

## Original Article

# Consumption of purple sweet potato leaves decreases lipid peroxidation and DNA damage in humans

Chiao-Ming Chen RD MSc<sup>1,2,3</sup>, Ya-Ling Lin RD MSc<sup>2</sup>, C-Y Oliver Chen PhD<sup>4</sup>  
Ching-Yun Hsu RD MSc<sup>5</sup>, Ming-Jer Shieh PhD<sup>2</sup>, Jen-Fang Liu RD PhD<sup>2</sup>

<sup>1</sup>Graduate Institute of Pharmacy, Taipei Medical University, Taipei, Taiwan

<sup>2</sup>School of Nutrition and Health Sciences, Taipei Medical University, Taipei, Taiwan

<sup>3</sup>Department of Dietetics, Taipei Medical University Hospital, Taipei, Taiwan

<sup>4</sup>Jean Mayer USDA Human Nutrition Research Center on Aging, Tufts University, Boston, MA, USA

<sup>5</sup>Chang-Gung Institute of Technology, Taoyuan, Taiwan

Consumption of polyphenols is associated with reduced risk of chronic diseases, possibly via a variety of bio-mechanisms, including antioxidation and anti-inflammation. Purple sweet potato leaves (PSPL) commonly consumed in Asia possess polyphenols. In this study, we aim to investigate antioxidant effect of 200 g/d PSPL containing 902 mg polyphenols in a clinical trial. This randomized, crossover clinical study included 16 healthy adults (7 M, 9 F; aged 20-22 y). After a 1-wk run period, subjects were assigned randomly to receive either PSPL or low polyphenol diet (LPD) for 2 wks, followed by a 2-wk washout period before crossing over to the alternate diet. Fasting blood and 24-h urine samples were collected from each subject at day 0, 7 and 14 of each phase. Our data showed PSPL consumption enhanced urinary total phenol excretion by 24.5% at day 14 as compared to day 0, while the LPD decreased total phenol content in plasma and urine by 3.3 and 16.3%, respectively ( $p \leq 0.05$ ). Low-density lipoprotein lag time and glutathione concentration in erythrocytes at day 14 was significantly enhanced by 15.0 and 33.3% by PSPL as compared to day 0, respectively, while their values were not altered by the LPD. Urinary 8-hydroxy-deoxyguanosine (8-OHdG) excretion decreased significantly by PSPL consumption by 36.7% at day 7 as compared to day 0, yet unchanged by the LPD ( $p \leq 0.05$ ). In conclusion, our results suggest that polyphenols in 200 g PSPL were bio-available and could enhance antioxidant defense and decrease oxidative stress in young healthy people.

**Key Words:** purple sweet potato leaves, polyphenols, lipid peroxidation, 8-hydroxydeoxyguanosine, DNA damage

## INTRODUCTION

Evidence from epidemiological studies suggested a strong, inverse association between incidence of chronic diseases and intake of plant foods, possibly due to their high nutrient density and low fat contents.<sup>1-5</sup> Thereby, consumption of plant foods has been strongly promoted and promulgated in the dietary guidelines by the public health authorities and regulatory agencies.<sup>6</sup> Nevertheless, contribution of nonessential phytonutrients ubiquitous in plant foods to reduced the risk of health problems via an array of putative mechanism of bioactions, including anti-inflammation, antioxidation, anti-proliferation, and induction of phase II enzymes has been gradually recognized.<sup>7-9</sup> In particular, there is growing interest in polyphenolic compounds because of their prevalence in plants, as well as potent antioxidant activity.<sup>10</sup>

Leaves of sweet potato (*Ipomoea batatas*) have been consumed commonly in Asian countries and are rich in micronutrients.<sup>11</sup> Because this plant tolerates well against diseases, pest infestation, and flooding,<sup>12</sup> leaves of sweet potato can provide health benefits to people residing in resource poor areas. Like other plant foods, grapes, green tea, onions, these leaves contain polyphenols ranging from 2-14 g/100g dry weight and exhibit antioxidant<sup>13,14</sup>

and anti-mutagenic activity.<sup>15</sup> Recently, we observed in a clinical trial that a 2-wk supplementation of 200 g/d cooked purple sweet potato leaves (PSPL) increased Con A-activated proliferation and IL-2 and -4 secretions in peripheral blood mononuclear cells and elevated lytic activity of NK cells.<sup>16</sup> In other human trial, we also found that 200 g/d PSPL for 2 wks enhanced total phenol content in plasma and LDL resistance against oxidation and decreased urinary 8-hydroxydeoxyguanosine (8-OHdG) in elite basketball players.<sup>17</sup>

While health benefits of polyphenolic compounds could be mediated via a wide spectrum of bioactions, the effect of PSPL incorporated into daily diets on antioxidant defenses and biomarkers of oxidative stress in health individuals remains to be examined. Thus, in this study,

**Corresponding Authors:** Dr. Jen-Fang Liu and Dr. Ming-Jer Shieh, School of Nutrition & Health Sciences, Taipei Medical University, 250 Wu-Shing Street, Taipei 110, Taiwan  
Tel: +886- 2-27361661 ext. 6546 and 6500; Fax: +886- 2-27373112.

Email: liujenfa@tmu.edu.tw; clark@tmu.edu.tw.

Manuscript received 22 April 2008. Initial review completed 18 June 2008. Revision accepted 22 August 2008.

we aim to investigate whether addition of 200 g/d PSPL to a low polyphenol diet (LPD) for 2 wk can enhance antioxidant defenses and thereby decrease oxidative stress in a cross-over clinical trial. The information gathered from this study is useful for promoting inclusion of sweet potato leaves for health promotion and prevention in resource poor areas.

## MATERIALS AND METHODS

### Preparation of purple sweet potato leaves

Purple sweet potatoes were planted at the Taoyuan District Agriculture Improvement Station, Taipei Branch, Taiwan, which is 1 hour away from the Taipei Medical University. Fresh PSPL were shipped daily to our metabolic research unit, weighted, washed, stir fried in soy bean oil, and then provided to subjects.

### Subjects

Sixteen non-smokers (7 M, 9 F, age: 20-22 yrs, BMI: 20.6-21.4 kg/m<sup>2</sup>) in good health condition, based on results from a medical history questionnaire, physical examination, electrocardiogram test, and standard clinical biochemistries. Exclusion criteria included: 1) history of cardiovascular, hepatic, gastrointestinal, and renal disease; 2) alcoholism; 3) use of antibiotics or multi-vitamin and mineral for  $\geq 4$  wk prior to the study. Volunteers were asked not to take any vitamin supplement or medication during the whole study period. The study was approved by the Medical Ethical Committee of the Institutional Review Board from Taipei Medical University, and written consent was obtained from each participant.

### Study design

A randomized, crossover design was employed in this study. The duration of the whole study was 7 wks, including 1-wk run-in and 2 phases of 2-wk dietary treatment with a 2-wk washout (Figure 1). During the whole study, all subjects were asked to follow a low polyphenol diet (LPD) that excluded berries, apples, pears, citrus fruits, fruit juices, onions, gynura, basil, bok choy, spinach, rabbit milkweed, brassica napus, chocolate, wine, coffee, tea, beans, nuts, soy related products, and most spices.<sup>18</sup> Following the run-in phase, 16 volunteers were assigned randomly to either the PSPL or LPD diet (n = 8). Lunch and dinner meals were provided to all subjects during the study, and were designed by a registered dietitian of the

Department of Dietetics in the Taipei Medical University Hospital. They were prepared daily under supervision of the registered dietitian. Meals for one day contained 2000  $\pm$  200 Kcal with 18, 30, and 52% of calories from protein, fat, and carbohydrate, respectively. Typical Chinese lunch and dinner meals consisted of a meat (pork or chicken) dish, a low polyphenols vegetable dish, steamed rice, and a low polyphenols fruit. Two hundred grams of cooked PSPL were divided equally into lunch and dinner meals. In order to ensure good compliance, all participants ate meals in the hospital cafeteria under supervision of the study dietitian. Breakfast was not provided to the subjects in the study, but a list of recommended food items that are low in polyphenols was provided. Further, to monitor compliance to the low polyphenol diet, 3-day dietary records were collected from the subjects every week. Total body fat was assessed using a body fat impedance analyzer at the end of each phase (Inbody 3.0, Biospace, Seoul, Korea).

### Sample collection and storage

Six fasted venous blood samples were collected from each subject between 7-9 AM in the study (Fig. 1). Following centrifugation at 1000 x g for 10 min at 4°C, aliquots of plasma samples were snap frozen in liquid nitrogen and stored at -80°C. One aliquot of fresh plasma was used immediately for the LDL oxidation assay on the same day. After washed with ice-cooled saline three times and hemolyzed using ice-cooled distilled water, erythrocytes were stored at -80°C for glutathione (GSH) determination. A total of six 24-h urine samples were collected from each subject on the same day of blood collection. Urine was collected into an amber plastic container and stored at 4°C before it was brought back to the lab. After the volume was recorded, aliquots of urine samples were stored at -20°C for determinations of total phenolic content and 8-OHdG.

### Biomarkers of antioxidant defense and oxidative stress

Total phenolic contents in urine and plasma were measured via the Folin-Ciocalteu's reaction, according to the method of Singleton.<sup>19</sup> Results were expressed as gallic acid equivalents (GAE)  $\mu$ mol/L.

Plasma  $\alpha$ -Tocopherol was measured using a HPLC method of Milne and Botnen.<sup>20</sup> Total antioxidant status (TAS) in plasma was assessed using a commercial enzymatic assay (Randox, UK). Reduced GSH in erythrocytes was determined using a commercial enzymatic assay (Calbiochem Co., CA, USA). Plasma malondialdehyde (MDA) and 4-hydroxy-2-nonenal (4-HNE), products of lipid peroxidation, were measured using a commercial enzymatic assay (Calbiochem Co., USA). Urinary 8-OHdG was determined using an ELISA assay (Japan Institute for the Control of Aging, Japan). The resistance of LDL against Cu<sup>2+</sup>-induced oxidation was determined according to the slightly modified method of Chen et al.<sup>21</sup> Briefly, following a 24-hour dialysis against saline containing Na-EDTA (1 mmol/L), LDL protein was quantified using a Bio-Rad protein assay kit (Bio-Rad Laboratories, Inc., Hercules, CA, USA). Subsequently, LDL (182 nmol/L) was oxidized by 10  $\mu$ mol/L CuSO<sub>4</sub> in a final volume of 1.0 mL. Formation of conjugated dienes was

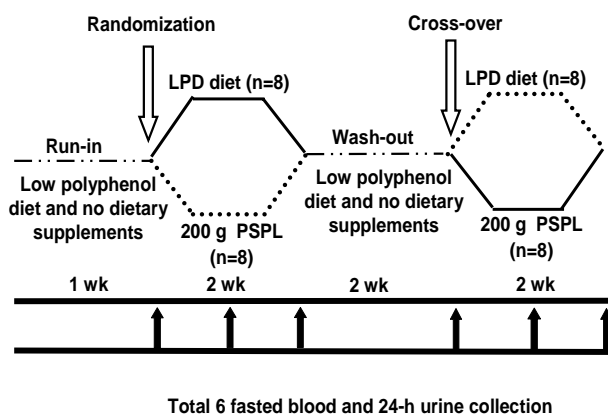


Figure 1. Study design

monitored by absorbance at 234 nm at 37°C over 6 hour using a UV3000 spectrophotometer (Hitachi, Japan) equipped with a 6-position automated sample changer. The results of the LDL oxidation are expressed as lag time (defined as the intercept at the abscissa in the diene-time plot).

### Statistical analysis

All results were reported as mean  $\pm$  SD. Repeat ANOVA (mix-model) analysis was performed to evaluate changes in parameters in the same dietary group over three time points, student's *t* test was performed to evaluate changes from the d 0 value (D7-D0 and D14-D0) between LPD and PSPL dietary group. *p* value  $\leq$ 0.05 was considered significant. The SAS statistical software package (SAS Institute Inc., Cary, NC) was used to perform all statistical analyses.

### RESULTS

During the study period, a balanced diet provided to the subjects contained 2000  $\pm$  200 Kcal, 95  $\pm$  10 g protein, 250  $\pm$  25 g carbohydrate, and 69  $\pm$  7 g fat. 200 g cooked PSPL contained 60 Kcal, 6.6 g protein, 1.2 g fat, 9.2 g carbohydrate, 38 mg vitamin C, 170 mg Ca, 40 mg Mg, 902 mg total phenols, and 47.5 mg carotenoids.<sup>22</sup> All 16 subjects completed the 7-wk study and were fully compliant to the LPD, based on the results of dietary records. No significant changes in their BMI, total body fat and clinical biochemistries were observed (Table 1).

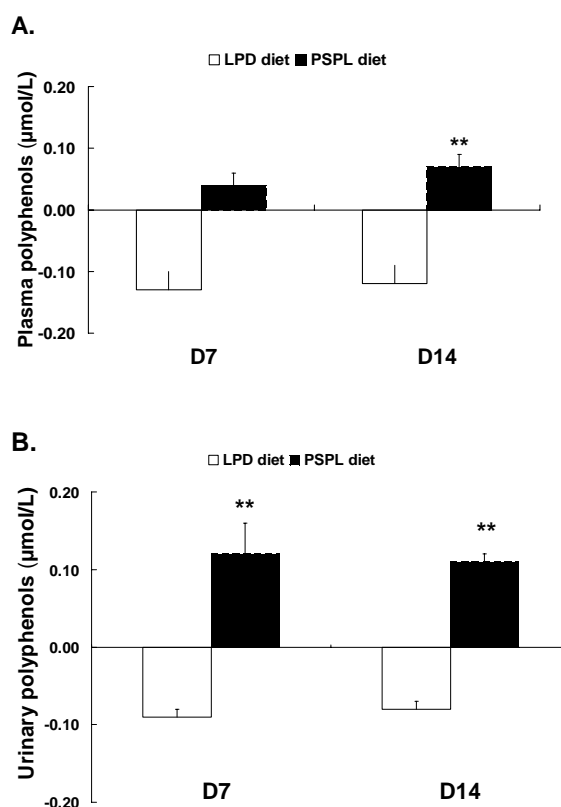
PSPL addition to the LPD maintained total phenolic content in plasma while the LPD alone led to a significant 3.3% decrease from 3.59  $\pm$  0.11 to 3.47  $\pm$  0.08  $\mu$ mol/L at

**Table 1.** Demographic characteristics and clinical biochemistries of subjects<sup>1</sup>

	Before	After
Age (yr)	20.4 $\pm$ 1.8	
Height (cm)	167.8 $\pm$ 9.1	
Body weight (kg)	58.7 $\pm$ 9.2	59.6 $\pm$ 9.2
BMI (kg/m <sup>2</sup> )	20.8 $\pm$ 2.3	21.1 $\pm$ 2.2
Body fat (%)	22.3 $\pm$ 6.8	23.8 $\pm$ 7.0
Creatinine (mg/dL)	0.89 $\pm$ 0.15	0.92 $\pm$ 0.15
GOT (IU/L)	19.7 $\pm$ 6.16	19.0 $\pm$ 4.40
GPT (IU/L)	14.80 $\pm$ 8.58	13.7 $\pm$ 5.94
Triglyceride (mg/dL)	62.2 $\pm$ 21.3	73.4 $\pm$ 55.8
Cholesterol (mg/dL)	156 $\pm$ 24.9	156.0 $\pm$ 33.4
HDL-Cholesterol (mg/dL)	58.3 $\pm$ 15.8	58.6 $\pm$ 17.9
LDL-Cholesterol (mg/dL)	86.1 $\pm$ 17.9	82.3 $\pm$ 20.2

<sup>1</sup>Results were expressed as mean  $\pm$  SD (n=16).

\*\*Means significantly differ, tested using pair-t test (*p*  $\leq$ 0.05).



**Figure 2.** Changes in plasma (A) and urinary (B) polyphenols. Total phenolic content in plasma at day 0 was 3.59  $\pm$  0.11 and 2.87  $\pm$  0.10  $\mu$ mol/L for LPD and PSPL, respectively, as well as 0.49  $\pm$  0.07 and 0.49  $\pm$  0.09  $\mu$ mol/L in urine. The results were reported as mean  $\pm$  SD, n=16. \*\*Means significantly differ between two groups, tested using student's *t* test (*p*  $\leq$ 0.05).

d 14 as compared to that at d 0 (*p*  $\leq$ 0.05). Similarly, urinary total phenolic excretion in the LPD group was decreased significantly by 16.3% from 0.49  $\pm$  0.07  $\mu$ mol/L at d 0 to 0.41  $\pm$  0.08  $\mu$ mol/L at d 14 (Figure 2). However, urinary total phenol excretion in the PSPL group was significantly augmented by 24.5% at d 14 as compared to that at d 0 (*p*  $\leq$ 0.05). Further, the increased plasma total phenolic content from d 14 to d 0 in the PSPL was significantly different from the slightly decreased value in the LPD (*p*  $\leq$ 0.05). Similarly, increases in urinary phenolics in the PSPL group at day 7 and 14 as compared day 0 were significantly different from those in the LPD group.

At day 14, PSPL and LPD both decreased plasma  $\alpha$ -tocopherol by 31.7 and 15.8% as compared to day 0, respectively (Table 2). Further, the decrease was larger in subjects consuming PSPL than LPD. Erythrocyte GSH status was not significantly altered by the LPD from day 0 to 14, while its concentration was enhanced significantly by PSPL consumption by 33.3% at day 14 vs. day 0. Further, the increase in erythrocyte GSH from day 0 to day 14 was significantly 72% larger as a result of PSPL intake than the LPD. Total antioxidant status was not significantly altered by the LPD and PSPL from day 0 to 14.

Plasma concentrations of MDA+HNE in the subjects consuming the LPD were significantly decreased by 4.0% after 1 wk (*p*  $\leq$ 0.05). The addition of PSPL into the LPD led to a significant decrease in MDA+HNE by 6.4% and 5.1% at day 7 and day 14 as compared to that at day 0. However, MDA+HNE concentration at day 14 in the

**Table 2.** The status of antioxidants in subjects<sup>†</sup>

	D0	D7	D14
Plasma $\alpha$ -tocopherol ( $\mu\text{mol/L}$ )			
LPD	6.2 $\pm$ 0.8	7.3 $\pm$ 0.8 <sup>a</sup>	5.2 $\pm$ 0.6 <sup>ab</sup>
PSPL	11.4 $\pm$ 1.4	9.8 $\pm$ 1.4 <sup>a</sup>	7.8 $\pm$ 0.8 <sup>ab</sup>
Erythrocyte GSH ( $\mu\text{mol/L}$ )			
LPD	18.5 $\pm$ 7.9	18.2 $\pm$ 7.0	23.5 $\pm$ 6.6
PSPL	25.9 $\pm$ 11.7	25.4 $\pm$ 10.4	34.5 $\pm$ 7.4 <sup>a</sup>
Plasma total antioxidant status ( $\text{mmol/L}$ )			
LPD	1.1 $\pm$ 0.1	1.1 $\pm$ 0.1	1.1 $\pm$ 0.2
PSPL	0.8 $\pm$ 0.1	0.9 $\pm$ 0.2	0.9 $\pm$ 0.2

<sup>†</sup>Results were expressed as mean  $\pm$  SD (n = 16).

<sup>a</sup>Means significantly differ as compared with D0, <sup>b</sup>Means significantly differ between D7 and D14, tested using mix model analysis ( $p \leq 0.05$ ).

**Table 3.** The status of oxidative stress<sup>†</sup>

	D0	D7	D14
MDA+4HNE ( $\mu\text{mol/L}$ )			
LPD	7.5 $\pm$ 0.1	7.2 $\pm$ 0.2 <sup>a</sup>	7.4 $\pm$ 0.3 <sup>b</sup>
PSPL	7.8 $\pm$ 0.3	7.3 $\pm$ 0.3 <sup>a</sup>	7.4 $\pm$ 0.2 <sup>a</sup>
8-OHdG ( $\text{ng/mL}$ )			
LPD	10.2 $\pm$ 5.8	9.9 $\pm$ 4.8	8.5 $\pm$ 3.7
PSPL	8.1 $\pm$ 5.6	5.1 $\pm$ 4.5 <sup>a</sup>	6.9 $\pm$ 3.1
LDL lag time (min)			
LPD	73.6 $\pm$ 13.9	74.7 $\pm$ 10.0	78.1 $\pm$ 14.0
PSPL	78.0 $\pm$ 12.9	87.1 $\pm$ 31.0	89.7 $\pm$ 16.5 <sup>a</sup>

<sup>†</sup>Results were expressed as mean  $\pm$  SD (n = 16).

<sup>a</sup>Means significantly differ as compared with D0, <sup>b</sup>Means significantly differ between D7 and D14, tested using mix model analysis ( $p \leq 0.05$ ).

PSPL group was not different from those at day 7. Urinary 8-OHdG, a systematic biomarker of DNA damage, was employed to reveal antioxidant action of constituents in PSPL (Table 3). The LPD did not alter urinary 8-OHdG value from day 0 to day 14 while PSPL consumption significantly decreased urinary 8-OHdG by 36.7% at day 7 as compared to that at day 0 ( $p \leq 0.05$ ). Further, the decrease in 8-OHdG by PSPL consumption from day 0 to day 7 was significantly larger than that by the LPD. The resistance of LDL against  $\text{Cu}^{2+}$ -induced oxidation significantly increased by 15% after consumption of PSPL for 2 weeks while no significant changes were found in the LPD group. (Table 3).

## DISCUSSION

Polyphenols in plant foods may contribute to decreased risk of chronic diseases because of an array of their putative mechanism of actions, i.e., antioxidation, anti-inflammation, and anti-proliferation.<sup>23</sup> Purple sweet potato leaves have been commonly consumed in Asian

countries. Since they are rich in various nutrients,<sup>24</sup> incorporation of PSPL into the daily diet may provide benefits in health promotion and prevention. In this study, we observed that the incorporation of 200 g/d PSPL into the LPD for 2 wks enhanced antioxidant defense and decreased oxidative stress in healthy subjects.

Since polyphenols are ubiquitous in plant foods, they are an integral part of our daily diets. It has been estimated that average polyphenol intake probably reaches 1 g/d in people who eat several serving of fruit and vegetables per day.<sup>25</sup> In this study, 902 mg polyphenols from 200 g PSPL added to the LPD provided a comparable quantity of polyphenol intake to the reported value. Consistent with no adverse effect reported in human studies, there were no apparent adverse effects after the consumption of 200 g/d PSPL for 2 wks, according to results of unaltered values of clinical biochemistries, serum creatinine, glutamic oxaloacetic transaminase (GOT), glutamic pyruvic acid transaminase (GPT), triglyceride, and cholesterol, as well as no reported gastrointestinal discomforts (diarrhea, abdominal pain or bloating).

Bioavailability of polyphenols has been documented commonly in humans.<sup>26</sup> Our results showed that 902 mg of polyphenols derived from 200 g/d PSPL consumption enhanced plasma total phenolic content and urinary phenolic excretion suggested bioavailability of polyphenols present in PSPL. However, it is a limitation of this study that we were unable to quantify the bioavailability of individual polyphenols due to unavailability of sophisticated instruments at the time of analysis. Nevertheless, this result was consistent to our unpublished observation that, following 2-hours food deprivation, concentrations of quercetin and caffeic acid in plasma were enhanced in rats fed a PSPL diet. On the other hand, a relatively smaller increase in plasma total phenolic content than changes in urinary phenolic excretion after consuming PSPL for 2 wk might be a result of a rapid clearance of polyphenols because half-lives of polyphenolic compounds are generally shorter than 12 h.<sup>27</sup>

Reactive oxidant species are believed to play an etiological role in pathogenesis of chronic diseases and aging.<sup>28</sup> Well documented antioxidant actions of polyphenols may partially account for decreased risk of oxidative stress-related chronic diseases.<sup>18, 29</sup> Because subjects in this study are healthy and may experience a low degree of oxidative stress, we did not observe the impact of PSPL polyphenols on plasma  $\alpha$ -tocopherol status, an outcome consistent to results of our rat study that flavonol quercetin could not prevent decreases in plasma and tissue  $\alpha$ -tocopherol in rats fed a vitamin E deplete diet.<sup>30</sup> On the contrary, PSPL polyphenols elevated glutathione status possibly through up-regulating glutathione synthesis and/or preventing glutathione use from consequences of their radical scavenging actions.<sup>31, 32</sup> However, exact mechanism(s) by which PSPL polyphenols modulate glutathione status in humans remain to be investigated. Various total antioxidant capacity assays have been commonly employed to reveal overall antioxidant efficacy of antioxidants in any given specimens. It was observed in a study by McAnlis et al. that 225 g fried onions rich in polyphenols increased plasma total antioxidant activity in humans,<sup>33</sup> while we observed that PSPL poly-

phenols didn't enhance plasma total antioxidant status. The direct radical quenching activity of polyphenols *in vivo* has been questioned because of their relatively low circulating concentrations as compared to plasma uric and ascorbic acid.

In addition to being annihilated by antioxidant defense, escaped reactive oxidant species can attack macromolecules and thereby cause pathogenesis of some diseases. For example, radical-mediated DNA damages are associated with carcinogenesis and oxidized LDL involve in atherogenesis. A growing body of evidence from *in vitro*, preclinical, and clinical studies suggested that polyphenols including flavonoids could protect LDL, DNA, protein, and lipid against oxidation.<sup>18, 21, 34, 35</sup> In this study, neither LPD nor 200 g/d PSPL altered the magnitudes of *in vivo* lipid peroxidation in apparently healthy individuals. In this study, the TBARS assay employed to assess MDA+4-HNE might be inadequate to reveal magnitude of *in vivo* lipid peroxidation because of appreciated interferences from bilirubin, sugar, and other factors. Interestingly, PSPL antioxidants decreased urinary excretion of DNA oxidation products temporarily in subjects in the PSPL group after the first week, but not after the second week. Similarly, polyphenols in onions and green tea diminished urinary 8-OHdG excretion in humans.<sup>39, 41</sup> Although these results of decreased urinary 8-OHdG excretion could be interpreted as a decrease in oxidant-induced DNA damage via antioxidative actions of polyphenols, it might simply suggest decreased capacities of DNA repairing mechanisms. In contrast to evidence from *in vitro* studies indicating antioxidant activity of polyphenols, our results suggested that antioxidant actions of PSPL polyphenols or other constituents might not be effective to diminish magnitudes of DNA and lipid oxidation in young healthy individuals when their endogenous antioxidant defense system is adequate to minimize *in vivo* oxidant-induced damages.<sup>35-37</sup> *In vitro* studies revealed, that polyphenols act as an oxygen radical scavenger as they enhance the resistance of LDL against Cu<sup>2+</sup>-induced oxidation<sup>38-43</sup>. Our study showed significantly increased LDL lag time after PSPL consumption for 2 weeks. Further, antioxidant actions of PSPL polyphenols that might be too subtle to be detected in a Cu<sup>2+</sup>-induced *ex vivo* oxidation model could be unmasked with *in vitro* addition of antioxidants.<sup>21</sup> The interactive effects among PSPL constituents, such as polyphenols and carotenoids, on antioxidant defense and oxidative stress remain to be investigated.

In conclusion, 902 mg of polyphenols in 200 g/d PSPL could be bioavailable and enhance glutathione status and decrease LDL oxidation. However, their antioxidant actions might not be sufficiently potent to modulate overall antioxidant defense in young healthy individuals.

#### ACKNOWLEDGEMENT

We gratefully thank the staff of the schools as well as the volunteers for their participation in the study. Support was provided by the grant 94TMU-TMUH-05 from the Taipei Medical University & Hospital and NSC 94-2320-B-038-045 from the National Science Council of Taiwan.

#### AUTHOR DISCLOSURES

Chiao-Ming Chen, Ya-Ling Lin, C-Y Oliver Chen, Ching-Yun Hsu, Ming-Jer Shieh and Jen-Fang Liu, no conflicts of interest.

#### REFERENCES

- Arts IC, Hollman PC, Feskens EJ, Bueno de Mesquita HB, Kromhout D. Catechin intake might explain the inverse relation between tea consumption and ischemic heart disease: the Zutphen Elderly Study. *Am J Clin Nutr.* 2001;74(2):227-32.
- Hertog MG, Feskens EJ, Hollman PC, Katan MB, Kromhout D. Dietary antioxidant flavonoids and risk of coronary heart disease: the Zutphen Elderly Study. *Lancet.* 1993;342(8878):1007-11.
- Huxley RR, Neil HA. The relation between dietary flavonol intake and coronary heart disease mortality: a meta-analysis of prospective cohort studies. *Eur J Clin Nutr.* 2003;57(8):904-8.
- Jiang R, Manson JE, Stampfer MJ, Liu S, Willett WC, Hu FB. Nut and peanut butter consumption and risk of type 2 diabetes in women. *JAMA.* 2002;288(20):2554-60.
- Tobias M, Turley M, Stefanogiannis N, Vander Hoorn S, Lawes C, Mhurchu CN, et al. Vegetable and fruit intake and mortality from chronic disease in New Zealand. *Australian and New Zealand Journal of Public Health.* 2006;30(1):26-31.
- USA C, WHO. Promoting fruit and vegetable consumption around the world. <http://www.who.int/dietphysicalactivity/fruit/en/index.html> 2007; Visit on June 18.
- Rice-Evans CA, Miller NJ, Paganga G. Structure-antioxidant activity relationships of flavonoids and phenolic acids. *Free Radic Biol Med.* 1996;20(7):933-56.
- Middleton E, Jr. Effect of plant flavonoids on immune and inflammatory cell function. *Adv Exp Med Biol.* 1998;439:175-82.
- Duthie SJ, Johnson W, Dobson VL. The effect of dietary flavonoids on DNA damage (strand breaks and oxidised pyrimidines) and growth in human cells. *Mutat Res.* 1997;390(1-2):141-51.
- Chu YF, Sun J, Wu X, Liu RH. Antioxidant and antiproliferative activities of common vegetables. *J Agric Food Chem.* 2002;50(23):6910-6.
- Mosha TC, Gaga HE. Nutritive value and effect of blanching on the trypsin and chymotrypsin inhibitor activities of selected leafy vegetables. *Plant Foods Hum Nutr.* 1999;54(3):271-83.
- Yan-Hwa Chu C-LCaH-FH. flavonoid content of several vegetables and their antioxidant activity. *J Sci Food Agric.* 2000;80:561-6.
- Islam MS, Yoshimoto M, Yahara S, Okuno S, Ishiguro K, Yamakawa O. Identification and characterization of foliar polyphenolic composition in sweetpotato (*Ipomoea batatas* L.) genotypes. *J Agric Food Chem.* 2002;50(13):3718-22.
- Philpott M, Gould KS, Lim C, Ferguson LR. *In situ* and *in vitro* antioxidant activity of sweetpotato anthocyanins. *J Agric Food Chem.* 2004;52(6):1511-3.
- Yoshimoto M, Yahara S, Okuno S, Islam MS, Ishiguro K, Yamakawa O. Antimutagenicity of mono-, di-, and tricaffeoylquinic acid derivatives isolated from sweetpotato (*Ipomoea batatas* L.) leaf. *Biosci Biotechnol Biochem.* 2002;66(11):2336-41.
- Chen CM, Li SC, Lin YL, Hsu CY, Shieh MJ, Liu JF. Consumption of purple sweet potato leaves modulates human immune response: T-lymphocyte functions, lytic activity of natural killer cell and antibody production. *World J Gastroenterol.* 2005;11(37):5777-81.

17. Chang WH, Chen CM, Hu SP, Kan NW, Chiu CC, Liu JF. Effect of purple sweet potato leaf consumption on the modulation of the antioxidative status in basketball players during training. *Asia Pac J Clin Nutr.* 2007;16(3):455-61.
18. Chen CY, Milbury PE, Collins FW, Blumberg JB. Avenanthramides are bioavailable and have antioxidant activity in humans after acute consumption of an enriched mixture from oats. *J Nutr.* 2007;137(6):1375-82.
19. Singleton VL RJ. Colorimetry of total phenolics with phosphomolybdic-phosphotungstic acid reagents. *Am J Enol Vitic.* 1965;16:144-58.
20. Milne DB, Botnen J. Retinol, alpha-tocopherol, lycopene, and alpha- and beta-carotene simultaneously determined in plasma by isocratic liquid chromatography. *Clin Chem.* 1986;32(5):874-6.
21. Chen CY, Milbury PE, Lapsley K, Blumberg JB. Flavonoids from almond skins are bioavailable and act synergistically with vitamins C and E to enhance hamster and human LDL resistance to oxidation. *J Nutr.* 2005;135(6):1366-73.
22. Department of Health EY, R.O.C., editor. *Nutrients database in Taiwan.* Taiwan; 1998.
23. Fang YZ, Yang S, Wu G, editors. *Free radicals, antioxidants, and nutrition.* Nutrition (Burbank, Los Angeles County, Calif; 2002 Oct.
24. Tang S-C. *Studies on the Antioxidative Capacity of Taiwan Indigenous Red Vegetables Extracts.* Master Thesis Taipei Medical University. 2000.
25. Manach C, Scalbert A, Morand C, Remesy C, Jimenez L. Polyphenols: food sources and bioavailability. *Am J Clin Nutr.* 2004;79(5):727-47.
26. Manach C, Williamson G, Morand C, Scalbert A, Remesy C. Bioavailability and bioefficacy of polyphenols in humans. I. Review of 97 bioavailability studies. *Am J Clin Nutr.* 2005;81(1 Suppl):230S-42S.
27. Scalbert A, Williamson G. Dietary intake and bioavailability of polyphenols. *J Nutr.* 2000;130(8S Suppl):2073S-85S.
28. Prentice RL, Willett WC, Greenwald P, Alberts D, Bernstein L, Boyd NF, et al. Nutrition and physical activity and chronic disease prevention: research strategies and recommendations. *J Natl Cancer Inst.* 2004;96(17):1276-87.
29. Luceri C, Caderni G, Sanna A, Dolara P. Red wine and black tea polyphenols modulate the expression of cyclooxygenase-2, inducible nitric oxide synthase and glutathione-related enzymes in azoxymethane-induced f344 rat colon tumors. *J Nutr.* 2002;132(6):1376-9.
30. Ameho CK, Chen CY, Smith D, Sánchez-Moreno C, Milbury PE, Blumberg JB. Antioxidant activity and metabolite profile of quercetin in vitamin E depleted rats. *J Nutr Biochem.* 2008 ;19(7):467-74
31. Rimbach G, Gohil K, Matsugo S, Moini H, Saliou C, Virgili F, et al. Induction of glutathione synthesis in human keratinocytes by Ginkgo biloba extract (EGb761). *Biofactors.* 2001;15(1):39-52.
32. Scharf G, Prustomersky S, Knasmuller S, Schulte-Hermann R, Huber WW. Enhancement of glutathione and g-glutamylcysteine synthetase, the rate limiting enzyme of glutathione synthesis, by chemoprotective plant-derived food and beverage components in the human hepatoma cell line HepG2. *Nutr Cancer.* 2003;45(1):74-83.
33. McAnlis GT, McEneny J, Pearce J, Young IS. Absorption and antioxidant effects of quercetin from onions, in man. *Eur J Clin Nutr.* 1999;53(2):92-6.
34. Boyle SP, Dobson VL, Duthie SJ, Kyle JA, Collins AR. Absorption and DNA protective effects of flavonoid glycosides from an onion meal. *Eur J Nutr.* 2000;39(5):213-23.
35. Kim HY, Kim OH, Sung MK. Effects of phenol-depleted and phenol-rich diets on blood markers of oxidative stress, and urinary excretion of quercetin and kaempferol in healthy volunteers. *J Am Coll Nutr.* 2003;22(3):217-23.
36. Moller P, Vogel U, Pedersen A, Dragsted LO, Sandstrom B, Loft S. No effect of 600 grams fruit and vegetables per day on oxidative DNA damage and repair in healthy non-smokers. *Cancer Epidemiol Biomarkers Prev.* 2003;12 (10): 1016-22.
37. Jacob RA, Aiello GM, Stephensen CB, Blumberg JB, Milbury PE, Wallock LM, et al. Moderate antioxidant supplementation has no effect on biomarkers of oxidant damage in healthy men with low fruit and vegetable intakes. *J Nutr.* 2003;133(3):740-3.
38. Pietta PG. Flavonoids as antioxidants. *J Nat Prod.* 2000; 63(7):1035-42.
39. Miura Y, Chiba T, Miura S, Tomita I, Umegaki K, Ikeda M, et al. Green tea polyphenols (flavan 3-ols) prevent oxidative modification of low density lipoproteins: an ex vivo study in humans. *J Nutr Biochem.* 2000;11(4):216-22.
40. Hirano R, Osakabe N, Iwamoto A, Matsumoto A, Natsume M, Takizawa T, et al. Antioxidant effects of polyphenols in chocolate on low-density lipoprotein both in vitro and ex vivo. *J Nutr Sci Vitaminol (Tokyo).* 2000;46(4):199-204.
41. Chen YY, Liu JF, Chen CM, Chao PY, Chang TJ. A study of the antioxidative and antimutagenic effects of *Houttuynia cordata* Thunb. using an oxidized frying oil-fed model. *J Nutr Sci Vitaminol (Tokyo).* 2003;49(5):327-33.
42. Mursu J, Voutilainen S, Nurmi T, Helleranta M, Rissanen TH, Nurmi A, et al. Polyphenol-rich phloem enhances the resistance of total serum lipids to oxidation in men. *J Agric Food Chem.* 2005;53(8):3017-22.
43. Chang S, Tan C, Frankel EN, Barrett DM. Low-density lipoprotein antioxidant activity of phenolic compounds and polyphenol oxidase activity in selected clingstone peach cultivars. *J Agric Food Chem.* 2000;48(2):147-51.

## Original Article

## Consumption of purple sweet potato leaves decreases lipid peroxidation and DNA damage in humans

Chiao-Ming Chen RD MSc<sup>1,2,3</sup>, Ya-Ling Lin RD MSc<sup>2</sup>, C-Y Oliver Chen PhD<sup>4</sup>  
Ching-Yun Hsu MSc<sup>5</sup>, Ming-Jer Shieh PhD<sup>2</sup>, Jen-Fang Liu RD PhD<sup>2</sup>

<sup>1</sup>Graduate Institute of Pharmacy, Taipei Medical University, Taipei, Taiwan

<sup>2</sup>School of Nutrition and Health Sciences, Taipei Medical University, Taipei, Taiwan

<sup>3</sup>Department of Dietetics, Taipei Medical University Hospital, Taipei, Taiwan

<sup>4</sup>Jean Mayer USDA Human Nutrition Research Center on Aging, Tufts University, Boston, MA, USA

<sup>5</sup>Chang-Gung Institute of Technology, Taoyuan, Taiwan

### 攝取紅甘藷葉飲食可以降低健康成年人體內的脂質過氧化作用及 DNA 的損傷

攝取富含多酚類食物，與減少慢性疾病的風險有關，其可能的生化機制包括增強抗氧化及抗發炎等。紅甘藷葉 (Purple sweet potato leaves, PSPL) 富含豐富的多酚類，是亞洲人經常食用的蔬菜。在這個交叉試驗中，提供 16 位健康受試者 (年齡 20-22 歲，7 男 9 女)，每日 200 公克紅甘藷葉 (包含 902 毫克總多酚類)，以探討其對人體抗氧化的影響。於一星期的適應期之後，將所有受試者隨機分成 2 組，實驗組每日攝取 200 公克紅甘藷葉，對照組則攝取低多酚類飲食，持續 2 週之後再進行 2 星期的排空期，之後 2 組飲食對調，再進行持續 2 週的試驗。分別收集受試者於每個試驗期的第 0、7、14 天的空腹血液及 24 小時尿液進行分析。結果顯示，攝取紅甘藷葉 14 天後，明顯增加受試者尿中多酚類的排泄，比第 0 天增加了 24.5%；但攝取低多酚類飲食 2 週之後，受試者的血漿及尿液中總多酚類的含量明顯減少，分別為 3.3 % 及 16.3 %。攝取紅甘藷葉 14 天後，低密度脂蛋白氧化遲滯時間 (Low-density lipoprotein lag time) 及紅血球中 glutathione 的濃度，分別比第 0 天時明顯提升了 15% 及 33.3%；而攝取低多酚類飲食組則沒有明顯變化。尿中 8-hydroxy-deoxyguanosine (8-OHdG) 的排泄方面，在攝取紅甘藷葉 7 天後，明顯比第 0 天減少了 36.7%；而攝取低多酚類飲食組則沒有明顯變化。綜合以上的結果，200 公克紅甘藷葉中的多酚類是可被人體吸收利用的，且可以降低體內脂質過氧化作用、DNA 的損傷及增加抗氧化防禦能力，以減少健康年輕人的氧化壓力。

**關鍵字:** 紅甘藷葉、多酚類、脂質過氧化作用、DNA 損傷