
REVIEW

Alcohol and lactation: A systematic review

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Abstract

The aim of the present paper is to critically review the current literature on the effect of alcohol intake during lactation on the hormonal control of lactogenesis; breast milk and infant blood alcohol concentration; and on the breastfeeding infant. The databases PubMed, CINAHL, Proquest Health and Medical Complete, ScienceDirect and ISI Web of Knowledge were searched for articles published between 1990 and 2005. We found limited research investigating the effect of alcohol intake on the infants of lactating women, with most being conducted using animal models. Results consistently show a decrease in lactational performance in both animal and human studies of alcohol intake and breastfeeding. Alcohol intake by lactating mothers in amounts recommended as 'safe' for non-lactating women may have a negative effect on infant development and behaviour. Clear guidelines for alcohol consumption are required for lactating women and health professionals to guide breastfeeding mothers to make educated choices regarding alcohol intake during this critical period of infant development.

Key words: alcohol, breastfeeding, lactation.

INTRODUCTION

Breastfeeding is the safest and best method for nurturing and optimising infant growth and health. In 2001 the World Health Organization Expert Consultation recommended exclusive breastfeeding for six months, with continued breastfeeding until two years of age together with complementary foods, a position now adopted in Australia.^{1,2} Alcohol is an important part of most human societies and mothers need advice on its use during lactation. The term 'alcohol' describes a series of organic chemical compounds; however, only one type, *ethyl alcohol* or *ethanol*, is found in significant quantities in drinks intended for human consumption.

Alcoholic beverages are a source of great enjoyment in many societies, but alcohol problems are an important public health concern.³ Considerable research has been

conducted into the effects of alcohol on the developing embryo, and foetal alcohol syndrome has become recognised as the foremost preventable, non-genetic cause of intellectual impairment.⁴ The literature regarding foetal alcohol syndrome will not be addressed in the present paper, but it is important to note that there are well documented recommendations to restrict or limit alcohol intake during pregnancy.^{3,5} Many studies report a reduced maternal alcohol intake during pregnancy and a return to prepregnancy levels, or at least higher intakes than during pregnancy, shortly following birth.^{6–8}

A report from the United States Institute of Medicine National Academy of Sciences concluded that alcohol consumption by lactating women in excess of 0.5 g/kg of maternal weight may be harmful to the infant, partly because of a potential reduction in milk volume.⁹ Without giving specific recommendations, the American Academy of Pediatrics stated that alcohol intake is 'compatible with breastfeeding'. However, the following effects are noted on the infant: 'with large amounts, drowsiness, diaphoresis, deep sleep, weakness, decrease in linear growth, abnormal weight gain; and maternal ingestion of 1 g/kg daily decreases milk ejection reflex' (p. 780).¹⁰

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The Health Council of the Netherlands states in their most recent report that alcohol use during breastfeeding has adverse effects on the infant. The Council recommends that mothers who have consumed a standard measure (10 g ethanol) of an alcoholic beverage can avoid exposing the nursing child to ethanol by abstaining from breastfeeding for a period of three hours from when the alcohol was consumed or using expressed milk. If the mother has consumed a higher amount, the Council suggests that the period until the next breast-feed should be longer, and can be calculated by multiplying the three-hour period by the number of standard measures of alcohol consumed.¹¹

The United States Department of Health and Human Services recommends that on the basis of alcohol being transferred into the breast milk, alcohol intake should be limited to protect the health of the mother and infant.¹²

The most recent Australian alcohol guidelines published by the National Health and Medical Research Council (NHMRC)³ provide a guideline for alcohol consumption for pregnant, or soon to be pregnant women (Guideline 11).

Guideline 11 states 'Women who are pregnant or might soon become pregnant (11.1) may consider not drinking at all; (11.2) most importantly, should never become intoxicated; (11.3) if they choose to drink, over a week, should have less than 7 standard drinks (spread over at least two hours); should note that the risk is highest in the earlier stages of pregnancy, including the time from conception to the first missed period' (p. 16).

An appendage to this guideline is some 'prudent' advice for lactating women not to exceed the levels of drinking recommended during pregnancy, and to consider not drinking at all.

The aim of the present paper is to review the literature on the physiological process and hormonal control of lactogenesis, the milk ejection reflex ('let down'), and the effect of alcohol on these processes in both the short term and long term. These three questions will be addressed:

- 1 What is the effect of alcohol intake on the hormonal control of lactogenesis?
- 2 What effect do blood alcohol levels have on the breast milk concentration of alcohol and subsequent infant blood alcohol levels?
- 3 What is the effect of alcohol intake on the breastfeeding infant?

METHODS

A systematic literature review was conducted using the electronic databases PubMed, CINAHL, Proquest Health

and Medical Complete, ScienceDirect and ISI Web of Knowledge from 1990 to 2005. The search terms were 'breastfeeding', 'breast feeding', 'breastmilk', 'breast milk', 'lactation', 'alcohol' and 'ethanol'. The search was limited to English language journals.

The US Department of Health and Human Services defines a standard drink as containing approximately 14 g (approximately 0.6 fluid ounces) of pure alcohol.¹³ The NHMRC standard drink contains 10 g (12.5 mL) of alcohol.³ All references to alcohol volumes in the present paper have been converted to Australian standard drink equivalents (unless stated otherwise).

References used in the present paper (at first use) have been classified using the NHMRC guide, 'How to use the evidence: assessment and application of scientific evidence' (Table 1).¹⁴ Although originally developed for clinical guidelines, the guidelines can be used in public health assessments recognising that in research on maternal and infant alcohol intakes, ethical restraints on human experimentation limit the types of research that can be undertaken. In the present review paper expert consensus statements and evidence from experimental studies with animals and/or cells may provide valuable adjunct information and are given a rating of level V.

Table 1 NHMRC levels of evidence

| <i>NHMRC level of evidence</i> | |
|--------------------------------|--|
| I | Evidence obtained from a systematic review of all relevant randomised controlled trials. |
| II | Evidence obtained from at least one properly designed randomised controlled trial. |
| II-1 | Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method). |
| III-2 | Evidence obtained from comparative studies (including systematic reviews of such studies) with concurrent controls and allocation not randomised, cohort studies, case-control studies, or interrupted time series with a control group. |
| III-3 | Evidence obtained from comparative studies with historical control, two or more single arm studies, or interrupted time series without a parallel control group. |
| IV | Evidence obtained from case series, either post-test or pretest/post-test. |
| V | Evidence provided by expert consensus statements, experimental animal and cell studies. |

Source: National Health and Medical Research Council (NHMRC).¹⁴

THE PHYSIOLOGY OF LACTATION

Lactogenesis

Specialised glands that secrete breast milk are already present at birth. However, it is not until puberty that they develop further and during pregnancy they become fully functional. The development of these mammary glands and the initiation of milk secretion from the numerous alveoli containing the milk secreting cells within the gland are regulated by hormonal control. The commencement of this secretory differentiation during pregnancy is referred to as 'lactogenesis stage I'. However, the gland will remain inactive until activated hormonally, initiating 'lactogenesis stage II', the onset of milk secretion occurring during the first four days postpartum.¹⁵

The most important hormones for the initiation and maintenance of lactation are prolactin and oxytocin. Prolactin levels rise throughout pregnancy controlling the final development of the mammary gland secretory mechanism. At the same time high levels of placental progesterone prevent the prolactin from initiating lactation. It is not until the baby is born and the placenta delivered that levels of progesterone fall allowing prolactin to exert its effects on the mammary tissue and initiating stage II of lactogenesis.¹⁶

Once lactation has been established, prolactin is also essential for the maintenance of lactation. In response to the infant's suckling, prolactin is released from the anterior pituitary gland and enables the mammary gland to produce milk before the next feed. Oxytocin, also released in response to the suckling stimulus, promotes the milk ejection reflex and emptying of the breast; however, it is the actual removal of milk from the breasts, in a constant favourable hormonal environment, which controls milk production.^{17,18} The lactating mammary gland exercises a local feedback inhibitory control over milk synthesis, autocrine control, based on a supply = demand feedback loop of control. The frequency or completeness of milk removal from the breast regulates the rate of milk secretion and there does not appear to be a direct relationship between prolactin and milk yield as the autocrine control 'downregulates' milk synthesis to match the mother's supply of milk to the infant's appetite.¹⁹

Milk ejection reflex

The 'milk ejection reflex' or 'milk let down' is responsible for expelling the milk from the alveoli into small ducts leading to the nipple. It is under the hormonal control of oxytocin, which is secreted into the blood stream from the posterior pituitary gland.²⁰ Like prolactin, oxytocin is

released in response to suckling or other stimuli (e.g. hearing the baby cry) and ensures effective emptying of the breast by the infant.^{21,22} Ultrasound imaging of milk ejection indicates that infant milk intake is positively related to the number of milk ejections.²³

THE EFFECT OF ALCOHOL ON THE MOTHER

Maternal blood alcohol concentration

Alcohol enters breast milk by passive diffusion and reflects levels in maternal blood within 30–60 minutes after ingestion (evidence level Lawton; Kesaniemi—NHMRC IV; evidence level Mennella and Beauchamp—NHMRC III-1).^{24–26} Factors that influence the blood alcohol concentration of the mother include body weight, amount of adipose tissue, stomach contents at the time of alcohol ingestion, rate at which alcohol beverages are consumed, and the amount and strength of alcohol in the drink (evidence level—NHMRC V, expert authority).²⁷

Ho and colleagues (evidence level—NHMRC V, experimental) developed a nomogram (Table 2) to guide lactating women who drink alcohol on how to avoid exposure of their infant to ethanol through breast milk.²⁸ Taking into account total body water, blood alcohol concentration and body weight, the average maximal elimination rate of 15 mg/dL/hour ($V_{\max} \times V_d$) was used. Time is calculated from the beginning of drinking, alcohol metabolism is assumed constant at 15 mg/dL, height of the woman is 162.5 cm and one drink is a standard Australian drink serve of 10 g of alcohol. At the end of each time period it is proposed that the alcohol content of the milk will be zero.

Effect of alcohol on lactogenesis and lactational performance

Milk ejection reflex

In many parts of the world folklore suggests that women should drink alcohol (particularly beer) to enhance breast milk supply and promote breastfeeding success. For example in Germany women drink malt beer. In Mexico women are encouraged to drink a local plant fermented juice called pulque daily during pregnancy and lactation, and Indochinese women in California drink wine steeped in herbs to promote successful lactation (evidence level—NHMRC V, review).²² However, it seems the evidence to support this enhanced effect of any source of alcohol on breastfeeding is limited and unsupported.²⁶

Table 2 Alcohol and breastfeeding: time (hour:minute) until the zero level in milk is reached for women at different body weights

| Maternal weight (kg) | No. standard drinks | | | | | | | | | |
|----------------------|---------------------|------|------|------|------|-------|-------|-------|-------|-------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| 45 | 1:54 | 3:50 | 5:45 | 7:40 | 9:36 | 11:31 | 13:27 | 15:22 | | |
| 47 | 1:52 | 3:44 | 5:37 | 7:29 | 9:22 | 11:14 | 13:07 | 14:59 | | |
| 50 | 1:51 | 3:43 | 5:35 | 7:27 | 9:18 | 11:11 | 13:03 | 14:54 | 16:52 | |
| 52 | 1:48 | 3:37 | 5:26 | 7:15 | 9:05 | 10:53 | 12:42 | 14:31 | 16:47 | |
| 54 | 1:46 | 3:32 | 5:19 | 7:05 | 8:52 | 10:38 | 12:25 | 14:11 | 16:21 | |
| 57 | 1:45 | 3:31 | 5:17 | 7:02 | 8:48 | 10:34 | 12:20 | 14:05 | 15:58 | |
| 59 | 1:42 | 3:26 | 5:09 | 6:52 | 8:36 | 10:19 | 12:02 | 13:45 | 15:52 | |
| 61 | 1:40 | 3:21 | 5:02 | 6:43 | 8:24 | 10:05 | 11:46 | 13:28 | 15:29 | 16:50 |
| 63 | 1:38 | 3:17 | 4:56 | 6:34 | 8:13 | 9:52 | 11:30 | 13:10 | 15:09 | 16:27 |
| 66 | 1:37 | 3:15 | 4:53 | 6:31 | 8:10 | 9:48 | 11:26 | 13:04 | 14:48 | 16:20 |
| 68 | 1:35 | 3:12 | 4:47 | 6:24 | 8:00 | 9:36 | 11:12 | 12:48 | 14:42 | 16:00 |
| 70 | 1:33 | 3:07 | 4:41 | 6:15 | 7:50 | 9:24 | 10:57 | 12:31 | 14:24 | 15:40 |

Adapted from the study by Ho *et al.*²⁸

The effect of alcohol in suppressing lactation through its effect on oxytocin was first identified in early studies in rats and later humans (evidence level Fuchs—NHMRC V, animal study; Cobo—NHMRC II).^{29,30} In a study of 40 women Cobo found that ethanol blocks the release of oxytocin and that the degree of inhibition is dose-dependent with ethanol doses between 0.5 and 2 g per kilogram of body weight.³⁰ (Note: 1 g/kg is six standard drinks for a 60-kg woman and results in a blood alcohol level of 0.15 if consumed in one hour.)

Cobo postulates that it is possible that doses higher than 2 g/kg (equivalent to approximately 12 standard drinks) in a 60-kg woman could completely inhibit the suckling-induced oxytocin release in humans. This is a central effect of ethanol as the mammary gland response to exogenous oxytocin was not changed by ethanol. On the basis of comparative studies both Fuchs and Subramanian suggest an inhibitory dose of 1.1–1.5 g/kg ethanol for women (evidence level Subramanian—NHMRC V, animal study).^{29–31}

Beer in quantities ranging from 800 mL to 1 L has been shown to increase serum prolactin secretion in normal men and non-lactating women as beer is reported to have different effects as a galactagogue, unlike ethanol alone (evidence level Carlson *et al.*—NHMRC IV; DeRosa *et al.*—NHMRC IV).^{32,33} However, it has been demonstrated in both human and animal studies that the effect of alcohol regardless of the source (e.g. beer) is at the posterior pituitary, through the effect of oxytocin on milk ejection from the mammary gland in response to suckling, rather than prolactin levels, which are responsible for milk biosynthesis (evidence level Heil and Subramanian—NHMRC V, animal study; Mennella *et al.*—NHMRC II).^{26,31,34,35}

Lactational performance

Lactational performance has been shown to decrease in both animal and human studies of alcohol intake and breastfeeding. Animal model research demonstrates a graded inverse response between alcohol intake and milk yield in alcohol-treated dams (evidence level Tavares do Carmo *et al.*—NHMRC V, animal study; Murillo-Fuentes *et al.*—NHMRC V, animal study).^{31,34,36,37}

Using a within-subjects study design where lactating women are tested with or without alcohol, on two days separated by one week, Mennella and colleagues have consistently shown a diminished milk yield in response to alcohol consumption in lactating mothers (evidence level Mennella and Beauchamp 1991—NHMRC III-1; Mennella 1998—NHMRC III-1).^{26,35,38–40}

Effect of alcohol on breastfeeding initiation and duration

Howard and Lawrence present data on drug use during pregnancy and breastfeeding from the United States 1988 National Maternal and Infant Health Survey (evidence level—NHMRC V, review article).^{41*} Drinking alcohol more than six times per week was equally associated with breast or formula feeding, whereas consuming less than six drinks per week doubled the likelihood of a mother breastfeeding. Early cessation of breastfeeding was most often reported by women with the highest frequency of all drinking patterns, including binge

*The data analysis presented on the National Maternal and Infant Health Survey is part of the review paper by Howard and Lawrence.

drinking, at three months postpartum than women who were continuing to breastfeed, even after preconception habits were taken into account.

A study by Little *et al.* (evidence level—NHMRC III-2) investigated the relationship between levels of maternal smoking and drinking of 463 women at preconception, during pregnancy, and in the postpartum period.⁸ Approximately 80% reported drinking some alcohol in the month before conception, with alcohol use dropping after conception, and only 40% of subjects reporting drinking in the last trimester. After delivery, drinking rose and by the end of the third month postpartum, 69% of the total sample reported some drinking, however, not to the level reported at preconception. Breastfeeding at three months postpartum was generally associated with less drinking, especially less binge drinking (Table 3).

THE EFFECT OF ALCOHOL ON THE INFANT

Infant alcohol absorption

Ethanol is a water-soluble non-polar compound that easily passes through biological membranes to be distributed proportionally throughout the water compartments of the body. The average water content of breast milk is 87.5% and that of blood is 85%. For this reason it is expected that the ethanol concentration at equilibrium would be slightly higher in breast milk.²⁴

The rate of absorption and elimination of alcohol in the breast milk, and level attained in the baby's blood through extrapolation from maternal blood alcohol levels, was investigated by Lawton.²⁴ Eight mothers consumed amounts of alcohol between 0.56 g and 1.5 g per kilogram of body weight. With moderate-to-high intakes, alcohol levels were higher in breast milk than in blood. At lower alcohol intakes, blood and milk alcohol levels were similar. The rate of elimination of alcohol from breast milk and blood were similar. The level of alcohol in breast milk falls as blood alcohol levels fall because retrograde diffusion of alcohol from the milk back to the blood stream occurs. Any alcohol present in milk stored in the breast returns to the blood supply to maintain equilibrium during elimination, regardless of emptying the breasts.^{24,25}

Using the baby of 'subject one' from the Lawton study as an example, the maximum blood alcohol value of the baby can be calculated.²⁴ This baby was six months old and weighed 6.5 kg. This is equivalent to the fifth percentile for boys and the 25th percentile for girls. During the experiment, if the infant consumed 180 mL of breast milk while the mother was near her maximum blood

alcohol level (119 mg alcohol/dL blood, 0.119%) the baby would have consumed 245 mg of alcohol (37 mg/kg body weight). However, taking into consideration the body water content of approximately 0.60 g per kilogram of body weight then the blood alcohol level would rise to approximately 6 mg alcohol/dL blood (0.006%).²⁴

Maternal alcohol intake and infant development

For ethical reasons there are limited human intervention studies on the effect of alcohol on the behavioural state of infants; however, observational studies provide some information in this area. Most research has been performed using small amounts of alcohol consumed by the mother and the subsequent behavioural effect on the infant is then evaluated.

A case report by Binkiewicz *et al.* (evidence level—NHMRC V, case report) documents the effect of chronic excessive alcohol intake by a breastfeeding mother on her four-month-old baby.⁴² A random sample of expressed breast milk contained 100 mg/dL of alcohol and her reported intake was approximately 10 Australian standard alcoholic drinks per day, over a one-week period.

Symptoms evident in the infant at four months were an increased weight gain and a simultaneous slowing in rate of growth. Her length for age was below the third percentile, she was obese, and her facial appearance was 'balloon shaped'. Alcohol increases cortisol levels in the blood and can give rise to a clinical pattern that closely resembles Cushing syndrome. Confirmation of the condition was established by impaired suppressibility of cortisol secretion by dexamethasone and increased excretion of cortisol in the urine. With no other problems she was eventually diagnosed with pseudo-Cushing syndrome, subsequently reversed with the removal of alcohol from the mother's diet.

In a landmark epidemiological study by Little *et al.* (evidence level—NHMRC III-2) 400 infants were investigated to determine the relationship between the mother's use of alcohol during breastfeeding and the infant's development at one year of age.⁴³ The Bayley Mental Development Index was used to measure mental development and the Psychomotor Development Index (PDI) measured motor development. There was a strong inverse linear relationship between chronic exposure to ethanol in breast milk and the PDI. At a clinical level the motor effect was small (4–5% decrease in test scores) with moderate alcohol intake of 1.4–2.8 standard Australian drinks per day. In the small number of infants whose mothers were heavy drinkers (≥ 8.4 standard Australian drinks) there was a 15% decrease in PDI test scores. At a

Table 3 Key articles evidence table; the effect of alcohol on the mother

| <i>NHMRC level</i> | <i>Reference</i> | <i>Key findings</i> |
|-----------------------------------|---|---|
| Effect on blood alcohol | | |
| IV | Kesaniemi (1974) ²⁵ | Ethanol reaches human milk in almost the same concentration as in the blood at 30 minutes after administration. |
| IV | Lawton (1985) ²⁴ | Alcohol appeared in both fore- and hind-breast milk at a level equivalent to or higher than the corresponding blood samples within an hour. |
| Effect on lactational performance | | |
| V | Fuchs (1969) ²⁹ | Lactating dams alcohol intake <ul style="list-style-type: none"> • 1.0 g/kg body weight—no effect on milk removal. • 2.0 g/kg body weight—significant reduction on milk removal. • >2.0 g/kg body weight further reductions in milk yield. • 5 g/kg body weight—complete inhibition of the milk ejection reflex. Ethanol inhibits oxytocin release in the rat. |
| Animal study (rat) | | |
| II | Cobo (1973) ³⁰ | Maternal alcohol intake <ul style="list-style-type: none"> • <0.5 g/kg body weight—no effect on milk ejection reflex. • 0.5–1 g/kg body weight—varying individual effect from no effect to complete block of milk ejection reflex. • 1.5–2 g/kg body weight—decreased milk ejection reflex (average decrease 80%). • >2 g/kg body weight—complete inhibition of the milk ejection reflex. |
| III-1 | Mennella and Beauchamp (1991) ³⁸ | Maternal alcohol intake of 0.3 g/kg body weight (in orange juice) decreases milk intake in infants and is proposed to be a result of a decrease in the milk ejection reflex. ^(a) |
| III-1 | Mennella and Beauchamp (1993) ²⁶ | Maternal alcohol intake of 0.3 g/kg body weight (in beer) decreases milk intake in infants and is proposed to be a result of a decrease in the milk ejection reflex. |
| III-1 | Mennella (1998) ³⁹ | Maternal alcohol intake of 0.3 g/kg body weight (in orange juice) resulted in decreased expressed breast milk yield. |
| V | Subramanian (1999) ³¹ | Alcohol administration in lactating dams (1.0 g/kg body weight and 2.0 g/kg body weight) inhibited the suckling-induced oxytocin release. All pups from alcohol-treated dams had reduced milk intakes. |
| Animal study (rat) | | |
| V | Tavares do Carmo <i>et al.</i> (1999) ³⁶ | Lactating dams alcohol intake <ul style="list-style-type: none"> • Alcohol treated, 20% ethanol diluted in drinking water and food ad lib—decreased milk yield lower than pair fed. • Pair fed, nutritional control receiving a solid diet per day and per 100 g body weight to give an equivalent daily caloric intake as the alcohol rats—decreased milk yield. |
| Animal study (rat) | | |
| V | Heil and Subramanian (2000) ³⁴ | Alcohol administration in lactating dams (1.0 g/kg body weight and 2.0 g/kg body weight). Pups of the 2.0 g/kg groups reduced milk intakes despite elevated suckling-induced prolactin release suggesting alcohol's primary impact is through oxytocin. |
| Animal study (rat) | | |
| V | Murillo-Fuentes <i>et al.</i> (2001) ³⁷ | Three experimental nutritional treatments with ethanol concentration increasing 5% each week over a four-week period starting at 5% week 1. <ol style="list-style-type: none"> 1. Pups exposed to ethanol during gestation only 2. Pups exposed to ethanol during lactation only 3. Pups exposed to ethanol only during lactation All alcohol-exposed pups had decreased milk intake compared with controls. |
| Animal study (rat) | | |
| III-1 | Mennella (2001) ⁴⁰ | Maternal alcohol intake of 0.3 g/kg body weight (in orange juice). Infants consumed approximately 20% less breast milk compared with control conditions. Compensatory intake was observed during the period 8–16 hours after exposure when mothers refrained from drinking alcohol. |
| II | Mennella <i>et al.</i> (2005) ³⁵ | Maternal alcohol intake of 0.4 g/kg body weight (in orange juice) decreased oxytocin levels and increased prolactin levels. The result was a decrease in milk yield and milk ejection. |

Table 3 Continued

| NHMRC level | Reference | Key findings |
|---|--|---|
| Effect on breastfeeding initiation and duration | | |
| Review article | Howard and Lawrence (1998) ⁴¹ | United States 1988 National Maternal and Infant Health Survey. Women who drink less than six alcohol drinks per week almost twice as likely to choose breastfeeding (odds ratio 1.9; $P < 0.05$). Women who weaned early reported the highest frequency of all drinking patterns, and more likely to report binge drinking (RR = 4.1; 99% CI 1.72–9.62), at three months postpartum than women who were still nursing. Women who never breastfed tended to be intermediate or more likely to be nursing women. |
| III-2 | Little <i>et al.</i> (1990) ⁸ | Investigated levels of maternal drinking and smoking ($n = 463$) women prior to, during and post pregnancy. Women breastfeeding at three months postpartum reported less drinking, and less binge drinking than women who never breastfed or breastfed for less than one month. |

^(a) This amount of alcohol approximates the ethanol content of approximately 1.5 standard drinks.

NHMRC = National Health and Medical Research Council; RR = relative risk.

population level these effects could have a considerable impact on community vitality and development. The association persisted even after controlling for over 100 potentially confounding variables (including maternal tobacco, marijuana and heavy caffeine use). No relation was apparent between the infant's exposure to ethanol and the Bayley Mental Development Index.

With the intake of six Australian standard drinks by a 60-kg lactating mother, in one sitting, the ingestion of ethanol through the breast milk is estimated (using the Kesaniemi method)²⁵ to be 232 mg in a 5-kg infant and can be harmful.⁴³ Little *et al.* propose that the ethanol is detrimental possibly because the developing brain is extremely sensitive to ethanol even in very small quantities; or the small quantities ingested during lactation are accumulated in the infant because it is metabolised or excreted more slowly than in adults.⁴³ The authors suggest that serial doses of ethanol accumulate in the infant as supported by the association between an 'absolute alcohol' score (representing the average daily exposure that could accumulate in the infant) and the PDI found in the study by Little *et al.* There was no significant association between the infant's exposure to maternal binges during lactation (which would be less likely to result in an accumulation of ethanol in the infant) and the PDI.

Lawton suggests that occasional exposure of a six-month-old 6.5-kg infant to 245 mg of alcohol (119 mg/dL in mother's blood resulting 37 mg/kg body weight in the infant) is unlikely to have an effect even after taking into account the body water content and low alcohol dehydrogenase activity of the infant.²⁴

Kesaniemi²⁵ concurs with Lawton²⁴ as to the level of maternal alcohol intake suggested not to cause harm to the infant. Kesaniemi states that mothers receiving

approximately 0.6 g/kg body weight ethanol orally would result in maternal blood and milk ethanol levels of $18.2 \pm 2.5 \mu\text{mol/mL}$ (83.7 mg/dL blood) and $16.9 \pm 2.5 \mu\text{mol/mL}$, respectively. At these levels a 5-kg infant receiving 200 mL of milk would receive approximately 180 mg of ethanol or approximately 36 mg/kg body weight, which Kesaniemi states is 'unlikely to have harmful effects on the infant' (p. 84).²⁵ However, both studies used small numbers of women and the alcohol was given very rapidly after fasting conditions.

Despite Lawton and Kesaniemi stating that these levels would not affect the infant, it should be noted that these levels are higher than that in many of the studies found to inhibit the milk ejection reflex^{26,30,38–40} (evidence level Mennella⁴⁰—NHMRC III-1), higher than the level at which motor development in the infant was affected,⁴³ and higher than that recommended by the Institute of Medicine (evidence level—NHMRC V, expert authority).⁹

Maternal alcohol intake and infant (feeding and sleeping) behaviour

The effect of alcohol-flavoured expressed breast milk and unaltered breast milk on the suckling response of infants was tested. The milk was bottle-fed to infants on demand and the pattern of suckling, the amount of milk consumed, and the suckling responses were recorded (evidence level—NHMRC IV).⁴⁴ The alcohol-flavoured breast milk contained 32 mg ethanol/100 mL, the average concentration detected in human milk approximately one hour after lactating women drank an acute dose of 0.3 g/kg alcohol.

Results showed that infants consumed significantly more and sucked more frequently when drinking the

alcohol-flavoured milk. This is inconsistent with the diminished intake by infants of breast milk immediately following mother's exposure to alcohol as reported previously,^{26,38,40} however, in the study by Mennella infants were able to bottle-feed on demand and may have been stimulated by the sweet flavour of the ethanol in the milk to consume and suck more. Mennella's study indicates that infants can readily detect flavours in breast milk and show a distinct preference for the alcohol-flavoured milk over and above the unaltered milk.⁴⁴

Using a within-subject study design described previously,³⁸ Mennella demonstrated a compensatory increase in the number of demand breastfeedings by infants that occurred post exposure to alcohol.⁴⁰ Consistent with previous findings,^{26,38} the infants consumed approximately 20% less breast milk during the first four hours after exposure to alcohol in the mother's milk and then compensated for this diminished intake during the 8–12 hours by increasing the number of breastfeedings that occurred in this time.

Mennella and Beauchamp tested the effect of alcohol ingestion by lactating women, on the odour of breast milk and the subsequent behaviour of the infant.³⁸ The ingestion of alcohol (0.3 g/kg body weight; equivalent to 1.5 Australian standard drinks) significantly altered the odour of breast milk as perceived by a panel of adults. Results demonstrated that the infants sucked significantly more frequently during the first few minutes of an alcohol-exposed breastfeed, and slept for shorter periods and more often, on the day when their mothers consumed alcohol.

Because of the common folklore belief that maternal alcohol consumption can promote sleep in breastfeeding infants, Mennella and Gerrish (evidence level—NHMRC III-1) further tested the effect of exposure to alcohol in breast milk on infants' sleep and activity levels in the short term.⁷ Exposure to alcohol through expressed breast milk (32 mg/100 mL) resulted in definite changes in infant's sleep–wake patterning. All infants slept for the same number of times during each test session; however, there was a significant reduction in the length of time spent sleeping after they consumed the alcohol-flavoured milk compared with the breast milk alone. The reduction in sleep was attributable to a shortening in the longest sleeping bout and the amount of time spent in active sleep. There was no significant difference in the amount of time spent in active sleep during the first half of the 3.5-hour testing session; however, infants spent significantly less time in active sleep during the second half of the testing session (i.e. 1.75–3.5 hours) following alcohol exposure. There was no significant difference in the number of times the infants

breastfed or the amount of milk consumed during these breastfeeds after alcohol exposure.

These results build on previous findings³⁸ in which exposure to alcohol in breast milk altered the infants' sleep–wake patterning such that the infants slept for shorter periods but more often during the day when exposed to alcohol.

To determine if these effects on infant sleep behaviour were a result of the experience to the flavour of the breast milk the authors repeated the study on another group of breastfed infants using non-alcohol-based vanilla in place of alcohol.⁷ However, results show that there was no significant difference in the amount of time the infants spent in active sleep during the 3.5-hour testing session in which they ingested their mothers' breast milk flavoured with vanilla compared with breast milk alone. Nor were there significant differences in the number of sleeping bouts, amount of time spent in quiet or total sleep, latency to sleep, longest sleep bout, or activity levels during wakefulness after exposure to the vanilla-flavoured milk. This suggests that it is not the flavour per se that is responsible for the disruptions in the sleep–wake patterning exhibited after alcohol exposure in breast milk.

Mennella and Garcia-Gomez (evidence level—NHMRC III-1) repeated the alcohol and sleep patterning study by Mennella and Gerrish,⁷ with the exception of extending the monitoring period to 24 hours.⁴⁵

During the first half of the centre 3.5-hour testing session there was no significant difference in the amount of time spent in active sleep. However, during the second half of this session (1.75–3.5 hours) infants exposed to alcohol in the mother's milk spent less time in active sleep, compared with the control condition. Infants then compensated for such decreases in the following 20.5 hours when mothers refrained from drinking alcohol, by exhibiting an increase in active sleep.

Mothers were unaware of changes in their infants' behaviour following exposure to alcohol and it is likely that the decrease in active sleep would go unnoticed as infants tended to fall asleep immediately following alcohol exposure but then woke up shortly afterwards resulting in a decrease in the amount of time spent in active sleep in the hours immediately following exposure to alcohol in the mother's milk.

Together these studies demonstrate that exposure to small amounts of alcohol in the mothers' milk has a direct, although subtle effect, on infant sleep patterning and the infants' ability to modulate behaviours in response to acute ethanol exposure.^{7,38,45} The mechanism for this effect on sleep patterning remains to be explained;^{7,38,45} however, Mennella and Gerrish⁷ propose based on their results and that of others^{43,46} that the

slight deficit identified in the motor development of the children exposed to chronic alcohol intake may be a result of continued disruption of active sleep subsequent to regular alcohol intake (evidence level Ioffe and Chernick—NHMRC III-2).

Animal model studies and experimental studies in humans suggest that pre- and postnatal experiences with the smell and taste of ethanol can affect later responsiveness to ethanol. Breastfed infants (6–13 months old) exposed to ethanol (determined from questionnaires about maternal and paternal alcoholism and alcohol intake) exhibited different behaviours in the presence of ethanol-scented toys compared with less exposed infants. Exposed infants demonstrated increased ‘mouthing’ of the scented toy (evidence level—NHMRC IV).⁴⁷ Whether mouthing the flavour scented toy indicates familiarity with the flavour of ethanol, which in turn leads to a greater willingness to accept ethanol-flavoured substances remains to be investigated.

Growth indices

For ethical reasons animal studies are the only way to determine the long-term effect of alcohol intake on infant development, body weight and metabolism.

The effects of maternal alcohol intake in lactating dams on the development of their offspring were studied using a rat model by Detering *et al.* (evidence level—NHMRC V, animal study).⁴⁸ Results from the study conclusively show that those pups whose dams received ethanol during either the pre- and postnatal period or only in the postnatal period had retarded physical growth that was more severe than that observed as a result of simple malnutrition.

These results are supported in a study by Vilaro *et al.* (evidence level—NHMRC V, animal study) in which the pups of alcohol-treated dams demonstrate a significant reduction in combined weight compared with control pups.⁴⁹ This decrease is associated with reduced milk production in the alcohol-fed dams despite their milk having a higher energy content due to a greater lipid concentration.

In a later study the physical activities, physical growth and the histological appearance of the cerebellum control pups nursed by non-alcohol consuming dams were compared with pups nursed by alcohol-consuming dams (evidence level—NHMRC V, animal study).⁵⁰ Pups exposed to alcohol opened their eyes several days later than pups in the control groups and had a lower average litter weight and brain weight that was evident until alcohol was removed from the diet. These degenerative changes were independent of the pups’ weight. That

study highlights the considerable growth and developmental problems occurring in pups as a result of alcohol intake in the lactating dams and the potential similar harm that could take place in humans with continued alcohol intake during lactation.

Lactational performance, brain and liver composition, circulating metabolites, plasma nutrients and metabolites were investigated in pups fed by ethanol-treated lactating dams.³⁶ The dams in the alcohol-treated group had a decreased milk yield that was associated with a decreased collective weight gain of their pups. These pups also exhibited a decreased brain weight and brain protein. The amount of DNA indirectly reflects the number of cells, and when expressed as DNA per total brain weight the alcohol-exposed pups had reduced values, possibly indicating a lower number of brain cells. This was also apparent in the liver of the alcohol-exposed pups, who also experienced a lower liver weight, lower liver protein and liver glycogen concentration than the control pups.

It is proposed that these lower levels of protein and glycogen are metabolic adaptations in response to the malnutrition being experienced by the alcohol-exposed pups. It is known that the lipid content increases in the milk of alcohol-treated rats.⁴⁹ This high lipid content partially compensates for the alcohol-induced malnutrition occurring in the alcohol-exposed pups and allows the proper metabolic adaptations to prevent severe hypoglycaemia and maintain minimum liver stores of glycogen. However, these adaptations are not enough to protect against impaired brain development, evident in the alcohol-exposed pups.³⁶

These results are supported in a later study by Oyama *et al.* (evidence level—NHMRC V, animal study) who found that pups suckled by alcohol lactating dams (5%, 10% and 20% ethanol) had significantly lower body weights compared with controls.⁵¹ However, only pups of lactating dams exposed to higher alcohol levels experienced a significant decrease in brain weight suggesting a preservation of the pups’ brain or a profound reduction in overall body growth as possible hypotheses for the difference between alcohol groups.

Liver weight of the 5% and 10% alcohol-exposed pups was significantly decreased. ATP-citrate lyase activity is indicative of liver lipogenesis and affected by the composition of the diet. Similar to previous results,³⁶ all alcohol-exposed pups experienced a decrease in liver weight, and there was a decrease in ATP-citrate lyase activity, which could be related to an increased milk lipid content in the alcohol-treated rats.⁴⁹

Results from that study indicate that the effects of maternal alcohol intake on pups’ development and metabolism are dose-dependent and although the low

Table 4 Key articles evidence table; the effect of alcohol on the infant

| NHMRC level | Reference | Key findings |
|---|--|--|
| Effect on infant alcohol absorption | | |
| III-1 | Mennella and Beauchamp (1993) ²⁶ | Estimated by multiplying the milk intake by the concentration of ethanol detected in breast milk and taking into account infant body weight. Estimated dose ranged from 2.3 to 8.4 mg/kg, which is approximately 0.8–2.8% of the maternal dose (0.3 g/kg body weight). |
| Effect on infant development | | |
| Case study | Binkiewicz <i>et al.</i> (1978) ⁴² | Long-term high-level alcohol intake causes pseudo-Cushing syndrome in an infant, subsequently reversed with alcohol withdrawal. |
| III-2 | Little <i>et al.</i> (1989) ⁴³ | Maternal alcohol intake of approximately 0.8 g/kg body weight has detrimental effect on infant motor development. |
| Effect on infant (feeding and sleeping) behaviour | | |
| III-1 | Mennella and Beauchamp (1991) ³⁸ | Maternal alcohol intake of 0.3 g/kg body weight (in orange juice). Infants initially sucked more frequently when mothers had consumed alcohol ($P < 0.008$). No significant difference between the total number of sucks on the two days of testing (control vs alcohol: 856.7 ± 103.4 vs 877.2 ± 102.3). The number of times the infants slept increased on the days when the mother consumed alcohol (6.6 ± 0.7 vs 7.8 ± 0.9 , paired t (11 df) = 2.31, $P < 0.05$). |
| IV | Mennella (1997) ⁴⁴ | Infants consumed significantly more and sucked more frequently when drinking alcohol-flavoured breast milk compared with unaltered breast milk. |
| III-1 | Mennella and Gerrish (1998) ⁷ | Infants bottle-fed the mother's milk alone (control condition) on one test day and the mother's milk containing 32 mg of ethanol per 100 mL on the other, and sleep and activity patterning monitored for next 3.5 hours using an actigraph. Alcohol ingested by the infants was estimated to range from 4.0 to 6.41 mg/kg (mean 5.24 ± 0.2), which is similar to what would be experienced at the breast after the consumption of 0.3 g/kg dose by the mother. All infants slept for the same number of times during each test session; however, there was a significant reduction in the length of time spent sleeping after they consumed the alcohol-flavoured milk compared with the breast milk alone (on average a 25% reduction; 78.2 minutes compared with 56.8 minutes after feeding with alcohol in breast milk). No significant difference in the amount of time spent in active sleep during the first half of the 3.5-hour testing session (control vs alcohol, 18.2 ± 3.8 vs 17.0 ± 4.2 minutes; $P = 0.84$), however, infants spent significantly less time in active sleep during the second half of the testing session (i.e. 1.75–3.5 hours) following alcohol exposure (control vs alcohol, 25.2 ± 5.5 vs 8.6 ± 2.6 minutes; $P = 0.09$). |
| III-1 | Mennella and Garcia-Gomez (2001) ⁴⁵ | Study design as previous ⁷ with testing time extended to 24 hours. During the first half of the centre 3.5-hour testing session there was no significant difference in the amount of time spent in active sleep. During the second half of this session (1.75–3.5 hours) infants exposed to alcohol in the mother's milk spent less time in active sleep, compared with the control condition. Infants exposed to alcohol then compensated for such decreases in the following 20.5 hours when mothers refrained from drinking alcohol, by exhibiting a $22.4 \pm 7.0\%$ increase in active sleep. |
| III-1 | Mennella (2001) ⁴⁰ | Maternal alcohol intake of 0.3 g/kg body weight (in orange juice). For the following four hours infants were videotaped during breastfeeding and were weighed immediately before and after each feeding. Infants demonstrated a compensatory increase in the number of demand breastfeedings. |
| Effect of alcohol on growth indices | | |
| V Animal study (rat) | Deterring <i>et al.</i> (1979) ⁴⁸ | Dams were fed a regular stock diet (control), liquid diet containing 35% of the energy as ethanol (50 g/L resulting in a blood alcohol level of 61 ± 6 mg%), or a liquid diet containing dextrin substituted for the calories supplied by ethanol (isoenergetic = IE). Pups whose dams received ethanol during either the pre- and postnatal period or only in the postnatal period had retarded physical growth that was more severe than that observed as a result of simple malnutrition (the IE diet alone). |

Table 4 Continued

| NHMRC level | Reference | Key findings |
|----------------------------|---|---|
| V Animal study (rat) | Hekmatpanah <i>et al.</i> (1994) ⁵⁰ | Four groups of lactating dams: (I) control with limited food; (II) receiving 5% alcohol and limited food; (III) receiving 10% alcohol and limited food; (IV) control with unlimited food. Pups exposed to alcohol opened their eyes several days later than pups in control groups and had a lower average litter weight and brain weight independent of malnutrition. Myelin formation and the appearance of the Purkinje cells ^(a) was delayed and failed to be as prolific as that of the controls at day 30. |
| V Animal study (rat) | Tavares do Carmo <i>et al.</i> (1999) ³⁶ | Three groups of lactating dams: (I) alcohol treated, received 20% ethanol and food ad lib (AL); (II) pair fed, as a nutritional control received an equivalent daily caloric intake as group I (PF); (III) control rats received a solid diet and tap water ad lib (C). The AL pups had a decreased collective weight gain. The brain weight was significantly reduced in the AL and PF animals ($P < 0.05$) compared with the C group and the brain protein content was decreased in AL pups compared with the other two groups ($P < 0.05$). When corrected for body weight (g/100 g body weight), the brain was heavier in the AL and PF litters than in the controls. The amount of DNA indirectly reflects the number of cells and when expressed as DNA per total brain weight the AL pups had lower values than those of the C or PF pups ($P < 0.05$), possibly indicating a lower number of brain cells. This was also apparent in the liver of the AL pups with the total amount of DNA per liver being significantly ($P < 0.05$) lower in the pups of both the AL and PF dams, suggesting that the liver of these animals had less cells than the C group despite the cell size being the same. The AL and PF pups had a lower liver weight ($P < 0.05$), a lower liver protein ($P < 0.05$) and liver glycogen ($P < 0.05$) concentration than the control pups. |
| V Animal study (rat) | Vilaro <i>et al.</i> (1987) ⁴⁹ | Ethanol-treated dams received ethanol diluted in drinking water with ethanol concentration increasing 5% each week over a four-week period starting at 10% week 1. Pups of alcohol-treated dams demonstrate a significant reduction in combined weight compared with control pups. |
| V Animal study (rat) | Heil and Subramanian (2000) ³⁴ | Lactating dams alcohol intake of 1.0 kg/kg body weight and 2.0 kg/kg body weight. Pups of the 2.0 g/kg groups exhibit lower body weights. |
| V Animal study (rat) | Oyama <i>et al.</i> (2000) ⁵¹ | Pups suckled by alcohol-treated lactating dams (5%, 10%, 20% ethanol solution groups) had significantly lower body weights compared with controls ($P < 0.05$). Only pups of lactating dams exposed to higher alcohol levels (10% and 20%) experienced a significant decrease in brain weight ($P < 0.05$). Liver weight of the 5% and 10% alcohol-exposed pups was significantly decreased ($P < 0.05$). The ingestion of the 5% ethanol solution by the dams decreased pups' brain lipogenesis rate from glucose. |
| V Animal study (rat) | Chen and Nyomba (2004) ⁵² | Offspring of rats exposed to alcohol (36%) during lactation exhibit insulin resistance regardless of having normal birth weight and growth pattern. |

^(a) A specific type of nerve cell that carries each and every piece of information outputted by the cerebellum. These cells possess a great deal of control over the refinement of motor activities.

NHMRC = National Health and Medical Research Council.

intake of ethanol (5%) did not have an effect on brain or liver weight it did have an effect on brain metabolism.

The phenomenon of insulin resistance has more recently been an area of investigation with regard to alcohol intake during lactation. In a study by Chen and Nyomba (evidence level—NHMRC V, animal study)

maternal alcohol consumption during lactation and its effect on glucose homeostasis in rat pups was investigated.⁵² Results demonstrate that the offspring of rats exposed to alcohol during lactation exhibit insulin resistance regardless of having normal birth weight and growth pattern. Despite a lack of clarity in determining

the mechanism for this effect, the study highlights the importance of lactation as a vulnerable period for the future metabolic homeostasis of the infant (Table 4).

CONCLUSION

Alcohol is almost ubiquitous in Australian society and is commonly consumed, including during lactation. The evidence available to give advice to lactating mothers is less than ideal and must rely on a combination of experiments, observational studies and animal data. The evidence supporting severe limitations on the consumption of alcohol during pregnancy is abundant⁴ and robust guidelines outlining recommendations for alcohol intake during this time are well documented.³ However, there is a paucity of scientific information about the effect during lactation making it harder to give definitive recommendations.

In animal and human studies alcohol has been shown to disrupt the hormonal control of lactation by decreasing the milk ejection reflex through the inhibition of oxytocin. Doses as low as 0.3 g/kg body weight (equivalent to 1.5 standard Australian drinks) have been reported to have an inhibitory effect with a subsequent decrease in milk intake by infants. Most often undetected, this decrease in intake with regular low-level alcohol consumption over an extended period of time could contribute to a significant decrease in milk intake and a resulting decline in infant body weight, growth and other vital developmental indices.

Ethanol is water-soluble and enters the breast milk by passive diffusion, reflecting maternal blood levels (or higher) within 30–60 minutes. The removal of alcohol from breast milk and blood are similar and the level of alcohol in breast milk will fall as blood alcohol levels fall because of retrograde diffusion of alcohol from the milk back into the blood stream.

Despite the popular folklore belief that consuming alcohol when breastfeeding will promote lactation,⁵³ and relax the infant and mother, the available research provides evidence to the contrary. Exposure to small amounts of alcohol in the mothers' milk has a direct effect on infant sleep patterning resulting in significantly less time spent in active sleep immediately after exposure to alcohol in breast milk. It is important for mothers to establish sound breastfeeding patterns in the first month and if a mother has a restless baby (as most are in the first few weeks) the introduction of alcohol may exacerbate this restlessness, prompting her to discontinue breastfeeding at this critical time. The authors advise nursing mothers to restrict all alcohol intake during this first month in an effort to provide the most optimal environment to support continued breastfeeding.

Table 5 Suggested advice for alcohol intake of breastfeeding mothers

-
1. No alcohol in the first month.
 2. After that—limit alcohol intake.
 - a. Preferable 1–2 standard drinks per day
 - b. Drinking just after breastfeeding
 3. If wanting to drink more than 2 then expressing milk in advance and skipping one feed may be an option to consider.
-

Early cessation of breastfeeding has been associated with a high frequency of alcohol consumption during lactation, even after controlling for confounders. Promoting a recommendation to reduce or eliminate alcohol intake during lactation would help foster the mindset of abstinence that appears to be so easily maintained during pregnancy. By preparing women for a continued abstinence of alcohol following pregnancy, women may be more inclined and mentally prepared to maintain this behaviour throughout lactation therefore possibly promoting prolonged breastfeeding duration.

Based on the available evidence the authors suggest the prudent use of alcohol and strongly recommend that lactating mothers consume only one to two standard drinks after breastfeeding. Advice restricting alcohol consumption during the first month of breastfeeding and providing direction on levels of consumption and timing of intake will enable lactating women to consume alcohol in quantities and conditions conducive to the optimal development of their young infant while supporting successful breastfeeding (Table 5).

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