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Relation of DDT residues to plasma retinol, α-tocopherol, and β-carotene during pregnancy and malaria infection: A case–control study in Karen women in northern Thailand

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Abstract

Populations living in endemic malaria areas maybe exposed simultaneously to DDT and malaria infection. DDT may impair status of vitamins, which are implicated in the immunity and pathophysiology of malaria. To explore possible interactions, DDT residues, retinol, α-tocopherol, β-carotene and cholesterol were measured in plasma samples of malaria-infected pregnant women (cases, $n=50$) and age matched malaria-free controls ($n=58$). DDT residues were found in all samples: mean (sd) total DDT levels of 29.7 and 32.7 ng/ml in cases and controls, respectively. Mean (sd) p,p′-DDT was higher in the controls than the cases (13.5 vs. 9.5 ng/ml, $p=0.006$). Malaria infection was associated with lower mean (sd) plasma retinol (0.69 vs. 1.23 μmol/L) and cholesterol (2.62 vs. 3.48 mmol/L) compared to controls ($p<0.001$). Mean (sd) plasma α-tocopherol (7.65 vs. 15.58 μmol/L) and α-tocopherol/cholesterol ratio (2.3 vs. 6.7 μmol/L/mmol/L) were significantly lower among the controls ($p<0.001$). Mean (sd) plasma β-carotene was low (<0.3 μmol/L) in both groups, but higher among malaria cases (0.19 vs. 0.15 μmol/L).

Plasma retinol among the controls showed highly significant positive correlations with individual DDT compounds, particularly with p,p′-DDT ($r=0.51$, $p<0.001$). Plasma α-tocopherol and β-carotene seemed not to be affected by DDT residues.

Keywords: DDT; Retinol (vitamin A); α-tocopherol (vitamin E); β-carotene; Pregnancy; Malaria

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1. Introduction

Over the span of 50 years, malaria rates have been dramatically decreased in Thailand as a result of successful vector control based primarily on the use of DDT for residual house spraying. International pressure and perceived adverse impact on environment and human health contributed to the decision to replace DDT by synthetic pyrethroids (phase out period 1995–1999). Nevertheless, the remaining stock of DDT is being used until recently in some areas of Thailand and it still serves as the primary chemical for mosquito vector control (Chareonviriyaphap et al., 2000). High amounts of DDT residues were detected in serum and human milk of mothers originating from different rural districts of the northern Thai provinces (Prapamontol et al., 1995; Stuetz et al., 2001).

According to the Stockholm Convention on persistent organic pollutants (POP), the production and use of DDT is limited to control disease vectors such as malarial mosquitoes (UNEP/UNDP, 2001). Recent return to DDT house spraying in Madagascar and South Africa have contributed to the control of resurgent malaria (Curtis, 2002). There is still the debate on whether DDT should still be used for the control re-emerging malaria despite its persistence in the environment. If proven, its associations to adverse effects in human could cause the final ban of the most effective insecticide ever used for in antimalarial campaigns (Attaran and Maharaj, 2000; Turusov et al., 2002). Much of the debate over DDT has focused on effects such as cancer and impact on reproduction. In recent studies exposure to DDT was associated with premature birth, lower birth weight, and spontaneous abortion (Korrick et al., 2001; Longnecker et al., 2001; Siddiqui et al., 2003).

Another implication by DDT might be its effect on vitamin status, and would be of great concern among population groups with highest risk for malaria; children under 5 years of age and pregnant women. Field studies and results from experimental malaria studies indicate that multiple specific nutrients, such as vitamin A and vitamin E, may affect the course of malaria infection and pathology (Shankar, 2000; Nussenblatt and Semba, 2002). Vitamin A deficiency increases susceptibility to malaria whereas deficiency of vitamin E or vitamin C may protect against malaria infection. Effects of DDT on vitamin A status have already been shown in animal and human studies. DDT exposed rats and pigs have reduced liver storage of vitamin A (Phillips, 1963; Graillot et al., 1976) and studies in volunteers occupationally exposed to DDT showed positive relationships between blood levels of DDT, its metabolites and vitamin A (Keil et al., 1972; Nhachi and Kasilo, 1990). Further, DDT as an organochlorine pesticide (OCP) might have a consuming effect on antioxidants such as vitamin E. Exposure to OCPs in animals and in human induces an oxidative stress (elevated lipid peroxidation) that could be reduced by antioxidants (Bagchi et al., 1993; Bachowski et al., 1998; Koner et al., 1998; Banerjee et al., 1999; Krieger and Loch-Caruso, 2001).

There are more than 100,000 Karen, Mon, Karenni living in a string of refugee camps along the Thai–Burmese border. This area is endemic for multi-drug resistant *Plasmodium falciparum* malaria and a particular burden in pregnant women (McGready and Nosten, 1999). DDT was sprayed in the camps as a vector control agent until the year 2000. Possible effects by DDT residues on vitamins which may affect the acquisition and course of malaria infection during pregnancy would be of great concern. The objective of this investigation was to measure the prevalence of DDT residues in Karen pregnant women from Maela refugee camp, the largest (40,000) of the camps on the border, and study their relationship to malaria and vitamin levels. DDT residues, retinol (vitamin A), α-tocopherol (vitamin E), and β-carotene were measured in anonymous plasma samples of pregnant women with and without malaria in a case–control study.

2. Subjects and methods

2.1. Study population, sample collection

Plasma samples (1–1.5 ml) of 108 pregnant Karen women (with and without malaria) from Maela refugee camp, collected between 1998 and 2000 and stored at −80 °C at the S.M.R.U base in Mae Sot, were used for the respective determinations. These anonymous specimens were from studies already completed and approved by the ethical committee of the Faculty of Tropical Medicine of Mahidol University.

Samples cooled on dry ice were transferred to the Toxicology laboratory of the Research Institute for Health Sciences (RIHES) in Chiang Mai and stored at −18 °C until analysing for OCP residues. Fractions of these plasma samples, again frozen at −18 °C and cooled on dry ice, were further transferred to the Institute of Biological Chemistry and Nutrition of the University of Hohenheim for the analysis of vitamins.

2.2. OCPs in plasma

The determination of OCPs was done by a method described by Prapamontol and Stevenson (1991) with modification. Plasma-aliquots of 1 ml, spiked with Aldrin (10 ng/ml) as an internal standard, were extracted with 5 ml ethyl-acetate/methanol/acetone (20:40:40 vol/vol) mixture by 15 min ultrasonication. After centrifugation (2000 rpm, 15 min), the supernatants were cleaned-up and enriched by solid-phase extraction using octadecyl (C_{18})-bonded silica cartridges (Accu Bond®). Compounds were eluted with isooctane (2 × 0.5 ml) and directly transferred to vials in order to be analysed. Reagents used were from J. T. Baker, Hayes, UK and Labscan, Dublin, Ireland. Standards of OCPs were obtained from the US Environmental Protection Agency, NC (γ-HCH, p,p′-DDE, p,p′-DDT), Labor Dr. Ehrenstorfer, Augsburg (heptachlor; aldrin; o,p′-DDE; o,p′-DDT), and Promochem, Wesel, Germany (HCB; α-, β-HCH; p,p′-DDD).

A Hewlett-Packard model, 5890 A series II gas chromatograph equipped with an automatic sampler (HP 7673), a fused silica capillary column (HP-1:25 m × 0.32 mm i.d., with 1.05 μm film thickness), a ^{63}Ni electron-capture detector and a computerised data handling system (HP 3365 Series Chemstation) were used for the analysis. Operating conditions: injection port: 200 °C; temperature programming (column): 80 °C for 1 min, then changed to 190 °C with 30 °C/min ramp rate, then changed to 250 °C with 4 °C/min ramp rate and held at 250 °C for 10 min; detection temperature: 300 °C. Oxygen free nitrogen (99.999%) as make up gas and helium as carrier gas (99.993%) were from TIG, Bangkok, Thailand.

The detection limit of the method (LDM) was 0.5 ng/ml plasma for respective OCPs (α/β/γ-HCH, HCB, heptachlor, dieldrin, o,p′-DDE, p,p′-DDE, p,p′-DDD, o,p′-DDT, p,p′-DDT). One series of spiked plasma for the intra-batch mean (n = 4) and one spiked plasma sample along with each batch of plasma samples (n = 8) was analysed for internal quality control. Mean recoveries of individual DDT compounds ranged from 96% for p,p′-DDT to 102% for p,p′-DDE while intra-batch and inter-batch variation coefficients were below 8% and 10%, respectively. Results were not corrected for recovery and values less than the LDM were excluded from statistical analysis.

2.3. Vitamins (retinol, α-tocopherol, β-carotene) in plasma

The analysis was performed according to a method described by Erhardt et al. (2002) using modifications: For extraction 30 μl of plasma were transferred to 0.5 ml vessels (Eppendorf tubes) with 150 μl ethanol–butanol (50:50 vol/vol), containing 5 mg BHT/ml and 4 μmol/L Tocol (Internal standard). After vigorous mixing and centrifugation (5 min at 12,000 × g) 20 μl of supernatants were analysed by using reversed-phase HPLC. The HPLC (Varian pro Star, Modell 210) was equipped with an automatic sampler (Dynamax, AI-200), chromatography workstation (Varian, version 5.31) and an UV–VIS Detector (Waters, 2487). The detector was programmed as follows: 325 nm from 0 to 4 min (for retinol), 292 nm from 4 to 10 min (for α-tocopherol and tocol), and 450 nm from 10 to 21 min (for β-carotene). The separation was achieved using a Sperisorb column (ODS-2, 3 μm, 250 × 4 mm, CROM, Herrenberg, Germany). An isocratic mobile phase consisting of acetonitrile (82%), dioxan (15%), and methanol (3%, containing 100 mM ammonium acetate and 0.1% triethylamin) was used as HPLC solvent in recirculation mode with a flow rate of 1.2 ml/min. International certified NIST standards (National Institute of Standards and Tech-

Table 1
Characteristics of study population

	With malaria (n = 50)	Controls (n = 58)	Total (n = 108)
Age (y)[a]	24.7 (15–40)	25.2 (16–40)	25.0 (15–40)
Gravidity[b]	3 (1–9)	3 (1–14)	3 (1–14)
Gravidity ≥ 3[c]	52	52	52
History of malaria[c]	32	33	32

[a] Mean, range in parentheses; [b] median, range in parentheses; [c] percentage (%) of subjects.

Table 2
DDT residues [ng/ml] in plasma of malaria patients and controls

	Malaria patients ($n=50$)				Controls ($n=58$)			
	N	Mean (sd)	Median	Min–max	*N*	Mean (sd)	Median	Min–max
p,p′-DDE	50	17.7 (17.5)	12.0	1.3–69.3	57	17.0 (15.8)	12.1	1.4–84.8
p,p′-DDD	44	2.6 (1.7)	2.1	0.6–6.5	50	2.1 (1.1)	1.9	0.7–4.7
p,p′-DDT	50	9.5 (6.6)[a]	8.3	0.5–26.0	58	13.5 (8.1)[a]	12.9	1.0–40.3
DDE/DDT	50	2.04 (1.29)[b]	1.67	0.25–6.28	57	1.28 (0.76)[b]	1.04	0.14–3.54
Total DDT[c]	50	29.7 (24.8)	22.2	2.0–99.6	58	32.7 (23.4)	27.4	1.0–117.1

[a,b] Significantly different between malaria patients and controls: [a] $p=0.006$, [b] $p<0.001$. [c] Total DDT is the sum of o,p′-DDE, p,p′-DDE, p,p′-DDD, o,p′-DDT, and p,p′-DDT.

nology, Gaithersburg, MD, USA) were used for quantification of respective vitamin concentrations. The detection limits of the method were 0.1 μmol/L for retinol, 0.7 μmol/L for α-tocopherol and 0.05 μmol/L for β-carotene.

Plasma cholesterol was measured for the determination of α-tocopherol/cholesterol ratios by spectrophotometric analysis using cholesterol reagent (Sigma Diagnostics, St. Louis, USA) and control serum (Medizintechnik Frank Golder Diagnostic) for quantification.

2.4. Statistical analysis

Characteristics of the women and concentrations of DDT isomers and vitamins were analysed using SPSS for Windows (version 10.0). For the comparison of DDT isomers and levels of vitamins between groups (malaria vs. controls) the *t*-test for independent comparison (and the Mann Whitney *U* test) was applied. Individual DDT compounds, vitamins, cholesterol and continuous characteristics were tested for bivariate correlations within the groups using Pearson (normal distribution) and Spearman correlation coefficients (nonparametric); a *p*-value <0.05 was assumed to be significant.

3. Results

Characteristics of the women assessed in this analysis are summarized in Table 1. Age, gravidity (number of pregnancies) and percentage of subjects with a history of malaria did not differ between malaria-infected ($n=50$) and control subjects ($n=58$), but there were more primigravidae in the malaria group (18 vs. 11). Length of residence in Maela camp was not routinely recorded for women who provided the samples. Before 1995 a great part of the women had their residence in smaller camps along the Thai–Burmese border. In Maela camp DDT was sprayed on a yearly basis as an indoor insecticide until 1995 and again in 1998 and 2000. The origins of most women are small villages in remote hill tribe areas of the eastern border of Burma where the use of DDT for agricultural pur-

Table 3
Retinol [μmol/L], α-tocopherol [μmol/L], β-carotene [μmol/L] and cholesterol [mmol/L] in plasma of malaria patients and controls

	Malaria patients ($n=50$)				Controls ($n=58$)			
	N	Mean (sd) [%]	Median	Min–max	*N*	Mean (sd) [%]	Median	Min–max
Retinol	50	0.69 (0.35)[a]	0.62	0.18–1.65	58	1.23 (0.31)[a]	1.19	0.54–2.05
Retinol <0.7	30	60%	0.52	0.18–0.67	1	1.7%	0.54	–
α-Tocopherol	50	15.6 (6.3)[a]	14.7	0.91–30.1	58	7.7 (3.8)[a]	7.6	0.38–18.2
α-Tocoph. <11.6	12	24%	8.9	0.91–11.5	52	90%	7.5	0.38–11.1
β-Carotene	38	0.19 (0.12)	0.15	0.05–0.48	23	0.15 (0.12)	0.11	0.05–0.52
Cholesterol	50	2.62 (1.08)[a]	2.51	0.99–5.43	58	3.48 (1.08)[a]	3.16	1.79–6.61
Tocoph./Cholest.	50	6.7 (3.8)[a]	5.9	0.4–22.5	58	2.3 (1.1)[a]	2.1	0.1–5.8

[a] Significantly different between malaria patients and controls: $p<0.001$.

Table 4
Correlations between DDT residues and plasma retinol among the controls

	p,p′-DDE	p,p′-DDD	p,p′-DDT	Total DDT
Retinol (controls, $n=58$)	0.43[S]	0.43	0.51	0.47
Sig (2-tailed)	0.001	0.002	<0.0001	<0.0001

Pearson correlations, except [S] for Spearman's rank correlation.

poses or to control malaria seems unlikely but cannot be excluded.

3.1. DDT residues and vitamins in plasma

Residues of p,p′-DDT (technical/commercial DDT) and its metabolites were found in all samples (Table 2). The predominant forms of DDT were p,p′-DDE, p,p′-DDT and p,p′-DDD. Women who were of gravida ≥3 ($n=52$) had lower mean (sd) p,p′-DDE (13.1 vs. 22.0 ng/ml, $p=0.005$) and lower mean (sd) total DDT (26.9 vs 36.1 ng/ml, $p=0.046$) than women of gravida <3 ($n=56$).

The comparison of groups shows no difference in the mean (sd) total DDT (29.7 vs. 32.7 ng/ml) but a significantly higher mean (sd) p,p′-DDT (13.5 vs. 9.5 ng/ml, $p=0.006$) and therefore lower DDE/DDT ratio (1.28 vs. 2.04, $p<0.001$) in the controls. Controls with a history of malaria ($n=19$) had higher mean (sd) p,p′-DDT (16.9 vs. 11.8 ng/ml, $p=0.022$) and total DDT (41.8 vs. 28.3 ng/ml, $p=0.037$) than the controls with no history of malaria ($n=39$). The time (days) from the date of previous DDT spraying in the camp till the date of blood draw showed inverse relationships to p,p′-DDE ($r=-0.347$, $p=0.013$) and total DDT ($r=-0.301$, $p=0.034$) in patients and to p,p′-DDT ($r=-0.227$, $p=0.087$) in the controls. Additionally, o,p′-DDT could be detected in 11 cases of malaria and in 32 controls with a mean of 1.1 and 1.3 ng/ml, respectively, while o,p′-DDE was only found in a single sample with 0.7 ng/ml. Other OCPs such as heptachlor or HCH were either not detectable or found in exceptional cases with concentrations close to the LDM of 0.5 ng/ml and were excluded from further analysis.

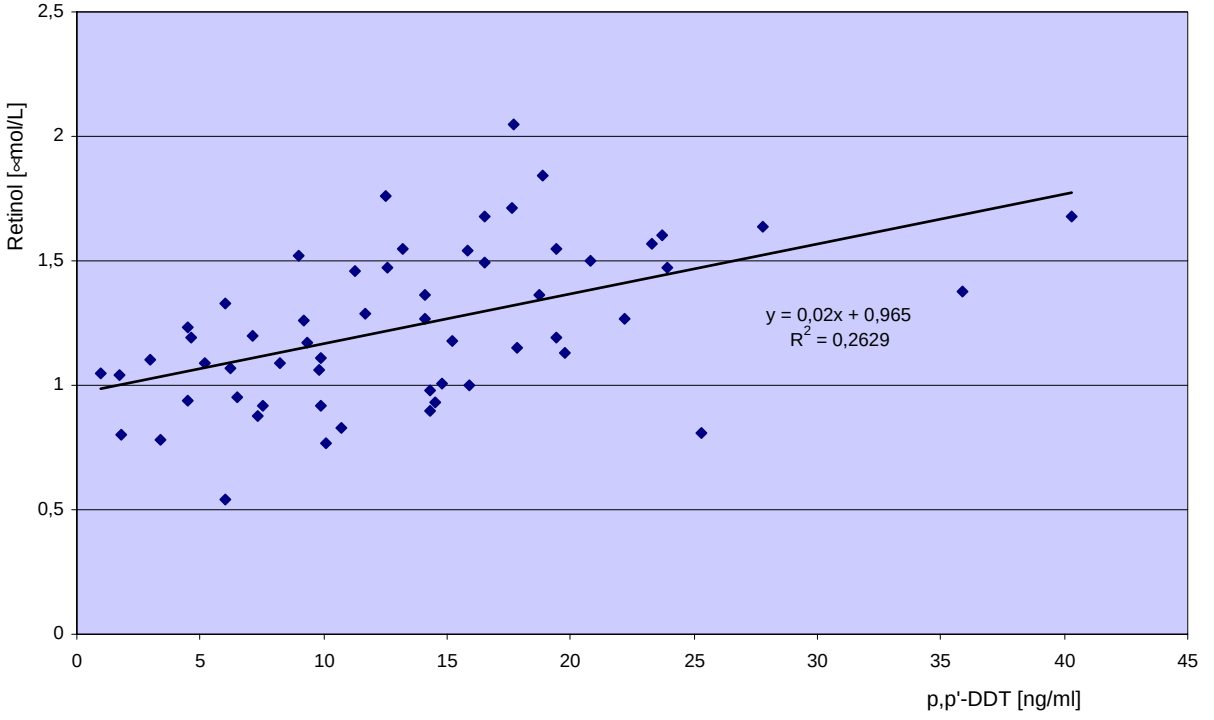

Fig. 1. Association between p,p′-DDT and plasma retinol among the controls.

Concentrations of plasma vitamins and cholesterol are summarized in Table 3. Plasma retinol among malaria cases showed a positive relationship to age ($r=0.44$, $p=0.002$) and gravidity ($r=0.31$, $p=0.031$). The comparison between the groups shows a lower mean (sd) retinol (0.69 vs. 1.23 μmol/L) and cholesterol (2.62 vs. 3.48 mmol/L) but higher α-tocopherol (15.58 vs. 7.65 μmol/L) and tocopherol/cholesterol ratio (6.7 vs. 2.3 μmol/L/mmol/L) in the cases (all variables $p<0.001$) than in the controls. 60% of the women with malaria had a prevalent vitamin A deficiency (<0.7 μmol/L) while 90% of the controls ($n=52$) had α-tocopherol levels less than 11.6 μmol/L, the threshold value for vitamin E deficiency (Sauberlich et al., 1974). Cholesterol correlated positively with α-tocopherol in patients ($r=0.32$, $p=0.022$) and controls ($r=0.40$, $p=0.002$) as well as with retinol ($r=0.41$, $p=0.003$) and β-carotene ($r=0.43$, $p=0.007$) in malaria cases. β-carotene was undetectable (<0.05 μmol/L) in 60% of the control samples and in 24% of the cases; levels of detectable β-carotene were low in both groups (<0.3 μmol/L), but higher in the women with malaria.

3.2. Associations between DDT residues, malaria and vitamins

Plasma retinol among the controls showed a highly significant correlation with DDT compounds (Table 4), in particular with p,p′-DDT (Fig. 1). This correlation was not observed in the cases. To explore this further we tested the following model: retinol = $a + b$ * ppddt + c * malaria + d * ppddt * malaria. * ppddt + c * malaria + d * ppddt * malaria. The results of the analysis of variance (ANOVA) shows an interaction term between p,p′-DDT and malaria ($p=0.04$) explaining 46% of the variance in plasma retinol. In both groups, neither α-tocopherol nor β-carotene was associated with DDT isomers.

4. Discussion

4.1. DDT residues

Considerable amounts of DDT residues were detected in plasma of pregnant Karen women from Maela refugee camp. Concentrations of p,p′-DDE were comparable to levels reported in the USA in the 1960s (Longnecker et al., 1999, 2001). Total DDT, p,p′-DDE and in particular p,p′-DDT were higher than in Vietnamese women and in pregnant women of China and India (Schecter et al., 1997; Korrick et al., 2001; Siddiqui et al., 2003). Higher p,p′-DDE but lower p,p′-DDT than in Karen women were detected postpartum in serum of mothers from districts of the Chiang Mai Province, in northern Thailand (Prapamontol et al., 1995).

In the present study, p,p′-DDE and total DDT were lower in women of gravida >3. The decline of DDT compounds is likely to be due to their loss by previous breast-feeding. The negative association between DDT in human milk and increasing number of pregnancies or length of previous lactation periods has been reported (Slorach and Vaz, 1983; Bouwman et al., 1990; Stuetz et al., 2001).

However, the high proportion of p,p′-DDT and therefore low DDE/DDT ratio as well as the negative correlations between DDT residues and the number of days from previous DDT spraying till individual blood sampling in both groups, indicate a recent exposure which can be attributed to DDT usage in vector control programs. The influence of women's different duration of stay in Maela and in previous camps on DDT residues could not be assessed. The higher p,p′-DDT levels found in the malaria-free women could reflect a higher contamination by a more effective DDT house spraying that in turn protected them against malaria vectors. However this is unlikely because DDT indoor spraying twice a year had little impact on the host–vector contacts in the refugee camps (Nosten et al., 2000). Alternatively malaria infection itself may be associated with altered liver metabolism and change in pharmacokinetics and responsible for a different distribution of DDT isomers. Only a prospective study in women with different exposure to DDT could help to understand the relation (if any) between DDT residues in the blood and malaria.

4.2. Retinol (vitamin A)

Mean retinol concentrations in this study were comparable to those measured in pregnant women of developing countries such as Indonesia, Nepal, Malawi and Zimbabwe, in whom a high prevalence of vitamin A deficiencies were accompanied by anaemia, parasitic

infections (hookworms, malaria), HIV, and combined micronutrient deficiencies (iron, folate, B12) (Suharno et al., 1992; West et al., 1999; van den Broek and Letsky, 2000; Friis et al., 2001; Tanumihardjo, 2002).

In this study, plasma retinol levels were higher in women with higher levels of DDT residues. However this was significant in the controls and confounded by malaria. The positive association between vitamin A and DDT was previously reported (Keil et al., 1972; Nhachi and Kasilo, 1990). It has been suggested that DDT induces an increase of plasma retinol by depletion of the liver reserves as observed in animal studies (Phillips, 1963; Graillot et al., 1976). A further study to assess vitamin A liver reserves by using dose–response tests (Underwood, 1990) would be necessary to reconsider this supposition. Reduced liver reserves and simultaneous low dietary intake would result in vitamin A deficiency which therefore could increase the risk for malaria infection. An increased susceptibility to *Plasmodium berghei* infection has been shown in vitamin A deficient rodents (Krishnan et al., 1976; Stoltzfuß et al., 1989) and low vitamin A status in children was associated with increased risk of parasitemia (Stuerchler et al., 1987). Vitamin A is well known as the anti-infective vitamin and is essential for normal immune function (Semba, 1998, 1999). Studies in animal models and cell lines have shown that vitamin A and related retinoids play a major role in antibody responses and cell-mediated immunity. Antibody mediated immunity, cell-mediated responses and non-specific immunity comprise the immunity response against *P. falciparum*. High-dose vitamin A supplementation reduced the frequency of *P. falciparum* episodes by 30% in preschool children (Shankar et al., 1999).

60% of the malaria cases in this study were deficient in vitamin A (below 0.7 μmol/L), indicating inadequate liver stores. Several studies described that inflammation and the resulting acute phase response during malaria infection is associated with low concentration of serum retinol (Thurnham and Singkamani, 1991; Hautvast et al., 1998; Rosales et al., 2000). It has been suggested that low plasma retinol is the consequence of malaria-induced changes in hepatic protein syntheses including retinol transport proteins (RBP and transthyretin) rather than of a true deficiency of vitamin A (Ross, 2000). During malaria, vitamin A may also be lost by excretion. Large amounts of vitamin A and RBP were detected in urine samples of patients with pneumonia and sepsis, with a strong relationship of fever to retinol excretion (Stephensen et al., 1994). It seems likely that the high prevalence of vitamin A deficiency in patients in this study is caused by malaria and not by the presence of DDT residues. Nevertheless the question remains whether DDT induces an increase of plasma retinol depleting liver stores and in turn decreases natural defences against malaria.

4.3. α-tocopherol (vitamin E) and β-carotene

Plasma α-tocopherol among Karen women was lower than in pregnant women of Nigeria and Tanzania (Ziari et al., 1996; Mulokozi et al., 2003). 90% of the controls in the present study were deficient in α-tocopherol (<11.6 μmol/L). Mean α-tocopherol as well as α-tocopherol/cholesterol ratio in the controls was significantly lower than in the women with malaria. This is in contrast to studies which reported significantly lower serum vitamin E in malaria patients and no difference in α-tocopherol between the groups when expressed as α-tocopherol/cholesterol ratio (Thurnham et al., 1990; Davis et al., 1994; Das et al., 1996; Adelekan et al., 1997). Thurnham and Das suggested that α-tocopherol is partly consumed as a consequence of oxidative stress and lipid peroxidation induced during malarial infection. However, the pregnant women in this study were all actively detected and few were symptomatic, with less oxidative stress related damages. Slightly higher plasma α-tocopherol and a significant reduction in erythrocyte membrane α-tocopherol concentration than in the controls was measured in Kenyan children with *P. falciparum* malaria (Griffiths et al., 2001). The study cautions the use of plasma α-tocopherol concentrations in isolation as an indicator of α-tocopherol status in malaria. Acute malaria infection is associated with haemolysis, therefore higher plasma α-tocopherol in patients than in controls as found in Karen pregnant women might be explained by the release of membrane α-tocopherol during destruction of parasitized red blood cells.

Plasma β-carotene was either undetectable or low (<0.3 μmol/L) in both groups of Karen women. The influence of different dietary intake by season on low plasma β-carotene and α-tocopherol as reported among rural pregnant women of Nepal (Jiang et al., 2005) seems to play an insignificant role in Maela camp.

The overwhelming majority of dietary nutrients are provided by ration foods (rice, mung beans, fermented fish, soybean oil) which are adequate in energy and protein, but low in micronutrient content (Banjong et al., 2003). Low levels of α-tocopherol and β-carotene, however, seemed not to be affected by DDT residues; neither α-tocopherol nor β-carotene were associated with any of the DDT isomers. Nevertheless, vitamin E deficiency might protect against malaria (Levander and Ager, 1993; Davis et al., 1994; Taylor et al., 1997), but might be simultaneously responsible for low birth weights and adverse pregnancy outcomes (Ramakrishnan et al., 1999).

5. Conclusion

All the pregnant women in this study had significant circulating levels of DDT residues. This is most likely to be the consequence of the indoor spraying of this insecticide in the camps until recent years. DDT residues and malaria had an influence on the levels of retinol, α-tocopherol and β-carotene. A prospective study is needed to elucidate these interactions and their potential impacts on pregnancy outcomes.

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