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Effects on Vitamin A Deficiency Disorder in Rural Areas Special Reference of Barjora Block in the District of Bankura in West Bengal

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Abstract

A fat-soluble factor present in butter was essential for the growth of rats on synthetic diet. Carotene is the precursor of vitamin A retinol is vitamin A alcohol found in mammals and is abundant in the liver oils of salt water fish. Fenretinide is a synthetic along of retinol. Vitamin A contents a beta ionane ring. Vitamin A plays a critical role in vision in dim light by the visual cycle. The important deficiency States due to lack of vitamin A in the diet are night blindness, Xerosis conjunctiva, Xerosis cornea, Bitot'spot and keratomalacia. Mild moderate cases of vitamin A deficiency can be treated by the daily oral dose of 10000 microgram of fat-soluble vitamin A for a period of 20 days. The study has conducted by sampling survey, diet survey by diet recall method and clinical assessment of nutritional status.

Food habit of the population of the survey area is greatly determined by their economic condition and nutrition education. The diet consumed by the majority of the population consist of predominantly of rice and moderate number of vegetables and negligible amount of egg, meat and fish. Most of the persons of this area are daily labourers, mason, maid servant and the helper of shopkeeper. They are economically backward and due to lack of knowledge about nutrition, they choose im proper diet. As a result, by the clinical assessment suffer from night blindness and 3.12% suffer from Bitot'spot, which are major clinical manifestation of vitamin A deficiency as clinical assessment evaluate the long-term nutritional history. So, we may say that 4.99% of population suffer from long term deficiency vitamin A.

Keywords: Synthetic Diet; Carotene; Fenretinide; β-Ionone; Visual Cycle; Night Blindness; Xerosis Conjunctiva; Xerosis Cornea; Bitto's Spot; Keratomalacia

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Introduction

Mc Collum and Devis [1] and Osborne and Mendel [2] showed that a fat-soluble factor present in butter was essential for the growth of rats on synthetic diet. The former group of workers called the factor as fat-soluble A'.

Steen bock (1919) discovered the vitamin A activity of carotenoids. Letter he (1920) found that vitamin A was present in the unsaponifiable fraction of fish liver oils. Moore (1930) made the important discovery that when carotene was fed to vitamin A deficient rats, vitamin A was found in the liver. In 1931 Karrer obtained a highly concentrated vitamin A preparation and determined the structure of the vitamin. In 1937, Khun and Morris announced a method of synthesis of vitamin A. Holmes and Corbet [3] obtain vitamin A in a crystalline form.

Carotene is the precursor of vitamin A. In nature carotene are synthesized exclusively by photosynthetic microorganisms. The general term vitamin A includes all compounds that have vitamin A activity. Retinol is the generally accepted chemical name for vitamin A. Retinol is vitamin A alcohol found in mammals and is abundant in the liver oils of saltwater fish. It was the empirical formula $C_{20}H_{29}OH$. The transport form is retinal while it's exter is stored in the tissues. Only 11 cis retinol is necessary for the formation of rhodopsin, which has high photo sensitivity. Retinal palmitate is water miscribe and thus suitable for intra muscular injection. In premature infants, retinyl palmitate supplement with intravenous lipids appears to be an excellent adjuvant to routine vitamin A supplement [4].

Fenretinide is a synthetic analog of retinol. It is not stored in the liver [5].

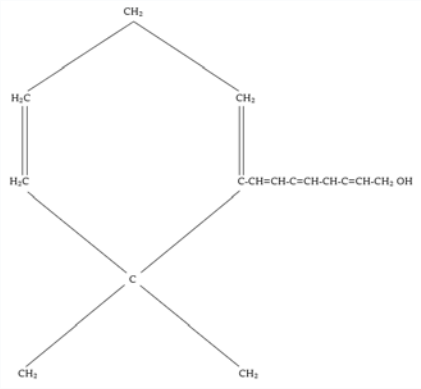

Figure 1.1: Vitamin A contents a beta -ionone ring. The side chain contents two isoprene units, 4 double bonds and one alcoholic group. Due to the presence of 4 double bonds, vitamin A is destroyed easily by oxidation, vitamin A2 is differs from vitamin A in having one more double bond in the beta ionone ring.

Carotene (Pro-vitamin A drives its name) from the plant pigment in carrots. It is a precursor of vitamin in the body. It is abundant in green and yellow vegetables as well as in yellow fruits, but is never produced in animals. More than 600 carotenoids have been described [6].

Chemical structure

The chemical structure of vitamin A (Retinal) is given below in Figure 1.

Source of vitamin A

Vitamin A is widely distributed in animal and plant food. In animal food as performed vitamin A (retinal) and in plant foods as Pro vitamins (carotenes).

a) Animal foods: Foods rich in retinal are liver, eggs, butter, cheese, whole milk, fish and meat, fish liver oils are the richest natural sources of retinal.

b) Plant foods: The cheapest source of vitamin A is given leafy vegetables such as spinach and amaranth which are found in great abundance in nature throughout the year.

Fortified foods: Foods fortified with vitamin A (e.g. vanaspati, margarine, milk) can be an important source. Vitamin A content food is given in table 1.1, 1.2.

Physiological and Biochemical functions of vitamin A

Vitamin A and vision: vitamin A plays a critical role in vision in dim light. The vitamin A visual cycle in soon in figure 1.2.

According to Wald (1935) the characteristic pigments of the retinal rods and cones rhodopsin. They differ only in respect of the protein moieties (called Scot opsin and photopsin respectively). The specific pigment common to both is a cis- isomer of retinene (vitamin A aldehyde) light initiates a series of photochemical changes in rhodopsin, beginning with bleaching of the purple pigment and ending with the formation of all trans retinene and its isomerization. The synthesis of rhodopsin is isomer specific requiring the 11cis-isomer of retinene. Morton and coworkers (1953) demonstrated the generation of rhodopsin with all trans vitamin A alcohol, phosphate buffer, magnesium isos and excised rat retinas.

Retinal phosphate has been shown to be formed in mammalian

Table 1.1: Retinal contains of selected food.

Halibut liver oil	900000
cod liver	18000
liver ox	16500
Butter	325
Margarine	900
Cheese	350
Egg	140
milk(cow)	38
Fish	40
Carrot	1167
Spinach	607
Amaranth	515
green leaves	300
mango ripe	313
Papaya	118
Orange	25
Tomato	84

Sources: Swaminathan. M; Food and nutrition, Vol.1, BUPPCO, Bangalore,1999.

Table 1.2: Vitamin content of various foods.

Food	Vitamin A (IU/100gm)	Retinal (micro grams or retinal equivalent)	Micromole
Halibut liver oil	4000000	120000	4400
Cod liver oil	200000	60000	220
Liver, sheep	45000	13500	49.5
Liver, ox	15000	4500	16.5
Liver, pig	5000	1500	5.5
Liver, calf	4000	1200	4.4
Butter	3500	1050	3.9
Cheese (whole fat)	1500	450	1.7
Egg, hen	1100	330	1.2
Kidney, ox	1000	200	1.1
Salmon, canned	250	75	0.28
Milk, summer	150	45	0.17
Herrings, fresh	100	30	0.11
Milk, winter	75	22	0.08
Beef, mutton	20	6	0.02

Sources: Swaminathan. M; Food and nutrition, Vol.1, BUPPCO, Bangalore,1999.

cells both *in vitro* and *in vivo* and has been identified as a constituent of mammalian membranes. Mannosyl retinyl phosphate has been shown to be formed both *in vitro* and *in vivo* in lever cells. The exact role of this compound in the formation epithelial tissues is not known [7].

Effects of vitamin A deficiency

Experimental deficiency is produced when diet is deficient in both retinol and carotene. The resulting hyper keratosis and impaired dark adaptation are corrected by 150 micrograms of retinol or 300 micrograms of beta carotene.

Deficiency occurs when the intake of vitamin A or its precursor carotene, is poor. It is common in economically less privileged

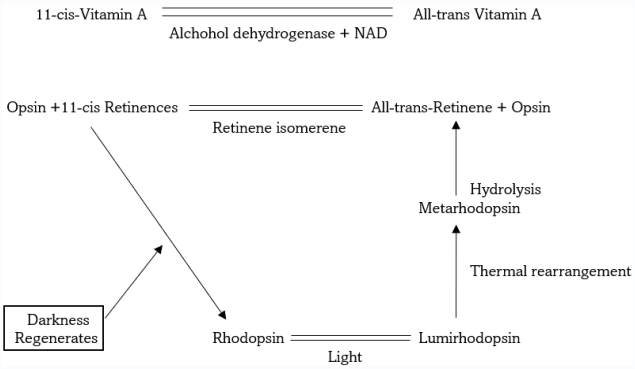

Figure 1.2: Vitamin A visual cycle.

countries, especially in children, due to inadequate intake.

Clinical manifestation

Eye: xeropthalmia is the comprehensive term now used by the WHO is denote all vitamin A deficiency manifestations effecting the structure of function of the eyes, including the conjunctiva, cornea and retia.

The following manifestation are included under the categories:

The important deficiency States due to lack of vitamin A in the diet are:

i) Night blindness

ii) Xerosis conjunctiva

iii) Xerosis cornea

iv) Bitot'spot and

v) Keratomalacia

Night blindness: The role of vitamin A in dim vision has been discussed earlier. In early stages of vitamin A deficiency, the individual cannot see well in dim light. Deficiency in reading or driving the car in dim light is experienced. In advanced deficiency, the subject cannot see objects in dim light. Night blindness is fairly common in regions where the vitamin A intake is inadequate. Night blindness is the earliest symptoms of deficiency [7].

Xeropthalmia: this condition refers to Xerosis of conjunctiva and cornea. These are described below:

Xerosis conjunctiva: the conjunctiva is dry, thickened, wrinkled and pigmentation gives the conjunctiva a smokey appearance. This condition is extremely common among all age group in India and other developing countries where the vitamin A intake is low.

Xerosis Cornea: when dryness spreads to cornea, it takes on a dull, hazy, lusless appearance.

This is due to the keratinisation of the epithelial over cornea.

Bitot's Spots: Bitot described this condition in 1863. Greyish or glistening white plaques formed of desquamated thicken conjunctival epithelium, usually triangular in shape and firmly adhering to the conjunctiva are frequently found in children having other signs of vitamin A deficiency.

Keratomalacia: When Xerosis of conjunctiva and cornea is not treated, itne develop into the condition known as 'Keratomalacia.'

Table 1.3: Prevalence criteria for determining the xerophthalmia problem.

Criteria	Prevalence in population at Risk (6 months to 6 years)
Night blindness	More than 1%
Bitot's spots	More than 0.5%
Corneal xerosis / corneal ulceration / Keratomalacia	More than 0.01%
Corneal ulcer	More than 0.05%
Serum retinol (less than μg/dl)	More than 5%

Sources: Park K; Preventive and Social Medicine.

Metaplasia and degeneration of the corneal epithelium occur, producing opacities and the cornea becomes vascularized.

Oedematous and infiltrated with leucocytes, necrosis, leading to ulceration and bacterial invasion brings about the distraction of the cornea. Blindness often results from corneal scarring of perforation.

Xeropthalmia fundus: The eye shows white or yellow spots along the course of blood vessels. Of the above, the outstanding signs are corneal Xerosis or keratomalacia, conjunctival Xerosis and Bitot's spot [8].

Vitamin A deficient children develop t-cell function abnormalities which are reversible with supplements [9].

Maternal vitamin A deficiency is associated with increased mother to child transmission of the Human Immuno deficiency Virus (HIV) [10] (Table 1.3).

Preventive of vitamin A deficiency

Mild up moderate cases of vitamin A deficiency can be treated by the daily orldoz of 10000 microgram of fat-soluble vitamin A for a period of 20 days.

In severe cases, larger dosage of 20000 microgram of fat-soluble vitamin A will have to be administered Delhi for one week, followed by 10000 micrograms for a father period of 20 days. Studies carried out by Swaminathan and coworkers at Hyderabad (1970) have shown that a single massive those of 50000 I.U. of above 6 months [11].

Treatment: Adequate supply of vitamin A decreases morbidity mortality in children [12, 13]. Access to health care and immunization other factors which determined morbidity [14]. Vitamin A supplement, either as a small weekly dose (8.7 micromol., 8333 I U) or a large dose at 4 monthly intervals (60000 micrograms retinol, equivalent to 2000000 I U vitamin A), [15] reduces childhood mortality [16] (Table 1.4)).

Vitamin A requirements

The recommended daily intake of vitamin A is 750 micrograms for adults.

Table 1.4: Suggested recipes for Vitamin A deficiency.

Recipe	Reasons
Boiled egg, egg custard	Rich in vitamin A and good quality protein
Coriander and mint chutney with bread / chapathi	Not only rich in beta carotene but also rich in other nutrients
Carrot halwa, carrot kheer, carrot juice	Rich in beta carotene
Papaya	Rich in beta carotene
Orange juice, tomato juice, mango juice	Rich in beta carotene and Vitamin C
Palak dal, Palak paneer	Rich in beta carotene and protein

Source: B. Sreelakshmi, Dietetics, New Age International (P) Ltd., New Delhi

Table 1.5: FAO/WHO Expert Group on Vitamin Requirements and I.C.M.R. Expert Groups are given.

Age	ICMR Expert Group Vitamin 'A' Retinol (μg)	ICMR Expert Group Carotene (μg)	FAO Expert Group Vitamin 'A' Retinol (μg)	FAO Expert Group As Carotene (μg)
Upto 1 year	300–400	1,200–1,600	300	1,800
1–3 years	250	1,000	250	1,500
4–6 years	300	1,200	300	1,800
7–9 years	400	1,600	400	2,400
10–12 years	600	2,400	575	3,450
13–15 years	750	3,000	725	4,350
Adolescents	750	3,000	750	4,500
Adults (Men and Women)	750	3,000	750	4,500
Pregnancy	750	3,000	750	4,500
Lactation	1,150	4,600	1,200	7,200

Source: Swaminathan M.; 1999.

The vitamin A requirements for the different age groups recommended by the FAO/WHO Expert Group on vitamin requirements and ICMR Expert Groups are given in Table 1.5.

Materials and Methods

This study has conducted by using the following methodology:

A. Sampling: To conduct the survey, I have use stratified random sampling method. In this method I have classified the total population into four groups or strata:

a. Pre-school children for 1-3- and 4-6-years age group.

b. School children for 7-9 and 10 - 12 years age group.

c. Adults for 19-45 years age group.

d. Adults for above 45 years age group (B. Srilakhsmi; Dietetics, New Delhi)

B. Diet Survey by diet recall method: In this method a trained interviewer asks the responded to recall in detail all the food and drink consumed during a time period in the previous 24 hours. Thus, the method is most commonly known as 24 hours recall. In some cases, the time period is the past 48 hours, the past 7 days or in some rear cases, the previous months. As memories of food intake may fed rather quickly beyond the most recent day or two the accuracy is lesser in long recall methods.

Not only the food items consumed by the respondent in past 24 hours are recorded the interviewer of assists the respondent in estimating portion sizes of food consumed. The interviewer may begin by asking for the respondent first at or drunk on the last awakening. Then he is asked to recall the food items he eats from morning to the current moment.

The interviewer then begins at the point exactly 24 hours in the past and works forward to the time of awaking. Some interviewer ask respondent to recall their diet from midnight to midnight of the previous day. Asking question about the activities of the respondent during the day, the interviewer can help in recalling intake.

The omission and mistake in recalling may be checked by reviewing the previous day's eating episodes several times. In some situation a quick list of food eaten in the previous day is initially recorded in the next phase, a detailed description of foods on the quick list is obtained. He may ask questions hold measures like cups, glasses, spoons, bowls, the geometric shapes of the food portion (e.g. circle, rectangle etc. and levels are also important to measure portion size.

In this method the respondent may have to be contacted letter by telephone or mail to clarify any point or to obtain information such as brand name, preparation method and serving sizes. This recall can then be analysed using a computerised diet analysis programme. A number is assigned to each food item for computer analysis.

This code is entered into the analysis software along with the quantity to calculate the nutrients of that food. The nutrient may be calculated manually from the food composition tables.

C. Clinical Assessment of Nutritional Status: Clinical examination is the most essential part of all nutritional surveys. Since, the ultimate objective is to assess of health of individual and population groups as influenced by the diet they consume the numerous signs and symptoms of dietary deficiencies have been classified by several individual scientists or expert committees.

Overlapping of deficiency States: By and large mal nourished individuals show signs and symptoms due to multiple deficiencies in the diet. Some of the signs and symptoms may be common in several deficiencies States. For example, follicular hyperkeratosis may be due to lack of essential fatty acids or a vitamin A of both. Other associated signs and symptoms present will give a clue to the dietary deficiencies causing glossitis or follicular hyper keratosis.

Classification of clinical signs and symptoms used in nutrition surveys: the signs and symptoms commonly observed in malnourished subject have been classified by the FAO/WHO committee under three heads.

Group I signs related to malnutrition and non to be value in nutrition surveys, Group II signs that need further investigation and Group III signs not related malnutrition. These are presented in APPENDIX -B.

Standardization of techniques of clinical signs and symptoms. The need for standardization of technique for clinical assessment of nutritional status was pointed out by the FAO/WHO committee in 1951. With a view to minimizing the errors in clinical assessment, FAO/WHO committee prepared a table showing the signs APPENDIX -C.

D. Methods of statistical analysis:

1. Mean is calculated by using the following formula: $\bar{X} = \sum X / n$

2. Standard error is calculated by using the following formula:

$$\partial\delta\bar{X} = \delta_s / \sqrt{n}$$

Where δ_S = standard deviation of the sample and is worked out as under.

$$\delta_S = \sqrt{\Sigma(\bar{X}i - \bar{X})^2 / n - 1}$$

n = number of items in the sample.

3. 't' is calculated by using the following formula: $t = (\bar{X} - \delta) / \partial\bar{X}$

Results and Discussion

This survey is conducted on 160 samples. It shows that only 33 persons (20.63%) are taken required amount of Vitamin A as suggested by ICMR. 96 persons (60%) are taken extra amount of vitamin A.

Table 3.1: Results of dietary assessment.

S.L. No.	Age Group (in years)	Sex	No. of Sample	Dietary intake of Vitamin A (in μg/day) Mean ± S.E		RDA of Vitamin A (μg/day)	't' Value
1	Pre-School Children						
	1–3	M	10	1610.9	±16.817	1600	0.648
		F	10	1600.7	±5.325	1600	0.131
	4–6	M	10	1571.8	±42.681	1600	0.660
		F	10	1628.5	±16.946	1600	1.681
2	School Children						
	7–9	M	10	2398.2	±26.439	2400	0.068
		F	10	2415.2	±38.610	2400	0.393
	10–12	M	10	2421.7	±16.462	2400	1.318
		F	10	2429.3	±17.568	2400	1.667
3	Adolescents						
	13–15	M	10	2417.9	±17.174	2400	1.042
		F	10	2355.7	±44.582	2400	0.993
	16–18	M	10	2414.2	±28.720	2400	0.494
		F	10	2421.6	±24.885	2400	0.867
4	Adults						
	19–45	M	10	2455.2	±28.342	2400	1.947
		F	10	2456.7	±22.603	2400	2.508
	Above 45	M	10	2456.0	±27.220	2400	2.057
		F	10	2447.8	±22.717	2400	2.104

Explanation: After computing 't' value, I have tested the significance of difference between means, dietary intake of vitamin A and RDA. There is no significance difference at different age group except adults age group.

Table 3.2: Prevalence of dietary deficiency of Vitamin A in different age group.

S.L. No.	Age Group (in years)	Sex	No. of Sample	Vitamin A deficiency detected	Percentage
1	Pre-School Children				
	1–3	M	10	3	30%
		F	10	2	20%
	4–6	M	10	3	30%
		F	10	2	20%
2	School Children				
	7–9	M	10	1	10%
		F	10	2	20%
	10–12	M	10	2	20%
		F	10	1	10%
3	Adolescents				
	13–15	M	10	2	20%
		F	10	2	20%
	16–18	M	10	2	20%
		F	10	2	20%
4	Adults				
	19–45	M	10	2	20%
		F	10	2	20%
	Above 45	M	10	2	20%
		F	10	1	10%

Table 3.3: Prevalence of dietary deficiency of vitamin A in males and females.

Gender	No. of Sample	Vitamin A deficiency detected	Percentage
Male	80	17	21.25%
Female	80	14	17.5%

Table 3.4: Results of clinical assessment.

Sl. No.	Age Group (in years)	Sex	No. of Sample	No. of cases of Vitamin A deficiency detected according to age and sex
1	Pre-School Children			
	(1–3)	M	10	3
		F	10	2
	(4–6)	M	10	3
		F	10	2
2	School Children			
	(7–9)	M	10	1
		F	10	2
	(10–12)	M	10	2
		F	10	1
3	Adolescents			
	(13–15)	M	10	2
		F	10	2
	(16–18)	M	10	2
		F	10	2
4	Adults			
	(19–45)	M	10	2
		F	10	2
	(Above 45)	M	10	2
		F	10	1

Table 3.5: Percentage of male & female are affected in Bitot's spot by the deficiency of Vitamin A.

Sex	No. of Samples	Percentage
Male Affected by Bitot's spot	3	3.75%
Female Affected by Bitot's spot	2	2.5%

Table 3.6: Percentage of male & female affected by night blindness.

Sex	No of Samples	Percentage
Male affected by night Blindness	2	2.5%
Female affected by night blindness	1	1.25 %

Table 3.7: Prevalence of dietary deficiency of Vitamin A.

Total No. of Sample	Normal intake of Vitamin A	Less amount intake of Vitamin A	Percentage of normal intake of Vitamin A	Percentage of less amount intake of Vitamin A
160	31	129	19.37%	80.63%

Table 3.8: Graphical representation of intake of Vitamin A.

Intake of Vitamin A	No. of Samples	Percentage
< Normal	96	60%
> Normal	31	19.37%
Normal	33	20.63%

The intake of Vitamin A of 31 persons (19.37%) is below the required amount as recommended by ICMR. The result of clinical assessment shows that only 1.87% suffer from night blindness and 3.12% persons suffer from Bitot'spot, which are major clinical manifestation of Vitamin A deficiency as clinical assessment evaluate the long-term nutritional history. So, we may say that 4.99% of population suffer from long term deficiency vitamin A (Table 3.1-3.8, Figure 3.1-3.8).

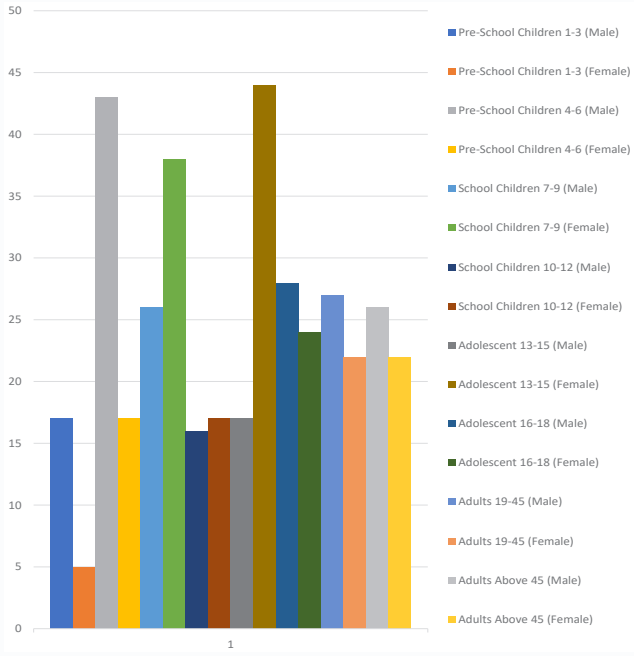

Figure 3.1: Mean Dietary intake of Vitamin A different age group.

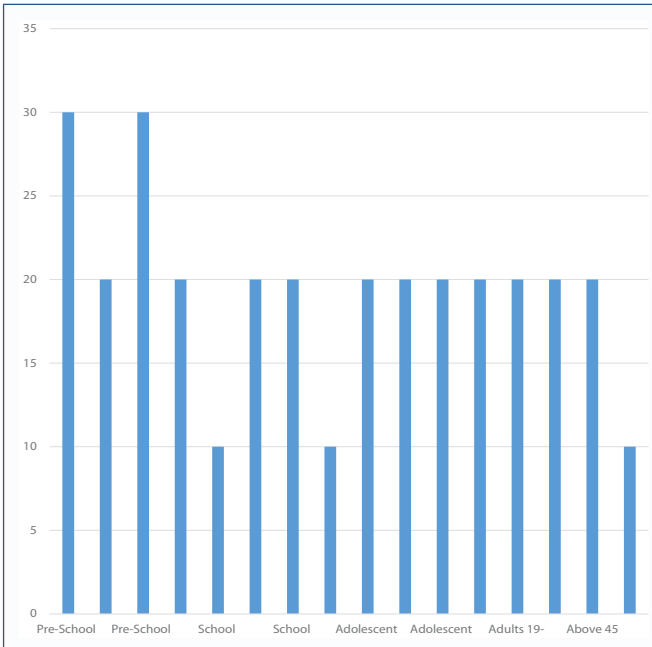

Figure 3.2: Prevalence of vitamin A deficiency of different age group.

Suggestion for prevention of Vitamin A deficiency disordered

A comprehensive strategy simultaneously addressing the following issues is crucial to achieve the goals of elimination of Vitamin A deficiency.

Nutrition Education: the education of the people to promote

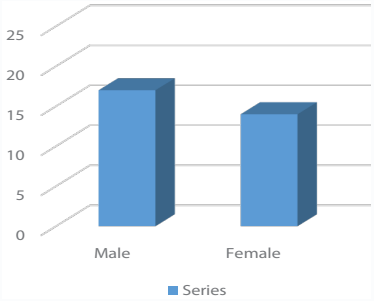

Figure 3.3: Prevalence of dietary deficiency of vitamin A in males and females.

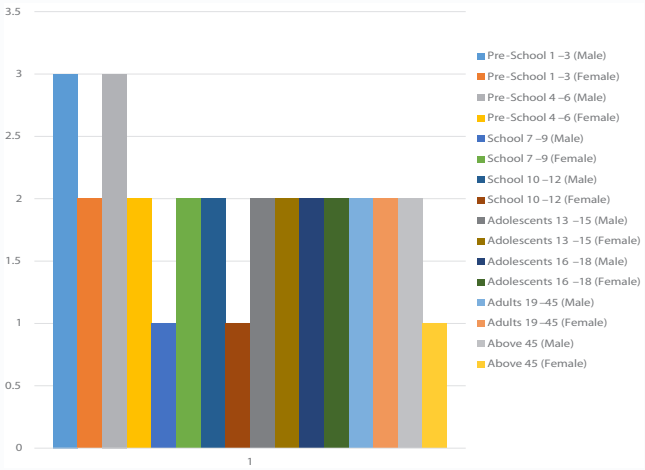

Figure 3.4: Vitamin A deficiency detects clinically.

Figure 3.5: Percentage of Male & Female are affected in Bitot's spot by the deficiency of Vitamin A.
Male: 3.75% and Female: 2.5% (Affected by Bitot's spot).

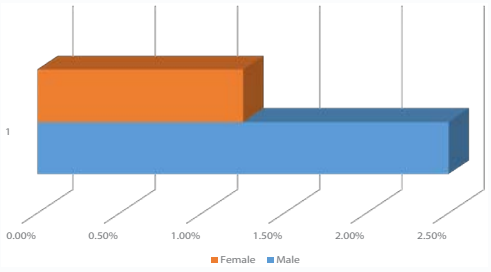

Figure 3.6: Percentage of Male and Female are affected by night blindness.

dietary intake of vitamin A and beta carotene rich foods is the foremost requirement for elevating the problem of Vitamin A deficiency in all age groups.

The strategy though it sounds simple is a complex one needing inputs from various sectors of the Government, Research Institutions, Home Science Colleges, NGOs and the private sector.

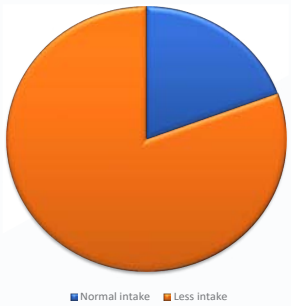

Figure 3.7: Prevalence of normal and abnormal intake of Vitamin A.
A → Population who consumed normal amount of vitamin A.
B → Population who consumed less amount of vitamin A.

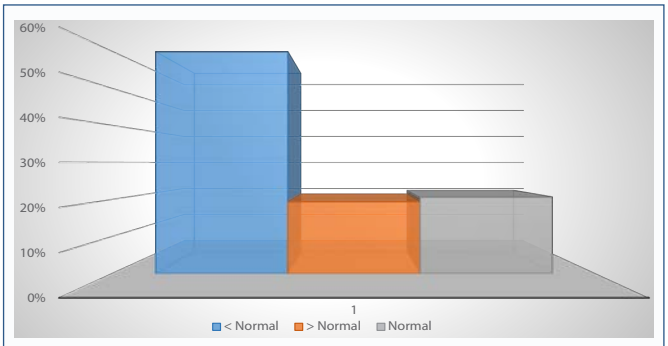

Figure 3.8: Graphical representation of intake of Vitamin A.

It should be recovered by the activities of ICDS.

Nutrient supplementation

"The national prophylaxis programme Against Nutritional Blindness is being implemented in the country since 1970 through health and family infrastructure. This involves oral administration of a massive dose of 200000 IU of vitamin A in oil every 6 months to preschool children. Since, vitamin A can be stored in the liver for a prolonged period, it is possible to build up adequate stores of vitamin A through periodic administration of massive dose of vitamin A. This approach has been found to be feasible in extensive field studies carried out at the National Institute of Nutrition. Studies in preschool children have shown that following a massive dose of 200000 IU of vitamin A, there is significant reduction in the prevalence of ocular science of Vitamin A deficiency.

Since 1992, the programme is included in the 'package of services' provided to children under the Child Survival and Safe Motherhood (CSSM) programme and it addresses child in the age group of 9 months to 3 years only. The first dose of 100000 IU Vitamin A is administered at 9 months of age along with measles vaccine. The subsequent doses of 200000 IU are given at 18 months along with DTP booster dose, at 24, 30 and 36 months. It would be better if children in the age group of 3-6 years are also covered under the vitamin A deficiency control programme.

Conclusion

Nutrition is recognised as major determinant of a wide range of disease. Numerous deficiency diseases continue to persist special in rural areas, as a result of essential nutrients deficiencies in the diet. Vitamin A deficiency is a common nutritional deficiency in our community as a result of poor socio-economic condition in lakh of nutritional knowledge.

By conducting diet survey in a rural area of the population of Barjora Block in the District of Bankura. We found that 20.63% people take the required amount of vitamin A, 60% people take adequate amount of vitamin A and 19.37 % people take inadequate amount of vitamin A with respect to their RDA. The result of clinical assessments shows that 1.87 people suffer from night blindness and 3.12% people suffer from Bitot'spot which are a major clinical manifestation of vitamin A. This result indicates that 4.99% people suffer from long term vitamin A deficiency.

The above result of nutritional assessment gives us a picture of nutritional status of our community. Low socio-economic condition is nor the only cause of nutritional deficiency in our community. Lack of awareness about health and nutrition is another important cause of poor nutritional status of the people of our community.

Vitamin A deficiency leads to xerophthalmia and may result in night blindness in children. It is a wide spread a problem of our community. The government should take initiation to combat with this situation.

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