

Alcohol levels in Chinese lactating mothers after consumption of alcoholic diet during postpartum “doing-the-month” ritual

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Received 25 August 2005; received in revised form 14 February 2006; accepted 14 February 2006

Abstract

This study examined the effects of exposure to ethanol through cultural practices by lactating mothers. Specifically, the pharmacokinetics of alcohol in Chinese lactating mothers was investigated after they consumed chicken soup flavored with sesame oil and rice wine (CSSR), a typically prescribed diet during the postpartum “doing-the-month” period. Experimental findings were employed to estimate the potential ethanol dose to neonates and determine associated health risks. Twenty-three lactating mothers were examined. Informed consent was obtained from each subject. The target alcohol dosage was 0.3 g/kg. Milk and blood samples were collected at fixed time intervals from each subject following exposure to CSSR, and alcohol levels were determined. Acute health risks to infants were estimated by comparing the potential infant dosage to an established criterion dose. Blood alcohol level peaked at 20 min after exposure to CSSR and decreased almost linearly thereafter. Alcohol in milk reached a plateau roughly at 20–40 min after exposure to CSSR and then decreased. Alcohol pharmacokinetics among subjects varied widely. The coefficients of variation in subject alcohol concentrations were 16.5–46.2% (mean, 30.0%) for blood and 32.8–57.6% (mean, 44.4%) for milk. Mean maximal alcohol concentration in blood (30.2 ± 5.0 mg/dl) was achieved at 23.5 ± 7.6 min and in milk (31.6 ± 10.3 mg/dl) at 31.7 ± 12.7 min. Potential infant doses were 3.0–58.8 mg (mean, 13.4 mg), and the predicted time required for milk alcohol level to return to zero level was 175 min. The acute health risks for infants exposed to alcohol through their mothers’ milk under the current exposure scenario are low (hazard index < 0.2). Nursing infants at least 3 h after ingesting a diet containing alcohol would further reduce potential health risks. © 2005 Elsevier Inc. All rights reserved.

Keywords: Alcohol; Ethanol; Milk; Infant; Health risk; Pharmacokinetics; Doing-the-month

1. Introduction

Maternal milk is recognized as the optimal source of nourishment for infants (DHHS, 2000; WGB, 1997). However, breast-fed infants are also exposed to environmentally persistent, bioconcentrated contaminants and industrial chemicals stored in the fat in mother’s milk (Coveney, 1985; Rogan, 1996; Somogyi & Beck, 1993; Sonawane, 1995). Additionally, therapeutic drugs (Ito, 2000) and recreational agents, that is, alcohol, caffeine, nicotine, and marijuana (Liston, 1998), may also enter breast milk. These agents can negatively affect milk production, volume, flavor, and composition and, thereby, affect nursing infant health.

Cultural factors, that is, maternal traditions, may also expose infants to harmful chemicals. For example, specific ethnic traditions have encouraged lactating mothers to drink alcoholic beverages as galactagogue (Flores-Heurta et al.,

1992; Walter, 1975) or to sedate their infants (Adams & Davidson, 1987). In such cases, alcohol imbibed by breast-feeding mothers is passed to nursing infants through breast milk (Argote-Espinosa et al., 1992; Mennella & Beauchamp, 1991). Chicken soup flavored with sesame oil and rice wine (CSSR) consumed by Taiwanese (ethnically Chinese) lactating mothers during the traditional Tso-Yueh-Tzu ritual is such an example, in which the mother-milk–infant pathway exposes an infant to alcohol. Tso-Yueh-Tzu, also known as “doing-the-month,” is a 30-day culturally defined postpartum period of prescribed behaviors deemed beneficial to convalescing mothers. The Tso-Yueh-Tzu ritual has many “should do” activities, such as eating certain foods, and a number of “should not do” behaviors, such as bathing, reading, or lifting heavy objects (Cheung, 1997; Liu-Chiang, 1995; Pillsbury, 1982). Based on Chinese folk medicine, the ingredients of CSSR are believed to benefit new mothers by enhancing energy and nutrient/protein intake, increasing peripheral blood circulation and facilitating recovery by redressing the “hot–cold”

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imbalance resulting from pregnancy (Liu-Chiang, 1995). Although CSSR is typically ingested during the Taiwanese Tso-Yueh-Tzu practice, its potential effect on mother alcohol pharmacokinetics and subsequent health risks to the nursing infant have not been investigated.

Experimental studies, which examined the pharmacokinetics of alcohol in milk and blood of mothers after they consumed alcoholic beverages, indicated that alcohol levels in milk are generally indicative of total body alcohol burden as alcohol concentrations in milk are very close to those in blood (Argote-Espinosa et al., 1992; da-Silva et al., 1993; Kesaniemi, 1974; Lawton, 1985). Large variations in body alcohol levels exist among individuals due to variability in alcohol absorption, body composition, and metabolism (Holford, 1987; Jones & Jonsson, 1994; Li et al., 2000; Norberg et al., 2003; Patrick et al., 1995; Ramchandani et al., 2001), especially when food is in the stomach (Jones et al., 1997). Chemicals excreted in milk are modified by numerous factors, such as milk fat content (Casey & Hambidge, 1983), parity (Noren, 1983), duration of lactation (Rogan et al., 1986), a mother's metabolism (Mizoi et al., 1987), absorption in the gastrointestinal (GI) tract (Drummer, 2001; Gentry, 2000), and, based on animal study, and based on rat study, number of pups suckling (Ring et al., 1990). Although variation in milk alcohol levels after acute alcohol dosing is expected, relevant studies are scarce.

This study examined the concentration–time relationship of alcohol in Chinese lactating mothers after they consumed CSSR. The experimental results were used to assess the effect of CSSR ingestion on alcohol pharmacokinetics of lactating mothers, estimate the potential ethanol dose to neonates, and evaluate the associated health risks to neonates.

2. Methods

2.1. Study design

The study protocol in general followed previously published studies (Mennella, 1998; Mennella & Beauchamp, 1991). Each experiment was performed approximately 15 days after delivery and was conducted at a commercial “Tso-Yueh-Tzu” center, a maternity health care setting, in which subjects resided for roughly 1 month after being discharged from hospitals. Blood and milk samples were obtained from each subject at fixed time intervals during a 3-h test period after they had imbibed CSSR. Alcohol levels in samples were then determined. Infant risk associated with alcohol exposure through breast milk was assessed using a hazard index, which is the estimated worst-case infant dose divided by a reference dose.

2.2. Ethics

The study enrollment criterion was that CSSR should be part of subjects' normal diet after delivery. The study protocol was reviewed and approved by The Committee on

Human Study at Taipei Medical University. Informed consent was obtained from each subject.

2.3. Subject selection

Twenty-three healthy, nonsmoking, pregnant Chinese women were recruited prior to delivery from gynecology and obstetrics clinics at Taipei Medical University Wan-Fang Hospital (Taipei, Taiwan). The test began approximately 15 days after delivery. Subject age, height, weight, body mass index, body adipose rate, expressed as mean \pm S.D., were 24.5 ± 3.4 years, 158.8 ± 6.5 cm, 62.5 ± 9.6 kg, 24.6 ± 2.6 kg/m², and $37.1 \pm 6.6\%$, respectively. Nineteen mothers were primiparous and four were multiparous.

2.4. Preparation of CSSR

The CSSR, the sole source of alcohol in this study, was prepared using commercially published recipes. The soup comprised black sesame oil, deboned chicken breasts, aged ginger, and rice wine (alcohol 19.5%, Taiwan Tobacco and Liquor Co., Taiwan). The alcohol level in CSSR decreased with cooking time (the alcohol concentrations were approximately 128, 122, 122, 116, 97, 74, 60, 33, 19, 15, 13 and 9 mg/ml for cooking times of 0, 10, 20, 30, 40, 50, 60, 70, 80, 90, 100 and 110 min, respectively) and reached approximately 40 mg/ml after cooking for 65 min, which was adopted for further CSSR preparation. Alcohol concentrations in CSSR were found to be stable (variable within $\pm 4\%$ of the initial level) when frozen (-10°C) for 1 month. Suitable amounts of CSSR were then prepared and stored in a freezer in small portions (soup liquid only) for experimental use. Since the entire study was conducted over a few months, CSSR was prepared on three different occasions, with the alcohol levels and other macronutrients being analyzed after each preparation. The CSSR alcohol level was determined to be 40.6 ± 1.8 mg/ml in all three preparations. A target alcohol dosage of 0.30 g/kg of body weight was achieved by administering ~ 8 ml of soup for each kg of subject body weight. This alcohol level was comparable with that used in other studies and within an acceptable taste range.

The average macronutrient levels in CSSR, that is, water, crude ash, crude protein, crude fat, and carbohydrates, and energy were measured using the standard methods of the from Association of Official Analytical Chemists (AOAC, 1980). In particular, water and crude ash were determined by the heat-evaporation/weighting method; crude protein was analyzed using Kjeldahl-H₂SO₄ titration; crude fat was determined by the hexane-extraction and heat-evaporation/weighting method; and carbohydrates were estimated by subtracting the weights of water, crude ash, crude protein, and crude fat from the sample weight; and energy level was calculated based on the formula energy (kcal) = crude protein (g) \times 4 + carbohydrates (g) \times 4 + crude fat (g) \times 9. The concentrations of these ingredients were 83.72 ± 0.00 , 0.41 ± 0.01 , 1.39 ± 0.01 ,

1.95 ± 0.12 , and 1.51 ± 0.03 g/dl, respectively, and the energy level was 28.8 ± 1.10 Kcal/dl.

2.5. Sampling protocol

Subjects were asked to refrain from imbibing alcohol for 3 days prior to the experiment to ensure a low baseline alcohol level; compliance was confirmed by 3-day dietary records (Mennella & Beauchamp, 1991).

On the morning of the sampling day, each subject was weighed, blood sample was taken, and milk from each breast was emptied with an electric breast pump, which was operated under the same intensity throughout the experiment. The alcohol levels in these milk and blood samples were used as baseline levels (time 0). Two servings of a cereal snack (~150 kcal) were then administered to each subject before CSSR intake. One hour later, the CSSR (after heating in a microwave oven) was consumed by the subjects within 15 min after experiment initiation. Subjects were allowed to tend to their infants but not breast-feed.

After the subjects consumed CSSR, milk samples (2 ml) were obtained from each of them at 10, 20, 30, 40, 60, and 90 min using an electric breast pump. At 120 min post-CSSR exposure, milk was emptied from both breasts using electric breast pumps. Volumes excreted were measured for infant alcohol dose calculations. Since this procedure was 30 min long (15 min for each breast), the midpoint (135 min) was adopted as the sampling time in further analysis. Venous blood samples (2 ml) were obtained using an indwelling catheter before alcohol dosing and at 20, 40, 60, 90, and 150 min after subjects consumed CSSR. The samples were drawn into Vacutainer tubes containing heparin salt. Catheter tubes were flushed with diluted heparin solution after each sampling (Jones et al., 1997). Blood and milk samples were centrifuged at 2,000 and 4,000 rpm, respectively, and upper layers were then stored at -80°C for further analysis.

2.6. Analysis of alcohol

Alcohol levels in milk and CSSR were analyzed using a gas chromatograph (GC) equipped with a flame ionization detector (Model 6890, Hewlett Packard Inc., DE, USA). One microliter of sample was injected directly into the GC. A capillary column (Part CP-WAX 52CB, 30 m \times 0.53 mm internal diameter, 1 μm thickness; Varian Chrompack Inc., CA, USA) with nitrogen as the carrier gas running at 6 ml/min. The oven temperature program comprised an initial temperature of 70°C for 4 min, increased to 100°C at a rate of $10^{\circ}\text{C}/\text{min}$ and maintained at 100°C for 3 min. Milk samples were analyzed within 7 days as a laboratory test (spiked sample) demonstrated that samples were stable (variable within $\pm 5\%$ of the initial level) during this period. The method detection limit was 2.47 mg/dl, and the average analytical precision was 2.16 (coefficient of variation [CV], %) across a dynamic range (2.5–50 mg/dl).

Blood (serum) alcohol levels were analyzed using a commercial test kit (Vitros ALC Slides, Ortho-Clinical Diagnostics, Inc., NY, USA). Ethanol concentration in each sample was determined by measuring the increase in NADH (reduced form of nicotinamide adenine dinucleotide) concentration at 340 nm after a 5-min incubation at 37°C . The lower limit of the reportable range was 10 mg/dl, and the average analytical precision was 1.79 (CV, %) across a dynamic range (10–300 mg/dl).

2.7. Data analysis

Summary statistics were expressed as mean \pm S.D. Concentration–time profiles of ethanol in blood and milk were plotted for average levels, as well as for each subject. Peak levels and time required to peak after alcohol exposure were recorded. Differences in time to peak and peak levels between blood and milk alcohol concentrations were analyzed using a nonparametric Wilcoxon signed-rank test (Ammon et al., 1996). A value of $P < .05$ was considered statistically significant. The areas under the concentration–time curve for milk (AUC_m) were determined via a linear trapezoidal method (Matthews et al., 1990). Correlations between blood and milk alcohol levels for each subject were determined using linear regression analysis. The correlation coefficients were obtained on the basis of the data collected at 20, 40, 60, and 90 min. Correlation analysis using pooled data for all subjects was also performed. Alcohol doses potentially available to infants were estimated on the basis of total milk yielded in 30 min and the highest alcohol levels in mother's milk with complete absorption assumed. Time required for the milk alcohol level to return to zero level was estimated using linear least-square extrapolation based on descending phase data as an earlier study has demonstrated a zero-order (linear) kinetics during this phase (Mumenthaler et al., 2000). The ethanol disappearance rate was obtained from the slope of the regression line.

3. Results

Fig. 1 presents the mean blood and milk alcohol levels for 23 subjects. Mother blood alcohol levels peaked at 20 min after ingestion of CSSR and decreased almost linearly to zero level (i.e., zero-order kinetics) after roughly 3 h. However, milk alcohol levels followed a different pattern, that is, they peaked at around 20–40 min and decreased linearly thereafter. At 135 min post-CSSR consumption, alcohol concentrations in milk were 9.0 ± 5.2 mg/dl, significantly higher than the pre-CSSR consumption level. The mean time required for milk alcohol levels to return to zero level, defined herein as half the analytical detection limit, was estimated at approximately 175 min based on the following linear regression equation: milk alcohol level = $-0.193 \times \text{time} + 35.1$ ($r^2 = 0.999$, $P < .05$). The mean ethanol disappearance rate

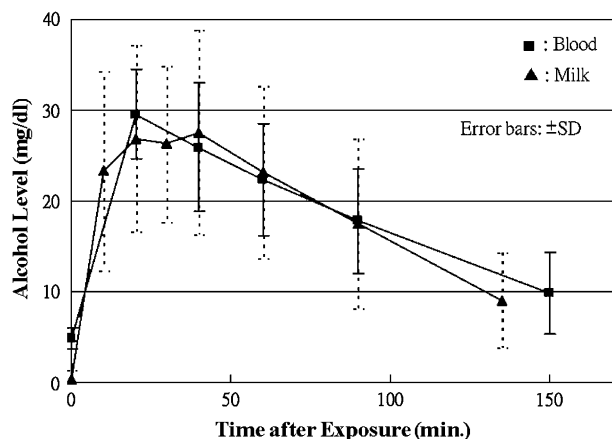


Fig. 1. Mean concentration–time curves for alcohol levels in blood (closed squares) and milk (closed triangles) of 23 lactating women after they consumed alcohol at a dosage of 0.3 g/kg in soup.

in milk was 0.193 mg/dl/min or 116 mg/l/h. Conversely, alcohol concentrations in blood at 150 min post-CSSR consumption (9.8 ± 4.5 mg/dl) were below the detection limit and, therefore, indistinguishable from 0. The mean blood ethanol disappearance rate was 90 mg/l/h, based on the following equation: blood alcohol level = $-0.15 \times \text{time} + 31.9$ ($r^2 = 0.994$, $P < .05$).

Fig. 2 shows individual blood and milk alcohol concentration profiles following CSSR consumption for 23 subjects. Time to peak milk alcohol levels varied among subjects; average peak time for milk alcohol was 31.7 ± 12.7 min postexposure. The time to peak blood alcohol levels also varied among subjects. Average peak time for blood alcohol was 23.5 ± 7.6 min, occurring statistically faster than ($P < .05$) that in milk. Mean maximal milk alcohol concentration in this study was 31.6 ± 10.3 mg/dl, and not statistically significantly different ($P > .05$) from the mean maximal blood alcohol concentration (30.2 ± 5.0 mg/dl). The AUC_m for the 23 subjects were $2,621 \pm 924$ min \times mg/dl (range, 1,395–4,678 min \times mg/dl).

The correlation coefficients between blood and milk alcohol levels, determined for each individual, were variable (range, -0.96 to 0.99 ; median, 0.79 ; mean, 0.62). Six of the correlation coefficients (subjects 1, 4, 13, 14, 16, 22) reached significant level ($P < .05$). The correlation coefficient between blood and milk alcohol levels based on pooled data from all subjects was 0.769 ($P < .05$).

4. Discussion

4.1. Alcohol pharmacokinetics in lactating mothers

4.1.1. Alcohol pharmacokinetics curves

Alcohol levels in milk after CSSR consumption peaked at roughly 20–40 min and decreased nearly linearly thereafter (Fig. 1). Blood alcohol levels followed a similar pattern as in milk, but increased to a maximum at 20 min.

Individual milk alcohol concentration curves (Fig. 2) demonstrated that large variations existed among subjects, as evidenced by individual kinetics patterns, for example, multiple peaks (cases 3, 10, and 12), fast increase/decline (cases 8 and 14), and plateau (cases 2, 19, and 21). On the other hand, alcohol concentration–time curves for blood were less variable than those for milk. It is evident that the summary curve (Fig. 1), though easier for interpretations and comparisons, comprised widely ranging curves and, thus, likely oversimplified results (Matthews et al., 1990).

An earlier study, which employed the same dose level as this study and examined the effects from different food contents on alcohol pharmacokinetics, demonstrated an average CV of 36.7% in areas under the concentration–time curve for breath alcohol levels (Jones et al., 1997). In this study, large variations were also observed in the AUC_m, which represented the alcohol dose available to infants. The coefficient of variation for AUC_m was 35.3% (range, 1,395–4,678 min \times mg/dl). Such variations were closely related to the complexity of alcohol concentration–time curves among subjects (Fig. 2).

4.1.2. Time to peak and peak levels

The time required for milk alcohol levels to peak varied among subjects (Fig. 2), that is, $\sim 39\%$ ($= 9/23$) of subjects had maximal milk alcohol levels at 40 min after consuming CSSR, followed by 30.4% ($= 7/23$) at 20 min and 21.7% ($= 5/23$) at 30 min. Average peak time for milk alcohol was 31.7 ± 12.7 min post-CSSR consumption. This analytical finding was comparable to the peak times of 30–60 min obtained by Mennella and Beauchamp (1991). Notably, peak times determined in experimental studies may be affected by the times at which samples are obtained. For example, a study by Argote-Espinosa et al. (1992) determined a peak time for milk of 60 min, approximately 30 min later than that obtained in this study. This discrepancy likely resulted from the different sample collection schemes in this study and the study by Argote-Espinosa et al., which collected samples only at 60 and 120 min post-alcohol exposure. This study collected milk samples more frequently, that is, 10, 20, 30, 40, and 60 min, in the first hour after subjects consumed CSSR and, thus, achieved superior resolution in determining peak levels.

Time required for blood alcohol levels to peak also varied among subjects: blood alcohol levels peaked in 82.6% ($= 19/23$) of subjects at 20 min and in 17.4% ($= 4/23$) of subjects at 40 min (Fig. 2). Average time to peak for blood alcohol (23.5 ± 7.6 min) was significantly less ($P < .05$) than that for milk alcohol (31.7 ± 12.7 min).

Mean maximal milk alcohol concentration in this study was 31.6 ± 10.3 mg/dl. This finding is consistent with that (~ 30 mg/dl) obtained in a study by Mennella and Beauchamp (1991) that employed a similar dose. It should be noted that peak blood alcohol concentrations are in general proportional to the dose administered (O'Neill et al., 1983; Wilkinson et al., 1977); however, time to peak may be

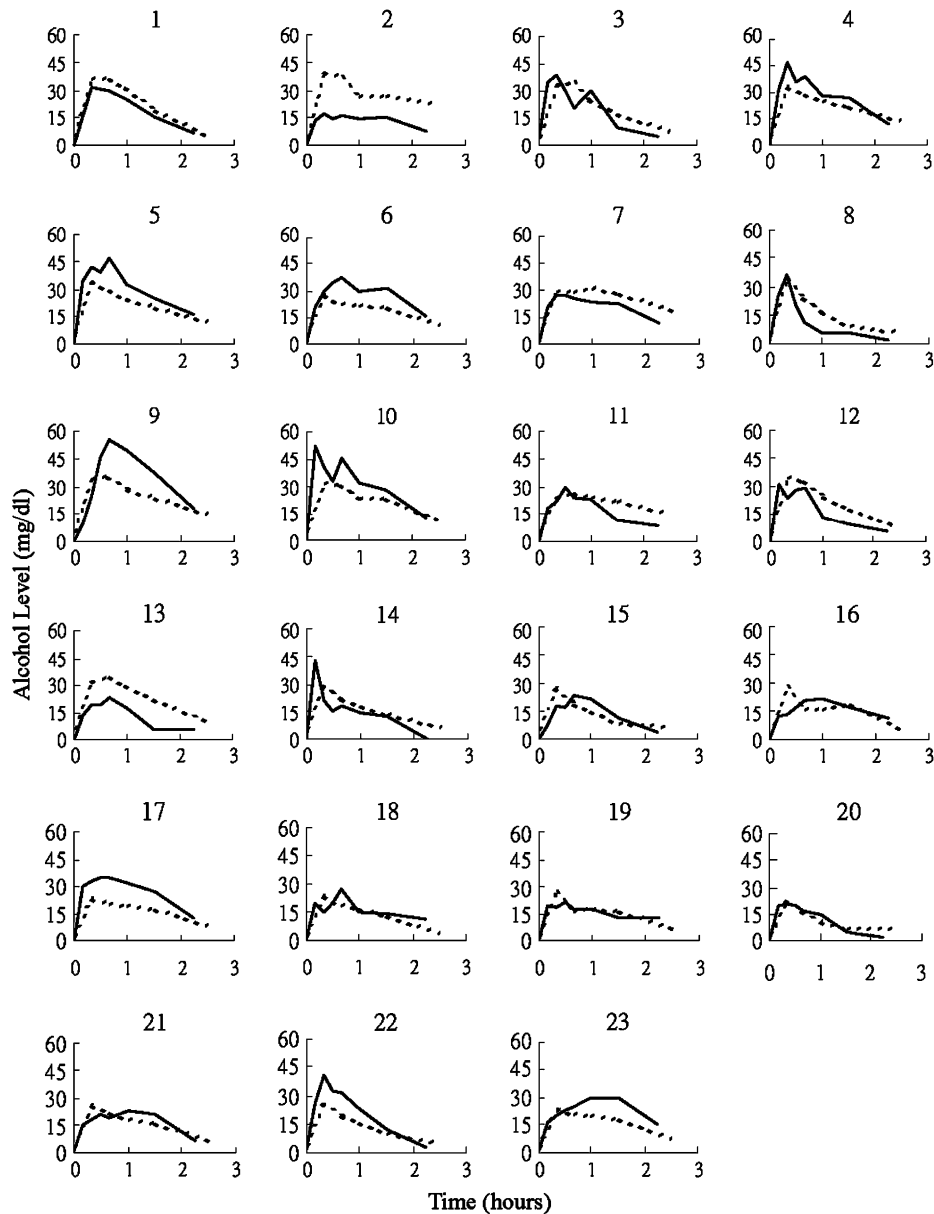


Fig. 2. Alcohol levels in blood (dotted line) and milk (solid line) over time for the 23 lactating women after they consumed alcohol at a dosage of 0.3 g/kg in soup.

delayed after a larger alcohol dose (Dubowski, 1985; Wilkinson et al., 1977). No significant difference ($P > .05$) was noted between mean maximal blood (30.2 ± 5.0 mg/dl) and milk alcohol concentrations in this study.

A study by Patrick et al. (1995) found that shorter time to peak for breath alcohol levels was indicative of a larger maximal breath alcohol level. However, in the current study, time to peak blood and milk alcohol levels versus maximal levels were not significantly correlated.

After peaking, alcohol levels in milk and blood decreased following zero-order kinetics. At 135 min post-CSSR consumption, alcohol concentrations in milk (9.0 ± 5.2 mg/dl) remained significantly higher than those at preconsumption. The disappearance rates of alcohol in

blood and breath were found to be proportional to the doses received (Martin et al., 1984; O'Neill et al., 1983). The mean blood ethanol disappearance rate in this study was 90 mg/l/h, which was in good agreement with the linear extrapolation result from higher dose levels (Martin et al., 1984). On the other hand, the mean ethanol disappearance rate in milk was 0.193 mg/dl/min (or 116 mg/l/h).

The mean time required for milk alcohol level to return to zero level was estimated at approximately 175 min based on linear extrapolation. This prediction was similar to the estimate in a recent study in which almost 160 min were required for milk alcohol level to reach 0 for a 62-kg (mean weight of subjects in this study) mother who consumed 0.3 g/kg of alcohol (Ho et al., 2001). Thus, despite

interindividual variation, this time frame (160–180 min) should prove effective in assisting women in minimizing neonate exposure to alcohol in maternal milk when receiving similar alcohol doses.

4.1.3. Correlation between milk and blood alcohol levels

Correlations between blood and milk alcohol levels were analyzed to determine whether milk alcohol level was a good indicator of alcohol body burden (blood alcohol level). The correlation coefficient between blood and milk alcohol levels based on pooled subject data was 0.769 ($P < .05$), suggesting that alcohol concentrations in milk were generally closely indicative of those in blood. This finding is consistent with previous alcohol pharmacokinetics results after alcohol consumption in humans (Argote-Espinosa et al., 1992; da-Silva et al., 1993; Kesaniemi, 1974; Mennella & Beauchamp, 1991) and in rats (Pepino et al., 1998). However, large between-subject variations in correlation coefficients were observed when examining individually; that is, they ranged from -0.96 to 0.99 (median, 0.79) with six of them being statistically significant. This variation was due to large differences in alcohol concentrations in samples collected at the same time points. Specifically, the CVs in subject alcohol concentrations were 16.5–46.2% (mean, 30.0%) for blood and 32.8–57.6% (mean, 44.4%) for milk. And such variability was comparable to the previous findings on mother blood and milk alcohol levels (Argote-Espinosa et al., 1992; da-Silva et al., 1993).

Milk is not a homogeneous matrix and, consequently, chemicals excreted in milk are affected by numerous factors, as described earlier. This study accounted for three factors impacting alcohol pharmacokinetics: alcohol doses were administered based on subject body weight; time alcohol remained in the GI tract was controlled by administering two servings of cereal prior to alcohol consumption; and the rate at which alcohol was consumed was controlled as subjects were required to finish their soup in 15 min. However, other uncontrolled factors may have contributed to interindividual variation in alcohol concentrations. For example, one subject's skin flushed red after ingesting CSSR. This subject's blood alcohol levels were comparable to those in other subjects; however, this subject's milk alcohol levels were lower compared to those in other subjects (Fig. 2, subject 2). And preparation and serving of CSSR may have contributed to some variation in mother alcohol doses, even though the alcohol levels in CSSR remained stable during storage.

4.2. Infant dose and health risk

The long-term health risk for infants, nursed by mothers who ingest small amounts of alcohol, remains unclear despite some critical findings. For example, earlier studies indicated that alcohol in mother's milk impacted infant sleep patterns by decreasing total and active sleep time (Mennella & Gerrish, 1998) and detrimentally hindered infant

psychomotor development (Little et al., 1989). Little (1990) believed that future studies with improved measurement tools and enhanced follow-up periods and sample sizes would elucidate the long-term health effects of alcohol on infants. On the other hand, research on short-term effects of maternal alcohol consumption on lactational performance found that human infants consumed approximately 20% less breast milk during the immediate hours after their mothers consumed an acute dose of alcohol (Mennella & Beauchamp, 1991). Less milk produced after maternal alcohol consumption was the cause of such reduction (Mennella, 1998).

The acute health risks for infants exposed to low levels of alcohol were estimated using a hazard index approach, that is, by comparing the potential infant dose under worst-case scenario to a reference dose. The potential infant doses in this study were 3.0–58.8 mg (mean, 13.4 mg), based on the amount of milk yielded in 30 min (data not shown) and peak alcohol levels in mother's milk with complete absorption assumed. The reference doses that cause acute neuronal dysfunction and death for 3-kg infants were estimated at 450 mg and 9 g, respectively (American Academy of Pediatrics, Committee on Drugs, 1984). Thus, the acute health risks under the exposure scenario in this study are low, as hazard indices were <0.2 . The alcohol body burden in mothers increases as the result of continued consumption of CSSR; however, it is unlikely that the resultant infant dose would reach even 50% of the reference level.

4.3. Implications of findings

Mother's milk contaminated with alcohol deserves further attention as this pathway is controlled by mothers, in contrast to involuntary environmental and occupational exposures. Although not supported by scientific evidence, alcoholic beverages have been prescribed by various cultural customs, and even by some health professionals, to primarily increase milk yield, facilitate milk excretion, and sedate infants. Ironically, alcohol consumption by breast-feeding mothers has been shown to reduce, rather than increase, milk production (Mennella, 1998), and lactogenesis is enhanced by a barley polysaccharide, rather than alcohol, in beer (Koletzko & Lehner, 2000). The consumption of CSSR by lactating mothers described in this study is part of the Taiwanese Tso-Yueh-Tzu ritual. The goal of Tso-Yueh-Tzu is to promote postpartum recovery and health. The practice of Tso-Yueh-Tzu, which can be traced back to the Sung Dynasty (960–1279 A.D.), comprises a ~30-day postpartum period of prescribed behaviors deemed beneficial for convalescing mothers. Translated into English, Tso-Yueh-Tzu means "implementing a one-month ritual"; the practice requires new mothers to stay in one location (either a home or a commercial Tso-Yueh-Tzu center). Although contemporary Taiwanese (Chinese) women question certain taboos in Tso-Yueh-Tzu, such as not washing the hair for an entire month (Leung et al., 2005; Liu-Chiang,

1995), eating prescribed foods, such as CSSR, remains a crucial practice in Tso-Yueh-Tzu (Chan et al., 2000; Holroyd et al., 1997; Liu-Chiang, 1995; Whang, 1981). Unfortunately when a mother's alcohol body burden is elevated as a result of alcohol intake, the opportunity for infant exposure through breast milk increases.

Maternal alcohol ingestion has no obvious benefit to infants. Likewise, very small alcohol doses consumed by infants during their early months can negatively affect short-term physiological responses and long-term development, even when the infant alcohol dose is low and less than the quantitation limit in most laboratories (Jones, 1992). Thus, it is crucial that use of alcoholic drinks/foods is not advocated during lactation.

Mother's milk is a valuable food source with confirmed merits; however, breast milk may not be suitable in all instances. This study and previous studies have clearly demonstrated that, although a mother's alcohol levels continually declined after acute consumption of alcohol-based foods or drinks, alcohol levels remained elevated in breast milk for a few hours before returning to zero level. Thus, lactating Taiwanese mothers should only breast-feed their infants at least 3 h after ingesting CSSR or other alcohol-containing foods or collect/store their breast milk for feeding during this period to ensure that milk is free of alcohol to minimize infant alcohol exposure and its associated health risks.

Acknowledgment

This study has been supported by the funding from the HungKuang Committee of Academic and Research Development (HKC-89-B-004) and internal funds from Taipei Medical University.

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