

Pregnancy increases urinary loss of carnitine and reduces plasma carnitine in Korean women

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(Received 27 July 2004 – Revised 15 November 2004 – Accepted 20 December 2004)

This study compared plasma and urinary carnitine concentrations in pregnant and non-pregnant Korean women. The subjects were fifty pregnant women and thirty non-pregnant women aged 24–28 years. During the first trimester, dietary carnitine intakes in the pregnant women were much lower than in non-pregnant women (70.00 (SD 29.22) $\mu\text{mol/d}$), but over the course of pregnancy carnitine intake increased from 44.64 (SD 24.84) $\mu\text{mol/d}$ during the first trimester to 96.11 (SD 36.56) $\mu\text{mol/d}$ during the third trimester. Pregnant women had a significantly lower plasma carnitine concentration than non-pregnant women. Plasma concentrations of non-esterified carnitine, acid-soluble acylcarnitine and total carnitine were significantly lower during the second and third trimesters than the first. Plasma acid-insoluble acylcarnitine levels, which tended to be higher in the non-pregnant women compared with the pregnant women, increased significantly as gestation proceeded. The urinary excretion of non-esterified carnitine, acid-soluble acylcarnitine and total carnitine was significantly higher in the pregnant women during the first and second trimesters than in non-pregnant women and decreased significantly as gestation proceeded. We found that there was a significant decrease in plasma carnitine level even though dietary carnitine intake increased as gestation proceeded. The low urinary excretion of carnitine in late pregnancy may be caused by an increased demand during pregnancy.

Carnitine: Pregnant women: Gestation: Trimesters: Plasma: Urinary excretion

Carnitine performs a crucial role in energy supply by controlling the influx of long-chain fatty acids into the mitochondria (Borum, 1995). Factors such as sex, age (DiMauro *et al.* 1973; Cederblad, 1976), nutritional status (Khan-Siddiqui & Banji, 1980), fasting (Frohlich *et al.* 1978; Hoppel & Genuth, 1980) and diseases (Chen & Lincoln, 1977; Rudman *et al.* 1977; Fuller & Hoppel, 1983) have been cited as influencing plasma carnitine levels in humans. Carnitine can be obtained by humans from either dietary sources or *de novo* synthesis from the amino acids lysine and methionine with the help of several vitamin and mineral co-factors (Sachan *et al.* 1997).

In infancy and in situations of high energy need, such as pregnancy and breast-feeding, the need for carnitine can exceed production by the body. It is well known that, throughout gestation, the mammalian fetus gains most of its energy from maternally derived carbohydrates, and that at birth there is a sudden interruption of the maternal–fetal transfer of carnitine, as well as of the amino acids lysine and methionine (Bremer, 1983). At that time, energy substrate use shifts from primarily carbohydrate to a greater use of lipids (Bayes *et al.* 1990; Campoy *et al.* 1991, 1992, 1996), with a fasting period followed by a slow, progressive introduction of feeding.

The low supplies of carnitine and its precursors, together with the immaturity of the liver and a reduced ability to perform enzymatic reactions, suggest that carnitine may be essential for newborn infants (Giovannini *et al.* 1991; Rebouche, 1992). Studies in various animal species have demonstrated a transfer of carnitine from the mother to the fetus during pregnancy

(Hahn & Skala, 1975; Robles-Valdes *et al.* 1976; Hahn *et al.* 1977). The carnitine status of pregnant women is very important for enabling mothers to supply adequate carnitine to maintain the infant's carnitine status. The amount of carnitine transferred across the placenta varies with different species, but the precise mechanism by which carnitine is supplied to the human fetus is not known. Carnitine deficiency during pregnancy can lead to pre-term and low birth weight infants with intrauterine growth retardation (Mete Akisu *et al.* 2001).

In humans, plasma carnitine concentrations have been shown to decline gradually during pregnancy and reach about half the pre-pregnancy values by parturition (Scholte *et al.* 1978; Bargen-Lockner *et al.* 1981). In contrast, other researchers have found that only the serum-free carnitine is decreased and that the concentration of esterified carnitine, which ordinarily makes up about 20% of the total, is increased in pregnant women (Novak *et al.* 1981). Korean diets are typically lower in carnitine than Western diets, but no studies have examined the carnitine status of pregnant Korean women. The inconsistencies of previous studies of carnitine status in pregnant women and the low carnitine intake of traditional Korean diets suggest the possibility that Korean women could be at risk of an impaired carnitine status during pregnancy.

This study estimated the dietary intake of carnitine using the carnitine content database of commonly used food stuffs recently established in our laboratory; we also evaluated the plasma and urinary concentrations of carnitine in pregnant Korean women compared with non-pregnant Korean women. This study

is the first to report the dietary intake, plasma concentration and urinary excretion of carnitine in pregnant Korean women; these results will serve as an index for future studies evaluating carnitine status in a diverse population within and outside Korea.

Subjects and methods

Study design

This study evaluated carnitine status and its effects on pregnancy outcome and lipid parameters in pregnant Korean women. The dietary intake of carnitine was estimated by dietary survey and plasma and urinary carnitine levels monitored during pregnancy. Plasma cholesterol and triacylglycerols were also monitored during pregnancy. Cord blood was collected when possible (eleven subjects). Correlations were made between carnitine status and pregnancy outcome for birth weight, and for plasma lipid levels in mothers.

Subjects

Sixty-five pregnant and fifty-five non-pregnant volunteers from Chonju, Korea, initially provided written informed consent to participate in this study. There were fifty pregnant and thirty non-pregnant subjects in each final group after subjects had dropped out because of moving, miscarriage, sickness or other private matters during the experimental period. This was the first pregnancy for all but three subjects. All subjects were screened for chronic and acute illnesses and use of medications. All were judged to be in good health. The non-pregnant women (n 30) were solicited by personal contact, with an attempt to choose non-pregnant women similar to the pregnant women in terms of age and body size. All the subjects were considered healthy since none was being treated for any illnesses, and no illness was reported during the study.

Survey of dietary intake

The dietary survey was conducted by the 24 h recall method for three consecutive days excluding weekends. All dietary intakes were assessed by the same researcher who conducted the initial survey. Direct interview for each subject was carried out with the aid of measuring instruments and books for eye measurement. Nutrient intakes were analysed using the Computer Aided Nutritional analysis program for professionals (CAN-PRO 2000; The Korean Nutrition Society, Seoul, Korea). For each subject, an average value of 3 d for a particular nutrient was used as the mean daily intake for that nutrient.

The dietary carnitine content of the diets was calculated using Lee's list of foods for which the carnitine content has been analysed (Lee *et al.* 2002).

Collection of blood and urine

Maternal blood samples were collected from the subjects during the first (up to week 13), second (weeks 14–27) and third (weeks 28–40) trimesters of pregnancy. From each subject, 12 h fasting blood samples were drawn into EDTA-treated vacuum tubes and centrifuged for 15 min at 3000 rpm; the plasma was separated and stored at -70°C until assay. In addition, 24 h urine samples from each subject were collected into plastic containers containing toluene as a preservative. Blood from the umbilical cord was obtained from eleven of the fifty subjects. Venous

cord blood (10 ml) was drawn into a syringe immediately following delivery, and plasma was prepared as described.

Serum lipid parameters and urinary creatinine

Total cholesterol, HDL-cholesterol and triacylglycerol in serum were enzymatically assayed with a commercial kit (Asan Pharmaceutical Co., Seoul, Korea). LDL-cholesterol was calculated by the equation: total cholesterol $-$ (triacylglycerol/5) $+ \text{HDL-cholesterol}$ (Friedewald *et al.* 1970). Urinary creatinine was determined by the Jaffe reaction method (Henry, 1967).

Analysis of carnitine

Carnitine was assayed using a modified radioisotopic method of Cederblad and Lindstedt (1972; Sachan *et al.* 1984). Acid-insoluble acylcarnitine (AIAC) was precipitated with perchloric acid and centrifugation, leaving the short-chain, acid-soluble acylcarnitine (ASAC) and the non-esterified free carnitine (NEC) in the supernatant. An aliquot of the supernatant was assayed to determine the NEC, and another aliquot was hydrolysed with 0.5 ml/l KOH to assay all the acid-soluble acylcarnitine (ASAC + NEC). ASAC was calculated as the difference between the NEC and the total acid-soluble carnitines. The pellets containing the AIAC were drained, washed and hydrolysed in 0.5 mol/l KOH for 60 min in hot water bath at 60°C . In each case, carnitine was assayed by using carnitine acetyltransferase (Sigma Chemical Co., St. Louis, MO, USA) to esterify the carnitine to a ^{14}C -acetylcarnitine from $1-^{14}\text{C}$ acetyl-CoA (Amersham, Little Chalfont, Buckinghamshire, UK). The radioactivity of the samples was determined in a Beckman LS-3801 liquid scintillation counter (Beckman Instruments, Palo Alto, CA, USA).

Statistical analysis

Significance of differences was determined by ANOVA with Duncan's multiple range test using SAS version 8 (SAS Institute, Cary, NC, USA). All values are expressed as group means together with their standard deviations, and $P < 0.05$ was considered significant. Pearson's correlation was used to determine correlations between the continuous variables.

Results

The subjects' anthropometric parameters are shown in Table 1. There were no significant differences between groups for parameters such as age, height, weight and BMI in the first trimester. However, weight and BMI increased as gestation proceeded in the pregnant group (Table 1). The mean daily intakes of energy, fat, carbohydrate, protein and other nutrients are shown in Table 2. Pregnant women had significantly lower nutrient intakes than non-pregnant women during the first trimester, but intakes increased significantly as gestation proceeded (Table 2).

The subjects' dietary carnitine intakes, which were calculated from Lee's list (Lee *et al.* 2002), are shown at the bottom of Table 2. The dietary carnitine intakes of the pregnant subjects were 44.64 (SD 24.84) $\mu\text{mol/d}$ in the first trimester, 56.58 (SD 34.46) $\mu\text{mol/d}$ in the second trimester, 96.11 (SD 36.56) $\mu\text{mol/d}$ in the third trimester and 70.00 (SD 29.22) $\mu\text{mol/d}$ for non-pregnant women. The level of dietary carnitine intake was positively correlated with the levels of dietary intakes of

Table 1. Anthropometric parameters of the subjects
(Mean values and standard deviations)

Variable	Non-pregnant women (<i>n</i> 30)		Pregnant women (<i>n</i> 50)					
			First trimester		Second trimester		Third trimester	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Initial age (years)	24.47	0.61	28.11	2.45				
Initial height (cm)	161.84	5.24	160.63	3.75				
Weight (kg)	54.79	5.39	55.64	8.15	59.78	8.18	67.45	9.05
BMI (kg/m ²)	20.93	1.90	21.57	3.15	23.07	2.9	26.15	3.14

Table 2. Subjects' nutrient intakes
(Mean values and standard deviations)

Nutrient	Non-pregnant women (<i>n</i> 30)		Pregnant women (<i>n</i> 50)					
			First trimester		Second trimester		Third trimester	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Energy (kcal)	1714.4	295.3 ^b	1420.1	391.6 ^c	1695.1	365.6 ^b	2037.4	380.8 ^a
Protein (g)	61.4	14.6 ^b	50.5	17.8 ^c	59.6	16.2 ^b	70.0	17.6 ^a
% energy	16.7	3.2	15.9	3.1	16.0	3.2	16.0	3.3
Fat (g)	22.6	8.5 ^b	15.7	7.1 ^c	20.4	8.8 ^b	26.7	9.3 ^a
% energy	11.8	3.4	9.7	2.9	10.5	3.3	11.9	3.8
Carbohydrate (g)	306.5	56.6 ^b	262.4	69.8 ^c	311.2	69.4 ^b	368.0	78.6 ^a
% energy	71.5	5.1	74.3	5.2	73.5	5.3	72.1	6.1
Calcium (mg)	516.7	161.2 ^{ab}	448.9	193.6 ^b	504.3	199.4 ^{ab}	593.5	217.2 ^a
Iron (mg)	12.5	3.4 ^b	9.9	4.4 ^c	11.7	3.3 ^{bc}	15.3	5.2 ^a
Vitamin A (RE)	419.6	164.9 ^b	297.4	164.9 ^c	430.6	170.3 ^b	619.2	274.0 ^a
Vitamin B ₁ (mg)	1.0	0.4 ^b	0.8	0.3 ^c	0.9	0.3 ^{bc}	1.3	0.6 ^a
Vitamin B ₂ (mg)	1.3	0.5 ^b	1.0	0.4 ^c	1.3	0.3 ^b	1.6	0.7 ^a
Niacin (mg)	15.1	6.2 ^{ab}	11.6	4.5 ^c	13.9	4.7 ^{bc}	17.3	7.2 ^a
Vitamin C (mg)	102.4	44.0 ^b	71.2	42.5 ^c	110.4	45.9 ^{ab}	128.7	54.5 ^a
Total carnitine (μmol/d)	70.00	29.22 ^b	44.64	24.84 ^c	56.58	34.46 ^{bc}	96.11	36.56 ^a

Values with different superscript letters are significantly different by ANOVA with Duncan's multiple range test at $P < 0.05$. RE, Retrol equivalent.

energy, protein, fat, iron, vitamin B₁ and niacin at $P < 0.01$, and with dietary intakes of carbohydrate and vitamin B₂ at $P < 0.05$, respectively (data not shown).

Plasma lipid data are summarised in Table 3. There were no significant differences in the levels of plasma total cholesterol, triacylglycerol or LDL-cholesterol between the non-pregnant and pregnant women in the first trimester, but each increased significantly with time in the pregnant women. The plasma triacylglycerol concentration (mg/dl) in each trimester was 89.83 (SD 25.13), 163.13 (SD 30.43) and 301.96 (SD 59.78), respectively. Plasma HDL-cholesterol concentrations (mg/dl) in each trimester were 34.51 (SD 7.97), 35.00 (SD 6.94) and 51.28 (SD 9.14), respectively, and were significantly higher during the third trimester than the first and second trimesters.

Plasma carnitine data are summarised in Fig. 1. Pregnant women had significantly lower plasma carnitine concentrations than non-pregnant women. Plasma concentrations of NEC, ASAC and total carnitine (TCNE) were significantly lower during the second and third trimesters than the first. Plasma AIAC levels, which also tended to be higher in the non-pregnant women compared with the pregnant women, significantly decreased as gestation proceeded and dropped significantly at each new trimester.

Urinary carnitine data are summarised in Fig. 2. Pregnant women had significantly higher urinary NEC, ASAC and TCNE levels during the first and second trimesters than non-pregnant

women, but the concentrations decreased significantly as gestation proceeded. Urinary AIAC concentrations were significantly lower in the third trimester, and acyl:free ratios were significantly higher in the third than in the first and second trimesters.

Plasma TCNE concentration during pregnancy was negatively correlated to maternal plasma triacylglycerol concentration ($r = -0.712$, $P < 0.01$; Fig. 3). Plasma TCNE was negatively correlated with dietary carnitine intake ($r = -0.270$, $P < 0.01$) and was positively correlated with urinary TCNE excretion ($r = 0.440$, $P < 0.01$). Cord blood was obtained from eleven of the subjects and used for correlations with pregnancy outcome. Cord blood TCNE concentration was positively correlated with birth weight ($r = 0.646$, $P < 0.05$), but there was no significant correlation between the plasma carnitine level in cord blood and pregnancy outcome other than birth weight (Fig. 4).

Discussion

Pregnancy is a dynamic, anabolic state (King, 2000). Women have higher energy needs during pregnancy because of the need to support the growth and development of the fetus, placenta and reproductive tissues such as the uterus and breasts. Additional energy needs reflect maternal fat storage and the general increase in metabolism that normally accompanies gestation (Pitkin, 1999). However, pregnant women have significantly lower

Table 3. Plasma lipid concentrations
Mean values and standard deviations

Lipid (mg/dl)	Non-pregnant women (n 30)		Pregnant women (n 50)					
			First trimester		Second trimester		Third trimester	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Total cholesterol (mg/dl)	165.80	32.60 ^c	150.61	20.96 ^c	209.20	33.55 ^b	258.95	48.62 ^a
Triacylglycerol (mg/dl)	76.94	15.46 ^c	89.83	25.13 ^c	163.13	30.43 ^b	301.96	59.78 ^a
HDL-cholesterol (mg/dl)	40.12	9.08 ^b	34.51	7.97 ^c	35.00	6.94 ^c	51.28	9.14 ^a
LDL-cholesterol (mg/dl)	106.13	17.86 ^c	96.43	19.35 ^c	138.64	26.31 ^a	150.74	33.75 ^a

Values with different superscript letters are significantly different by ANOVA with Duncan's multiple range test at $P < 0.05$.

nutrient intakes during the early stages of pregnancy because most pregnant women experience morning sickness, although energy intake increases significantly as gestation proceeds. Carnitine intake was positively correlated with dietary intakes of energy, protein, iron, vitamin B₁ and niacin at $P < 0.01$, and with dietary intakes of carbohydrate and vitamin B₂ at $P < 0.05$, respectively. This demonstrates that carnitine intakes are primarily a function of overall nutrient intake.

The marked increase in plasma lipid levels observed in this study is consistent with previous reports (Stipout *et al.* 1987; Chiang *et al.* 1995). Desoye *et al.* (1987) reported that plasma cholesterol and triacylglycerol concentrations were positively correlated with levels of progesterone, human placental lactogen and insulin throughout the entire period of gestation. The hypercholesterolaemia seen during pregnancy may be related to the increased production of sex steroids (Ordovas *et al.* 1984). However, the physiological significance of triacylglycerol elevation could be related to the maintenance of an adequate fuel supply to meet the energy demands of both the pregnant mother and the fetus (Butte, 2000).

Several authors (Scholte *et al.* 1978; Bargaen-Lockner *et al.* 1981; Cederblad *et al.* 1985) have reported that plasma NEC and TCNE levels at delivery are decreased to about half the concentration seen in non-pregnant women. It is noteworthy that the major decrease in plasma carnitine occurred during the first half of the pregnancy (Cederblad *et al.* 1986). In contrast, other workers have found that only the serum NEC level is decreased, and that the concentration of esterified carnitine is elevated in pregnant women (Novak *et al.* 1981). The increase in fatty acid oxidation, which takes place toward the end of gestation and during delivery, may be associated with the conversion of free carnitine to carnitine esters. However, we found that there is a significant decrease in TCNE level in the plasma as a result of a decrease in both free carnitine (or NEC) and acylcarnitine (ASAC and AIAC).

This observed decrease in plasma carnitine concentration during gestation may be due to a plethora of factors. By week 20 of pregnancy, however, levels had already fallen to 50% of control values. An increase in the renal clearance of carnitine is one possible factor responsible for the decreased plasma carnitine

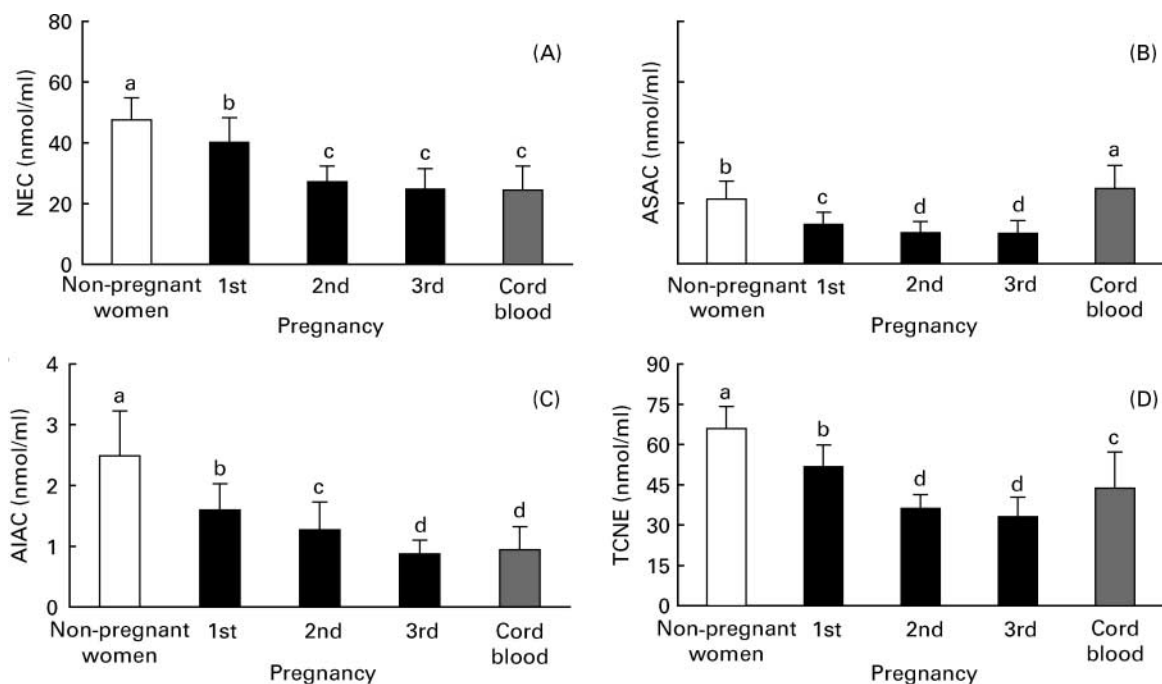


Fig 1. Plasma carnitine concentrations (Mean values with standard deviations shown by vertical bars). Values with different superscript letters are significantly different by ANOVA with Duncan's multiple range test at $P < 0.05$. (A) NEC, non-esterified carnitine; (B) ASAC, acid-soluble acylcarnitine; (C) AIAC, acid-insoluble acylcarnitine; (D) TCNE, total carnitine (NEC + ASAC + AIAC). 1st, 1st trimester; 2nd, 2nd trimester; 3rd trimester.

Carnitine profiles during gestation in Korean

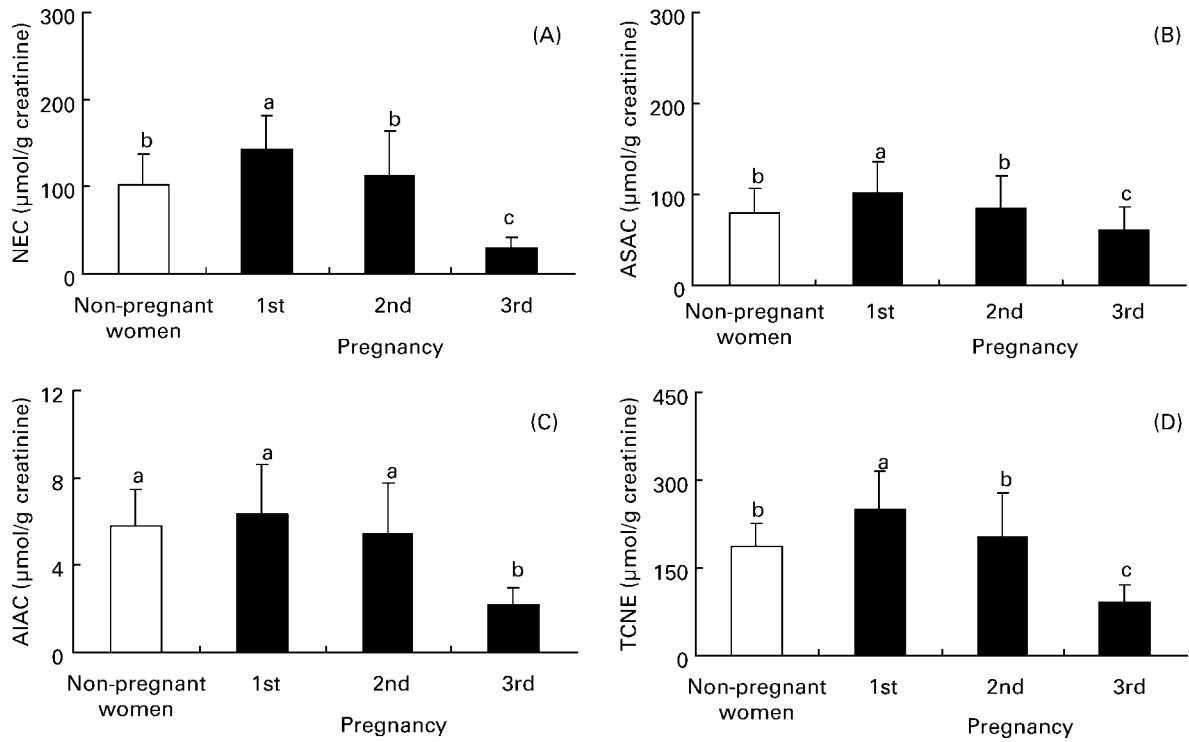


Fig. 2. Urinary carnitine concentrations (Mean values with standard deviations shown by vertical bars). Values with different superscript letters are significantly different by ANOVA with Duncan's multiple range test at $P < 0.05$. (A) NEC, non-esterified carnitine; (B) ASAC, acid-soluble acylcarnitine; (C) AIAC, acid-insoluble acylcarnitine; (D) TCNE, total carnitine (NEC + ASAC + AIAC). 1st, 1st trimester; 2nd, 2nd trimester; 3rd, 3rd trimester.

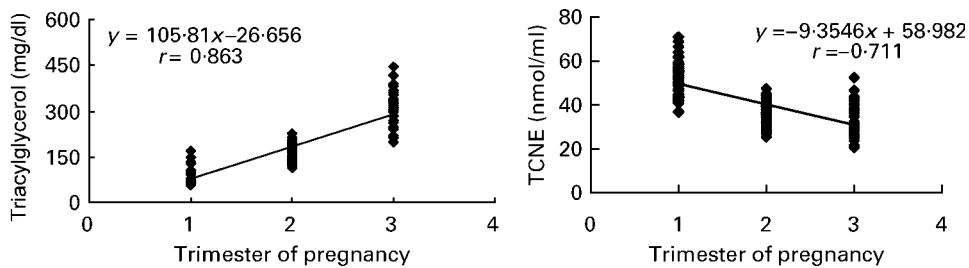


Fig. 3. Relationship between trimester of pregnancy and plasma triacylglycerol and total carnitine concentrations. Triacylglycerol was positively correlated, and total carnitine negatively correlated, with trimester of pregnancy by the Pearson correlation test at $P < 0.01$. TCNE, total carnitine.

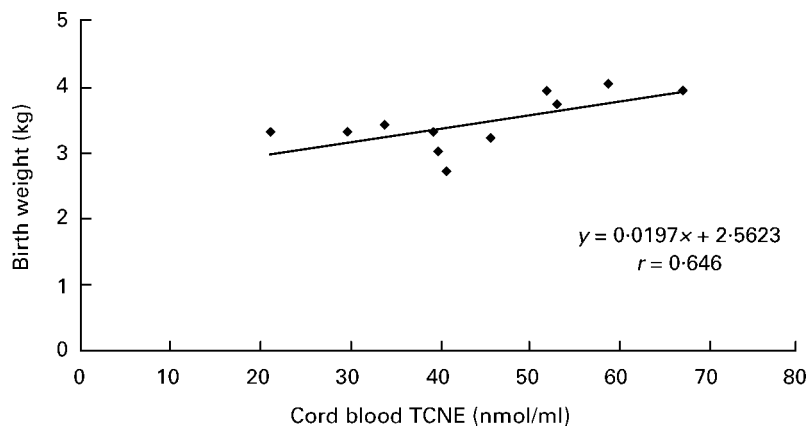


Fig. 4. Relationship between birth weight and cord blood total carnitine concentration. Significantly correlated by the Pearson correlation test at $P < 0.01$. TCNE, total carnitine.

level. Carnitine facilitates the removal of excess and potentially toxic acyl groups from the cell, these being excreted as acylcarnitine into urine (Chalmers *et al.* 1983). It is possible that there is an increased need for carnitine during pregnancy to perform this metabolic function. If so, it would decrease the free carnitine concentration and increase the clearance of acylcarnitine (Cederblad *et al.* 1986). It has, however, also been shown that plasma carnitine levels in rats are influenced by androgens and oestrogens; therefore, hormonal changes during pregnancy could also be responsible for the decreased plasma carnitine level seen during pregnancy.

ASAC and TCNE concentrations were higher in the cord blood than in plasma during the third trimester of pregnancy. In earlier reports, the carnitine level in cord blood has been found to be either similar to (Schmidt-Sommerfeld *et al.* 1981) or higher than (Bargen-Lockner *et al.* 1981; Novak *et al.* 1981; Cederblad *et al.* 1985) the value in the mother, in agreement with our findings (data not shown). Carnitine homeostasis in humans is maintained by dietary carnitine intake, a modest rate of endogenous carnitine synthesis and the efficient conservation of carnitine by the kidney (Rebouche *et al.* 1993). Since it is generally recognised that urinary carnitine profiles change with metabolic state (Frohlich *et al.* 1978; Hoppel *et al.* 1980), the higher NEC, ASAC and TCNE excretion in the first trimester of pregnancy may be a reflection of the metabolic state. Reduced carnitine excretion in late pregnancy is thought to occur because of the increased carnitine demand of the fetus. We found that there was a significant decrease in plasma carnitine levels even though dietary carnitine intakes increased as gestation proceeded, and the low urinary excretion of carnitine in late pregnancy may be caused by an increased demand during pregnancy.

Since the infant of a carnitine-deficient mother relies on the carnitine content of the mother's breast milk for its carnitine supply, carnitine deficiency could continue well into infancy and cause such problems as failure to thrive, hypotonia or even sudden infant death syndrome (Harpey *et al.* 1987; Lambert *et al.* 1988; Boulat *et al.* 1993). All infants in this study were born at term (37–42 weeks of gestation), and their birth weights were appropriate for gestational age. Cord blood levels were highly correlated with birth weight (r 0.646, $P < 0.05$), showing that birth weight might be affected by maternal status. There was, however, no significant correlation between the plasma carnitine level in cord blood and the pregnancy outcome except for birth weight (Fig. 4). The observation of a correlation between cord blood TCNE and birth weight was made in only eleven subjects but was still statistically significant. Since all the infants were of a normal birth weight, it is not yet possible to conclude that low cord blood carnitine is related to low birth weight. These observations deserve to be confirmed in larger numbers of infants, including infants with a wider range of birth weight.

In humans, plasma carnitine concentration and urinary carnitine excretion are affected by the macronutrient content of the diet. High-fat diets result in higher plasma carnitine concentrations and higher carnitine clearances than do low-fat diets (Cederblad, 1987). Among individuals consuming vegetarian diets, which tend to be high in carbohydrate and low in fat and carnitine, plasma carnitine concentration and urinary carnitine excretion are significantly lower than in individuals consuming omnivorous diets (Lombard *et al.* 1989).

In addition, Mitchell and Synder (1991) reported that urinary carnitine output was related only to dietary carnitine and protein,

although this was not seen not in our study. Our results showed that plasma carnitine concentration in pregnant women is significantly lower than in non-pregnant women, but urinary carnitine excretion, except in the third trimester, is significantly higher than in non-pregnant women (Figs. 1 and 2). We found that carnitine intake, plasma carnitine level and urinary excretion of carnitine were altered by pregnancy. Dietary carnitine intake was in fact negatively correlated with plasma carnitine concentration ($P < 0.01$).

The negative correlation between dietary intake and plasma total carnitine might be interpreted as a depletion of carnitine by dietary intake, but we believe that would be an incorrect interpretation of the data. The negative correlation is more likely to be an artefact of normal metabolic processes during pregnancy. It is known that plasma carnitine concentrations typically decrease to approximately 50% of pre-pregnancy levels (Bargen-Lockner *et al.* 1981), regardless of dietary intake. The increased dietary intake may actually protect against even greater carnitine losses in late pregnancy.

Plasma carnitine concentration was positively correlated with urinary excretion, indexed to creatinine excretion, in this study. Plasma carnitine is regulated by sex hormones, as androgens and oestrogens influence the plasma carnitine levels in rats (Borum, 1980). It has also been shown that plasma levels of acylcarnitine are sharply elevated under physiological conditions of accelerated fatty acid oxidation (Osmundsen *et al.* 1991; Cha *et al.* 1997). Plasma carnitine concentration (except ASAC) during pregnancy was negatively related to maternal triacylglycerol at $P < 0.01$ (Fig. 3).

In conclusion, changes in carnitine status during pregnancy might necessitate an adaptation of energy requirement during pregnancy, switching to a greater use of carbohydrate rather than fatty acids. Previous studies have yielded inconsistent results for carnitine status, some showing declines only in free carnitine, others showing a decline in total carnitine. This study demonstrated a decline in total carnitine and all carnitine fractions in pregnant Korean women. The observation that low cord blood carnitine is associated with low birth weight (although all were within a normal range) may suggest that poor carnitine status impairs fetal development, and this deserves further study. Further study is also needed to determine whether carnitine supplementation is warranted for Korean women during pregnancy.

Acknowledgements

We thank Easy Bio System Inc. for their financial support. This work was also supported by grant No. 01-PJ1PG3-22000-0011 from the Health Planning Technology and Evaluation Board.

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