 84:1043-54.
- 13 Martin RM, Gunnell D, et al. Breastfeeding in infancy and blood pressure in later life: systematic review and meta-analysis. *Am J Epidemiol* 2005; 161:15-26.
- 14 Gdalevich M, Mimouni D, et al. Breast-feeding and the risk of bronchial asthma in childhood: a systematic review with meta-analysis of prospective studies. *J Pediatr* 2001; 139: .
- 15 Akobeng AK, Ramanan AV, et al. Effect of breast feeding on risk of coeliac disease: a systematic review and meta-analysis of observational studies. *Arch Dis Child* 2006; 91:39-43.
- 16 Klement E, Cohen RV, et al. Breastfeeding and risk of inflammatory bowel disease: a systematic review with meta-analysis. *Am J Clin Nutr* 2004; 80:1342-52.
- 17 Martin RM, Gunnell D, et al. Breast-feeding and childhood cancer: A systematic review with metaanalysis. *Int J Cancer* 2005; 117:1020-31.
- 18 Akobeng AK, Heller RF. Assessing the population impact of low rates of breast-feeding on asthma, coeliac disease and obesity: the use of a new statistical method. *Arch Dis Child* 2006.
- 19 Dietz WH. Breastfeeding may help prevent childhood overweight. *Jama* 2001; 285:2506-7.
- 20 Mustard JF. Early childhood development and experience based brain development - the scientific underpinnings of the importance of early child development in a globalised world. 2006; Accessed 2 February 2007. <http://www.brookings.edu/views/papers/200602mustard.pdf>



- 21 McCain M, Mustard JF. Early Years Study: Reversing the Real Brain Drain. Toronto, Canada: Children's Secretariat, 1999.
- 22 Halfon N, Shulman E, et al. Brain development in early childhood. In: Halfon N, Shulman E, Hochstein M, eds. *Building community systems for young children*. Los Angeles: UCLA Center for Healthier Children, Families and Communities, 2001.
- 23 American Academy of Paediatrics (AAP). Policy Statement: Breastfeeding and the Use of Human Milk. *Ped* 2005; 115:496-506.
- 24 Arenz S, Ruckerl R, et al. Breast-feeding and childhood obesity--a systematic review. *Int J Obes Relat Metab Disord* 2004; 28:1247-56.
- 25 Martin RM, Gunnell D, et al. Breast-feeding and childhood cancer: a systematic review with metaanalysis. *Int J Cancer* 2005; 117:1020-31.
- 26 Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and breastfeeding: collaborative reanalysis of individual data from 47 epidemiological studies in 30 countries, including 50302 women with breast cancer and 96973 women without the disease. *Lancet* 2002; 360:187-95.
- 27 Gdalevich M, Mimouni D, et al. Breast-feeding and the onset of atopic dermatitis in childhood: a systematic review and meta-analysis of prospective studies. *J Am Acad Dermatol* 2001; 45: .
- 28 Oddy WH. A review of the effects of breastfeeding on respiratory infections, atopy, and childhood asthma. *J Asthma* 2004; 41:605.
- 29 Der G, Batty GD, et al. Effect of breast feeding on intelligence in children: prospective study, sibling pairs analysis, and meta-analysis. *Bmj* 2006.
- 30 Jain A, Concat J, et al. How good is the evidence linking breastfeeding and intelligence? *Pediatrics* 2002; 109:1044-53.
- 31 Drane DL, Logemann JA. A critical evaluation of the evidence on the association between type of infant feeding and cognitive development. *Paediatr Perinat Epidemiol* 2000; 14:349-56.
- 32 Anderson JW, Johnstone BM, et al. Breast-feeding and cognitive development: a meta-analysis [see comments]. *Am J Clin Nutr* 1999; 70:525-35.
- 33 Reynolds A. Breastfeeding and brain development. *Pediatr Clin North Am* 2001; 48:159-71.
- 34 Burgard P. Critical evaluation of the methodology employed in cognitive development trials. *Acta Paediatr Suppl* 2003; 92:6-10.
- 35 Slykerman RF, Thompson J, et al. Breastfeeding and intelligence of preschool children. *Acta Paediatr* 2005; 94:832-837.
- 36 Rao MR, Hediger ML, et al. Effect of breastfeeding on cognitive development of infants born small for gestational age. *Acta Paediatr* 2002; 91:267-74.
- 37 Agostoni C. Small-for-gestational-age infants need dietary quality more than quantity for their development: the role of human milk. *Acta Paediatr* 2005; 94:827-9.
- 38 Smith MM, Durkin M, et al. Influence of breastfeeding on cognitive outcomes at age 6-8 years: follow-up of very low birth weight infants. *Am J Epidemiol* 2003; 158:1075-82.
- 39 McCann MF, Bender DE, et al. Infant feeding in Bolivia: a critique of the World Health Organization indicators applied to demographic and health survey data. *Int J Epidemiol* 1994; 23:129-37.
- 40 Feldman R, Eidelman AI. Direct and indirect effects of breast milk on the neurobehavioral and cognitive development of premature infants. *Dev Psychobiol* 2003; 43:109-19.
- 41 Gomez-Sanchiz M, Canete R, et al. Influence of breast-feeding on mental and psychomotor development. *Clin Pediatr (Phila)* 2003; 42:35-42.
- 42 Oddy WH, Kendall GE, et al. Breast feeding and cognitive development in childhood: a prospective birth cohort study. *Paediatr Perinat Epidemiol* 2003; 17:81-90.
- 43 Lawlor DA, Najman JM, et al. Early life predictors of childhood intelligence: findings from the Mater-University study of pregnancy and its outcomes. *Paediatr Perinat Epidemiol* 2006; 20:148-62.
- 44 Richards M, Hardy R, et al. Long-term effects of breast-feeding in a national birth cohort: educational attainment and midlife cognitive function. *Public Health Nutr* 2002; 5:631-5.
- 45 Mortensen EL, Michaelsen KF, et al. The association between duration of breastfeeding and adult intelligence. *Jama* 2002; 287:2365-71.
- 46 Montgomery SM, Ehlin A, et al. Breast feeding and resilience against psychosocial stress. *Arch Dis Child* 2006.
- 47 Oddy WH. Fatty acid nutrition, immune and mental health development from infancy through childhood. In: Huang JD, ed. *Frontiers in nutrition research*: Nova Science Publishers, 2006.
- 48 Sorensen HJ, Mortensen EL, et al. Breastfeeding and risk of schizophrenia in the Copenhagen Perinatal Cohort. *Acta Psychiatr Scand* 2005; 112:26-9.
- 49 Smith J. Formula for fatness: infant food marketing in Australia. In: Dixon J, Broom D, eds. *The seven deadly sins of obesity*. Sydney: University of New South Wales Press, 2007a.
- 50 Smith JP. The contribution of infant food marketing to the obesogenic environment in Australia *Breastfeeding Review* 2007b; accepted 23-1-07, in press.
- 51 Plagemann A, Harder T. Breast-feeding and the risk of obesity and related metabolic diseases in the child. *Metabolic Syndrome and Related Disorders* 2005; 3:222-232.
- 52 Gillman MW, Rifas-Shiman SL, et al. Risk of overweight among adolescents who were breastfed as infants. *Jama* 2001; 285:2461-7.
- 53 Singhal A. Early nutrition and long-term cardiovascular health. *Nutr Rev* 2006; 64:S44-9; discussion S72-91.
- 54 Dewey KG. Is breastfeeding protective against child obesity? *J Hum Lact* 2003; 19:9-18.
- 55 von Kries R, Koletzko B, et al. Breast feeding and obesity: cross sectional study. *Bmj* 1999; 319:147-50.
- 56 Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. *Pediatrics* 1998; 101:539-49.
- 57 Fomon SJ, Filmer LJ, Jr., et al. Influence of formula concentration on caloric intake and growth of normal infants. *Acta Paediatr Scand* 1975; 64:172-81.
- 58 Lucas A, Lucas PJ, et al. Pattern of milk flow in breast-fed infants. *Lancet* 1979; 2:57-8.
- 59 Agras WS, Kraemer HC, et al. Does a vigorous feeding style influence early development of adiposity? *J Pediatr* 1987; 110:799.
- 60 Ventura A, Savage J, et al. Early behavioural, familial and psychosocial predictors of overweight and obesity. In: Tremblay R, Barr R, Peters R, eds. *Encyclopedia on Early Childhood Development [online]*. Montreal, Quebec: Centre of Excellence for Early Childhood Development 2005.

- 61 Taveras EM, Scanlon KS, et al. Association of breastfeeding with maternal control of infant feeding at age 1 year. *Pediatrics* 2004; 114:e577-83.
- 62 Baughcum AE, Burklow KA, et al. Maternal feeding practices and childhood obesity: a focus group study of low-income mothers. *Arch Pediatr Adolesc Med* 1998; 152:1010-4.
- 63 Sullivan SA, Birch LL. Infant dietary experience and acceptance of solid foods. *Pediatrics* 1994; 93:271-7.
- 64 Dewey K, Peerson J, et al. Growth of breast-fed infants deviates from current reference data: a pooled analysis of US, Canadian, and European data sets. World Health Organization Working Group on Infant Growth. *Pediatrics* 1995; 96:495-503.
- 65 Whitehead RG. For how long is exclusive breast-feeding adequate to satisfy the dietary energy needs of the average young baby? *Pediatr Res* 1995; 37:239-43.
- 66 Heinig MJ, Nommsen LA, et al. Intake and growth of breast-fed and formula-fed infants in relation to the timing of introduction of complementary foods: the DARLING study. Davis Area Research on Lactation, Infant Nutrition and Growth. *Acta Paediatr* 1993; 82:999-1006.
- 67 Stettler N, Zemel BS, et al. Infant weight gain and childhood overweight status in a multicenter, cohort study. *Pediatrics* 2002; 109:194-9.
- 68 Lucas A. Programming by early nutrition in man. *Ciba Found Symp* 1991; 156:38-50; discussion 50-5.
- 69 Lucas A. Programming by early nutrition: an experimental approach. *American Society for Nutritional Sciences* 1998:401S.
- 70 Lucas A. Programming not metabolic imprinting. *Am J Clin Nutr* 2000; 71:602.
- 71 Baird J, Fisher D, et al. Being big or growing fast: systematic review of size and growth in infancy and later obesity. *Bmj* 2005; 331:929.
- 72 Singhal A, Lucas A. Early origins of cardiovascular disease; is there a unifying hypothesis? *Lancet* 2004; 363:1642-1645.
- 73 Hamosh M. Bioactive factors in human milk. *Pediatr Clin North Am* 2001; 48:69-86.
- 74 Hauner H, Rohrig K, et al. Effects of epidermal growth factor (EGF), platelet-derived growth factor (PDGF) and fibroblast growth factor (FGF) on human adipocyte development and function. *Eur J Clin Invest* 1995; 25:90-6.
- 75 Petruschke T, Rohrig K, et al. Transforming growth factor beta (TGF-beta) inhibits the differentiation of human adipocyte precursor cells in primary culture. *Int J Obes Relat Metab Disord* 1994; 18:532-6.
- 76 Casabiell X, Pineiro V, et al. Presence of leptin in colostrum and/or breast milk from lactating mothers: a potential role in the regulation of neonatal food intake. *J Clin Endocrinol Metab* 1997; 82:4270-3.
- 77 Singhal A, Farooqi IS, et al. Early nutrition and leptin concentrations in later life. *Am J Clin Nutr* 2002; 75:993-9.
- 78 Lyle RE, Kincaid SC, et al. Human milk contains detectable levels of immunoreactive leptin. *Adv Exp Med Biol* 2001; 501:87-92.
- 79 Miralles O, Sanchez J, et al. A physiological role of breast milk leptin in body weight control in developing infants. *Obesity (Silver Spring)* 2006; 14:1371-7.
- 80 Agostoni C. Ghrelin, leptin and the neurometabolic axis of breastfed and formula-fed infants. *Acta Paediatr* 2005; 94:523-5.
- 81 Lucas A, Sarson DL, et al. Breast vs bottle: endocrine responses are different with formula feeding. *Lancet* 1980; 1:1267-9.
- 82 Lucas A, Boyes S, et al. Metabolic and endocrine responses to a milk feed in six-day-old term infants: differences between breast and cow's milk formula feeding. *Acta Paediatr Scand* 1981; 70:195-200.
- 83 Ailhood G, Guesnet P. Fatty acid composition of fats is an early determinant of childhood obesity: a short review and an opinion. *Obes Res* 2004; 5.
- 84 Koletzko B, Agostoni C, et al. Long chain polyunsaturated fatty acids (LC-PUFA) and perinatal development. *Acta Paediatr* 2001; 90:460-4.
- 85 Ailhaud G, Massiera F, et al. Temporal changes in dietary fats: role of n-6 polyunsaturated fatty acids in excessive adipose tissue development and relationship to obesity. *Prog Lipid Res* 2006; 45:203-36.
- 86 Martin RM, Ebrahim S, et al. Breastfeeding and atherosclerosis: intima-media thickness and plaques at 65-year follow-up of the Boyd Orr cohort. *Arterioscler Thromb Vasc Biol* 2005; 25:1482-8.
- 87 Tu MT, Lupien SJ, et al. Measuring stress responses in postpartum mothers: perspectives from studies in human and animal populations. *Stress* 2005; 8:19-34.
- 88 Gustafsson P, Duchon K, et al. Breastfeeding, very long polyunsaturated fatty acids (PUFA) and IQ at 6 1/2 years of age. *Acta Paediatr* 2004; 93:1280-1287.
- 89 Barnett WS. Long term effects of early childhood programs on cognitive and school outcomes. *The future of children* 1995; 5:25.
- 90 Webb K, Marks G, et al. Towards a national system for monitoring breastfeeding in Australia: recommendations for population indicators, definitions and next steps. Canberra: National Food and Nutrition Monitoring and Surveillance Project, Commonwealth Department of Health and Aged Care, 2001.
- 91 Bogen DL, Hanusa BH, et al. The effect of breast-feeding with and without formula use on the risk of obesity at 4 years of age. *Obes Res* 2004; 12:1527-35.
- 92 Grummer-Strawn LM, Mei Z. Does breastfeeding protect against pediatric overweight? Analysis of longitudinal data from the Centers for Disease Control and Prevention Pediatric Nutrition Surveillance System. *Pediatrics* 2004; 113:e81-6.
- 93 Gillman MW, Rifas-Shiman S, et al. Breastfeeding and overweight in adolescence. *Epidemiology* 2006; 17:112-114.
- 94 Mayer-Davis EJ, Rifas-Shiman SL, et al. Breast-feeding and risk for childhood obesity: does maternal diabetes or obesity status matter? *Diabetes Care* 2006; 29:2231-7.

## Appendix I

### *Methodological problems in infant feeding research.*

There are uncertainties in this area of health research, and some are quite important.

**Firstly**, there are **very few randomized control trials which compare the effects of breastfeeding versus artificial-feeding**, because this is generally unethical. It would be unethical to deliberately deprive infants of breastfeeding as breastfeeding is known to be important and desirable in nearly all cases.

Therefore **most studies are observational**, that is they compare the prevalence of chronic disease observed in a population group which was breastfed, with the disease incidence for a population group that was not. It is difficult for such studies to show 'causation' and the results can also be 'confounded' by differences in the groups' characteristics, other than infant feeding method, which also affect chronic disease risk.<sup>viii</sup> This difficulty is especially so if the studies are comparing later life outcomes, when many environmental or behavioural factors could affect the outcome. Hence, a single study may not prove a result. However, if a number of studies get similar results, the findings are strengthened. Meta-analyses and systematic reviews are studies which compare and combine the results of several studies, and this approach is very useful to show the broad picture, and give more confidence that a statistically significant link exists.

It is **difficult for observational studies to show 'causation'**. Findings are more persuasive if there are biologically plausible ways in which an 'exposure' (such as artificial feeding) could result in a higher incidence of a subsequent condition. It is also more conclusive if there is a dose-dependent relationship, that is, if earlier or more complete artificial feeding shows larger differences or effects than when artificial baby milk is introduced later, or when artificial baby milk is combined with breastfeeding.

A further problem in breastfeeding research is that studies often compare **feeding groups which are defined inconsistently or inappropriately. Many studies compare babies who were 'ever' breastfed, with those who were 'never' breastfed.** Not surprisingly, such studies rarely find a difference - unless the study size is very large, or is for conditions like gastrointestinal illness or necrotizing enterocolitis where the effects of even a small period of breastfeeding are very powerful. The strongest effects would show in studies comparing infants exclusively breastfed to six months with those exclusively artificially-fed to six months. However, such studies are rare because exclusive breastfeeding is uncommon in most western countries, making it expensive to recruit enough study subjects. The result is that many of the existing studies underestimate the true differences between 'breastfed' and 'not breastfed', and it is important to look carefully at how 'breastfed' and 'not breastfed' is defined in each study.

It is also important to consider whether the study is large enough to show an effect. Many studies have **too few infants** who are exclusively breastfed and exclusively artificially-fed for the study to show any statistically significant differences between the infant feeding methods. Where there are also problems in how the feeding groups are defined, such small studies showing no difference are even more unreliable.

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<sup>viii</sup> For example the artificial feeding group may be less well educated than the breastfeeding group. Less well educated groups may be more likely to smoke, and this will affect their risk of chronic disease for reasons which have nothing to do with whether they were breastfed as infants.

In summary, it is hard to find high quality studies in this area. High quality studies are expensive as they require recruitment of large numbers of infants for whom breastfeeding and artificial feeding is carefully and specifically defined. Hence, meta-analyses and systematic reviews provide the best way to get an overview of the findings of research to date, and until more high quality studies are funded, conducted, and available.

#### *Dose response studies indicate causation in obesity studies*

A number of studies have found a dose-response of obesity to early life artificial feeding. A meta-analysis of 17 different studies measuring duration of breastfeeding, with around 121 000 participants, found that the probability of overweight/obesity in later life was reduced by 4% for each month of breastfeeding, with breastfeeding for less than a month increasing obesity risk by nearly 50% compared to breastfeeding for more than six months<sup>11</sup>. Likewise, there is evidence of a threshold effect for exposure to artificial feeding. Several studies have found that a significant protective effect of breastfeeding against obesity arose only for infants who had been breastfed exclusively for around four months or for a substantial duration. In two such studies, infants who were breastfed longer than six months or breastfed exclusively for more than four months had a reduced odds of obesity in childhood or adolescence of around 30%<sup>91, 92</sup>. In other studies measuring duration, breastfeeding reduced later life obesity by between around 20%<sup>52</sup> and 45%<sup>55</sup>. The 2005 meta-analysis by Owen and co-workers found the association between early feeding and later obesity was stronger in studies where breastfeeding was sustained beyond two months, or where initial feeding was exclusive.

These greater observed effect sizes in studies with more precise measurement of duration or intensity of exposure to artificial feeding is important, because it suggests pooled estimates from meta-analyses which includes studies with ambiguous measures of exposure may understate the magnitude of effects. Many studies included in recent meta-analyses defined breastfeeding without minimal criteria for exclusivity or duration, and categorised 'mixed fed' infants as either 'breastfed' or 'formula fed'.<sup>ix</sup> More precise categorization 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 factors in obesity studies*

While imprecise exposure measurement may understate 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<sup>24</sup>, and it is difficult to accurately assess parental attributes or family environments associated with alternative lifestyles such as dietary habits or physical activity levels<sup>54</sup>.

A recent study contributed to this question of confounding by comparing siblings who had discordant breastfeeding duration. This within-family study of 2709 families included 2372 discordantly fed sibling pairs and showed that those breastfed for a longer duration (four months longer on average) had a 6–8% lower odds of overweight in adolescence<sup>93</sup>. 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. Furthermore, another very recent study of 15 253 adolescents from relatively homogenous socioeconomic and parental occupational background found that exclusive breastfeeding reduced obesity by around 25–35% 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.<sup>94</sup>

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<sup>ix</sup> This definition was often necessary to obtain sufficiently large sample sizes of breastfed infants in populations where exclusive or sustained breastfeeding beyond the early weeks is rare.