Table 2. Weight gain, energy intake and Nitrogen balance in rats fed various palm oil fractions.

<table>
<thead>
<tr>
<th>Dietary treatment</th>
<th>Weight gain (g)</th>
<th>Energy intake (MJ)</th>
<th>Nitrogen intake (g)</th>
<th>Nitrogen retained B-B (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPO</td>
<td>258</td>
<td>29.2</td>
<td>11.1</td>
<td>1.52</td>
</tr>
<tr>
<td>RPO</td>
<td>236</td>
<td>19.9</td>
<td>10.5</td>
<td>1.05</td>
</tr>
<tr>
<td>RPO</td>
<td>260</td>
<td>44.1</td>
<td>10.6</td>
<td>1.31</td>
</tr>
<tr>
<td>RPS</td>
<td>244</td>
<td>55.4</td>
<td>10.6</td>
<td>1.81</td>
</tr>
<tr>
<td>RPS</td>
<td>240</td>
<td>37.0</td>
<td>10.2</td>
<td>1.56</td>
</tr>
<tr>
<td>OO (control)</td>
<td>255</td>
<td>34.1</td>
<td>10.6</td>
<td>1.88</td>
</tr>
</tbody>
</table>

Average starting weight approximately 107 g/male

and RPO-fed groups were respectively, 13% higher and 8% lower than the present results. A direct comparison of those results with the present ones is not justified since both the design and duration of the two studies were different.

The practical implication of our studies may be summarised as follows:

Millions of children around the world suffer from protein-energy malnutrition. Such children are characterised by muscle wasting, loss of adipose tissue and oedema. The routine administration of supplementary foods may help in the rehabilitation of malnourished children, which may be a practical outcome of these studies.

Influence of palm oil and palm oil fractions on protein utilisation

Chijioke Jeyakumar Henry, Amal Ghassou-Choueiri, Michael I Chaur

Aspects of palm oil and palm oil fractions on protein utilisation

Chijioke Jeyakumar Henry, Amal Ghassou-Choueiri, Michael I Chaur

Diet-derived and topically applied tocopherols accumulate in skin and protect the tissue against ultraviolet light-induced oxidative stress

Maret G Traber, Maurizio Podda, Christine Weber, Jens Thiele, Michalis Rallys, Lester Packer
Dept. Molecular and Cell Biology, University of California, Berkeley

To evaluate the tissue-specific distribution of lipophilic antioxidants including various vitamin E forms (tocopherols and tocotrienols) and oxidized and reduced forms of Coenzyme Q (ubiquinone and ubiquinol) a procedure was developed using gel electrophoresis and U.V. detection. A unique distribution of these antioxidants in skin and heart muscle was found, suggesting that their distribution was dependent on tissue selective mechanisms for maintaining antioxidant defences. Ubiquinone-9 was highest in kidney (81 ± 29 nmol) and in liver (42 ± 16 nmol), while the highest ubiquinone-9 concentrations were found in kidney (306 ± 123 nmol) and heart (284 ± 23 nmol). Liver contained nearly identical amounts of ubiquinone-9 (41 ± 16 nmolo and ubiquinone-9 (46 ± 18 nmol). These mice were fed a commercial Chow diet containing α-tocopherol (38 mg/kg diet), α-tocotrienol (10 ± 1.1), α-tocopherol (3.1 ± 0.7) and γ-tocopherol (7.4 ± 1.7). Of the vitamin E forms, brain contained only α-tocopherol (5.4 ± 0.1 nmol); 99.9% and no detectable tocotrienols. In other tissues, the α-tocopherol content was higher (30 nmol), while each of the other forms represented about 1% of the total (α-tocopherol 0.2 ± 0.4 nmol, α-tocotrienol 0.1, γ-tocopherol 0.2). Remarkably, skin contained nearly 15% tocotrienols and 1% γ-tocopherol. The unique distribution of tocotrienols in skin suggested that they might have superior protection against environmental stressors. Therefore, the penetration of topically applied vitamin E (tocotrienol enriched fractions of palm oil, TRP) and vitamin E homologue concentrations before and after exposure of skin to U.V. light was assessed. 20% of 5% TRP in polyethylene glycol-400 (PEG) was applied to 2 skin sites and 20 μL PEG to 18 sites. After 2 h, the skin was washed and half of the sites exposed to U.V.-irradiation using a Campbell Light Unit (280 w/m2 for 20 min). The vitamin E content of hairless mouse skin was: α-tocopherol 0.0 ± 1.0 mg/kg skin, γ-tocopherol 0.44 ± 0.03, α-tocopherol 0.48 ± 0.07, γ-tocopherol 0.92 ± 0.03. Topical TRP enriched skin vitamin E: α-tocopherol 0 ± 70 μmol skin, γ-tocopherol 37 ± 15, α-tocotrienol ± 52 ± 24. After U.V.-irradiation, concentrations of all vitamin E homologues from both treatments areas decreased significantly (p<0.01), but the TRP-treated skin contained vitamin E at concentrations 7-10 fold higher than control treatment values. These findings provide provocative clues on the uptake and regulation of lipophilic antioxidants. The unique distribution of these antioxidant substances suggest their distribution may be dependent upon tissue-specific selective mechanisms.

Introduction

The major lipophilic antioxidant in plasma, membranes and tissues is vitamin E, an essential nutrient that protects essential polyunsaturated fatty acids in membranes and tocopherols and tocotrienols from a tocotrienol-rich palm oil fraction (TRF), and evaluated the protection conferred by these various forms of vitamin E against oxidative stress.

This paper reviews our recent findings concerning the tissue distribution of vitamin E homologues in hairless mouse tissues.

We also present our findings on the protective effects of topical applied TRF to hairless mouse skin.

Methods and materials

Materials

Highest purity reagents and reagents were used. Tocopherol and tocotrienol were from Hemdall Corp. (Orange, B). TRF was kindly provided by Palm Oil Research Institute of

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References

Malaysia (PORIM Kuala Lumpur, Malaysia). Tocotrienols for use as standards were purified from TRF by Dr. Atsaf A Qureshi, University of Wisconsin (Madison, WI). Ubiquinones-9 and 10 standards were a gift from Nisshin Flour Mill Co., Ltd (Tokyo, Japan). Animals Handling and housing. Experimental procedures for animal handling were approved by the Animal Care and Use Committee of the University of California, Berkeley. Female hairless mice (SHR, 8-12 weeks old) were purchased from Charles River Laboratories (Wilmington, MA, USA) and were kept under standard light and temperature conditions. Food (Hatsus Tekb Rodent Diet 8656, WI, USA) and water were provided ad libitum. Tissues were obtained from three mice, which were anesthetized and killed by cervical dislocation.

TRF application to skin. Mice were anesthetized by an intraperitoneal injection of sodium pentobarbital (50 mg/kg body weight) and remained anesthetized during the entire experiments. Four polypropylene plastic rings (1 cm) were glued onto the animals' backs, then 20 ml of a 5% w/v mixture containing TRF in polyethylene glycol-400 (PEG). Six to eight mice per group were used. The PEG was applied to the skin circumference by 2 rings or PEG alone to the other 2 rings. After 2 hours, the skin was washed as described by Dupart et al., the position of the application site was marked, the plastic rings removed, and the mice exposed to UV-irradiation. Skin samples were then discarded out in 15 min. UV-irradiation. The mice were placed under an Oriel 1000 W solar simulator (Oriel, Stratford, CT) with an output of 2.8 mW/cm² of UV-A and UVB light (290-400 nm) and irradiated for 29 minutes (corresponding to 3 MED) on either the upper or lower back, while the other part was shielded from UV-light by covering unexposed regions of the skin with paper and aluminium foil. After exposure, the mice were killed by neck dislocation, and the skin with subcutaneous fat were excised from the 4 exposure sites and the specimens immediately frozen in liquid nitrogen. All tissues were extracted as described by Burton et al., with the exception of skin and diet samples, which were handled as described.

Figure 1. Percent distribution of α-tocopherol, γ-tocopherol, α-tocotrienol, and γ-tocotrienol in various hairless mouse tissues. The fraction of the total vitamin E represented by each of the various vitamin E homologues is shown. Mice were fed chow diet.

HPLC analysis. The details of the method are reported elsewhere. The HPLC system consisted of a Hewlett Packard 1050 series gradient pump, a SCL-10A Shimadzu system controller with a SILC-RIA autoinjector with sample cooler, a Beckman Ultravision ODS C-18 column, a Hewlett Packard 1050 diode array detector and an BioRad LKB 2026 HPLC-IT-600 pen-tocopherol detector with a glassy carbon electrode (0.5 V potential, full recorder scale at 50 nA). The detectors were setup in line, the eluent passing first through the diode array detector (275 nm).

Anchovies and sesame oil as standards were purified from TRF by Dr. Atsaf A Qureshi, University of Wisconsin (Madison, WI). Ubiquinones-9 and 10 standards were a gift from Nisshin Flour Mill Co., Ltd (Tokyo, Japan). Handling and housing. Experimental procedures for animal handling were approved by the Animal Care and Use Committee of the University of California, Berkeley. Female hairless mice (SHR, 8-12 weeks old) were purchased from Charles River Laboratories (Wilmington, MA, USA) and were kept under standard light and temperature conditions. Food (Hatsus Tekb Rodent Diet 8656, WI, USA) and water were provided ad libitum. Tissues were obtained from three mice, which were anesthetized and killed by cervical dislocation.

TRF application to skin. Mice were anesthetized by an intraperitoneal injection of sodium pentobarbital (50 mg/kg body weight) and remained anesthetized during the entire experiments. Four polypropylene plastic rings (1 cm) were glued onto the animals' backs, then 20 ml of a 5% w/v mixture containing TRF in polyethylene glycol-400 (PEG). Six to eight mice per group were used. The PEG was applied to the skin circumference by 2 rings or PEG alone to the other 2 rings. After 2 hours, the skin was washed as described by Dupart et al., the position of the application site was marked, the plastic rings removed, and the mice exposed to UV-irradiation. Skin samples were then discarded out in 15 min. UV-irradiation. The mice were placed under an Oriel 1000 W solar simulator (Oriel, Stratford, CT) with an output of 2.8 mW/cm² of UV-A and UVB light (290-400 nm) and irradiated for 29 minutes (corresponding to 3 MED) on either the upper or lower back, while the other part was shielded from UV-light by covering unexposed regions of the skin with paper and aluminium foil. After exposure, the mice were killed by neck dislocation, and the skin with subcutaneous fat were excised from the 4 exposure sites and the specimens immediately frozen in liquid nitrogen. All tissues were extracted as described by Burton et al., with the exception of skin and diet samples, which were handled as described.

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Quantification was carried out by comparison of peak areas to the area of standard curves obtained with authentic compounds. For vitamin E, α- and γ-tocopherols were used as standards because the chromanol nucleus is the same in α-tocotrienol and α-tocotrienol, and in γ-tocopherol and γ-tocotrienol, respectively.

Statistical analysis. A statistical significance was carried out using SuperAnova for the Macintosh (Berkeley, CA). A p-value <0.05 was considered statistically significant.

Results Tissue antioxidants. Tissue concentrations were measured in various mouse tissues and in diet. In all tissues, α-tocopherol was the major form of vitamin E. As shown in Figure 1, the brain contains 90.8% α-tocopherol, while skin contains nearly 15% tocotrienols and 1%-γ-tocopherol. The associated tocopherol was not the source of these vitamin E forms because in skin samples which had the fat removed, the tocotrienol concentrations were actually higher (compared to skin with skin and subcutis). In other tissues (heart, kidney, liver), each of these forms represents about 1% of the total vitamin E.

The diet contained α-tocopherol (29.7 ± 6.2 mg/kg diet), γ- tocopherol (10.3 ± 1.1), α-tocotrienol (3.1 ± 0.7) and γ-tocotrienol (7.4 ± 1.7). It is likely that the tocotrienols found in mouse skin arise from the mouse diet and the samples immediately frozen in liquid nitrogen. All tissues were extracted as described by Burton et al., with the exception of skin and diet samples, which were handled as described.

The left panel shows the vitamin E concentrations (mean ± SD, n=5) in skin from non-irradiated or UV-irradiated 21-day-old mice. The right panel shows the vitamin E concentrations in skin from hairless mice before and after UV-irradiation following topical application of TRF (PEG). Note the different scales on the right and left y-axes. Significant decreases in each vitamin E homologue were observed after UV-irradiation both in PEG-treated and in TRF-treated skin. By least-squares methods: PEG versus TRF: α-tocopherol, p<0.001; α-tocotrienol, p<0.01; γ-tocopherol, p<0.001; and γ-tocotrienol, p<0.001; TRF versus TRF+UV: α-tocopherol, p<0.001; α-tocotrienol, p<0.001; for γ-tocopherol, p<0.001; and γ-tocotrienol, p<0.001.

After topical application of TRF, all the vitamin E forms were readily penetrated into the skin and present skin and were present in concentrations for exceeding the baseline levels. Norkus et al. have also demonstrated that application of α-tocopherol aceate into hairless mouse skin results in penetration of high concentrations into skin. Tocopherols and tocotrienols in murine skin, applied topically or derived from the diet, were significantly depleted by UV-
Malaysia (PORIM Kuala Lumpur, Malaysia). Tocotrienols for use as standards were purified from TRF by Dr Asaf A Qureshi, University of Wisconsin (Madison, WI). Ubiquinones-9 and 10 and standards were a gift from Nissin Flavour Milling Co, Ltd (Tokyo, Japan).

Animals
Handling and housing. Experimental procedures for animal handling were approved by the Animal Care and Use Committee of the University of California, Berkeley. Female hairless mice (SKH-1, 8–12 weeks old) were purchased from Charles River Laboratories (Wilmington, MA, USA) and were kept under standard light and temperature conditions. Food (Harlan Teklad Rodent Diet 8656, WI, USA) and water were provided ad libitum. Tissues were obtained from three mice, which were anaesthetized and killed by cervical dislocation.

TRF application to skin. Mice were anaesthetized by an intraperitoneal injection of sodium pentobarbital (50 mg/kg body weight) and remained anaesthetized during the entire experimental period. Four polypyrrole plastic rings (1 cm²) were glued onto the animals’ backs, then 20 µl of a 5% v/v mixture containing TRF in polyvinyl alcohol-400 (PEG) was applied to each site. Six ST mice (Los, MO) was applied to the skin crosslinked by 2 rings or PEG alone to the sites. After 2 hours, the skin was washed as described by Dutta et al.²⁸ The position of the application site was marked, the plastic rings removed, and the mice exposed to UV-irradiation. Skin specimens were removed at 0, 15, and 30 min.

UV-irradiation. The mice were placed under an Oriel 1000 W solar simulator (Oriel, Stratford, CT) with an output of 2.8 mW/cm² of UV-A and UV-B light (290–400 nm) and irradiated for 29 minutes (corresponding to 3 MED) on either the upper or lower back, while the other part was shielded from UV-light by covering it with uncolored regions of the skin with paper and aluminum foil. After exposure, the mice were killed by neck dislocation, and the skin with subcutaneous fat were excised from the 4 exposure sites and the samples immediately frozen in liquid nitrogen. All tissues were extracted as described by Burton et al.²⁹ with the exception of skin and diet samples, which were handled as described.

Figure 1. Percent distribution of α-tocopherol, ω-tocopherol, α-tocotrienol, and γ-tocotrienol in various hairless mouse tissues.

The fraction of the total vitamin E represented by each of the various vitamin E homologues is shown. Mice were fed chow diets.

HPLC analysis
The details of the method are reported elsewhere.²⁹ The HPLC system consisted of a Hewlett Packard 1050 series gradient pump, a SCL-10A Shimadzu system controller with a SIL-10A autoinjector with sample cooler, a Beckman Ultraviolet ODS C-18 column, a Hewlett Packard 1050 diode array detector and an Biospectroscopy (Cupertino, CA) HPLC flow injection autochemical detector with a glassy carbon electrode (0.5 V potential, full recorder scale at 50 nA). The detectors were setup in line, the eluent passing first through the diode array detector (275 nm). The mobile phase consisted of a mixture of A (80/20 v/v methanol/water and 0.2 percent w/v lithium perchlorate) and B (ethanol, reagent grade with 0.2 percent w/v lithium perchlorate). Mobile phase A was delivered at 0.615 µl/min and B at 39 Å, A after 16 minutes the mobile phase was changed linearly over a 2 min. to 100% B, which was continued for 10 more min. at 1 ml/min. to the initial composition; total run time was 40 minutes.

Quantitation was carried out by comparison of peak areas to the area of standard curves obtained with authentic compounds. For vitamin E, α- and γ-tocopherols were used as standards because the chromin nucleus is the same in α-tocotrienol and α-tocotrienol, and in γ-tocotrienol and γ-tocotrienol, respectively.

Statistical analyses
A p-value of <0.05 was considered statistically significant.

Figure 2. α-tocopherol, ω-tocopherol, γ-tocopherol and γ-tocotrienol contents of murine skin.

The left panels show the vitamin E concentrations (mean ± SD, n = 10) in skin from non-irradiated or UV-irradiated (5 MED, 30 min) topical application of PEG (vehicle alone). The right panels show the vitamin E concentrations in skin of hairless mice before and after UV-irradiation following topical application of TRF (in PEG). Note the different scales on the right and left y-axes. Significant decreases in each vitamin E homologue were observed after UV-irradiation both in PEG-treated and in TRF-treated skin. By least square means comparisons: PEG versus TRF, α-tocopherol, ω-tocopherol, γ-tocopherol, p<0.001; α-tocopherol, p<0.001; ω-tocopherol, p<0.001; γ-tocopherol, p<0.001; TRF versus TRF+UV for α-tocopherol, p<0.001; α-tocopherol, p<0.001; γ-tocopherol, p<0.001; and γ-tocopherol, p<0.001.

After topical application of TRF, all the vitamin E forms rapidly penetrated the skin and the increase skin were present in concentrations far exceeding the baseline levels. Nicklas et al.²⁹ have also demonstrated that application of α-tocopherol acetate onto hairless mouse skin results in penetration of high concentrations into skin. Tocopherols and tocotrienols in murine skin, applied topically or derived from the diet, were significantly depleted by UV-
response to an oxidative stress, suggests that these vitamin E forms protect similarly against UV-irradiation induced damage. A larger percentage of the various vitamin E forms remained after UV-irradiation of the PEO-treated compared with the TRF-treated skin (Figure 2). This implies a greater destruction of the various vitamin E forms in the TRF-treated skin. Whether this is due to increased free radical formation remains to be clarified. Localisation of TRF nearer to the upper epidermal layers in the TRF-treated skin could allow increased destruction during UV-irradiation. Alternatively, the TRF vitamin E may have penetrated the lipid components surrounding cells and thus may not be accessible to aqueous antioxidants which can recycle the vitamin E. Thus, the applied TRF may have a different behaviour during UV-exposure than the vitamin E naturally present. It should be emphasized that the skin was washed with ethanol and dried before exposure to UVC-light; therefore, the vitamin E forms we have measured are not on the skin surface, but have penetrated into the skin. Ubiquinol is the first line of defence in response to an oxidative stress. It may readily react with the tocopheroyl radical and be oxidised, or it may react directly with peroxyl radicals. Tissues involved in detoxification, both the liver and the kidney, have extraordinarily high concentrations of ubiquinol, perhaps to protect them from radicals escaping from 450. In addition, these tissues have high concentrations of mitochondria, which could also account for their high coenzyme Q contents.

Coenzyme Q (ubiquinol/ubiquinone) was chosen as a marker for oxidative damage because ubiquinol is the most labile, lipid soluble antioxidant and is not present in TRF. Ubiquinol is oxidised prior to co-occurrence during UV-irradiation of skin and in its substantially decreased before co-occurrence concentrations are affected. The levels of ubiquinol detected in murine skin are low; nonetheless, following UV-irradiation ubiquinol, ubiquinone and total Q all significantly decreased. Regardless of TRF application, UV-irradiation caused a loss in the total Q pool, thus depleting the skin of a vital component. In summary, the data presented here give provocative clues to the uptake and regulation of tissue lipophilic antioxidants. This paper demonstrates not only that a variety of antioxidants are present in skin, but that topical application provides efficient means of enriching the tissue in protective antioxidants, such as vitamin E. Furthermore, those vitamin E homologues consumed during UV-light induced oxidative stress.

Acknowledgements
Both Koh and Kenneth Tsang provided excellent technical assistance. We gratefully acknowledge the efforts of Dr Aarif Anwar, University of Wisconsin, Madison, WI, who isolated tocotrienols for use as standards for this study. This study was supported by grants from the NIH (CA 14797) and the Palm-Oil Research Institute of Malaysia. JF was supported by a fellowship of the Fritz Thyssen Stiftung, Germany (AZ 219590B).

References
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In summary, the data presented here give provocative clues to the uptake and regulation of tissue lipophilic antioxidants. This paper demonstrates not only that a variety of antioxidants are present in skin, but that topical application provides an efficient means of enriching the tissue in protective antioxidants, such as vitamin E. Furthermore, these vitamin E homologues are consumed during UV-light induced oxidative stress.

Acknowledgments
Both Koh and Kenneth Trang provided excellent technical assistance. We gratefully acknowledge the efforts of Dr Aqas A Qandali, University of Wisconsin (Madison, WI), who isolated tocopherols for use as standards for this study. This work was supported by grants from the NIH (CA 101973) and the Palm Oil Research Institute of Malaysia. J.T. was supported by a fellowship of the Fritz Thyssen Stiftung, Germany (AZ 219505B).

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