Selenium Status in an Iodine Deficient Population of the West Ivory Coast

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Abstract Selenium is an essential trace element which is part of the active site of seleno-dependent glutathione peroxidase and type 1 deiodinase. Therefore, it plays a key role in thyroid hormone metabolism. The present work was undertaken in order to evaluate selenium status in two Ivory Coast populations: the first with high (Glanlé) and the second with low (Abidjan) prevalence of iodine deficiency. Selenium, glutathione peroxidase, glutathione reductase, glutathione and diglutathione were determined in blood and/or urine. In plasma and erythrocytes, selenium and glutathione peroxidase were dramatically low in Glanlé. Compared to Abidjan, selenium, glutathione peroxidase, vitamin E and riboflavin status were decreased whereas diglutathione was increased in Glanlé. The results clearly demonstrate a selenium deficiency and suggest an oxidant stress in Glanlé. Causes and consequences of this selenium deficiency and oxidant stress remain to be determined. *J Physiol Anthropol* 20 (2): 81-84, 2001 <http://www.jstage.jst.go.jp/en/>

Keywords: selenium, deficiency, humans, iodine status

Introduction

Selenium was discovered in 1817 by Berzelius. It was considered for a long time to be a toxic element for humans and animals. Its essential role in animals was proven in 1957. Briefly, this trace element may prevent cancer, heart diseases and toxic manifestations due to xenobiotics or toxic elements, it delays the ageing process and enhances humoral, cell-mediated and non-specific immunity (Nève and Thérond, 1991; Nève, 1991a).

There is a large variation in selenium concentration and availability in soil and food all over the world. These

differences are explained by differences in soil composition (basalt has been reported to contain more selenium than granite (Goyens et al., 1987)), soil acidity and redox function (Nève and Thérond, 1991), both of which modify selenium availability for plants. Therefore, human selenium dietary intake and status differ considerably among geographical area (Table 1). Contrary to other trace elements, i.e. iron, zinc or copper, selenium is highly available from foods. Moreover, selenium bioavailability from foods of plant origin is higher than from animal products (Goyens et al., 1987; Combs, 1988; Fairweather-Tait, 1997). Therefore, family income is not a significant determinant of selenium status.

Selenium is part of the active site of seleno-dependent glutathione peroxidase (GPX) and type 1 deiodinase (Larsen and Berry, 1995; Körlle, 1995; Diplock, 1992; Dumont et al., 1994). Therefore, selenium status plays a key role in thyroid hormone metabolism. Interaction between iodine and selenium biological function greatly depends on the status of these two trace elements. Review articles have been written on that topic (Larsen and Berry, 1995; Körlle, 1995). Combined selenium and iodine deficiency causes an irreversible loss of thyroid function. Iodine deficiency is a necessary condition of endemic goitre. However, the lack of type 1 deiodinase and increase in oxygen toxicity, as a consequence of selenium deficiency, contribute to irreversible thyroid damages (Goyens et al., 1987; Vanderpas et al., 1993; Untoro et al., 1999). Thyroid metabolism modifications during the association of selenium and iodine deficiencies are summarised in Fig. 1. Moreover, in Africa, thiocyanate, originating from cassava contributes also to goitrogenesis and could amplify the effects of selenium deficiency (Goyens et al., 1987; Levander, 1982).

In the West part of Ivory Coast Republic, goitre and cretinism prevalence is high (Kouamé et al., 1998;

Table 1 Selenium dietary intake and status in different countries

Countries	Se intake $\mu\text{g/d}$	Serum Se $\mu\text{mol/l}$	Whole blood Se $\mu\text{mol/l}$
China (Keshan area)	11	<0.30	0.13–0.38
New Zealand	28–32	0.38–0.63	0.57–1.14
Finland (Before supplementation)	30	0.38–0.89	0.63–1.01
Belgium	50–60	0.76–1.33	1.20–2.09
France	35–48	0.70–1.35	
The United States of America	60–220	1.27–2.53	1.90–2.85
Canada	110–220	1.27–2.53	1.90–3.16
Venezuela	>220	1.16–1.45	>4.43

Fig. 1 Coupled iodine and selenium deficiencies and thyroid metabolism

Zimmermann et al., 2000) but blood serum thyroid hormones remain in the international reference ranges (Kouamé et al., 1998). The present study was conducted in order to evaluate selenium status in a village (Glanlé) located in the west part of Ivory Coast Republic and in its capital (Abidjan).

Material and Methods

The subjects recruited for this study included 50 subjects living in the west part of Ivory Coast Republic (Glanlé) and 47 apparently healthy control subjects living in the capital of Ivory Coast Republic (Abidjan). In the first area, the prevalence of goitre and cretinism is high whereas in the second area, goitre and cretinism are not encountered. Glanlé is in a mountain area, whereas Abidjan is close to the sea. In Glanlé, families produce their own foods, whereas in Abidjan, consumed foods could be produced outside.

Blood was collected by venipuncture in Vacutainer® tubes containing heparin as anticoagulant. Samples were immediately stored in a refrigerated box and kept refrigerated until sample treatment. For glutathione determination, one volume of whole blood was deproteinized with 9 volumes of 6% (w/v) metaphosphoric acid within 2 hours. Supernatant was isolated by

centrifugation at 4000 rd/min for 20 min and stored in cryovials at -70°C within one hour and kept frozen until analysis. The other blood sample was centrifuged at 4000 rd/min for 20 min at 4°C . Erythrocytes and plasma were separated and frozen at -70°C until analysis. Urine was collected in polypropylene vials in the morning (7.30–9.30 a.m.). Aliquot was frozen at -70°C until analysis.

Selenium was determined in plasma (P Se), erythrocytes (E Se) and urine (U Se), after sample dilution, using electrothermal atomic absorption spectrometry with Zeeman background correction. Seleno-glutathione peroxidase was performed in plasma (P GPX) and erythrocytes (E GPX) by a modified method of Gunzler et al. (Gunzler et al., 1974) using a RA 1000 autoanalyzer. Glutathione reductase was determined in erythrocytes (E GR) using the method of Thurnham and Rathakette (Thurnham and Rathakette, 1982). Whole blood total glutathione (GSHt), glutathione (GSHr) and diglutathione (GSSG) were determined using the method of Akerboom and Sies (Akerboom and Sies, 1981). Plasma vitamin E and retinol determinations were performed by high performance liquid chromatography (Arnaud et al., 1991). Riboflavin status was assessed by erythrocyte glutathione reductase activation coefficient (E GR AC) (Nichols et al., 1974; Bayoumi and Rosalki, 1976). Plasma albumin measurements were performed by immunoprecipitation and urinary creatinin was determined by Jaffe's method using a Hitachi 917 autoanalyzer. Accuracy and precision of analysis were checked using control materials.

Results

Results are indicated in Table 2. Selenium status was lower in Glanlé than in Abidjan as demonstrated by the statistically significant lower levels in plasma, erythrocytes and urinary selenium as well as the decrease in glutathione peroxidase activity in plasma and erythrocytes. Moreover, when compared to international reference ranges, the population of Glanlé was selenium deficient. Albumin and vitamin E status were lower in Glanlé than in Abidjan, whereas diglutathione and total glutathione were higher. However, the albumin and vitamin E results remained

Table 2 Biological results obtained in 2 Ivory Coast Republic populations with high (Glanlé) and low (Abidjan) prevalence of iodine deficiency

Parameters	Abidjan n=47	Glanlé n=50	P (Student t test)
Plasma Selenium (P Se), $\mu\text{mol/l}$	1.25 ± 0.20	0.40 ± 0.15	<0.0001
Erythrocyte Selenium (E Se), $\mu\text{mol/l}$	2.97 ± 1.06	0.77 ± 0.33	<0.0001
E Se/P Se	2.39 ± 0.89	2.00 ± 0.50	0.0130
Urinary Selenium (U Se), $\mu\text{mol/l}$	0.80 ± 0.46	0.20 ± 0.11	<0.0001
U Se, $\mu\text{mol/mmol creatinine}$	42 ± 17	8 ± 4	<0.0001
Plasma Glutathione Peroxidase, UI/l	322 ± 45	173 ± 49	<0.0001
Erythrocyte Glutathione Peroxidase, UI/g Hb	61 ± 21	21 ± 7	<0.0001
Total Glutathione, $\mu\text{mol/l}$	689 ± 197	849 ± 253	0.0008
Diglutathione (GSSG), $\mu\text{mol/l}$	21 ± 7	64 ± 38	<0.0001
Glutathione (GSHr), $\mu\text{mol/l}$	648 ± 188	722 ± 229	NS
GSHr/GSSG	33 ± 10	15 ± 8	<0.0001
Erythrocyte Glutathione Reductase (E GR), UI/g Hb	2.18 ± 1.15	2.21 ± 1.06	NS
E GR Activation Coefficient	1.68 ± 0.43	1.95 ± 0.45	0.0028
Vitamin E, $\mu\text{mol/l}$	21.5 ± 4.3	17.7 ± 3.6	<0.0001
Albumin, g/l	44 ± 6	40 ± 5	<0.0001
Retinol, $\mu\text{mol/l}$	1.66 ± 0.44	1.57 ± 0.55	NS

Results are expressed as mean $\pm$ 1 standard deviation.

within the international reference ranges. On the contrary, riboflavin status was marginal and total glutathione was low both in Abidjan and Glanlé.

Discussion

The village of Glanlé was of interest because of the high incidence of goitre but also because most of the families produced much of their own foods. On the contrary, Abidjan was selected because of the low incidence of goitre, its seaside location and availability of food items produced outside the country.

Our results clearly demonstrate a selenium deficiency in the village of Glanlé (Van den berg et al., 1993). All selenium indices were dramatically lower in the Glanlé inhabitants than in the Abidjan residents. Moreover, 23 out of the 50 subjects living in Glanlé had plasma selenium concentration under $0.35 \mu\text{mol/l}$, the cut-off of severe selenium deficiency (Van den berg et al., 1993). Finally, in Glanlé, plasma selenium concentration was comparable to those reported in selenium deficient area i.e. China or New Zealand (Table 1). On the contrary, selenium status in Abidjan was adequate (Thorling et al., 1986; Nève, 1991b). Moreover, only one out of the 47 subjects had plasma selenium under $0.75 \mu\text{mol/l}$, considered to reflect marginal selenium status (Van den berg et al., 1993). Selenium deficiency in Glanlé is not the consequence of protein malnutrition as plasma albumin concentrations are in the international reference range. However, albumin was lower in Glanlé subjects than in the Abidjan residents. Unfortunately, no information about the selenium content of the soil and food of Glanlé is available. The higher selenium status of

the Abidjan population could be explained by difference in their consumption habits and by the availability of foods from different origins. A possible hypothesis for the very poor selenium status in Glanlé population could be attributed to genetic factors.

Low GPX activity in Glanlé could be explained by the decrease in selenium status but also in vitamin E and riboflavin status (Golden and Ramdath, 1987; Kuang, 1991). However, glutathione reductase activity was equal in Glanlé and Abidjan. Plasma vitamin E, selenium and riboflavin status and GSH/GSSG ratio were significantly lower in Glanlé than in Abidjan. These results suggest an oxidant stress in Glanlé (Golden and Ramdath, 1987; Kuang, 1991; Richard, 1996).

According to reference ranges of our laboratory (Arnaud and Preziosi, 1994), glutathione concentrations observed in Abidjan and also, but to a lesser extend, in Glanlé, were low. This observation suggests that cysteine intake is low in Ivory Coast Republic. The low glutathione reductase activity (Ceballos-Picot et al., 1992; Tessier et al., 1995) and the marginal riboflavin status observed in both groups could also contribute to these low concentrations (Golden and Ramdath, 1987; Kuang, 1991). Indeed, riboflavin deficiency (E GR AC > 1.70) was noted in 74% of Glanlé subjects and in 34% of Abidjan residents (Van den berg et al., 1993).

Causes and clinical consequences of the selenium deficiency in Glanlé west mountain Ivory Coast remained to be determined. Although a severe selenium deficiency in Glanlé, selenium supplementation should not be undertaken without iodine supplementation. Indeed, the aggravation of hypothyroxinemia after selenium supplementation is associated with an aggravation of

hypothyroidism (Goyens et al., 1987; Vanderpas et al., 1993).

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Received: February 3, 2001

Accepted: March 10, 2001

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