

## Original Communication

# The Potential of Orange-Fleshed Sweet Potato to Prevent Vitamin A Deficiency in Africa

Fekadu Gurmu<sup>1,2</sup>, Shimelis Hussein<sup>1</sup>, and Mark Laing<sup>1</sup>

<sup>1</sup>University of KwaZulu-Natal, African Center for Crop Improvement, Pietermaritzburg, South Africa

<sup>2</sup>South Agricultural Research Institute, Hawassa Research Centre, Hawassa, Ethiopia

Received: August 4, 2014; Accepted: January 9, 2015

**Abstract:** Purpose: Vitamin A deficiency is among major health problems worldwide that leads to blindness, retarded growth and death, particularly in developing countries. In these countries, vitamin A deficiency largely affects pre-school children, pregnant and lactating mothers, and the rural poor. For instance, the predicted prevalence of vitamin A deficiency for 36 sub-Saharan African countries is 19.1 %. Methods: Different strategies, including vitamin A supplementation, food fortification and dietary diversification, have been used to combat this problem. However, these strategies are not sustainable due to their high costs. Results: Orange-fleshed sweet potato (*Ipomoea batatas* L. Lam) is a low priced crop, which is part of staple foods in most of sub-Saharan Africa that can be a year-round source of vitamin A. Most of the orange-fleshed sweet potato varieties contain 3000–16000 µg 100 g<sup>-1</sup> of β-carotene and this contributes to 250 to 1300 µg 100 g<sup>-1</sup> Retinol Activity Equivalents (RAE). Therefore, by using orange-fleshed sweet potato, it is possible to improve vitamin A status, increase the bio-availability of different micro-nutrients such as Fe, Zn, Ca and Mg, reduce vitamin A deficiency and hence reduce child mortality rates by 23 to 30 %. Conclusion: The article highlights the significance of vitamin A for human nutrition, the effect of vitamin A deficiency, the different prevention methods and the potential of orange-fleshed sweet potato as a food crop to prevent vitamin A deficiency.

**Key words:** β-carotene, intervention strategies, *Ipomoea batatas*, orange-fleshed sweet potato, vitamin A deficiency

## Introduction

Vitamin A deficiency (VAD) is a major health problem globally. An estimated 140 to 190 million children aged under 5 years are reportedly known to have low serum retinol concentrations ( $<0.7 \mu\text{mol l}^{-1}$ ). Of these, nearly 100 million live in south Asia and sub-Saharan Africa (SSA) [1–3]. According to a World Health

Organization (WHO) report, most countries in SSA are categorized as having a public health problem concerning clinical and sub-clinical VAD [3]. VAD leads to blindness, retarded growth and death in many of the developing countries. It is estimated that some 3 million children in SSA under the age of 5 suffer from partial or total blindness as a result of VAD. It especially affects pre-school children, and pregnant and lactating mothers of the rural poor [3]. For instance,

the predicted prevalence of vitamin A deficiency for 36 sub-Saharan African countries is 19.1 %. [4]. Diets with too little bioavailable vitamin A that is needed to meet human physiological needs may result in VAD [2]. The recommended daily intakes of vitamin A range from 1000 to 3000 IU for children under 5 years of age and from 3000 to 10,000 IU for adults [5].

Different strategies have been used to control VAD. The strategies include vitamin A supplementation of large doses in the form of capsules, fortification of commonly consumed food items such as oil, sugar, breakfast cereals and grain flour, and dietary diversification which includes eating food items naturally rich in pro-vitamin A such as yellow/orange root crops, leafy vegetables and yellow/orange fruits.

Sweet potato (*Ipomoea batatas* L. Lam), especially the orange-fleshed ones, can be used for human dietary diversification as it is rich in  $\beta$ -carotene (pro-vitamin A) [2]. Orange-fleshed sweet potato (OFSP) is a good, low-priced and sustainable source of vitamin A. Orange flesh is among the different sweet potato storage root flesh colors which include white, cream, yellow, or red-purple [6]. The orange flesh, with various degrees of color intensity, reflects a high  $\beta$ -carotene content. Most of the orange-fleshed sweet potato varieties contain 3000–16000  $\mu\text{g}$  100  $\text{g}^{-1}$  of  $\beta$ -carotene and this contributes to 250 to 1300  $\mu\text{g}$  100  $\text{g}^{-1}$  retinol activity equivalents (RAE) [7]. Other major dietary pro-vitamin A carotenoids are alpha-carotene and beta-cryptoxanthin [8]. A carotenoid must contain a  $\beta$ -ionone structure, a ring structure containing a single double bond and the methyl group, to act as pro-vitamin A [9].

In addition to the orange flesh color, some sweet potato varieties have red or purple flesh color which reflects high anthocyanin content [10–12]. Sweet potato is also a good source of vitamins such as vitamin C, and B2, and minerals such as Fe, Zn, Ca and Mg [13, 14]. This review article highlights the significance of vitamin A in human nutrition, the effect of VAD, the different prevention methods and the potential of

OFSP to prevent vitamin A deficiency. The information outlined in this review may help nutrition specialists, organizations working in the area of health improvement and sweet potato breeders targeting the improvement of pro-vitamin A in the crop.

## The roles of vitamin A in human nutrition

Vitamin A is an essential compound for human health. Children require vitamin A for normal mental and physical development. It is an essential micronutrient for pregnant and lactating mothers as well as for adults for healthy eyesight [15]. Generally, it plays a role in metabolic functions, eyesight, regular growth and development, and the immune system [16]. According to the United States Institute of Medicine, vitamin A is a family of compounds including retinol, retinoic acid, and retinyl esters. Retinyl esters are the storage forms of vitamin A, usually as retinyl palmitate [17]. Retinyl palmitate is the most common storage form of vitamin A, although there are other common forms such as retinyl stearate, retinyl linoleate, and retinyl myristate [6].

Vitamin A plays an important role in the early embryonic development of all mammals, and in proper functioning of the immune system, rod cells in the retina of the eye and mucous membranes throughout the body [18]. It also helps with the maintenance of cell function for growth, epithelial integrity, red blood cell production, immunity and reproduction [18]. As mammals cannot synthesize vitamin A, they obtain it from food. Therefore, consumption of a diversified diet with adequate amount of vitamin A is vital. The major sources of vitamin A are 1) animal products such as milk, butter, cheese, eggs, chicken, kidney, liver, liver pate and fish oils and 2) plant products such as dark green and yellow vegetables (kale, yellow pepper), yellow fruits such as broccoli, spinach, turnip greens, carrots, squash, sweet

Table 1: Summary of some of the roles of vitamin A for human body.

For a healthy:

vision	embryonic development and reproduction
skin	bone metabolism
tooth growth	haematopoiesis
hair	antioxidant activity
tissues	reduction of the risk of degenerative diseases such as
new cell growth	cancer, cardiovascular diseases, cataract, and macular
mucous membranes	degeneration
gene transcription	
immune function	

Source: Adapted from [21, 41]

Table II: Summary of various sources of vitamin A.

Food items	Serving Size	RAE ( $\mu\text{g } 100 \text{ g}^{-1}$ )	% RDA men	% RDA women
Sweetpotato	1/2 C*	1400	155	200
Carrot	1 medium	1015	112	145
Kale, boiled	1/2 C	240	26.6	34.2
Mango	1/2 medium	200	22.2	28.5
Turnip Greens	1/2 C	200	22.2	28.5
Spinach, raw	1 C	185	20.5	26.4
Papaya	1/2 medium	150	16.6	21.4
Red Bell Pepper	1/2 medium	140	15.5	20
Apricot	3	135	15	19.2
Cantaloupe	1/2 C	130	14.4	18.5
Milk, Fat Free	1 C	150	16.6	21.4
Romaine	1 C	70	7.7	10
Egg, large	1	95	10.5	13.5
Milk, whole	1 C	75	8.3	10.7
Tomato, raw	1 medium	35	3.8	5
Broccoli	1/2 C	35	3.8	5
Green Bell Pepper	1/2 C	15	1.6	2.1
Orange	1 medium	15	1.6	2.1

\*C = Cup. RAE = retinol activity equivalents

Source: [40]

potatoes, pumpkin, cantaloupe, apricots, ripe mangos, and papaya in the form of  $\beta$ -carotene [15, 18, 19].

Vitamin A plays a role in a variety of functions throughout the body, such as: vision, gene transcription, immune function, embryonic development and reproduction, bone metabolism, hematopoiesis, skin and cellular health and antioxidant activity [20]. It has also a positive synergistic effect with iron and zinc bio-availability and hence, through the improvement of vitamin A status in children, it is possible to increase the bio-availability of different micro-nutrients and reduce child mortality rates by 23 to 30 % [3,20–23]. A summary of the roles of vitamin A and its sources is presented in Table I and Table II, respectively.

The vitamin A status of individuals can be clinically assessed using two indicators. The first is from the quality of eyesight, where different levels of night blindness (xerophthalmia) are used to estimate the status of vitamin A and the stage of VAD. Xerophthalmia is represented by a range of visual appearances of VAD that ranges from milder stages of night blindness to potentially blinding stages of corneal xerosis, ulceration and necrosis [3]. The second indicator is biochemically-determined concentrations of retinol in plasma or serum. Serum retinol levels below  $0.70 \mu\text{mol l}^{-1}$  represent VAD. If this concentration is below  $0.35 \mu\text{mol l}^{-1}$  it represents severe VAD [3].

For countries that do not have serological data, the frequency of abnormal conjunctival impression cytology or the distribution of vitamin A concentration in breast milk are roughly used as comparators of serum retinol to estimate the status of vitamin A [21].

## Vitamin A deficiency and its health consequences

Vitamin A deficiency (VAD) is a global problem of major concern. It causes childhood blindness and mortality, which would otherwise be preventable [1–3, 18, 24–32]. Around 250 million pre-school children in the world have VAD, and about 250,000 to 500,000 go blind every year. Of those children who go blind, half die within one year of losing their eyesight [33]. VAD is a major contributor to morbidity and mortality due to infections, especially from diarrhea and measles. Since vitamin A is essential to maintaining the normal differentiation of specialized epithelial tissues throughout the body, its deficiency causes the failure of this function and the tissue becomes keratinized in many epithelia [34]. Moreover, it has an influence on gene expression, immune function, embryonic development and reproduction, and bone metabolism [17].

*Table III:* Prevalence of night blindness and number of individuals affected among preschool-age children and pregnant women in populations of countries at risk of VAD 1995–2005, globally and by WHO region.

WHO regions	Preschool-age children		Pregnant women	
	Prevalence (%)	# affected (millions)	Prevalence (%)	# affected (millions)
Africa	2.0	2.55	9.8	3.02
Americas	0.6	0.36	4.4	0.50
South-East Asia	0.5	1.01	9.9	3.84
Europe	0.8	0.24	3.5	0.22
Eastern Mediterranean	1.2	0.77	7.2	1.09
West Pacific	0.2	0.26	4.8	1.09
Global	0.9	5.17	7.8	9.75

Source: [3]

VAD primarily affects children and pregnant women of poor communities in low and middle income countries [3, 26, 28]. The consequences of VAD are severe, especially in developing countries, where the poor cannot afford to purchase vitamin A food sources such as animal products, vegetables and fruits.

The World Health Organization's VAD estimates from 1995 to 2005 indicated the prevalence of night blindness in 45 countries for preschool-age children and 66 countries for pregnant women [3]. In Africa, the highest prevalence of VAD is found in preschool children, while in south-east Asia the highest prevalence is in pregnant women. Regarding biochemical measurements of VAD, 122 countries were classified as having a moderate to severe public health problem in preschool-age children and 88 countries in pregnant women. In this case, in south-east Asia, the highest incidence is seen in both preschool-age children and pregnant women, followed by Africa and the eastern Mediterranean [3]. South-east Asia and sub-Saharan Africa have high levels of VAD, with 40 % of preschool children estimated to be deficient [3, 21]. In these regions, VAD mainly affects the poor, young children (six months to six years of age) and pregnant women.

The clinical form of vitamin A deficiency, xerophthalmia, results when the eye is adversely affected, and is expressed as night blindness or as a total, irreversible blindness [34, 35]. When these clinical signs are observed, i.e., when the rod cells are damaged to the point of causing night blindness, many other bodily functions have already been weakened, resulting in increased susceptibility to enteric and respiratory diseases, particularly among infants and young children [18]. For instance, in 1995, xerophthalmia affected an estimated 3.1 million children world-wide and sub-clinical vitamin A deficiency affected an estimated 227.6 million [35].

According to a report by the World Health Organization, night blindness caused by VAD was estimated to affect 5.2 million preschool-age children and 9.8 million pregnant women. This corresponded to 0.9 and 7.8 % of the population being at risk of VAD, respectively (Table III). Similarly, low serum retinol concentration ( $<0.70 \mu\text{mol l}^{-1}$ ) affected an estimated 190 million preschool-age children and 19.1 million pregnant women globally. This corresponded to 33.3 % of this age group and 15.3 % of pregnant women being at risk of VAD (Table IV).

VAD is primarily caused by the inadequate dietary consumption of vitamin A sources and/or suboptimal use of the nutrient in the body. A number of secondary factors that contribute to insufficient dietary intake of vitamin A have also been reported [32]. Some of the factors include insufficient production of vitamin A-rich foods, lack of income to purchase vitamin A rich foods such as meat and fish, unavailability of vitamin A-rich foods in markets, a large family size, low level of maternal education, low levels of awareness of the importance of vitamin A and illness [32].

## Strategies to combat vitamin A deficiency

In order to combat the problem of VAD, three intervention strategies are generally considered [3, 36]: 1) vitamin A supplementation of large doses in the form of capsules; 2) fortification of commonly consumed food items; and 3) dietary diversification, i.e., eating food items naturally rich in pro-vitamin A such as dark green and yellow vegetables (kale, tomatoes, yellow pepper), yellow fruits such as broccoli, spinach, turnip greens, carrots, squash, sweet potatoes, pumpkin, cantaloupe, apricots, ripe mangos, and

papaya. The third approach should be coupled with nutrition education to change the dietary habits of the target population [3]. The strategies are briefly described below.

### Vitamin A supplementation

Provision of high doses of vitamin A in capsules to the people at risk is the first and fastest option, and is one that has been practiced for a long time. This option involves the periodic delivery of high-potency supplements, containing 200,000 IU of vitamin A, to children (<5 years), half of which is given to infants of 6–11 months of age [3]. The frequency of administration is once every 4–6 months for children from 6–24 months old and once for women within 6–8 weeks after delivery during the safe infertile period [36]. It is the most widely practiced approach to controlling VAD. This approach is supported by many governments and international agencies [6]. For example, Bangladesh has a universal vitamin A supplementation program that reaches approximately half of the target population [18]. The same report indicated that providing adequate vitamin A decreases the incidence, duration and severity of childhood diseases, such as measles. It also reduces the risk in children of routine infections that would otherwise lead to severe infections and death even before the signs of night blindness develop. Therefore, supplementation programs are saving the lives and sight of thousands of children.

However, the distribution of high doses of vitamin A has its own shortcomings. For example, in Indonesia, the distribution of high doses of vitamin A has been carried out through various delivery systems for over 15 years. However, it has proven too costly to expand beyond the highest risk areas and the coverage has dropped from an initial high percentage of  $\geq 80\%$  to 40–50% [24]. This approach is therefore reported to be very expensive, and it is also difficult to reach all children that are at risk, especially in the remote rural areas [30]. Many rural poor families do not adequately and regularly access the vitamin A supplements due to poor infrastructure in the remote areas, especially in SSA, where chronic VAD is widespread. Moreover, the Alpha-Tocopherol, Beta-Carotene Cancer Prevention trial (ATBC) indicated that in smokers who also consume alcohol,  $\beta$ -carotene supplementation (20 mg of  $\beta$ -carotene) promotes pulmonary cancer and, possibly, cardiovascular complications [38].

In many countries, the emphasis has been on supplementation programs [1]. The initial hope was that

VAD could quickly be solved via the distribution of vitamin A capsules. However, the supplementation must be repeated every six months, because their effects last for only 4–6 months. In many countries with poorly developed health and road infrastructure, it can be logistically difficult to implement this strategy. In developing countries, the supplementation program has mostly been carried out through financial support from donors and it is not a long-term solution. It is difficult to sustain this strategy and consequently the outreach to poor rural populations can be limited [4, 39] and hence about 100–250 million children still remain severely affected by VAD [27]. Therefore, other options of combating the deficiency, such as food fortification and dietary diversification, are chosen.

### Food fortification

Fortification of commonly consumed food items with vitamin A is a second approach to increasing the dietary intake of vitamin A. This method has been practiced in high income countries. It was started in Central and South America three decades ago and has been their primary strategy for reducing VAD [3]. It has been done through the fortification of sugar with vitamin A. The other food items that can be fortified with vitamin A are fats, oils, margarine, cereal products, flour and salt. For example, currently in the USA, most dairy products and some cereals are fortified with vitamin A [6]. For instance, fortified ready-to-eat cereals usually contain at least 25% of the U.S. RDA for vitamin A [40]. However, in lower income countries, fewer vitamin A fortification programs exist, for several reasons: 1) the difficulty in identifying appropriate foods to fortify especially where markets for foods are not well developed; 2) the problem of infrastructure; and 3) the affordability of the fortified food items. This hinders the aim of reaching those consumers who are most at risk [1]. Therefore, food fortification can be difficult to sustain [4]. Firstly, the fortified food should be consumed by almost everyone, including the poorest individuals. Secondly, there should be a balance in the consumption of fortified foods. That means it must be consumed with a narrow range of intakes so that it prevents VAD in most people, but does not cause toxicity in people who eat more than the average amount [6]. Due to the indicated limitations of the food fortification approach, the third approach, dietary diversification, has been given special focus and attention, especially for low income countries.

## Dietary diversification

Dietary diversification is the third approach to improving the vitamin A status of a population. The purpose of this approach is to achieve and maintain an adequate intake of micronutrient-rich foods [1]. Vitamin A is found in both animal and vegetables, either as the pre-formed vitamin (retinol), or as carotenoids ( $\beta$ -carotene) which can be converted to retinol [1, 15, 19, 34]. The major sources of vitamin A as retinol are animal products such as cod liver oil, liver, milk, eggs and butter [19, 34]. The other sources are plants that provide pro-vitamin A in the form of carotenoids. There are different types of food carotenoids in plants. These are  $\alpha$ -carotene,  $\alpha$ -cryptoxanthin,  $\beta$ -carotene,  $\beta$ -cryptoxanthin, lutein, lycopene and zeaxanthin, which account for more than 90 % of the carotenoids in humans. Among these,  $\alpha$ -carotene,  $\alpha$ -cryptoxanthin and  $\beta$ -carotene are pro-vitamin A carotenoids, while most other have no vitamin A activity.  $\beta$ -carotene is the predominant form and most efficient provitamin A carotenoid [8, 16, 41, 42]. It is a forty-carbon photosynthetic pigment found in all green plants in various amounts. The  $\beta$ -carotene gives (through central cleavage) two molecules of retinol. However, due to digestive inefficiencies, it takes six micrograms of  $\beta$ -carotene in the diet to equal one retinol equivalent. Surplus retinol is stored in the liver of animals, and liver products such as cod liver oil, and is an excellent source of vitamin A [6, 18]. Through the carotenoid cleavage enzyme 15, 15'- $\beta$ -carotene oxygenase (CMO-I), the recommended conversion factor of  $\beta$ -carotene to retinol is 1:6, while for all other pro-vitamin A carotenoids, it is 1:12 [43, 44]. However, considering the wide variation in the bioavailability of carotenoids in different foods, it is an oversimplification to have a single conversion factor for  $\beta$ -carotene and a single factor for the other pro-vitamin A carotenoids [45].

The most common vegetables that are good sources of vitamin A are yellow or orange vegetables such as sweet pepper, carrot, tomato, pumpkin and OFSP, and leafy greens such as kale, spinach and cabbage. It is also found in fruits such as mango, papaya, orange, cherry, jack fruit and guava [16]. In developing countries, more than 80 % of dietary vitamin A is supplied by carotenoids present in these plants [16]. Carrot and sweet potato are carotenoid-rich root crops where carrot contains both  $\beta$ -carotene and  $\alpha$ -carotene while the yellow to orange-fleshed sweet potato contains  $\beta$ -carotene as its principal carotenoid [41]. Consumption and further promotion of locally available vitamin A-rich foods is the most efficient strategy in developing countries. This approach is technically feasible and

cost-effective. The advantage of this approach over vitamin A supplementation and the food fortification approach is that it has a long-term sustainability since fruit and vegetable parts can be harvested and shared at a local level without the intervention of national programs [6].

In addition, bio-fortification of food crops that are not normally good sources of  $\beta$ -carotene with vitamin A is also being practiced. A variety of staple foods, such as maize, cassava and rice are being bio-fortified with  $\beta$ -carotene through conventional breeding or genetic engineering [6]. Golden rice is one of the best examples of bio-fortified food crops in which vitamin A content is enhanced through a transgenic approach [18, 46]. However, transgenic crops face limited consumer acceptance and strict regulatory measures. On the contrary, OFSP is developed by plant breeders through conventional plant breeding so that it adapts to local environments and preferences, including sensory traits [47–48]. Moreover, current varieties of OFSP contain more  $\beta$ -carotene than Golden Rice [49]. While the cultivation and consumption of sweet potato is traditionally part of the culture of African communities, and hence can be a year round source of vitamin A, rice may be more feasible in Asia, where rice production and consumption is a tradition [15]. Hence, OFSP is an efficient option that is much cheaper and quicker to introduce and has a greater chance of consumer acceptance.

## Orange-fleshed sweet potato and its potential to prevent vitamin A deficiency

The advantages of OFSP as a source of vitamin A over other strategies include: 1) it is a staple food that can supply significant amounts of vitamin A and carbohydrate simultaneously; and 2) it is an ideal source of vitamin A instead of the expensive animal products and vegetables that are beyond the reach of most people in the developing world [1, 18, 19]. Therefore, the promotion of OFSP is a better strategy to combat VAD [27]. In general, OFSP is the cheapest and most accessible potential source of vitamin A compared to other food items that are currently available [1, 2, 15, 19, 25, 50, 51]. OFSP is a major source of  $\beta$ -carotene for people in some parts of Africa and south-east Asia [52] and the poorest households in these continents normally obtain over 60 % of their energy needs from food staples, so this strategy is particularly suited to

poor rural households that cannot access fortified food products but can grow OFSP [50].

The intensity of the orange color of sweet potato roots roughly reflects the amount of  $\beta$ -carotene present in the sweet potato. Research has confirmed the existence of a strong positive correlation between flesh color and  $\beta$ -carotene content in sweet potato [1, 53–60]. A study of the relationship between  $\beta$ -carotene content and Hunter color values in sweet potato cultivars indicated a high correlation ( $r=0.89$ ) between the two traits [61]. Roots with orange, deep orange and intermediate orange as the primary flesh color, and orange and intermediate orange as the secondary colors, have a significant amount of  $\beta$ -carotene [57]. Similarly, roots with intermediate orange as the primary flesh color, and pale orange as the secondary colors, have a substantial amount of  $\beta$ -carotene. This is true only if the secondary color represents a small proportion of the primary color. Roots with pale orange as the primary flesh color, and orange and intermediate orange as the secondary colors that represent a large proportion of the primary color, also contain a significant amount of  $\beta$ -carotene. Roots with yellow orange, pale orange, yellow, intermediate yellow, pale yellow and cream do not have high levels of  $\beta$ -carotene [57]. These flesh color differences are attributed to varietal differences, growing, harvesting, and storage conditions [6]. Therefore, examining the intensity of root flesh color of sweet potato is crucial while breeding for the improvement of the crop for  $\beta$ -carotene (pro-vitamin A).

## Daily requirement of $\beta$ -carotene and OFSP for the human body

Vitamin A values of foods are measured in terms of retinol equivalents (RE) as a means of making standard comparisons among foods. Age and sex determine the human dietary vitamin A requirements [61]. Some of the recommended daily allowance (RDAs) by age and sex are 1–3 years, 400  $\mu\text{g}$  RE; 4–6 years, 500  $\mu\text{g}$  RE; 7–10 years, 700  $\mu\text{g}$  RE; non-pregnant female over 10 years, 800  $\mu\text{g}$  RE; and males over 10 years, 1000  $\mu\text{g}$  RE. Therefore, small quantities of OFSPs, which may contain from 300  $\mu\text{g}$  RE to over 3000  $\mu\text{g}$  RE per 100 g fresh weight, can easily provide such RDAs and also serve as a rich source of other vitamins and nutrients [14]. For example, the addition of 100 g OFSP in daily diet can prevent VAD in children and significantly reduce the death of mothers [15]. This is because 6  $\mu\text{g}$   $\beta$ -carotene is equivalent to 1  $\mu\text{g}$  of retinol [14, 18] and

the RDA of vitamin A in adults on average basis is 750  $\mu\text{g}$  retinol day<sup>-1</sup>. A 100 g OFSP can provide more than 6500  $\mu\text{g}$   $\beta$ -carotene [62–64], which is equivalent to more than 1000  $\mu\text{g}$  retinol. Therefore, 100 g OFSP per day exceeds the RDA to prevent VAD. As a result, depending upon the color intensity of the OFSP variety used, and taking into account losses of  $\beta$ -carotene during cooking, which accounts for approximately 20 % loss through boiling [42], 1/4 to 1 cup of boiled and mashed sweet potato meets the RDA of vitamin A for a young child [48]. OFSP is also a good source of energy, which is equivalent to 293–460 kJ/100 g [14, 63].

However, it is not only the amount of OFSP or  $\beta$ -carotene content consumed that is important; the bio-availability of the carotenoids also matters. Carotenoid bio-availability means the fraction of carotenoids becoming accessible for uptake by the intestinal mucosa [6]. Bio-availability of  $\beta$ -carotene in OFSP is higher than that in most dark-green leafy vegetables. This is because  $\beta$ -carotene in OFSP is more readily released during cooking, thereby enhancing bioavailability. A report by Tanumihardjo et al. (2010) also indicated that staple foods biofortified with provitamin A carotenoids such as sweet potato, cassava, maize, and rice, have shown to have more efficient bioconversion to retinol in the range of 3–6  $\beta$ -carotene for 1  $\mu\text{g}$  retinol compared to that generally observed for vegetables which ranges between 10–80  $\beta$ -carotene for 1  $\mu\text{g}$  retinol [65]. In mature plant tissues,  $\beta$ -carotene bioavailability is affected by chromoplast structure, where globular chromoplasts provide the best bioavailability and crystalline types provide the poorest bioavailability of  $\beta$ -carotene [66]. Hence, the better solubility may be related to less crystallinity of  $\beta$ -carotene in non-leafy vegetables. Further, the addition of 3 to 5 g fat per meal is required to ensure maximum carotenoid absorption [2]. The presence of fat in the food preparation increases bio-availability of  $\beta$ -carotene of OFSP.

## Factors affecting production and promotion of OFSP as strategic crop to prevent VAD

Although OFSP is a very valuable crop with dual purposes both in food security and health, its production is constrained by many factors. Some of the production constraints are diseases and pests, prolonged drought, poor soils, land shortage, labor shortage, lack of improved varieties with high dry

Table IV: Prevalence of serum retinol <0.7µmol-1 and number of individuals affected among preschool-age children and pregnant women in populations of countries at risk of VAD 1995–2005, globally and by WHO region.

WHO regions	Preschool-age children		Pregnant women	
	Prevalence (%)	# affected (millions)	Prevalence (%)	# affected (millions)
Africa	44.4	56.4	13.5	4.18
Americas	15.6	8.68	2.0	0.23
South-East Asia	49.9	91.5	17.3	6.69
Europe	19.7	5.81	11.6	0.72
Eastern Mediterranean	20.4	13.2	16.1	2.42
West Pacific	12.9	14.3	21.5	4.90
Global	33.3	190	15.3	19.1

Source: [3]

matter content, lack of planting materials and lack of production inputs [67]. The major postharvest and marketing constraints are storage pests, lack of processing tools, low price for the products and the lack of organized markets [67]. Therefore, new sweetpotato varieties should meet farmers' criteria in terms of quality, disease resistance, taste, flavor, early maturity, and texture (which depends on root dry matter content) [68]. Dry matter content (DMC) is a very important trait as sweetpotato varieties with high DMC are preferred by both farmers and industries [27, 69]. Many of the sweetpotato consumers in Africa prefer sweetpotato varieties with high DMC [70]. DMC has an influence on eating quality, shelf-life and processing quality, and therefore influences the acceptability of OFSP. This is because the OFSP with high  $\beta$ -carotene tends to have lower DMC. In most of the SSA, white-fleshed varieties are predominantly grown and are preferred by farmers due to their high DMC [50]. However, the white-fleshed sweet potato varieties have little or no  $\beta$ -carotene. Therefore, farmers-preferred OFSP with improved DMC should be developed in order to increase their adoption and large-scale production to combat VAD and achieve food security.

## Breeding for high $\beta$ -carotene: progresses and success stories

OFSP varieties that contain high levels of  $\beta$ -carotene and high root yield have been identified through conventional plant breeding, especially in developing countries [71]. For example, about 40 varieties of sweet potato high in both pro-vitamin-A and dry matter have been introduced into sub-Saharan Africa

[49]. These varieties have improved yield, disease and pest resistance, drought tolerance and quality characteristics [48, 49], and are therefore receiving good consumer acceptance. Specifically, the selection of OFSP with high  $\beta$ -carotene and yield has been conducted in Tanzania to develop new vitamin A-enriched sweet potato varieties [72]. Fifteen drought-tolerant OFSP varieties were released in Mozambique and contributed to reducing VAD through increased vitamin A intake and serum retinol concentrations [73, 74]. Breeding and selection of OFSP for high  $\beta$ -carotene and resistance to sweet potato virus disease (SPVD) was conducted in Uganda in 2002 and 2003 and promising varieties have been selected for further evaluation and released to farmers [19]. The varieties are included in Table V.

There has been good potential for the general acceptance of OFSP varieties in Kenya and Uganda both for consumption and marketing. Varieties such as *Japones tresmesino* and SPK 004 have been disseminated to farmers. *Japones tresmesino* was not preferred by adult consumers due to its low dry matter content (~22 %), but was preferred by children. SPK 004, which has a higher dry matter content (~28 %), was selected by adults due to its drier texture [75]. OFSP varieties such as Zapallo, SPK 004 and Salybolo were found to be high-yielding varieties with good root characteristics in Kenya [15, 76]. In Uganda, varieties Ejumula, Kala and SPK 004 have been identified for desirable consumer acceptance and for good adaptation in different agro-ecologies. Varieties Zapallo, *Japones tresmesino* and Tainung 64 have received better acceptance in Tanzania [15, 76]. In South Africa, varieties Resisto, W-19 and Excel have been selected for high dry matter, high  $\beta$ -carotene content and processing quality [15, 76]. On the other hand, more than twenty promising

Table V: List of some of the OFSP varieties released and produced in Africa and their  $\beta$ -carotene contents.

No.	Variety	Flesh color	$\beta$ -carotene content ( $\mu\text{g}/100\text{g}$ )	Country of release
1	Carrot C	Deep orange	12390–14370	Tanzania
2	Ejumula	Deep orange	7760–14370	Uganda
3	Jewel	Orange	11030	Mozambique
4	Kakamega	Intermediate orange	376–3760	Uganda, Kenya, Rwanda
5	K566632	Deep orange	700–800	Kenya
6	Mayai	Intermediate orange	11030	Grown by farmers in Zanzibar Island and coastal Tanzania
7	CN-1424-9	Orange	11030	Mozambique
8	Resisto	Deep orange	24900	Mozambique, South Africa, Madagascar
9	NASPOT 9 O	Deep orange	11030	Uganda
10	NASPOT 10 O	Deep orange	11030	Uganda
11	Tainung 64	Orange	3760–7230	Mozambique
12	W-151	Deep orange	10500–14370	Kenya
13	Zambezi	Deep orange	10900	Zambia
14	102027.02	Dark orange	11030	Kenya
15	102022.7	Orange	3760–7230	Kenya
16	10300.152	Orange	4920	Kenya
17	Caromex	Dark orange	11030	Mozambique
18	CN1448-49	Intermediate orange	4470–4920	Mozambique
19	Gaba Gaba	Deep orange	11030	Mozambique
20	Kandee	Orange	11030	Mozambique
21	Japones Tresmesino Select	Light orange	3760–7230	Mozambique
22	Lo-323	Intermediate orange	5490	Mozambique
23	Persistente	Dark orange	11030	Mozambique
24	Cordner	Deep orange	3760–7230	Mozambique
25	199062.1	Intermediate orange	3760–7230	Mozambique
26	CRI-Apomuden	Orange with slight yellow stripes	2000–4000	Ghana
27	Impilo	Pale orange	5091	South Africa
28	Khano	Deep orange	14036	South Africa
29	W-119	Orange	10464	South Africa

Source: Adapted from [7]

OFSP clones have been distributed to Madagascar, Mozambique, Ethiopia, Uganda, Tanzania, Zimbabwe, Egypt, Zanzibar, Malawi, and D.R. Congo [15]. The list of OFSP varieties released in Africa and their  $\beta$ -carotene contents is presented in Table V.

In addition to the OFSPs, current research is undergoing to enhance the  $\beta$ -carotene content of other staple foods, such as in maize and cassava. It is reported that maize varieties that are bred for

high  $\beta$ -carotene content are good sources of vitamin A [77]. In Nigeria, two new maize varieties with high levels of  $\beta$ -carotene have been released [78].  $\beta$ -carotene-rich bio-fortified yellow maize can be a good source of vitamin A for Africans who consume maize as their staple food [79]. In Mongolia, bio-fortified maize reportedly maintained the vitamin A need of the people and was found to be as effective as vitamin A supplementation [80].

$\beta$ -carotene enriched elite cassava varieties are being developed through conventional plant breeding and selection procedures by International Institute of Tropical Agriculture (IITA), International Center for Tropical Agriculture (CIAT) and National Root Crops Research Institute (NRCRI). The objective is to develop cassava gene pool with high  $\beta$ -carotene and to develop varieties that will be released to farmers [81, 82]. Bio-fortified cassava is also a viable intervention food crop to prevent vitamin A deficiency [83]. In general,  $\beta$ -carotene obtained from plant sources is a better choice since animal sources are expensive and also high in saturated fat and cholesterol. Therefore, OFSP is a potential crop to provide the RDA of vitamin A for the poor people in Africa and to prevent VAD.

## Conclusion

Vitamin A deficiency (VAD) is among the main health problems that are of major concern and can cause preventable childhood blindness and mortality. Three intervention strategies are generally considered: encapsulated vitamin A supplementation, fortification of commonly consumed food items and dietary diversification. Among the food crops that are used for dietary diversification, OFSP is a cheap and practical option that can be used as a source of both vitamin A and energy. It is an easily accessible source of vitamin A for poor people in developing countries. Most varieties of OFSP released so far in Africa can provide more than 100 % of the RDA of vitamin A. The consumption of these varieties is highest immediately after harvest and then gradually decreases. However, most African farmers harvest sweet potatoes normally in a piecemeal manner, so the harvesting and consumption can be extended for longer periods of time. Promotion of OFSP based on farmers' preference is therefore crucial to combat the problem of VAD in a cost effective and sustainable way.

## Acknowledgements

The authors acknowledge the Alliance for a Green Revolution in Africa (AGRA), International Foundation for Science (IFS) and Syngenta Foundation for financial support of the study. Gratitude also goes to the Southern Agricultural Research Institute (SARI) in Ethiopia for granting study leave for the first author.

## Conflicts of interest

On behalf of all authors, the corresponding author declares that there are no conflicts of interests.

## References

1. Low, J., Walker, T., Hijmans, R. (2001) The potential impact of orange-fleshed sweet potatoes on vitamin A intake in Sub-Saharan Africa. In: A regional workshop on food-based approaches to human nutritional deficiencies. The VITAA Project, vitamin A and orange-fleshed sweet potatoes in Sub-Saharan Africa, pp. 1–16, Nairobi, Kenya.
2. van Jaarsveld, P., Faber, M., Tanumihardjo, S.A., Nestel, P., Lombard, C.J., Benadé, A.J.S. (2005) Beta carotene-rich orange-fleshed sweet potato improves the vitamin A status of primary school children assessed with the modified-relative-dose-response test. *Am. J. Clin. Nutr.* 81, 1080–1087.
3. WHO. (2009) Global prevalence of vitamin A deficiency in population at risk from 1995–2005. WHO Global Database on Vitamin A Deficiency, pp. 1–57, World Health Organization, Geneva, Switzerland.
4. Victor, M.A. and Baker, S.K. (2005) Vitamin A deficiency and child survival in sub-Saharan Africa: A reappraisal of challenges and opportunities. *Food and Nutrition Bulletin* 26 (4), 348–355.
5. ConsumerLab.com. LLC (2014) Recommended Daily Intakes and Upper Limits for Nutrients. <https://www.consumerlab.com/rdas/>. Accessed on 5/11/2014.
6. Burri, B.J. (2011) Evaluating sweetpotato as an intervention food to prevent vitamin A deficiency. *Comprehensive review in Food Science and Food Safety* 10, 118–130.
7. Kapinga, R., Tumwegamire, S., Ndunguru, J., Andrade, M.I., Agili, S., Mwanga, R.O., Laurie, S., Dapaah, H. (2010) Catalogue of orange-fleshed sweetpotato varieties for Sub-Saharan Africa, pp. 40, International Potato Center (CIP), Lima, Peru.
8. Ohnishi, S.T and Kojinra, R. (1997) Antioxidant activities of aged garlic extract and cancer chemotherapy. In: P.A. Lachance. *Nutraceuticals: Designer Foods III. Garlic, Soy and Licorice*. Food and Nutrition Press, Inc. Trumbull, Connecticut, USA.
9. Kaneko, J.J., Harvey, J.W., Bruss, M.L. (2008) *Clinical Biochemistry of Domestic Animals*. Elsevier Inc., Burlington, USA.

10. Huaman Z. (1999) Sweetpotato (*Ipomoea batatas*) Germplasm Management. Training manual, pp. 218, International Potato Center (CIP), Lima, Peru,.
11. Yoshinaga, M., Yamakawa, O., Nakatani, M. (1999) Genotypic diversity of anthocyanin content and composition in purple-fleshed sweetpotato [*Ipomoea batatas* (L.) Lam.]. J. Plant. Br. Crop Sci. 49, 43–47.
12. Vimala, B. and Hariprakash, B. (2011) Variability of morphological characters and dry matter content in the hybrid progenies of sweetpotato [*Ipomoea batatas* (L.) Lam.]. Geneconserve 10, 65–86.
13. Grüneberg, W., Mwanga, R., Andrade, M., and Daapah, H. (2009) Sweetpotato breeding. In: Andrade, M., Barker, I., Cole, D., Daapah, H., Elliott, H., Fuentes, S., Grüneberg, W., Kapinga, R., Kroschel, J., Labarta, R., Lemaga, B., Loechl, C., Low J., Ortiz, O., Oswald, A., Thiele, G., Lynam, J. and Mwanga, R. eds. Unleashing the potential of sweetpotato in Sub-Saharan Africa: Current challenges and way forward, pp. 197, International Potato Centre (CIP), Lima, Peru.
14. Woolfe, J.A. (1992) Sweet potato: an untapped food resource. Cambridge University Press, Cambridge, United Kingdom.
15. Kapinga, R., Anderson, P., Crissman, C., Zhang, D., Lemaga, B., Opio, F. (2005) Vitamin A partnership for Africa: A food based approach to combat vitamin A deficiency in sub-Saharan Africa through increased utilization of orange fleshed sweetpotato. Hortic. Sci. Focus. 45, 12–14.
16. Bhaskarachary, K., Rao, D.S.S., Deosthale, Y.G., Reddy, V. (1995) Carotene content of some common and less familiar foods of plant origin. Food. Chem. 54, 189–193.
17. United States Institute of Medicine. (2000) Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Natl. Academy Press., Washington, D.C, USA;
18. Toenniessen, G.H., (2000) Vitamin A Deficiency and Golden Rice: The Role of the Rockefeller Foundation, pp. 1–5, The Rockefeller Foundation, New York, USA,
19. Mwanga, R., Odongo, B., Niringiye, C., Zhang, D., Yench, G.C., Kapinga, R. (2003) Orange-fleshed sweet potato breeding activities in Uganda. In: The 6th Conference of the African Crop Science Society (ACSS) Conference Proceeding, African Crop Science Society, pp. 103–107, Kampala, Uganda,
20. Graham, R.D. and Rosser, J.M. (2000) Carotenoids in staple foods: their potential to improve human nutrition. Food and Nutrition Bulletin 21,404–409.
21. West, K.P.J. (2002) Extent of vitamin A deficiency among preschool children and women of reproductive age. J. Nutr. 132, 2857–2866.
22. Beaton, G., Martorell, R., Aronson, K.J., Edmonston, B., McCabe, G., Ross, A.C. (1993) Effectiveness of vitamin A Supplementation in the control of young child morbidity and mortality in developing countries. Nutrition Policy Paper No. 13, pp. 166, World Health Organization, Geneva, Switzerland.
23. West, K.P.J. (2003) Vitamin A deficiency disorders in children and women. Food and Nutrition Bulletin 24, 78–90.
24. Muhilal, M., Murdiana, A., Azis, I., Saidin, S., Jahari, A.B., Karyadi, D. (1988) Vitamin A-fortified monosodium glutamate and vitamin A status: a controlled field trial. Am. J. Clin. Nutr. 48, 1265–70.
25. Hagenimana, V., Kosambo, L.M., Carey, E.E. (1997) Potential of sweetpotato in reducing vitamin A deficiency in Africa, pp. 287–294, CIP Program Report,
26. Haidar, J., Tsegaye, D., Mariam, D.H., Tibebe, H.N., Muroki, N.M. (2003) Vitamin A supplementation on child morbidity. E. Afr. Med. J. 80, 17–21.
27. Tumwegamire, S., Kapinga, R., Zhang, D., Crissman, C., Agili, S. (2004) Opportunities for promoting orange-fleshed sweet potato as a mechanism for combat vitamin A deficiency in sub-Saharan Africa. Afr. Crop. Sci. J. 12, 241–252.
28. Aguayo, V.M. and Baker, S.K. (2005) Vitamin A deficiency and child survival in sub-Saharan Africa: A reappraisal of challenges and opportunities. Food and Nutrition Bulletin 26, 348–355.
29. Maziya-Dixon, B., Akinyele, I.O., Sanusi, R.A., Oguntona, T.E., Nokoe, S.K., Harris, E.W. (2006) Vitamin A deficiency is prevalent in children less than 5 years of age in Nigeria. J. Nutr. 36, 2255–2261.
30. Anderson, P., Kapinga, R., Zhang, D., Hermann, M. (2007) Vitamin A for Africa (VITAA): An entry point for promoting orange-fleshed sweet potato to combat vitamin A deficiency in sub Saharan Africa. In: Proceedings of the 13th International Society for Tropical Root Crops (ISTRC) Symposium, pp. 711–720, Arusha, Tanzania.
31. Tofu, A., Anshebo, T., Tsegaye, E., Tadesse, T. (2007) Summary of progress on orange-fleshed sweet potato research and development in Ethiopia. In: Proceedings of the 13th International Society for Tropical Root Crops (ISTRC) Symposium, pp. 728–731, Arusha, Tanzania.
32. Demissie, T., Ali, A., Mekonnen, Y., Haider, J., Umata, M. (2009) Demographic and health-related

- risk factors of subclinical vitamin A deficiency in Ethiopia. *J. Health. Popul. Nutr.* 27, 666–673.
33. Rotondi, M.A. and Khobzi, N. (2010) Vitamin A supplementation and neonatal mortality in the developing world: a meta-regression of cluster-randomized trials. *Bulletin of the World Health Organization* 88, 697–702.
  34. Hicks, R.M. (1983) The scientific basis for regarding vitamin A and its analogues as anti-carcinogenic agents. In: *Symposium of Proceedings of the Nutritional Society*, pp. 83–93, London, United Kingdom.
  35. International Vitamin A Consultative Group. (1995) Two decades of progress: Linking knowledge to action. In: *Report of the XVI International Vitamin A Consultative Group Meeting*, Washington, D.C, USA.
  36. Ortiz, D., Sánchez, T., Morante, N., Ceballos, H., Pachón, H., Duque, M.C., Chávez, A.L., Escobar, A.F. (2011) Sampling strategies for proper quantification of carotenoid content in cassava breeding. *J. Plant. Br. Crop. Sci.* 3, 14–23.
  37. Beaton, G.H., Martorell, R., Aronson, K.A., Edmonston, B., McCabe, G.A., Ross C. and Harvey B. (1994) Vitamin A supplementation and child morbidity and mortality in developing countries. *Food and Nutrition Bulletin* 15(4), 282–289.
  38. The American Journal of Clinical Nutrition. (1999) Alcohol, vitamin A, and  $\beta$ -carotene: adverse interactions, including hepatotoxicity and carcinogenicity. *Am. J. Clin. Nutr.* 69 (6), 1071–1085.
  39. Semba, R., de Pee, S., Sun, K., Akhter, N., Bloem, M.W., Raju, V.K. (2010) Coverage of vitamin A capsule program in Bangladesh and risk factors associated with non-receipt of vitamin A. *J. Health. Pop. Nutr.* 28, 143–148.
  40. Keith, L.S. (2007) Bioavailability of vitamin A from fortified foods. Ohio State University Extension Fact Sheet. Department of Human Nutrition and OSU Extension, Ohio State University, Neil Avenue, Columbus.
  41. Rodriguez-Amaya, D.B. (2001) *A Guide to Carotenoid Analysis in Foods*. ILSI Human Nutrition Institute, Washington, D. C, USA.
  42. van Jaarsveld, P.J., De Wet, M., Harmse, E., Nestel, P., Rodriguez-Amaya, D.B. (2006) Retention of  $\beta$ -carotene in boiled, mashed orange-fleshed sweet potato. *Journal of Food Composition and Analysis* 19 (4), 321–329.
  43. Furr, H.C. (1989) Vitamin A concentrations in liver determined by isotope dilution assay with tetradeuterated vitamin A and by biopsy in generally healthy adult Humans. *Am. J. Clin. Nutr.* 49, 713–716.
  44. Ford, N.A. (2010) Carotenoid Metabolism in Mice and Prostate Cancer Risk. PhD Thesis, Graduate College of the University of Illinois at Urbana-Champaign, Urbana, Illinois.
  45. Rodriguez-Amaya, D.B. (1989). Critical review of pro-vitamin A determination in plant foods. *J. Micronutr. Anal.* 5, 191–225.
  46. Penelope, N., Bouis, H.E., Meenakshi, J.V., Pfeiffer, W. (2006) Biofortification of staple food crops. *J. Nutr.* 136, 1064–1067.
  47. Pray, C., Paarlberg, R., Unnevehr, L. (2007) Patterns of political response to biofortified varieties of crops produced with different breeding techniques and agronomic traits. *The J. Agrobiotec. Econ.* 10, 135–143.
  48. Prakash, C.S. (1994) Sweetpotato biotechnology: progress and potential. *Biotechnol. Dev. Monitor.* 18, 1819–1822.
  49. Ye, X., Al-Babili, K.A., Zhang, J., Lucca, P., Beyer, P., Potrykus, I. (2000) Engineering the provitamin A ( $\beta$ -carotene) biosynthetic pathway into (carotenoid-free) rice endosperm. *Science* 287, 303–305.
  50. Low, J., Kapinga, R., Cole, D., Loechl, C., Lynam, J., Andrade, M. (2009) Challenge theme paper 3: Nutritional impact with orange-fleshed sweet potato (OFSP). *Unleashing the Potential of Sweet potato in Sub-Saharan Africa*, pp. 73–105, CIP, Nairobi, Kenya.
  51. Kaguongo, W., Ortmann, G.F., Wale, E., Darroch, M.A.G., Low, J. (2010) Factors influencing adoption and intensity of adoption of orange flesh sweetpotato varieties: evidence from an extension intervention in Nyanza and Western province, Kenya. In: *The 48th Agricultural Economists Association of South Africa (AEASA) Conference*, pp.1–24, Cape Town, South Africa.
  52. Liao, Z., Chen, M., Yang, Y., Yang, C., Fu, Y., Zhang, Q., Wang, Q. (2008) A new isopentenyl diphosphate isomerase gene from sweet potato: cloning, characterization and color complementation. *Biologia* 63, 221–226.
  53. Takahata, Y., Noda, T., Nagata, T. (1993) HPLC determination of  $\beta$ -carotene of sweet potato cultivars and its relationship with color values. *Japanese J. Breed.* 43, 421–427.
  54. Cervantes-Flores, J.C. (2006) Development of a Genetic Linkage Map and QTL Analysis in Sweet potato. PhD thesis, North Carolina State University, Raleigh, USA.

55. Mcharo, M. and LaBonte, D. (2007) Genotypic variation among sweet potato clones for  $\beta$ -carotene and sugar content. In: Proceedings of the 13th International Society for Tropical Root Crops (ISTRC) Symposium, pp. 746–754, Arusha, Tanzania.
56. Gasura, E., Mashingaidze, A.B., Mukasa, S.B. (2008) Genetic variability for tuber yield, quality, and virus disease complex traits in Uganda sweet potato germplasm. *Afr. Crop. Sci. J.* 16, 147–160.
57. Burgos, B., Carpio, R., Sanchez, C., Paola, S., Eduardo, P., Espinoza, J., Grüneberg, W. (2009) A colour chart to screen for high  $\beta$ -carotene in OFSP breeding. In: The 15th Triennial Symposium of the International Society for Tropical Root Crops (ISTRC), pp. 47–52, Lima, Peru.
58. Vimalaa, B., Sreekanthb, A., Binua, H., Wolfgang, G. (2009) Variability in 42 orange-fleshed sweet potato hybrids for tuber yield, carotene and dry matter content. *Geneconserve* 40, 190–200.
59. Cervantes-Flores, J., Sosinski, B., Pecota, K.V., Mwanga, R.O.M., Catignani, G.L., Truong, V.D., Watkins, R.H., Ulmer, M.R., Yencho, G.C. (2010) Identification of quantitative trait loci for dry-matter, starch, and  $\beta$ -carotene content in sweet potato. *Mol. Breeding*. 28, 201–216.
60. Vimala, B. and Hariprakash, B. (2011) Variability of morphological characters and dry matter content in the hybrid progenies of sweet potato [*Ipomoea batatas* (L.) Lam.]. *Geneconserve* 10, 65–86.
61. Williams, S.R. and Worthington-Roberts, B.S. (1988) *Nutrition Throughout the Life Cycle*. Times Mirror/Mosby College Publishing, St. Louis, MO, USA.
62. Ezell, B.D. and Wilcox, M.S. (1958) Sweet potato pigments: variation in carotene content of sweet potatoes. *Agri. Food. Chem.* 6, 61–65.
63. Hagenimana, V., Low, J., Anyango, M., Kurz, K., Gichuki, S.T., Kabira, J. (2001) Enhancing vitamin A intake in young children in Western Kenya: orange-fleshed sweet potatoes and women farmers can serve as key entry points. *Food and Nutrition Bulletin* 22, 370–387.
64. Christina, S.L. (2007) Nutrient and sensory quality of orange-fleshed sweet potato. MSc thesis, University of Pretoria, Pretoria, South Africa.
65. Tanumihardjo, S.A., Palacios, N., Pixley, K.V. (2010) Provitamin A carotenoid bioavailability: what really matters? *Int. J. Vitam. Nutr. Res.* 80(4–5), 336–50.
66. Fleshman, M.K., Lester, G.E., Riedl, K.M., Kopec, R.E., Narayanasamy, S., Curley, R.W.Jr., Schwartz, S.J., Harrison, E.H. (2011) Carotene and Novel Apocarotenoid Concentrations in Orange-Fleshed *Cucumis melo* Melons: Determinations of  $\beta$ -Carotene Bioaccessibility and Bioavailability. *J. Agric. Food Chem.* 59, 4448–4454.
67. Mudioppe, J., Kindness, H., Hagenimana, V. (2000) Socio-economic constraints and the production, processing, and marketing of sweetpotato in Kumi District, Uganda. In: The Fifth Triennial Congress of the African Potato Association, Kampala, Uganda.
68. Ssebuliba, J., Muyonga, J.H., Ekere, W. (2006) Performance and acceptability of orange-fleshed sweet potato cultivars in eastern Uganda. *Afr. Crop. Sci. J.* 14, 231–240.
69. Kwach, J., Odhiambo, G.O., Dida, M.M., Gichuki, S.T. (2010) Participatory consumer evaluation of twelve sweet potato varieties in Kenya. *Afr. J. Biotechnol.* 9, 1600–1609.
70. Kapinga, R.E. and Carey, E.E. (2003) Present status of sweetpotato breeding for eastern and southern Africa. In: Rees, D., Oirschot, Q. and Kapinga, R. (Eds.) *Sweet potato Post-harvest Assessment: Experiences from East Africa*, pp. 3–8, Chatham: United Kingdom.
71. Nestel, P., Bouis, H.E., Meenakshi, J.V., Pfeiffer, W. (2006) Biofortification of staple food crops. *J. Nutr.* 136, 1064–1067.
72. Masumba, E., Kapinga, R., Tollan, S.M., Mary, O., Kitundu, C.D. (2007) Adaptability and acceptability of new orange-fleshed sweet potato varieties in selected areas of Eastern and Central zones of Tanzania. In: Proceedings of the 13th International Society for Tropical Root Crops (ISTRC) Symposium, pp. 737–745, Arusha, Tanzania.
73. Low, J., Arimond, M., Osman, N., Cunguara, B., Zano, F., Tschirley, D. (2007) A food-based approach introducing orange-fleshed sweet potatoes increased vitamin A intake and serum retinol concentrations in young children in rural Mozambique. *J. Nutr.* 137, 1320–1327.
74. International Potato Center. (2011) Sweet potato breeding in Africa for Africa. CIP Newsletter, Nairobi, Kenya.
75. Carey, E.E., Hagenimana, V., K'osambo, L., Oyunga, M.A., Smit, N., p'Obwoya, C.O., Turyamureeba, G., Ndolo, P.J., Low, J. (1999) Using orange-fleshed sweet potato varieties to combat vitamin A deficiency and enhance market opportunities for smallholder farmers in sub-Saharan Africa. In: Akoroda, MO and Teri JM, editors. *Food Security and Crop Diversification in SADC Countries: The Role of Cassava and Sweet potato*. Proceedings of the scientific workshop of the Southern African Root Crops Research Network (SARRNET), pp. 157–168, Lusaka, Zambia.

76. Kapinga, R., Zhang, D., Lemaga, B., Andrade, M., Mwanga, R., Laurie, S., Ndolo, P., Kanju, E. (2007) Sweet potato crop improvement in sub Saharan Africa and future challenges. In: Proceedings of the 13th International Society for Tropical Root Crops (ISTRC) Symposium, pp. 119, Arusha, Tanzania.
77. White, W., Curtis, C., McClafferty, B., Ewing-Blount, S. (2013) Iowa State study finds corn bred to contain beta-carotene is a good source of vitamin A. Iowa State University of Science and Technology, Iowa, USA.
78. International Institute of Tropical Agriculture. (2012) Vitamin A-enriched maize released in Nigeria, seen to benefit health of millions. CGIAR Consortium, pp. 1–16, Ibadan, Nigeria.
79. Muzhingi, T., Tendekayi, H.G., Andrew, H.S., Michael, A.G., Robert, M.R., Guangwen, T. (2011) Yellow maize with high  $\beta$ -carotene is an effective source of vitamin A in healthy Zimbabwean men. *Am. J. Clin. Nutr.* 94, 510–519.
80. Howe, A.J. and Tanumihardjo, S.A. (2006) Carotenoid-biofortified maize maintains adequate vitamin A status in Mongolian gerbils. *J. Nutr.* 136, 2562–2567.
81. Bafana, B. (2011) Not everyone in Nigeria pleased with new vitamin A-fortified cassava. Inter Press Service, Bulawayo, Zimbabwe.
82. Njoku, D.N., Vernon, G., Egesi, C.N., Asante, I., Offei, S.K., Okogbenin, E., Kulakow, P., Eke-okoro, O.N., Ceballos, H. (2011) Breeding for enhanced  $\beta$ -carotene content in cassava: constraints and accomplishments. *J. Crop. Imp.* 25, 560–571.
83. La Frano, M., Woodhouse, L.R., Burnett, D.J., Burri, B.J. (2013) Biofortified cassava increases  $\beta$ -carotene and vitamin A concentrations in the TAG-rich plasma layer of American women. *J. Nutr.* 21, 1–11.

---

Fekadu Gurmu

University of KwaZulu-Natal  
 African Center for Crop Improvement  
 Private Bag X01  
 Scottsville 3209, Pietermaritzburg  
 South Africa  
 Tel.: +251 911743625  
 Fax: +251 462204521  
 fekadugurmu@yahoo.com