The Effects of Maternal Passive Smoking on Maternal Milk Lipid

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Abstract- Passive smoking was long overlooked by those in the medical and legal professions as being harmful to one's health, but in recent years the negative effect of passive smoking has come to the fore in the media and laws have been changed so that less people are obliged to unwillingly suffer from passive smoking, particularly in the workplace and in indoor settings. To study the effects of environmental tobacco smoking exposure during the breast-feeding period on maternal milk lipids. This cohort study was conducted on 45 mothers environmental tobacco smoking exposure and 40 non-exposed post-partum mothers referred to the Shahid Ayat health center, Tehran, Iran. Socioeconomic conditions and the demographic characteristics of exposed and non-exposed groups were recorded. Milk samples were collected twice- at baseline (5-7 days after delivery) and four months after delivery. The samples were reserved at −20°C until assay. Milk lipids including cholesterol, triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) were evaluated. Dietary intake assessment was performed by means of the 24-hour dietary recall questionnaire both times. Maternal occupation status and education levels were significantly different between the two groups. Lipids profiles of milk were significantly higher 5-7 days after delivery in the non-exposed group and four months after delivery. Dietary intake was not significantly different between the two groups. Maternal environmental tobacco smoking exposure affects milk lipids which are essential for infant growth.

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Introduction

The number of cigarette smokers of all ages is increasing worldwide, and subsequently, many people are exposed to environmental tobacco smoking (1). Passive smoking was long overlooked by those in the medical and legal professions as being harmful to one's health, but in recent years the negative effect of passive smoking has come to the fore in the media and laws have been changed so that less people are obliged to unwillingly suffer from passive smoking, particularly in the workplace and in indoor settings. The harm caused to an unborn baby is now well documented and viewed as a complete no-no (2).

While most countries have policies to forbid smoking in public places, exposure is not avoidable at home. Over 4000 chemicals such as cotinine have been found in cigarette smoke, while most of them have negative effects (1). The chemicals from the tobacco smoke enter the mother's breast milk and will therefore be passed into the baby upon feeding. Breast milk will normally contain whatever is in the mother's body. Without a doubt, the more cigarettes the mother exposed to environmental tobacco smoking, the higher concentration of harmful substances is passed onto the child. Exposure of the fetus to passive and/or light active smoking involves a reduction of most axiological parameters and not only weight (3). As regards body composition, smoking appears to reduce fat mass. The prevention of smoking during pregnancy is therefore extremely important as intrauterine growth seems to be negatively influenced not only by active smoking, but

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also by passive and light active smoking (4). The amount of nicotine that is passed on to the baby depends on how many cigarettes the mother smoked, when she smokes in relation to feeding and the amount of time in between cigarettes. Nicotine is actually naturally present in breast milk and the baby will in fact, inhale more nicotine from breathing in the smoky air in a room rather than from the intake of the breast milk itself. Based on previous studies, nicotine and tar produced by cigarette burning is three times higher in the blood of passive smokers than active ones. In addition, nicotine could pass through the milk of post-partum mothers (2-4).

Both active and passive smoking influence maternal and neonatal health. Neonatal environmental tobacco smoking exposure may have lower birth weight; pre-term birth and being highly respiratory disturbed (4). Mothers who smoke tend to have less breastfeeding due to the effects of nicotine on dopamine and prolactin (5, 6). Human milk is considered the ideal food for healthy infants and meets the infants' nutrient requirements during the first 4–6 months of life. During this crucial period of rapid body growth and development, infants accumulate 1500-1600g of lipids, mainly fatty acids (FA), representing about 90% of all energy retained in their growing tissues breastfeeding which is recommended for neonates until six months of age, improves neonatal health by transmitting necessary antibodies and substances. Furthermore, previous studies have shown that smoking may change the lipid content of milk in mothers who smoke which may affect the infant's well being and also reduce anti-oxidant levels of mothers who smoke (7, 8). The aim of this study was to evaluate the effects of environmental tobacco smoking exposure during the breast-feeding period on post-partum maternal milk lipids.

Materials and Methods

This concurrent cohort study was held in the Shahid Ayat (Affiliated health center of Tehran University of Medical Sciences). Among post- partum women who referred to the health center 5-7 days after delivery based on exposure or non exposure as environmental tobacco smoking exposed, one hundred and two women were selected by simple random selection. Fifty one exposed and fifty one non exposed ones asked to sign informed consents before entering the study. Ethical committee of Tehran University of Medical Sciences approved the study protocol. Women with diseases influencing breastfeeding, special medications, exposure to other substances except for a cigarette and breast feeding less than four months were excluded from the study.

Exposure was evaluated by the number of cigarette used by other family members at home along with cotinine levels in milk at baseline of the study (5-7days after delivery) and four months after delivery.

Milk samples which collected two times, at the beginning and four month later were reserved at −20°C until assessment. The ELISA method was applied for cotinine measurement. Milk lipids including cholesterol, triglyceride (TG), high density lipoprotein (HDL), and low density lipoprotein (LDL) were determined by chemical kits with auto analyzer.

Seventeen women left the study (six women in exposed and eleven in non-exposed group). At the end, participants included forty-five exposed and 40 non-exposed women. Respondents were asked to report all foods consumed over the previous 24 hour period by means of valid and reliable 24 hour diet recall questionnaires (9). This instrument shows accounted intakes from midnight to midnight and is useful to estimate portion size of consumed food in the past 24 hours.

Statistical analysis

SPSS version 16.0 for Windows was used for data analysis. T-tests and Mann-Whitney U-test for continuous variables and Pearson X2 test with the Fisher exact test for categorical variables were applied for comparing the two groups.

Cholesterol, HDL and LDL quantities of maternal milk are presented in median values, but TG data presented by mean ± SD.

Result

Maternal educational level was higher in non-exposed while other demographic characteristics were not significantly different between the two groups (Table 1).

Non exposed mothers were more occupied than exposed (Table 2). Maternal occupation status and education levels were significantly different between the two groups. Lipid profiles of milk were significantly higher 5-7 days after delivery in the non-exposed group and four months after delivery. Dietary intake was not significantly different between the two groups.

Mean number cigarette used by other family members in the exposed group was 10 (2-40). As expected, concentration of cotinine in the milk of mothers in the exposed group was significantly higher

than that of non exposed group at baseline as well as the end of study (P<0.05)(Tables 3, 4).

Table 1. Demographic characteristics of exposed and non-exposed groups

	Exposed	Non-exposed	P- value
Mother's age(Year)	27.03±3.5	26.7±3.5	0.7
Father's age(Year)	32.5±4.6	31.05±5.5	0.1
Mother's education (Year)	9.3±3.1	10.8±3.1	0.01
Father's education (Year)	9.4±2.7	10.5±3.6	0.07
Mean parity	1.8±0.6	1.6±0.6	0.2
Mother's BMI			
Normal (19-24.9)	7(13.7%)	10(19.6%)	0.4
Obese (25-29.9)	44(86.3%)	41(80.4%)	
Father's BMI			
Normal (19-24.9)	36(70.5%)	33(64.7%)	0.5
Obese (25-29.9)	15(29.4%)	18(35.3%)	

Table 2. Socioeconomic condition of exposed and non-exposed groups

	Exposed	Non-exposed	P-value
Maternal employment	0	11(21.6%)	<0.001
Overpopulation index			
Less than one	22(43.1%)	21(41.2%)	
one	25(49%)	28(54.9%)	0.6
More than one	4(7.8%)	2(3.9%)	
Father's occupation			
Supervisors, simple and low ranking employees	4(7.8%)	2(3.9%)	
Skilled workers	1(2%)	1(2%)	0.8
Semi-skilled workers	38(74%)	40(78.4%)	
Simple workers	8(15.7)	8(15.7%)	

Table 3. Lipid profiles of breast milk in exposed and non-exposed groups (at baseline)

Lipid profiles of breast milk	Exposed	Non-exposed	P-value
Triglyceride (TG)(mg/dl)	503.7±288.33	706.3±284.4	<0.05
Cholesterol (TC)(mg/dl)	4(2-5)	2(1-3)	<0.05
LDL(ng/dl)	2(1-3)	2(1-3)	<0.05
HDL(ng/dl)	2.20±(2-2.6)	2.10±(1-2.40)	<0.05
Cotinine (ng/dl)	4.15±1.8	1.32±2.02	<0.05

Significant: (P<0.05)

Table 4. Lipid profiles of breast milk in exposed and non-exposed groups (4 months after delivery)

	Exposed	Non-exposed	P- value
Triglyceride(TG)(mg/dl)	375.4±146	579.8±230.7	<0.05
Cholesterol (TC)(mg/dl)	5(3-8)	4(2-5)	<0.05
LDL (ng/dl)	2(2-3)	1(1-2)	<0.05
HDL(ng/dl)	2.3(2-2.5)	2.3(2.2-3)	<0.05
Cotinine (ng/dl)	4.78±1.26	0.84±1.97	<0.05

Significant: (P<0.05)

Lipid profiles of milk in non exposed group were significantly higher than passive smokers at baseline (P<0.05)(Table 3).

Table 4 shows lipid profiles of breast milk in exposed and non-exposed groups (four months after delivery) that is significantly higher too (P<0.05).

Dietary assessment did not show any nutrient intakes difference between the two groups at baseline as well as the end of study (Tables 5, 6).

Table 5. 24 hour's dietary intakes at baseline

	Exposed	Non-exposed	P-vale
Protein (g/d)	54.6±17.9	52.5±28.3	*N.S
Lipid (g/d)	59.3±26.4	58.5±32.8	N.S
Cholesterol(mg/ d)	134 (97-196)	152 (97-380)	N.S
Calcium(mg/d)	624.7±349.1	533.2±303.6	N.S
Iron (mg/d)	10.2±3.08	10.4±3.9	N.S
Zinc (mg/d)	8.2±2.8	7.6±4.08	N.S

*N.S: non signifiant (p>0.05)

Table 6. 24 hour's dietary intakes after four month

	Exposed	Non-exposed	P-value
Protein(g/d)	54.5±17.2	56.3±21.8	*N.S
Lipid (g/d)	59.6±21.8	57.7± 21.6	N.S
Cholesterol (mg/d)	150(82-373)	152 (81-371)	N.S
Calcium (mg/d)	709.09±325.2	651.3±404.9	N.S
Iron (mg/d)	10.9±2.9	10.8±3.1	N.S
Zinc (mg/d)	8.05±2.4	8.02±3.2	N.S

*N.S: non-significant (p>0.05)

Discussion

Our study was the first study in Iran that done to evaluate the effects of environmental tobacco smoking exposure on postpartum maternal milk with assessment of cotinine as a biomarker of passive smoking. Passive smoking means inhalation of the cigarette burning substances in the presence of an active smoker. This study results showed that passive smoking affects maternal milk lipid profiles significantly. Previous findings have shown that passive smoking of post partum women is strongly associated with avoiding breast feeding (10,11). It can be due to the effects of smoking on prolactin and dopamine secretion in the brain (6).

Breastfeeding is believed to need high levels of commitment to be successful and also, socioeconomic status is considered as an important factor to initiate and continue breastfeeding. In the current study, maternal educational levels were significantly different between the two groups while levels of education were not different between fathers (12).

In a study by Weiser et al, the level of maternal education was significantly lower in post partum mothers who smoked (12). These findings were consistent with Nakamura et al findings that found higher education's among maternal smoking than others in pregnant women (13).

Although, according to our dietary assessment, lipid intakes of both groups was not different and increased mildly second time in the exposed group, our data showed that lipid components were less in exposed mothers than non-exposed ones while this difference observed in both study times. In agreement with our study, a previous study by Hopkinson et al illustrated low volume and fat components of milk in mothers who smoke than no smokers after delivery (14). The maternal dietary pattern in our study does not justify the differences in milk composition in the two groups.. Indeed, minimal differences in the quality of dietary intakes have been found.

Total lipid levels were similar in smoking and non-smoking mothers at colostrum period while fat levels decreased in smoking mother after the first month of delivery in another previous study (15). These findings can be explained by the increase in lipoprotein lipase activity and metabolism of lipoproteins induced by nicotine and its by-products in smoking mothers (16,17). Decreased fat components of maternal milk may affect the infant's health since infants supply important fatty acids which are essential for development by breast feeding (18,19). According to statistics, 20 - 30% of the babies who are born with a low birth weight do so because their mother smoked throughout pregnancy (20,21). Giving birth to a low-weight baby may have long-lasting negative effects on the child's growth and development. A premature birth can also result in a low birth weight baby and smoking increases this risk by around 30%. Smoking also causes many other unpleasant and potentially life-threatening illnesses including heart attacks, strokes, and other types of cardiovascular disease (20). In fact, smoking has an adverse effect on most organs and parts of the body and is linked to many other diseases including diabetes, osteoporosis, cataracts, ulcers and back pain. It is well known that milk fat content and composition affects the supply of individual fatty acids to breastfed infants, thereby modifying their energy intake and as a consequence developmental processes too (15). Quantity of fat and other nutrition intakes were not significantly different between smoking mothers and mothers who not smoke while it has been assumed that smoking mothers make lower use for energy intakes during first six months after delivery (15). Survey results suggest nursery renovations and related potential pollutant sources may be associated with differences in an urban city, income, and presence of older children with respiratory ailments, which could potentially confound health studies. While there are no standards for indoor

residential air quality, our findings suggest that additional research is needed to assess indoor pollution exposure for infants, which may be a vulnerable population (22).

Young persons (0-19yrs) had levels as high as or higher than other ages for all exposures except sulfate. Persons with lower socio-economic status had higher estimated exposures, with some exceptions (23). Neufeld et al illustrated that HDL cholesterol levels are lower in dyslipidemic children from households with smokers than in those without household smoke exposure (24) as we showed in lipid profiles of breast milk in exposed and non-exposed groups at baseline and 4 months after delivery(Tables 3 and 4). Comparing the changes in breast milk in exposed and non-exposed groups at baseline and 4 months after delivery, it appears, however that TG, TC and LDL cholesterol were higher and HDL cholesterol lower respectively in S vs. NS, at both time points. Considering the fact that HDL cholesterol is the main cardiac risk factor, it is probable that the total milk HDL in related to HDL cholesterol in such a way to consider a main factor in prevention of the cardiovascular disease in newborns (25).

These observations suggest a protracted effect of smoking before pregnancy throughout lactation, thus affecting both intrauterine fat accretion and milk fat content. Although, according to our dietary assessment, the quality of fat intake was not significantly different in exposed and non-exposed groups, some minor differences were present, even if significant only at 3 months. In general, exposed mothers showed a trend towards lower energy intake throughout the 0–6 month lactation period. Passive smoking may worsen the risk profile for later atherosclerosis among high-risk young persons. It can be conclude that maternal exposure, whether or not it is inhaled voluntarily, affects milk components which are necessary for the infant's developments so exposure to cigarette should be avoided for post-partum women.

Maternal environmental tobacco smoking exposure affects milk lipids which are essential for infant growth.

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