# Prevalence of hypercarotenaemia in nursery/kindergarten children in the Western province in Sri Lanka: a preliminary survey

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### Article history

### <u>Abstract</u>

Received: 22 May 2012 Received in revised form: 31 January 2013 Accepted: 4 February 2013

#### <u>Keywords</u>

Hypercarotenaemia Prevalence Children

# Hypercarotenaemia is seen more frequently among young infants and children. This condition develops mainly due to excessive intake of carotenoid bearing foods. Evidence shows that, not all infants develop hypercarotenaemia due to excessive intake of carotenoid containing foods. The objective of this study was to study the prevalence of hypercarotenaemia among nursery/kindergarten children in the Western province of Sri Lanka and to correlate the food intake with development of hypercarotenaemia. The occurrence of hypercarotenaemia among their siblings fed similar diets was also observed. A self administrated questionnaire was given to the parents (n = 780) of nursery/kindergarten children (2-5 years) in the Western province to collect information on the intake of carotenoid rich foods and development of hypercarotenaemia among the children attending these nurseries and their siblings. Among all the subjects investigated twelve (n = 12) had developed hypercarotenaemia. Children fed with high carotenoid bearing food/fruits were categorized in to two groups depending on the whether they have received a vitamin A mega dose (n = 287) or not (n = 328). The prevalence of hypercarotenaemia among children fed high quantity of carotenoid foods (n = 615) was 2%, while the group fed with vitamin A mega dose and not fed vitamin A mega dose were 1.4% and 2.5% respectively. We suggest a genetic effect that is probably recessive, involved with absorption or the metabolism of carotenoids in children who develop hypercarotenaemia. In conclusion, the study showed the prevalence of hypercarotenaemia to be <2%, irrespective of the amount of carotenoids ingested and vitamin A mega dosing.

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# Introduction

Hypercarotenaemia is commonly observed among young infants and children in Sri Lanka. The main cause for development of hypercarotenaemia is excessive ingestion of carotenoid bearing food and fruits more than three times per week (Priyadarshani et al., 2009; Wageesha et al., 2011). Due to the lipophilic nature, carotenoids deposit in the adipose tissue and impart a yellow colouration especially to palms, soles and areas where subcutaneous fat is abound. There is some evidence that not all infants fed high carotenoid diets develop this clinical feature of hypercarotenaemia. Hypercarotenaemia can be verified by elevated serum carotenoids level, a normal or slightly elevated vitamin A level and normal liver and thyroid function tests. Children with liver diseases, hypothyroidism or diabetes mellitus are easily distinguished from hypercarotenaemia unassociated with excessive intake of carotenoids.

It was proven that a mutation in carotenoid 15, 15' monooxygenase (CMO1) which catalyses the first step in the conversion of dietary pro-vitamin A carotenoids to vitamin A in the small intestine cause

symptoms of hypercarotenaemia (Lindqvist *et al.*, 2007). The possibility of a role of genetically based metabolic factor was postulated (Lindqvist *et al.*, 2007). The sequence of the metabolism of carotenoids involves the conversion of these hydrocarbons to mono-hydroxy metabolites and to poly-hydroxy metabolites. The liver cytochrome P450 system increases the polarity and thus the solubility by the addition of hydroxy groups, first to the hydrocarbon and then to the metabolites in order to promote excretion from the body (Rock, 1997).

There were no reported detailed studies on the prevalence of hypercarotenaemia globally except for a study which had investigated the prevalence of hypercarotenaemia among anorexia nervosa populations (Boland *et al.*, 2001). Since hypercarotenaemia among children is presenting an increasing prevalence (unpublished data) an attempt was made to find the frequency of occurrence of hypercarotenaemia among Sri Lankan children. Since it was practically difficult to conduct such a study in the whole island the Western province was selected.

The main objectives were to study the prevalence

of hypercarotenaemia among nursery/kindergarten children, to correlate the food intake with development of hypercarotenaemia and to observe the occurrence of hypercarotenaemia among siblings of above children who had been fed similar diets. The study instrument used was a pilot tested questionnaire.

# **Materials and Methods**

A questionnaire was administrated to the parents (n = 780) of nursery/kindergarten children in three international schools and 7 private nurseries in the Western province of Sri Lanka. The age of the children ranged from 2-5 years. The questionnaire was instrumental in obtaining information on the extent of feeding carotenoid rich foods and development of hypercarotenaemia among those children and their siblings. The questionnaire mainly consisted of; child's gender and age, age that solid food first given, food combinations that were frequently given to the child, number of times carotenoid rich foods were ingested per week and approximate quantity, vitamin A mega dosed or not, if the child had experienced hypercarotenaemia, number of siblings and their dietary patterns and if they have experienced hypercarotenaemia etc.

# Results

The questionnaire analysis indicated that among the 780 cases, 615 children have been fed high carotenoid diets, similar to the quantity a hypercarotenaemic child had ingested. From the 615 cases only 287 children had received the vitamin A mega dose (Figure 1). Four of the children who had received vitamin A mega dose had experienced hypercarotenaemia and one among them had a sibling who had a similar diet but had not shown any clinical features of hypercarotenaemia.

Eight cases out of 328 children not who had not been given a vitamin A mega dose had experienced hypercarotenaemia and only 5 out of the 8 had siblings. All five siblings had ingested the same amount of carotenoid bearing food/fruits for a similar duration as their hypercarotenaemic sibling and only one sibling had experienced hypercarotenaemia. From the remaining 320 children who were not hypercarotenaemic, 176 have siblings and 138 out of them have had similar diets, and only two among the 138, had experienced hypercarotenaemia (Figures 1 and 2).

## **Discussion and Conclusion**

On questioning the mothers as to why the







Figure 2. The relationship of hypercarotenaemia with high carotenoid diet and Vitamin A supplementation in the studied sample including siblings

infants and young children were fed high carotenoid containing diets (carotenoid bearing food/fruits more than 3 times per week) (Wageesha *et al.*, 2011), they revealed that the information was gathered through radio, television, medical and para-medical professionals who basically do not believe that hypercarotenaemia is a clinical problem and consider the condition to be rare.

The majority of the test group comprised children attending International schools at Wattala, Panadura and Kalutara in the Western province of Sri Lanka. These schools were chosen as the parents were well exposed to the media publicity and also could afford to provide their children copious quantities of carotenoid rich foods.

From among all the subjects investigated; approximately 1.5% had shown hypercarotenaemia.

Out of 780, 615 children have had high carotenoid diets similar to hypercarotenaemics (Priyadarshani *et al.*, 2009; Wageesha *et al.*, 2011) and 12 have had hypercarotenaemia, thus the prevalence of hypercarotenaemia among high carotenoid eaters is 2%. When considering the high carotenoid bearing food/fruits eaters, 47% had taken the vitamin A mega dose while the other 53% had not. From the group who had taken vitamin A mega dose 1.4% experienced hypercarotenaemia, while this was 2.5% in the non vitamin A mega dosed group.

Therefore, it can be said that even if the same amount of carotenoid bearing foods as given to hypercarotenaemics are given to most children they do not develop hypercarotenaemia. This indicates no major correlation between carotenoid intake and hypercarotenaemia clearly showing that a genetic factor is at play; the subjects appear to need to be genetically pre-disposed to hypercarotenaemia. Likewise, the questionnaire results also showed that the mega dosing with vitamin A also has had no major effect on development of the condition.

The study proves that the defective gene(s) involved in the absorption or metabolism of carotenoids is not common in the group tested. We propose a genetic effect that is probably recessive, especially in the absorption or the metabolism of carotenoids in hypercarotenaemics. In conclusion, the study showed the prevalence of hypercarotenaemia to be <2%, irrespective of the amount of carotenoids ingested and vitamin A mega dosing.

#### Acknowledgements

The authors thank the parents for providing the necessary information and IPICS Sri 07, Uppsala University, Sweden for financial support.

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