

PRODUCTION OF B-CAROTENE-RICH PRODUCT BY FOAM-MAT DRYING OF CARROT JUICE

Journal

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ABSTRACT

A new β -carotene-rich product was formed by concentration and foam-mat drying carrot juice extract. The drying process yielded β -carotene amounted in 111.3 mg/100g dehydrated carrot juice.

To evaluate the bioavailability of β -carotene from carrot juice powder, the change in concentration of retinol in blood serum and liver tissue of rats were determined, after supplementation with synthetic vitamin A and dried carrot juice. Male albino rats were fed a synthetic diet deficient in vitamin A for 4 weeks, 5 rats were continued on the vitamin A deficient diet for 4 more weeks and 16 were repleted with vitamin A (4000 IU/kg diet) in the form of vitamin A acetate (group A, n = 8), or dehydrated carrot juice (group B, n = 8) for 4 weeks. Whereas10 rats were continued on the vitamin A adequate diet for 4 (n = 5) and 8 weeks, (n = 5). A significant improvement was observed in the serum and liver retinol levels on repletion for 4 weeks in the dehydrated carrot juice group, compared to the vitamin A depleted rats. These results imply that beta-carotene from dehydrated carrot juice was effective in overcoming vitamin A deficiency. Retinol and β -carotene were measured by HPLC.

The study also aimed to fortify yoghurt with dehydrated carrot juice as a natural source of β -carotene (2000 IU/L skimmed milk) to control vitamin A deficiency. After processing, fortified yoghurt was stored at 7 °C for 15 days and analyzed for stability of β -carotene. Levels of β -carotene in fortified yoghurt decreased by about 18% after storage period. Fortification had no significant effect on taste, color, flavor and appearance of yoghurt.

Results suggest that in the developing countries like Egypt, sources of vitamin A such as dehydrated carrot juices are valuable in overcoming the problem of vitamin A deficiency.

Key words: β-carotene - foam-mat drying - Fortifyed yoghurt with dehydrated carrot juice

INTRODUCTION

Antioxidants are important in disease prevention in both plants and animals, inhibiting or delaying the oxidation of biomolecules by preventing the initiation or propagation of oxidizing chain reaction (Velioglu et al., 1998). Synthetic antioxidants require extensive and expensive tests to ascertain their safety for food applications and for this reason; there is interest in the use of naturally occurring antioxidants (Frankel, 1995). The consequent search for natural replacements for synthetic antioxidants has led to the evaluation of a number of plant sources (Heinonen et al., 1998). Fruits and vegetables contain numerous different compounds, many of which have antioxidant properties such as carotenes (ILSI, 1999).

Provitamin A carotenoids, in particular β-carotene in fruit and vegetables, are the major source of vitamin A (retinol) for a large proportion of the world's population (Bafidu, 1995 & Food and Nutrition Board, 1989). In their 1988 guidelines on the human requirement for vitamin A, the FAO/WHO proposed that 6 μg βcarotene in food has the same vitamin A activity as 1 µg retinol (FAO/WHO, 1988).

β-Carotene (BC) and retinol (or its metabolite, retinoic acid) have been found to reduce the risk of some types of cancer; therefore both have been suggested as cancer chemo preventive agents (Moon et al., 1994 and Ziegler, 1989). However, BC would seem the more appropriate, because of the toxicity of retinol. Consumption of retinyl ester at high level for prolonged periods can lead to liver cirrhosis and other toxic symptoms (Hathcock et al., 1990). BC, even though it is converted to retinol and retinyl esters in the small intestine is nontoxic under the same conditions (Bendich, 1992).

Many studies have showed that high doses of BC do not produce high levels of retinol storage in the tissue and do not cause hypervitaminosis A (e.g. Moore, 1957). Brubacher & Weiser (1985) and Chen et al. (1999) gave different doses of BC to rats and measured retinol storage in liver. They reported that the efficiency of the conversion to retinol decreased with increasing doses.

Bioavailability is defined as the fraction of an ingested nutrient from food that is available for absorption in the intestine and metabolic process and storage (Jackson, 1997). The bioavailability of BC from plant food sources generally is lower (about one-third to one fourth) than that from the pure compound (Brown et al., 1989, Micozzi et al., 1992, de Pee et al., 1995, Torronen et al., 1996, Castenmiller et al. 1999 and Huang et al., 2000). In carrots the carotenoids are present in crystalline form or associated with proteins embedded in chromoplasts (Byrant et al., 1992), which limit their release during the digestion process. Cooking may enhance the carotenoid release by softening or breaking down the cell walls and by dissociating the protein complex (Poor et al., 1993; Zhou et al., 1996; Rock et al., 1998). Another way of reducing matrix effects is by homogenization or particle size reduction (Gartner et al., 1997; Castenmiller et al, 1999; van het Hof et al., 1999). Dietary fat, as well, has a documented positive effect on bioavailability of carotenoids (Roels et al., 1958; Dimitrov et al., 1988; Shiau et al., 1994: Jalal et al., 1998).

The normal level of fortification in processed milk within the United States is 1163 μ g/L (2000 IU/L) of retinyl palmitate, which supplies 40% of the recommended daily intake (National Research Council, 1989). A according to FDA, all fluid milk products that are fortified with vitamin A cannot exceed 1744 μ g/L (3000 IU/L) to be in regulatory compliance for the usual claim of 1163 μ g/L (2000 IU/L) (FDA, 1999).

The objective of this research was performed to: (a) produce carrot juice powder by applying foam-mat drying; (b) compare between the bioavailability of β -carotene from dried carrot juice as well as synthetic vitamin A; (c) The possibility of utilization of this natural β -carotene in food.

MATERIALS AND METHODS

Materials:

Fresh carrots (Daucus carota L. Var. Chanteny) were obtained from the Vegetable Research Department of the Horticulture Research Institute ARC, Giza. Standard of \beta-carotene and retinol were purchased from Sigma Chemical Co. (USA). All solvents used for extraction and HPLC were analytical and HPLC grade purchased from Merck (Darmstadt, Germany), respectively. Egg albumin powder purchased from LOBA CHEMIE PVT. LTD, (India). Skimmed milk was obtained from the local market.

Processing of carrots:

Carrots were washed with water before peeling. The peeled carrots were cut into small pieces. The juice was extracted with a grinder (food preparation center CombiMax 700 "Braun" Germany): then pasteurized at 90 °C for 10 minutes, followed by concentration by using a rotary vacuum evaporator at 60 °C.

Production of foam-mat dried carrot juice:

The concentrated carrot juice was blended with 1.5% dried egg albumin as foaming agent and then whipped for 10 min to increase the volume to about three folds. A foamed of carrot juice with four cm thickness were spread over drying trays and dried in a laboratory air drier oven at 70 °C for 5 hr.

Fortification of voghurt with B-carotene:

Skimmed cow milk was warmed to 50-60 °C and cooled to 37 °C then starter organisms were added. Yoghurt was fortified with dehydrated carrot juice, in amount equivalent to 2000 IU vitamin A acetate/L skimmed milk, using the conversion 1 IU = 0.6 μg βcarotene. Fortified milk was poured in plastic containers, fallowed by incubation for 6 hrs to make yoghurt (as traditional method). The yoghurt was transferred to refrigerator at 7 °C for storage.

Chemical analysis:

Moisture, protein, fat, ash and carbohydrates were determined according to the methods described in the A.O.A.C. (1984).

Analysis of \(\beta\)-carotene and vitamin A:

Analysis was carried out using a Hewlett Packared 1050 liquid chromatograph equipped with a model HP1050 pump. The HPLC column was a ODS, HP, 250 X 4mm, 5µm) Spherisorb, and UV

detector (VWD) HP1050, the samples were injected by HP1050 auto sampler. Data were stored and analyzed by computer system (hp, HPLC Chem Station, software).

β-carotene in dehydrated carrot juice and fortified yoghurt was measured by HPLC (Sadler *et al.*, 1990). Mobile phase was methanol: THF: water (67: 27: 6), flow rate 2ml/min at a wavelength of 450 nm.

Retinol in plasma and liver was determined by HPLC (Bieri et al., 1979), mobile phase consisted of methanol only, which was used for retinol analysis by monitoring the absorbance at 325 nm, and flow rate 1 ml/min.

Experimental animals:

Fifty male albino rats (50-60g) were randomly divided into three main groups, baseline (n = 5); control (n = 10) and experimental (n = 35). The control rats (n = 10)was divided into two sub groups which (a) were fed a vitamin A adequate diet for four (n = 5) and (b) eight weeks (n = 5). At the end of the experiment, the animals were sacrificed to obtain data of serum and liver vitamin A level. The five rats in the baseline group were sacrificed at the beginning of the study to obtain reference values for serum and liver parameters at baseline.

The experimental group of rats (n = 35) was fed a vitamin A deficient diet for 4 weeks, during which 9 rats died. All others developed unmistakable clinical signs of vitamin A deficiency, which included hair loss, easy pluckability, discoloration in fur and corneal changes. The surviving (26) rats were divided randomly into four groups. The first group of five rats was sacrificed and autopsied immediately to obtain liver and serum vitamin A level. A second group of 5 rats was continued on the vitamin A deficient diet for 4 more weeks. The two other groups of 8 animals each were repleted with a) synthetic vitamin A (Group A), b) dehydrated carrot juices (Group B). Dehydrated carrot juice was analyzed for vitamin A and the quantities added corresponded to four times the amount of synthetic vitamin A (4000 IU/kg diet) given to animals in Group A. The repletion period was four weeks at the end of the experiment, which the animals were sacrificed and all analyses carried out.

During the course of the 8-week study period, food intakes and body weights were recorded every day. The animals were also observed for the presence of clinical signs and symptoms of vitamin A deficiency.

Diet:

Animals were fed a diet deficient in vitamin A and B-carotene to deplete the gastrointestinal tissue of retinoids and carotenoids. A synthetic diet consisting of 200g casein, 170g sucrose, 205g dextrose, 205g dextrin, 150g palm oil, 50g salt Mix, 20g vitamin Mix (complete except for choline, methionine and vitamin A), and 30g agar (Newberne and Suphakarn, 1977). The control group of rats received this diet complete with vitamin A and 3g, choline, During repletion, the same diet was fed with addition of synthetic vitamin A or dehydrated carrot juice such that the amount of dehydrated carrot juice added was equivalent to 4000 IU vitamin A acetate/kg diet, using a conversion factor of 4 from carotene to vitamin A. All the calculations were based on the \(\beta\)-carotene value of dehydrated carrot juice analyzed, 111.3 mg/100gm, using the conversion 1 $IU = 0.6 \mu g \beta$ carotene. The dehydrated carrot juice was mixed with the vitamin A deficient diet twice during the study.

Feeding and care of animals:

The animals were housed in individual cages. Food and water were given ad libitum. A 12-h dark/light cycle was maintained. Individual body weights and food intakes were recorded every other day during the 8-week study period.

Tissue collection:

After noting the food intake and body weight, the animals were lightly anaesthetized with chloroform, and severing the jugular vein collected blood. If inadequate blood was obtained from the jugular vein, the heart was punctured and blood was collected. The collected blood allowed to stand for clotting at room temperature. Thereafter, it was centrifuged for 10 min at 1500 rpm for the separation of the serum. The serum was stored in a freezer until the analysis of retinal, which was usually done within 24 hours of sacrifice. The liver was quickly removed. The non-hepatic tissues were removed from the liver, which was then blotted on a filter paper and weighted. Each liver was wrapped in aluminum foil and stored in the freezer until analysis of retinol content, which was usually done within 24 hrs of sacrifice. The kidney, and spleen were also removed, cleaned and weighed.

Parameters studied:

Food intake, weight gain and organs weight were recorded. Clinical signs and symptoms were recorded using the methods detailed by Dawson et al. (1980).

Organoleptic tests:

Organoleptic tests were carried out according to Amerine et al., (1965). Ten panelists were asked to evaluate samples for their characteristics (color, taste, odor and general appearance); the score used for each quality in the sheet was 10 while in general appearance was 20.

Statistical analysis:

All data were presented as the mean ± standard deviations (S.D.). Significant differences were determined according to Snedecor and Cochran (1967) by one-way ANOVA using MSTAT-C, (Michigan state university version 1.42, computer software), Duncan's multiple range tests was performed if differences were identified between means at P<0.05.

RESULTS AND DESCUTION

Food intake.

The average daily food intake is shown in Table (1). No significant (P<0.05) change between the control (vitamin A sufficient diet) and experimental group (vitamin A deficient diet) rats was observed until the 3rd week of the study period. Thereafter, a significant decrease in the food intake was seen in the experimental rats. Murthy *et al.* (1985) have reported that dietary vitamin A deprivation resulted in a gradual atrophy of the circumvallate papilla resulting in a loss of taste. Similar observations were found by Zile *et al.* (1981) who reported that, in animals, vitamin A deficiency manifested first by retardation in growth and loss of appetite followed by decrease in storage depots of vitamin A. As mentioned in the methods section 2 groups of 8 vitamin A deficient rats each were repleted with either synthetic vitamin A or β-carotene following the 4-week depletion period.

In the control group of rats, the average food intake stabilized at 14.06 g/day/rat from the 5th week onwards. In the group of experimental rats continued on the vitamin A deficient diet for 8

weeks, food intake declined from 7.3 g/day in the 5th week to an average of 4.06 g/day in the 7th week of the study, after which all the rats died. Therefore, for the 8-week depletion period, serum and liver vitamin A could not be obtained.

The food intake increased significantly on repletion with various sources of vitamin A. The highest change in food intake was recorded for Group B, in which the rats had an average daily intake of 11.83 g in the 5th week and 16.31 g by the end of 8 weeks of the repletion period. In comparison, the animals in Group A increased their food intake from 11.38 g in the 5th week to 15.96 g in the 8th week.

Thus, it can be interpreted that, β-carotene from dehydrated carrot juice was highly accepted by the vitamin A deficient animals; their appetite was returned to normal with no difference in the food intakes seen between the synthetic vitamin A group (Group A) and those fed the dehydrated carrot juice (Group B).

Body weight.

Also, as seen from Table (1), the control group of rats had an increase in weight from the initial mean of 53.07 g to the final of 131.97 g at the end of 8 weeks of study. The experimental group of rats depleted of vitamin A, showed a loss in weight significantly beyond the 3rd week of the study compared with control. Thereafter, the animals fed vitamin A deficient diets continued to lose weight and none in the group continued on the vitamin A deficient diet for 8 weeks survived.

The two groups of rats which were repleted after 4 weeks of depletion started gaining weight by the end of the 1st week of repletion, attained body weights similar to the control group by the end of the 4th week of repletion and surpassed the control group by 4 weeks of repletion. The present study thus corroborated the results of Anzano et al. (1979) and Nambiar & Seshadri, (2001), who have shown a similar pattern of body weight gain upon supplementation. It is clearly evident that dehydrated carrot juice was equally effective in supporting growth and restoring it to normal in vitamin A deficient animals.

Table (1): Effect of feeding trials of experimental rats.

_ 		Dietary groups			
Parameter	Period	Control	VAD	Gr A	Gr B
	(weeks)	(n=5)	(n=5)	(n=8)	(n=8)
	1	6.12±0.82*	5.83±1.05 a	5.69±0.28 a	5.81±0.28 a
	2	6.98±0.54 °	6.25±0.89 a	6.02±0.26 a	6.35±0.63 °
	3	8.86±0.66 a	6.62±0.68 b	6.30±0.52 b	6.43±0,55 b
Average daily food	4	11.56±0.89 a	6.91±0.94 6	6.90±0.64 b	7.10±0.32 b
intake (g/rat)*	5	12.96±0.67 a	7.30±0.85 b	11.38±0.48°	11.83±0.21 °
	6	14.06±0.45°	4.73±0.79 b	13.66±0.66 a	13.85±0.65 a
	7	14.96±0.38 a	4.06±0.62 b	14.59±0.85 a	14.98±0.74 a
	8	16.26±0.78 a		15.96±0.42 a	16.31±0.66 a
Total food intake (g)		91.75	_	80.50	82.66
	1	53.07±2.35°	51.13±1.26 ^b	52.08±1.12 a	53.38±1.39 °
	2	59.35±1.89 °	53.06±0.84 b	53.61±1.68 b	53.21±0.65 ^b
	3	65.69±0.98 a	51.81±0.69 b	52.96±0.96 b	52.93±0.95 b
Average body weight*	4	75.08±3.02 a	50.41±0.79 b	51.13±0.58 b	51.06±1.38 b
(g)	5	86.96±2.04 a	50.01±0.85 b	80.63±0.65°	82.96±1.09°
	6	100.83±1.56 a	48.93±1.64 b	98.96±0.78*	99.31±1.42 a
	7	115.39±1.66 a	45.12±1.55 b	116.69±0.69 a	117.38±2.69 a
	8	131.97±1.81 a		132.31±1.09 a	132.69±1.93 a
Body weight gain (g)		78.90 a		80.23 a	79.31 a
Food efficiency ratio		0.859		0.996	0.959

^{*} Values are mean ± SD. Values with different superscript letters in a horizontal row are significant different (P<0.05).

⁻ All the rats died.

Efficiency of food utilization.

Food efficiency ratio (FER) was calculated by noting the changes in the body weights of the rats in relation to the food intake. A marked increase was seen in the efficiency of food utilization values in the repletion phase in which the weight gain was better than in the control groups throughout the last 4 weeks of the study. Thus, the food efficiency ratios suggested that the \beta-carotene availabilities of dehydrated carrot juice (FER, 0.959) was very good and were similar to those of synthetic vitamin A (FER, 0.996) by the 8th week.

Organ weights.

The liver weight of the control group was 2.02 g at 4 weeks and 5.86g at 8 weeks compared to 0.75 g at baseline. The liver weight of the vitamin A depleted rats at 4 weeks was only 1.37 g. On repletion, a significant increase in the liver weights occurred, restoring them to the same weight as that seen in control rats at 8 weeks (Table 2). The weights of kidneys, and spleen followed a similar pattern.

Table (2): Organ weights of rats fed dehydrated carrot juice*.

Organ	Period	Dietary group				
weight (g)	(weeks)	Control (n=5)	VAD (n=5)	Gr A (n=8)	Gr B (n=8)	
	0**	$0.75 \pm 0.53^{\circ}$	0.75 ± 0.53^{a}	0.75 ± 0.53^{a}	0.75 ± 0.53^{a}	
Liver	4	2.02 ± 0.49^{a}	1.37 ± 0.29^{b}	1.40 ± 0.19^{b}	1.38 ± 0.41^{6}	
	8	$5.86 \pm 0.36^{\circ}$		5.90 ± 0.25^{a}	5.99 ± 0.22^{a}	
	0**	0.66 ± 0.48^{a}	0.66 ± 0.48^{a}	0.66 ± 0.48^{8}	0.66 ± 0.48^{a}	
Kidney	4	$0.81 \pm 0.32^{\circ}$	$0.73 \pm 0.40^{\text{ b}}$	0.74 ± 0.29^{b}	0.73 ± 0.35^{b}	
	8	0.97 ± 0.33^{a}		0.95 ± 0.36^{a}	$0.97 \pm 0.51^{\text{ a}}$	
	0**	0.23 ± 0.13^{a}	0.23 ± 0.13^{a}	0.23 ± 0.13^{a}	0.23 ± 0.13^{a}	
Spleen	4	0.38 ± 0.28^{a}	0.28 ± 0.37^{b}	0.30 ± 0.29^{b}	$0.29 \pm 0.37^{\text{ b}}$	
_ [8	$0.45 \pm 0.43^{\circ}$		0.43 ± 0.56^{a}	0.46 ± 0.28^{2}	

^{*} Values are mean ± SD. Values with different superscript letters in a horizontal row are significant different (P<0.05).

Clinical signs and symptoms:

Marked clinical signs and symptoms of vitamin A deficiency were observed by the end of the third week in the experimental groups, fed the vitamin A deficient diet. Apparent hair loss characterized by easy pluckability from the coat, discoloration in the fur and corneal

^{**} Baseline group.

⁻ All the rats died.

changes, loss of luster and small amounts of porphyrin were evident around the eyes, which progressed as the deficiency continued. A swelling of the eyelids was observed and the anterior segment of the eye had a characteristic dry appearance. A small white spot was observed in two of the experimental group rats, Dawson *et al.* (1980) reported that all mammalian species depleted of vitamin A show rod dysfunction, conjunctival and corneal xerosis, These changes occur only after liver and serum retinol levels are substantially depleted.

On either vitamin A or dehydrated carrot juice repletion, the ocular signs disappeared by 3 weeks of repletion indicating that the β -carotene from the dehydrated carrot juice was efficiently absorbed and was able to reverse the signs of exophthalmia. Mariath *et al.* (1989) have also shown a reversal of clinical exophthalmia after feeding fruits of palm tree *(Mauritiavinifera)* providing 134 μ g of retinol equivalents for 20 days to rats.

Serum vitamin A:

Serum retinol concentrations are normally maintained with in a narrow range in individuals with adequate liver vitamin A stores. Thus, serum retinol is a useful indicator of vitamin A status and can be used to identify of hypo- and hypervitaminosis A (Underwood, 1994). The serum retinol levels measured at the 4th week revealed that a rise than the baseline value of 27.78 µg/dL to 33.13 µg/dL was evident in the control group of rats, whereas a significant decline to 17.38 µg/dL was seen in the rats depleted of vitamin A (Table, 3). The rats continued on the control diet for another 4-week period showed a slight increase in serum retinol level. On repletion, the maximum rise was seen in Group A (given vitamin A acetate) (32.86 μg/dL), followed by Group B (given dehydrated carrot juice) (26.31 µg/dL). Though the serum retinol levels of the groups supplemented with \betacarotene in the form of dehydrated carrot juice was lower than the synthetic vitamin A group; they were higher than the serum retinol levels seen after 4 weeks of depletion. The serum retinol levels after 4 weeks of repletion in Group B approached the value seen in the animals at baseline (27.78 µg/dL) to support the argument that foods containing provitamin carotene are valuable as a source of vitamin A.

Liver vitamin A:

Since 90% of the body's reserves of the vitamin A are stored in the liver and these are readily depleted during vitamin A deficiency. measurement of hepatic vitamin A content yields the most accurate in formation about the vitamin A status (Underwood, et al., 1979). However, serum retinol concentrations decrease transiently during the acute phase response to infection (Filteau, et al., 1993 and Mitra, et al., 1998). Such decreases do not reflect changes in liver vitamin A stores and, thus, can interfere with the use of serum retinol as an indicator of vitamin A status (Klasing, 1988). Table (3), data reveals that the deposition of vitamin A in the control group of rats at 4 weeks (5.85 µg/g liver) and 8 weeks (5.04 µg/g liver), significantly exceeded that at baseline (2.97 µg/g liver). In the experimental group at 4 weeks, the liver retinol was 1.74-µg/g liver, less than 1/3 of the control rats. A decline in the hepatic retinol levels in vitamin A deficiency have been reported by Mejia et al. (1979) and Nambiar & Seshadri, (2001). Significant increases in the liver retinol levels were observed in both the repleted groups, where in group B had the maximal value of 5.16 µg/g liver, approaching the value seen in the control rats at 8 weeks (5.04 μ g/g liver).

Table (3): Plasma and liver retinol of rats fed dehydrated carrot juice.

Parameter	Period	Dietary group				
	(weeks)	Control (n=5)	VAD (n=5)	Gr A (n=8)	Gr B (n=8)	
Serum	0**	27.78 ± 0.82^{a}	$27.78 \pm 0.88^{\circ}$	27.78 ± 1.26. a	27.78 ± 0.91^{2}	
	4	33.13 ± 0.58^{a}	17.38 ± 0.89^{b}	17.38 ± 2.56^{b}	17.38 ± 0.52^{b}	
(μg/dL)	8	35.09 ± 0.96^{a}		32.86 ± 2.81^{2}	$26.31 \pm 0.76^{\mathrm{b}}$	
Liver	0**	2.97 ± 0.21^{a}	2.97 ± 0.36^{a}	2.97 ± 0.62^a	2.97 ± 0.36^{a}	
	4	5.85 ± 0.19^{a}	1.74 ± 0.12 b	1.74 ± 0.09^{b}	$1.74 \pm 0.21^{\mathrm{b}}$	
(μg/g)	8	5.04 ± 0.09^{a}		4.97 ± 0.19^{8}	5.16 ± 0.08^{a}	

^{*} Values are mean ± SD. Values with different superscript letters in a horizontal row are significant different (P<0.05).

^{**} Baseline group.

⁻ All the rats died.

Utilization of dehydrated carrot juice: Fortification of yoghurt with β-carotene:

It is a well known that the vitamin A content of milk supplied through dairies is reduced to a great extent due to removal of fat. Fortification of such milks with vitamin A is the only remedy to overcome the nutritive deficiency of these milks.

Table 4, showed the chemical composition of fortified yoghurt with dehydrated carrot juice, data showed that all groups have almost the same values of the main constituents of yoghurt.

Table (4): Chemical composition of fortified yoghurt with dehydrated carrot juice (g/100g)

Groups	H ₂ O	Protein	Fat	Carbohydrate	Ash
Control	88.2	3.1	0.02	8.02	0.95
Fortified	87.9	3.4	0.02	8.07_	1.36

Table 5, showed the stability of β -carotene during the processing of fortified yoghurt, the loss of β -carotene was very negligible (1.6%) which showed the stability of β -carotene during the processing of fortified yoghurt.

Table (5): Effect of processing on the stability of β -carotene of fortified voghurt.

Treatment	β-carotene content (IU/100ml)*
Unfortified milk	N.D.**
Fortified milk	200.59 ± 9.85
Fortified yoghurt	197.42 ± 12.85

^{*} Values are mean $(n=3) \pm SD$.

Table 6, shows the stability of β -carotene in the fortified yoghurt during storage for 15 days at 7 °C. It could be noticed that, there was a continual decease in β -carotene content during storage, level of β -carotene in fortified yoghurt decreased by about 18% after 15 days of storage. The loss in the β -carotene attributed to destroy by light, air or its combination. Carotenoids are easily oxidized because of the large number of conjugated double bonds (Fennema, 1996). Also, the degradation rate increased significantly during storage and this may be due to decreasing of PH values (Rizk *et al.*, 2002).

^{**}N.D. = not detected.

Table (6): Effect of storage at 7°C on the stability of β-carotene of

fortified voghurt.

Storage period (days)	Fortified yoghurt (IU/100ml)*	% Loss
0	197.44 ± 6.59 °	
3	190.29 ± 8.55 b	3.6
6	186.54 ± 10.36 °	5.5
9	175.69 ± 13.96 ^d	11.0
12	167.59 ± 15.12 f	15.1
15	161.87 ± 14.95 g	18.0

^{*} Values are mean $(n=3) \pm SD$.

Sensory evaluation:

Table 7, showed the effect of dehydrated carrot juice added to yoghurt on different organoleptic properties of yoghurt. The overall judgement degrees of fortified voghurt with a natural source of Bcarotene were around the same values of control group. These results ment that addition of dehydrated carrot juice in yoghurt fortification has no effect on general organoleptic properties.

Table (7): Effect of fortified milk with dehydrated carrot juice on

the organoleptic properties of voghurt*.

Yoghurt properties	Control	Fortified yoghurt
Taste (10)	9.6 ± 2.92^{a}	9.5 ± 1.89°
Color (10)	9.5 ± 2.05^{b}	9.5 ± 2.15^{b}
Odor (10)	9.3 ± 3.66^{c}	9.4 ± 2.64^{c}
General appearance (20)	19.1 ± 2.85^{d}	19.0 ± 3.88^{d}
Overall acceptapelity (50)	47.5 ± 2.87^{e}	47.4 ± 2.64^{e}

^{*} Values are mean ± SD.

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abcdefg Values on the same column not sharing the same superscript were significantly different (p<0.05).

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إنتاج منتج غنى بالبيتا كاروتين بتجفيف عصير الجذر بالرغوة

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- تم إنتاج منتج غنى بالبيتا كاروتين وذلك باستخلاص عصير الجذر وتركيزة ثم تجفيفه بالرغوة ، والمنتج الناتج أحتوى على ١١١،٣ مليجرام بيتا كاروتين لكل ۱۰۰ جرام عصير جذر مجفف.
- ولتقييم التمثيل الحيوي للبيتا كاروتين بعصير الجذر المجفف تم تقدير التغير في تركير الريتينول في كلا من سيرم الدم والكبد للفئران المغذاة على غذاء به فيتامين أ مخلق أو به عصير جزر مجفف. واستخدم في ذلك فئران ذكور امهق تم تغذیتهم علی غذاء به نقص فی فیتامین أ لمدُه ٪ أسبوع و استمر ۗ ٥ فئران منهم على هذا الغذاء لمدة ٤ أسابيع أخرى بينما تم تغذية ١٦ فأر على طعام به كمية زائدة عن احتياج الفأر من فيتامين أ (٤٠٠٠ وحدة دولية/كجم طعام) أما في صورة فيتامين أ أسيتات (مجموعة أ وعددها ٨ فأر) أو في صورة عصير جذر مجفف (مجموعة ب عددهم ٨ فأر). بينما استمر تغذية ١٠ فئران على طعام به احتياج الفأر من فيتامين أ لمدة ٤ أسبوع واستمر ٥ فئران منهم على هذا الغذاء لمدة ٤ أسابيع أخرى.
- لوحظ تحسن في مستوى ريتينول سيرم الدم والكبد في المجموعة التي غزيت على طعام به كمية زائدة عن احتياج الفأر من فيتامين أ وذلك مقارنة بالفئران المغذاة على غذاء به نقص في فيتامين أ وهذه النتائج تدل على أن البيتا كاروتين بعصير الجذر المجفف له تأثير فعال في التغلب على نقص فيتامين أ . استخدم جهاز HPLC لتقدير كلا من الريتينول والبيتا كاروتين.
- أيضا استهدفت الدراسة إلى إنتاج زبادي مدعم بالبيتا كاروتين (٢٠٠٠ وحدة دولية/لتر لبن منزوع الدسم) واستخدم عصير الجذر المجفف كمصدر طبيعي للبيتا كاروتين وذلك للتغلب على نقص فيتامين أ. وتم تقدير ثبات البيتا كاروتين في الزبادي المدعم أثناء تخزينه على ٧ م لمدة ١٥ يوم ، ووجد أن نسبة البيتا كاروتين في الزبادي المدعم انخفض بنسبة ١٨%من الكمية الأساسية بعد فترة التخزين وأيضا وجد أن عملية التدعيم لم تحدث أي تأثير على الطعم أو اللون أو النكهة وأيضا على مظهر الزبادي.
- ومن النتائج يمكن أن نتوصل إلى أنة للتغلب على مشكلة نقص فيتامين أفى الدول النامية مثل مصر يمكن استخدام عصير الجذر المجفف كمصدر طبيعي له.