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Red palm oil as a source of vitamin A for mothers and children: impact of a pilot project in Burkina Faso

NM Zagré[1], F Delpeuch[2], P Traissac[2] and H Delisle[3,*]

[1]Université de Montréal, Quebec, Canada and Health Science Research Institute, Ouagadougou, Burkina Faso: [2]UR 106 – Nutrition, Food, Societies, Institut de Recherche pour le Développement (IRD), Montpellier, France: [3]Department of Nutrition, Université de Montréal, PO Box 6128 Downtown Station, Montreal, Quebec, Canada H3C 3J7

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Abstract

Objective: To demonstrate the effectiveness of the commercial introduction of red palm oil (RPO) as a source of vitamin A (VA) for mothers and children in a non-consuming area, as a dietary diversification strategy.

Design: A pre–post intervention design (no control area) was used to assess changes in VA intake and status over a 24-month pilot project.

Setting and subjects: The pilot project involved RPO promotion in 10 villages and an urban area in east-central Burkina Faso, targeting approximately 10 000 women and children aged < 5 years. A random sample of 210 mother–child (12–36-months-old) pairs was selected in seven out of the 11 pilot sites for the evaluation.

Results: After 24 months, RPO was reportedly consumed by nearly 45% of mothers and children in the previous week. VA intake increased from 235 ± 23 μg retinol activity equivalents (RAE) to 655 ± 144 μg RAE in mothers (41 to 120% of safe intake level), and from 164 ± 14 μg RAE to 514 ± 77 μg RAE in children (36 to 97%). Rates of serum retinol < 0.70 μmol l^{-1} decreased from 61.8 ± 8.0% to 28.2 ± 11.0% in mothers, and from 84.5 ± 6.4% to 66.9 ± 11.2% in children. Those with a lower initial concentration of serum retinol showed a higher serum retinol response adjusted for VA intake.

Conclusions: Commercial distribution of RPO was effective in reducing VA deficiency in the pilot sites. While it is promising as part of a national strategy, additional public health and food-based measures are needed to control VA malnutrition, which remained high in the RPO project area.

Keywords
Red palm oil
Vitamin A nutrition
Food diversification
West Africa
Evaluation

The World Summit for Children[1] in 1990, the International Conference on Micronutrients[2] in 1991 and the International Conference on Nutrition[3] in 1992 all highlighted the need to reduce micronutrient deficiencies, in particular vitamin A (VA), iodine and iron deficiency. The momentum that gathered during the last decade resulted in vast programmes of VA supplementation through primary health care and mainly in conjunction with national immunisation days. Some African countries have recently started organising special micronutrient days[4]. Fortification of local foods with VA has also gained ground in West Africa, although at this time it is still at the feasibility stage.

Unfortunately, dietary diversification has not met with the same enthusiasm despite the fact that this approach is potentially effective and sustainable. Animal sources of VA are beyond the reach of poorer groups; however, controlled studies using green leafy vegetables in China[5], red palm oil (RPO) in India and Africa[6–9] and *gac fruit* in Vietnam[10] have shown that plant sources of provitamin A can improve the VA status of women and children. While some studies have expressed doubts about the efficacy of provitamin carotenoids from green leafy vegetables[11,12], other studies are more encouraging[5]. Moreover, results obtained with RPO have led to this source being cited as a potential solution for the future in areas where VA deficiency is a public health problem, notably in Africa[13]. However, the results pertained only to the biological efficacy of these foods. Little information is available on their effectiveness when people are free to choose what foods they buy and eat.

This was the rationale for setting up the 'Red Palm Oil Pilot Project' in Kaya district of Burkina Faso, in 1999. The objective was to test the feasibility and effectiveness of retailing RPO as a VA source for women and children in an area where RPO was not available and not normally eaten. RPO is extracted from the oil palm tree. The common species in Africa is *Elaeis guineensis*.

*Corresponding author: Email helene.delisle@umontreal.ca

Project activities were implemented in 10 villages and one urban sector, for a total target population of about 10 000 pre-school children and women of childbearing age. RPO promotion, in the hands of home economists trained in social marketing, included talks, food demonstrations, local theatre plays and inter-village contests. The project collected RPO from the south-western part of the country, where the oil is traditionally produced by some women's groups. The oil was shipped to Kaya; from there, it was dispatched to test villages where women sold it mainly at home and on weekly markets. Village committees were responsible for paying back the oil purchased on credit from the project, and for returning to retailers their profit (about 8%). The oil was retailed in various quantities, from 5 ml to 1 l. The (subsidised) price compares to that of groundnut oil and amounts to roughly US$0.013 for 10 ml, the recommended daily 'dose'. RPO is not promoted as a substitute for groundnut oil or shea butter, which are traditional fats in the area, but as a 'supplement' to be added in small amounts to the individual dish at mealtime. RPO is typically added to boiled beans, green leaves, or else to the soup to go with the staple food. More detailed information about the project was published previously[14].

This paper presents results evaluating the impact of the 2-year pilot project on the VA intake and status of women and children.

Methodology

Project evaluation design

The evaluation was based on a longitudinal pre–post design. There was no control area because of financial and practical constraints, and also because the evaluation was mainly of the adequacy assessment type[15,16]. Two main survey rounds were carried out, the first in May 1999 (baseline survey) and the second in May 2001. Both included the assessment of VA intake and status of mothers and pre-school children. The mother signed an informed consent form before she and her child were included in the study. The evaluation protocol was approved by the Ethics Committee of the Faculty of Medicine at the Université de Montréal and by the National Scientific and Technological Research Centre in Burkina Faso.

Sample

A sample size of 160 mother–child pairs was based on an alpha error of 0.05, a statistical power of 0.90 and an expected reduction of one-third[17] (from 60 to 40%, based on unpublished data from Niger) in the proportion of serum retinol concentration $<0.70\,\mu mol\,L^{-1}$. To account for potential missing data, a random sample of 210 pairs was included at baseline. The subjects were sampled from seven of the 11 pilot sites (including the urban sector) because of logistical constraints related to blood sample processing. Children and their mothers were selected from the list of eligible households, i.e. those having resided in the area for at least 6 months and including a child aged between 12 and 36 months.

Variables and measurement methods

Vitamin A intake

Food consumption was estimated using a food-frequency questionnaire (FFQ) based on the approach of the International Vitamin A Consultative Group (IVACG) and using normal household measures to estimate the portions. The FFQ comprised an assessment of weekly consumption of plant sources of VA and of monthly consumption of animal sources. A validation study of the method in Niger (Delisle *et al.*, unpublished data*) showed that assessment of monthly consumption of animal products is relevant since these food items are eaten only occasionally and stand out in the memory.

Conversion of provitamin A carotenoids into retinol activity equivalents (RAE) was based on recent recommendations by the US Institute of Medicine, i.e. 12 μg of β-carotene from mixed dishes for 1 μg RAE[18]. For β-carotene from RPO, we used the standard equivalent of 6 μg for 1 μg of RAE based on the presence of fat and the absence of a plant matrix. For breast-fed children, VA intake from breast milk was calculated on the basis of the average milk uptake for these age groups[19–21] and the average retinol content measured in 85 lactating women from our study zone, i.e. 37.8 μg of retinol per 100 ml of breast milk. VA intake was compared with recommendations of the World Health Organization/Food and Agriculture Organization[22]. Inadequate VA intake was defined as intake $<62.5\%$ of safe intake level, in agreement with the threshold of IVACG[23].

Serum retinol concentration

Serum retinol was used as indicator of VA status. Measurements were done in duplicate, by means of high-performance liquid chromatography (HPLC)[24], at the laboratory of the Unité de Formation et de Recherche en Sciences de la Santé in Burkina Faso. Standard cut-off points were used for very low and low serum retinol (0.35 and $0.70\,\mu mol\,L^{-1}$, respectively)[25].

Age, anthropometry and morbidity of children

Age of the child was based on the birth certificate or health record; otherwise, a calendar of local events helped to determine it as accurately as possible. Prior to blood sampling, children's height and weight were measured by the same investigator (N.M.Z.). The occurrence and duration of illness in the previous 15 days were documented by maternal recall, using a validated method[26].

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Socio-economic variables

Data included age, marital status and physiological status (pregnancy, lactation) of the mother, and the occupation and level of education of both the mother and the father. The number of siblings of the target child was also noted.

Data processing

All data analyses took into account the characteristics of the sampling design: stratification on survey sites, large sampling fractions and repeated measurements on the same subjects. In addition to the sampling weights, the final weights included a re-weighting according to strata to correct for different rates of missing data. Changes from baseline in serum retinol, VA intake and other characteristics of mothers and children were assessed using linear or logistic regression. In these models, the 'survey round effect' (or year effect) was tested by Wald's chi-square test[27]. The magnitude of the effect was assessed by the difference between year 2001 and 1999 surveys for continuous variables and by prevalence odds ratios for dichotomous variables. For polytomous variables, a sampling design-adjusted chi-square test was used.

Using serum retinol and VA intakes as key outcome variables, baseline stunting of children, number of siblings, main occupation of parents and baseline serum retinol were tested as potential modifiers of the 'year effect' by regression models including a survey round × variable interaction term. Most interactions did not change the relationship but only modulated its strength.

The subsequent analyses focused on serum retinol as the main response variable. VA intake (as % of safe intake level) was studied as the potential mediating variable explaining the 'year effect' on serum retinol concentration. A first model (model 1) tested the effect of the year of survey on retinol status. The magnitude of the effect is the difference of means (or prevalence odds ratio) between the two surveys. In a second step (model 2), potential confounding factors were entered. The difference between serum retinol (or prevalence odds ratio) of the two surveys then represents the year effect adjusted for the confounding factors. According to the same logic, in a third step (model 3), VA intake (as % of safe intake level) was added. The serum retinol difference between the two surveys in this third model represents the year effect, adjusted for the confounding factors and for VA intake change. The mediating effect of VA intake on changes in serum retinol can thus be assessed by comparing models 2 and 3.

Statistical analyses were performed using the software SUDAAN version 7.5.3 for Windows[27].

Results

Characteristics of the sample

The baseline survey sample included 210 mothers and 214 children (four sets of twins). In the 2001 survey, 184 mothers and 181 children were seen again. Blood samples were taken from 203 mothers and 203 children in the first round, and 147 mothers and 143 children in the last round. Apart from the death of seven children (mainly from diarrhoea and fever), the reasons for absence were moving home, travelling, illness and a few refusals to have blood samples taken. When the children seen again and those lost to follow-up were compared, baseline serum retinol concentrations were not different. Dropouts featured more educated parents (22.7% vs. 8.9%; $P = 0.007$) and non-stunted children (81.7% vs. 67.1%; $P = 0.033$). However, there was no significant relationship between education level and serum retinol change, and that of stunting was barely significant. Table 1 presents the baseline characteristics of the households and Table 2, changes in the anthropometric and health status of the children.

Changes in vitamin A intake

Total VA intakes of mothers and children are shown in Table 3. The increase over the 2-year period was significant both for the children and their mothers.

VA intake of children increased from 41% of safe intake level at baseline to 120% of safe intake level 2 years later. For mothers, it increased from 36% to 97%. The proportion of children and mothers at risk of inadequate intake thus decreased from 89% to 47%, and from 87 to 60%, respectively.

Table 4 lists the foods contributing to VA intake. Although the relative contribution of retinol (excluding

Table 1 Profile of households at study baseline ($n = 210$)

Indicator	%
Mother	
Physiological status	
Pregnant	4.3
Breast-feeding	69.5
Not pregnant, not breast-feeding	26.2
Educational level	
Nil or coranic school	84.5
Literacy or formal school	15.5
Main occupation	
Agriculture	51.9
Other (wages, trade, informal sector and craftsmanship)	48.1
Has an additional occupation	36.2
Number of pre-school children	
1	76.4
≥2	23.6
Total number of siblings of target child	
0–1	42.1
2–3	27.4
≥4	30.5
Head of household	
Educational level	
Nil or coranic school	72.2
Literacy or formal school	27.8
Main occupation	
Agriculture	40.7
Other (wages, trade, informal sector and craftsmanship)	59.3
Has an additional occupation	36.4

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Table 2 Anthropometric and morbidity status of the children according to the year of survey

	Baseline ($n = 210$) 05/1999		Final ($n = 181$) 05/2001	
	Mean	95% CI	Mean	95% CI
Age (months)	25.6	24.4, 26.8	50.1	48.9, 51.3
HAZ				
Mean	−1.15	−1.35, −0.95	−1.6	−1.8, −1.4
% with HAZ < −2.0 (stunting)	23.9	17.1, 30.7	31.3	21.9, 40.7
Illness in the last fortnight				
Mean duration of illness (days)	2.2	2.06, 2.4	0.6	0.5, 0.7
% with illness	49.4	40.8, 58.0	21.3	13.5, 29.1
Measles in the last year (%)	2.3	0.0, 5.1	10.0	9.4, 10.6
Received VA capsule 6 months earlier (%)	96.6	93.2, 99.8	78.4	74.3, 82.5

CI – confidence interval; HAZ – height-for-age *Z*-score; VA – vitamin A.

breast milk) to total VA intake of children decreased, absolute retinol intake increased significantly in both children and mothers. Before the intervention, carotenoids supplied 97% of total VA intake of mothers and a smaller amount (67%) in the children due to breast milk retinol. In the last survey, the proportion of carotenoids remained high, and came from (in descending order) green leafy vegetables, fruits and RPO. However, RPO was the first source in subjects who had consumed it during the preceding week, i.e. 42.5% of the children and 43.5% of the mothers. Mean daily consumption of RPO in the whole sample was 3.1 ± 0.1 g in children and 4.7 ± 0.2 g in mothers. Considering only those who reported consuming RPO, mean daily consumption was 10.8 ± 1.1 g in children and 14.6 ± 1.1 g in mothers, with a respective contribution of 1220 RAE and 1650 RAE.

Changes in serum concentrations of vitamin A

Table 5 shows changes in the mean concentration of serum retinol and in the proportion of low levels between pre- and post-survey. Mean serum retinol levels increased significantly in the children and their mothers, with a concurrent reduction in the proportion of low serum retinol values. However, 67% of the children and 28% of the mothers still had low serum retinol concentrations after the project.

Table 3 Change in total dietary vitamin A intake (retinol activity equivalents per day) of children and mothers

	Baseline 05/1999		Final 05/2001		Change		
	Value	SE	Value	SE	Value*	95% CI†	*P*-value‡
Children	$n = 210$		$n = 181$				
Mean intake	163.7	7.3	514.3	38.3	+350.0	274.3, 425.7	0.0001
25th percentile	107.1	6.9	177.0	28.4	+69.9	–	–
50th percentile	154.3	6.9	276.2	27.5	+121.9	–	–
75th percentile	205.2	7.4	544.8	69.0	+339.6	–	–
Mean intake as % of safe intake level	0.41	0.02	1.20	0.09	+0.79	0.61, 0.97	0.0001
% with intake <0.625	88.8	3.2	47.0	1.6	0.11	0.07, 0.19	0.0001
Distribution of intake as proportion of safe intake level (%)							
<0.30	30.2	3.6	20.7	4.0	–	–	–
0.30–0.624	58.7	4.3	26.3	4.3	–	–	–
0.625–0.99	9.6	2.9	20.0	4.2	–	–	–
≥1.0	1.6	1.4	33.0	4.3	–	–	–
Mothers	$n = 210$		$n = 184$				
Mean intake	235.4	11.5	655.4	72.1	+420.0	278.5, 561.5	0.0001
25th percentile	145.3	6.1	187.2	21.7	+41.9	–	–
50th percentile	202.7	12.6	301.7	26.1	+99.0	–	–
75th percentile	282.8	25.8	757.3	103.1	+474.5	–	–
Mean intake as % of safe intake level	0.36	0.02	0.97	0.14	+0.61	0.33, 0.89	0.0001
% with intake <0.625	87.3	3.4	59.5	4.3	0.21	0.12, 0.38	0.0001
Distribution of intake as proportion of safe intake level (%)							
<0.30	53.0	4.1	33.0	4.6	–	–	–
0.30–0.624	34.3	4.3	26.4	4.4	–	–	–
0.625–0.99	9.6	3.0	13.6	3.3	–	–	–
≥1.0	3.1	1.9	26.9	3.5	–	–	–

SE – standard error; CI – confidence interval.
* Difference of means (2001 minus 1999) for continuous variables; odds ratio (2001 vs. 1999) for dichotomous variables.
† 95% CI for survey round effect.
‡ Null hypothesis of no difference between 2001 and 1999: *t*-test.

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Table 4 Changes in the contribution of food categories to dietary vitamin A of children and mothers (as proportion of total intake)

	Baseline 05/1999		Final 05/2001		Change		
	Mean	SE	Mean	SE	Value*	95% CI†	*P*-value‡
Children	$n = 210$		$n = 181$				
Total dietary vitamin A intake	1.0		1.0				
Total retinol intake	0.33	0.02	0.07	0.01	−0.26	−0.31, −0.21	0.0001
From breast milk§	0.26	0.02	0.00	0.00	−0.26	−0.31, −0.21	0.0001
From animal food	0.07	0.01	0.07	0.01	<0.01	−0.02, 0.04	0.90
Total carotenoid intake	0.67	0.02	0.92	0.01	+0.25	0.20, 0.30	0.0001
From red palm oil	<0.01	<0.01	0.19	0.02	+0.19	0.16, 0.22	0.0001
From vegetables (green leaves primarily)	0.43	0.02	0.34	0.03	−0.09	−0.16, −0.02	0.012
From fruits	0.24	0.02	0.39	0.03	+0.15	0.09, 0.21	0.0001
Mothers	$n = 210$		$n = 184$				
Total dietary vitamin A intake	1.0		1.0				
Total retinol intake	0.03	0.01	0.05	0.01	+0.02	−0.01, 0.04	0.16
Total carotenoid intake	0.97	0.01	0.95	0.01	−0.02	−0.04, 0.01	0.16
From red palm oil	<0.01	<0.01	0.23	0.03	+0.23	0.18, 0.28	0.0001
From vegetables (green leaves primarily)	0.59	0.02	0.34	0.03	−0.25	−0.31, −0.19	0.0001
From fruits	0.38	0.02	0.38	0.03	<0.01	−0.05, 0.07	0.97

SE – standard error; CI – confidence interval.
* Difference of means (2001 minus 1999).
† 95% CI for difference of means.
‡ Null hypothesis of no difference between 2001 and 1999: *t*-test.
§ 59% of children were breast-fed in 1999; 0% in 2001.

Table 5 Changes in serum retinol concentration of children and mothers

	Baseline 05/1999		Final 05/2001		Change		
	Value	SE	Value	SE	Value*	95% CI	*P*-value†
Children	$n = 199$		$n = 140$				
Mean serum retinol concentration (μmol L^{-1})	0.55	0.02	0.64	0.02	+0.09	0.02, 0.16	0.012
Serum retinol concentration <0.70 μmol L^{-1} (%)	84.5	3.2	66.9	5.6	0.37	0.19, 0.73	0.004
Serum retinol concentration <0.35 μmol L^{-1} (%)	13.0	1.5	7.6	1.8	0.55	0.31, 0.97	0.04
Mothers	$n = 199$		$n = 144$				
Mean serum retinol concentration (μmol L^{-1})	0.69	0.02	0.95	0.04	+0.26	0.16, 0.34	0.0001
Serum retinol concentration <0.70 μmol L^{-1} (%)	61.8	4.0	28.2	5.5	0.24	0.12, 0.47	0.0001
Serum retinol concentration <0.35 μmol L^{-1} (%)	15.5	3.7	1.2	0.6	0.068	0.022, 0.21	0.0001

SE – standard error; CI – confidence interval.
* Difference of means (2001 minus 1999) for continuous variables; odds ratio (2001 vs. 1999) for dichotomous variables.
† Null hypothesis of no difference between 2001 and 1999: *P*-value for Wald chi-square test.

Modifiers of serum retinol changes

Baseline serum retinol was the principal modifier of pre–post serum retinol changes in children and their mothers. The effects are shown in Table 6.

Children whose initial retinol concentration was <0.70 μmol L^{-1} showed a significant increase, whereas in children with normal serum retinol at baseline, a declining trend was observed. As for mothers, only those with low baseline serum retinol (<0.70 μmol L^{-1}) showed a significant increase. The other potential modifiers – baseline stunting of children, number of siblings and main occupation of parents – also showed an interaction effect, but their impact could not be dissociated from that of baseline serum retinol.

Determinants of final retinol status

Table 7 shows non-adjusted means of serum retinol (and rates of low concentrations) and crude pre–post intervention changes (model 1). When all potential confounding factors are added (model 2), adjusted means and pre–post changes remain the same as in model 1, in both children and mothers. When VA intake (as % of the safe level of intake) is introduced as one of the independent variables (model 3), however, the pre–post change in serum retinol is no longer significant in the children, while remaining significant – although lower – in the mothers. The same applies to rates of low serum retinol and 2001 vs. 1999 odds ratios. These results demonstrate a mediating effect of VA intake changes on

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Table 6 Serum retinol changes according to baseline level

Serum retinol at baseline	Serum retinol (μmol L^{-1}) Baseline 05/1999 *n*	Mean	Final 05/2001 *n*	Mean	Change *P*-value	% with serum retinol < 0.70 μmol L^{-1} Baseline 05/1999 %	Final 05/2001 %	Change *P*-value
Children								
<0.70 μmol L^{-1}	167	0.48	116	0.62	$<10^{-4}$	100.0	68.4	$<10^{-4}$
≥ 0.70 μmol L^{-1}	32	0.91	20	0.74	0.17	0.0	55.1	0.0058
			Interaction: $P = 0.016$				Interaction: not computed	
Mothers								
<0.70 μmol L^{-1}	119	0.47	84	0.88	$<10^{-4}$	100	29.9	$<10^{-4}$
≥ 0.70 μmol L^{-1}	80	1.05	55	1.01	0.62	0.0	27.2	0.011
			Interaction: $P < 10^{-4}$				Interaction: not computed	

Table 7 Mediating effect of changes in dietary vitamin A (VA) intake on serum retinol changes

	Serum retinol concentration (μmol L^{-1}) *P*-value	1999 mean	2001 mean	Change	95% CI	Serum retinol concentration < 0.70 μmol L^{-1} *P*-value	2001 vs. 1999 odds ratio	95% CI
Children		$n = 198$	$n = 134$					
Model 1 – survey round	0.012	0.55	0.64	+0.09	0.02, 0.16	0.0053	0.37	0.19, 0.74
Model 2 – survey round adjusted for confounders*	0.012	0.55	0.64	+0.09	0.02, 0.16	0.021	0.42	0.20, 0.88
Model 3 – survey round adjusted for confounders and total VA intake (four classes)	0.12	0.56	0.63	+0.07	−0.02, 0.16	0.11	0.47	0.18, 1.20
Mothers		$n = 198$	$n = 142$					
Model 1 – survey round	0.0001	0.69	0.94	+0.25	0.16, 0.34	0.0001	0.26	0.13, 0.50
Model 2 – survey round plus confounders†	0.0001	0.69	0.95	+0.26	0.17, 0.35	0.0001	0.24	0.12, 0.45
Model 3 – survey round plus confounders plus total VA intake (four classes)	0.0001	0.71	0.93	+0.22	0.12, 0.32	0.0011	0.29	0.14, 0.61

CI – confidence interval.
* Sex of child, main occupation of head of household (agriculture, wages, informal sector, trade or craftsmanship), secondary occupation of head of household (yes/no), main occupation of mother (agriculture, wages, informal sector, trade or craftsmanship), secondary occupation of mother (yes/no).
† Main occupation of head of household (agriculture, wages, informal sector, trade or craftsmanship), secondary occupation of head of household (yes/no), main occupation of mother (agriculture, wages, informal sector, trade or craftsmanship), secondary occupation of mother (yes/no).

serum retinol changes in the children. This is not observed in mothers, which suggests that factors other than higher VA intake, that are not included in the model, are at play in explaining serum retinol changes in mothers.

Discussion

Our results show that the VA intake and status of children and their mothers improved between baseline and the end of the pilot project. Without the benefit of a control group, however, it is difficult to determine the independent contribution of the project. Setting up a control group is not simple[15,16]. In our particular case, there was also the unavoidable contamination of a control group, since it was impossible to prevent the spread of RPO sales to areas outside the pilot zone. The marked increase in VA intake of mothers and children related to RPO consumption, and the concomitant rise of serum retinol, may reasonably be attributed in large part to the project as RPO was not available in the study area prior to the project. Other factors might have influenced VA intake and status, in particular the severe food shortages during the lean season of year 2000, but in a negative way. The same goes for health and nutritional conditions, which tended to deteriorate throughout the country during the project period. Indeed, an increased prevalence of malnutrition as reflected in child stunting (from 29% to 37%) and under-five mortality rate (from 187 to 219 per 1000) was observed between the national surveys of 1993 and 1999[28,29].

Changes in vitamin A intake and serum retinol response

The larger reduction in the rate of low serum retinol in mothers (−33.6%) than in children (−17.6%) is puzzling,

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since VA intake increments were roughly similar. This may partly reflect the higher occurrence of infection and parasites in children than in their mothers, given that these conditions tend to reduce serum retinol[30–32]. The impact of the project may have been partially masked by the high-dosage VA supplement taken by the majority of the children 6 months before both surveys as part of national yearly supplementation campaigns. These data also suggest that VA supplement benefits are short-lived, considering the high rates of low serum retinol in our study children.

Methodological issues

The methods used to assess VA intake and VA status impinge on the association between these variables. In our study, food consumption was measured by means of a validated FFQ. A recent study on pre-school children in Indonesia showed that VA intake measured through a single 24-hour recall showed an association with serum retinol level, which was not the case when intake was assessed using the semi-quantitative FFQ developed by IVACG[23]. However, our FFQ differed from the IVACG instrument in that we assessed the monthly consumption of animal sources of VA. Furthermore, we computed pre-formed VA and provitamin A intake directly from a food composition table, which included data from HPLC analyses of a large number of green leafy vegetable specimens from Niger[33].

The estimated bioefficacy of provitamin A carotenoids may obscure the relationship between VA intake and serum retinol. Many studies[34–36] have challenged the assumed bioavailability of provitamin A from plant sources, particularly green leafy vegetables, and the standard conversion factor of 6 μg β-carotene for 1 μg retinol equivalent (RE)[22,37] was changed to 12 μg of β-carotene for 1 μg RAE[18] for mixed dishes in North America. Some authors[35,36] contend that the American recommendations are unsuitable for developing countries and suggest factors ranging from 15 to 21 μg of β-carotene per μg RAE, based on studies in Asia using stable isotopes. The bioefficacy of provitamin A carotenoids also varies within a given population. Controlled studies using double isotope labelling showed that only half the subjects responded to β-carotene supplements to any significant extent[38,39], although the proportion increased when large doses of β-carotene were given in oil[40,41].

The low sensitivity and specificity of serum retinol as an indicator of VA status remain of concern. It is well known that serum retinol concentration reflects liver stores only when these are nearly depleted. In addition, serum retinol decreases during infection and inflammation[30,31] due to the synthesis of inflammatory proteins at the expense of carrier proteins such as retinol binding protein (RBP)[42], and also due to the excretion of free retinol by the kidneys because of reduced formation of RBP-transthyretine–retinol complex. Zinc deficiency could also be involved, since the enzyme responsible for reducing retinal to retinol is zinc-dependent[43]. It is associated with low levels of serum retinol[44] and zinc supplements were shown to enhance the effect of VA supplements[45,46]. The impact of the pilot project could thus have been affected by several different health and nutrition factors, as well as by the assessment methods used.

Influence of baseline vitamin A status

Our results showed that changes in retinol status were significant only in subjects who were deficient at baseline. Similar findings were reported in several supplementation trials, showing that VA supplementation was most effective in deficient subjects[47,48]. We also observed an intriguing downward trend in the serum retinol status of subjects whose initial status was normal. This decrease seems too large merely to reflect regression to the mean. This decline cannot be explained by a higher rate of breast-feeding in the 'normal' group, since the rate was 53% as opposed to 60% in initially deficient children ($P = 0.4$). Neither can the phenomenon be explained by VA intake, the means of which increased from 165 μg RAE to 541 μg RAE in subjects with an initial deficiency compared with 165 μg RAE to 482 μg RAE in children with normal retinol status at baseline (data not shown). Our results are somewhat reminiscent of the negative effects of VA supplements reported in well-nourished children in Tanzania[49]. Further study is required to elucidate this question.

Ageing of children and improvement of vitamin A intake and status

In this longitudinal study, a pertinent question is how far the ageing of the children could have contributed to improved VA status through higher intake and reduced morbidity. However, several studies have shown that VA intake varies very little among children in this age group. In Niger children aged 2–4 years, VA intake did not increase significantly over a 2-year period, in contrast with macronutrient intake[50]. In southern India, there was a significant increase in VA intake (breast milk excluded) only between 12 and 24 months, after which time values remained stable until the children were 4 years old[51]. Similarly, the rate of low serum retinol appears to change little up to 5 years of age[9,52,53]. It is thus unlikely that the increase in age of the children played a significant role in changes in VA intake and status among children in our study.

Conclusion

This study has shown that RPO contributes to reducing VA deficiency in children and women of childbearing age. To our knowledge, it is the first study to check the feasibility and effectiveness of introducing RPO on a

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commercial basis. Our results show that it is possible to introduce a new food if a sufficiently persuasive approach is used, and if the product is easily available and its price affordable to the population. Based on the findings, scaling-up the promotion and sale of RPO appears relevant although other measures are essential to control VA malnutrition. A two-pronged programme of this sort is indeed under way, with ongoing promotion of the consumption of RPO in target groups and support to the RPO production and marketing system through women's groups.

In spite of improvements with the project, however, the prevalence of inadequate VA intakes and low serum retinol concentrations was still high. As less than half the target population had eaten RPO in the week preceding the last survey, further progress may be expected during the ongoing programme. None the less, additional approaches are needed, including VA fortification where relevant and public health measures, for the control of infection.

The potential benefit of RPO for VA nutrition and health is a good enough reason for integrating it in VA strategies at the national level in Burkina Faso, and possibly also in other regions of the Sahel. Additionally, RPO is a source not only of VA and fat[54] but also of antioxidants, in particular vitamin E, which may have a cholesterol-lowering effect[55]. Antioxidant carotenoids also appear to play an important role in the prevention of chronic diseases such as macular degeneration[56,57] and cancer[58,59]. The potential economic benefits of dietary diversification strategies should not be overlooked either, and, in the ongoing programme, the income effect for women involved in RPO production and retailing is being examined.

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