慢性腎臟病之營養治療與飲食技巧

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慢性腎臟病的營養治療

- 目標
  - 減少含氮廢物的堆積與尿毒性的代謝異常
  - 預防營養不良
  - 延緩腎臟疾病的進展
- 臨床應用
  - 在所有慢性腎臟病階段(Stages 1~5)維持足夠的熱量攝取
  - 遵從飲食蛋白質的限制並確保安全性
  - 評估、定期監測營養狀況
  - 積極營養諮詢

Handbook of nutrition and the kidney 2005

Association of level of GFR with nutritional status

- CKD病患發生營養不良的原因：
  - GFR < 60 mL/min/1.73 m^2, ↓食慾
    自發性 ↓蛋白質 and ↓熱量的攝取
  - 低蛋白飲食
  - 代謝性酸中毒
  - 慢性發炎-
    - Patients with elevated biomarkers of inflammation, such as CRP,tend to have a higher risk of uremic malnutrition and progressive atherosclerotic vascular disease.
    - In the literature a prevalence of malnutrition between 10 and 70% has been reported. (40-50% of all patients with ESRD are malnourished) Blood Purif 2008;26:49–53
  - 胰島素阻抗
- 當 GFR < 60 mL/min/1.73 m^2, 應評估營養狀況

腎臟專科營養師基礎訓練班, 2006

低蛋白質飲食

- ↓ renal blood flow
- ↓T cell ↓間質性腎炎
- ↓ acid ↓NH_4^+

  ↓NH_4^+ 可活化補體系統，引起腎發炎
- ↓磷攝取量
- 動物實驗顯示：低蛋白和/或低磷飲食可以延緩或預防腎衰竭的進展，脂肪的種類也會影響腎衰竭的進展

腎臟專科營養師基礎訓練班, 2006
臨床營養學-膳食療養2008

Restricted Protein Diet Is Associated With Decrease in Proteinuria: Consequences on the Progression of Renal Failure

Objective: Reduction of proteinuria is associated with a slower progression of renal failure. We questioned whether the change in proteinuria in response to a supplemented very low protein diet (SVLPD), which is known to reduce proteinuria, could function as a marker of the potential renoprotective effect of an SVLPD.

Design and Patients: In the 220 consecutive patients of our previously published cohort, the glomerular filtration rate (GFR) was assessed every 3 months using the ^{51}Cr-EDTA method. Seventy-eight patients (mean age 52 ± 17 years, body mass index 23 ± 3 kg/m^2, GFR 15 ± 6 mL/min) exhibited a proteinuria more than 1 g per day at the start of the regimen. Mean protein intake assessed by urinary nitrogen appearance was 0.42 ± 0.24 g/kg per day at 4 months. The median follow-up was 24 months.

Results: Proteinuria decreased significantly after patients were treated with an SVLPD. The maximum mean percent reduction was attained at 3 months (47% ± 27%), was not influenced by the levels of baseline proteinuria, and was similar in patients receiving or not receiving angiotensin-converting enzyme inhibition at the start of the study. The percent reduction and the residual proteinuria at 3 months predicted the rate of the later GFR decline. GFR decline was significantly lower in patients whose reduction in proteinuria at 3 months was higher than 50% (0.42 ± 0.37 mL/min/mo vs. 0.10 ± 0.15 mL/min/mo and 1.0 ± 0.6 mL/min/mo vs. 0.15 ± 0.19 mL/min/mo, $P < .001$ in patients with proteinuria higher or lesser than 3 g/d at start, respectively).

Conclusion: These results do not differ from those reported with therapies antagonizing angiotensin II formation and/or activity aiming at reducing proteinuria in chronic renal diseases.

Journal of Renal Nutrition, Vol 17, No 4 (July), 2007: pp 250–257

Dietary protein and the progression of CKD

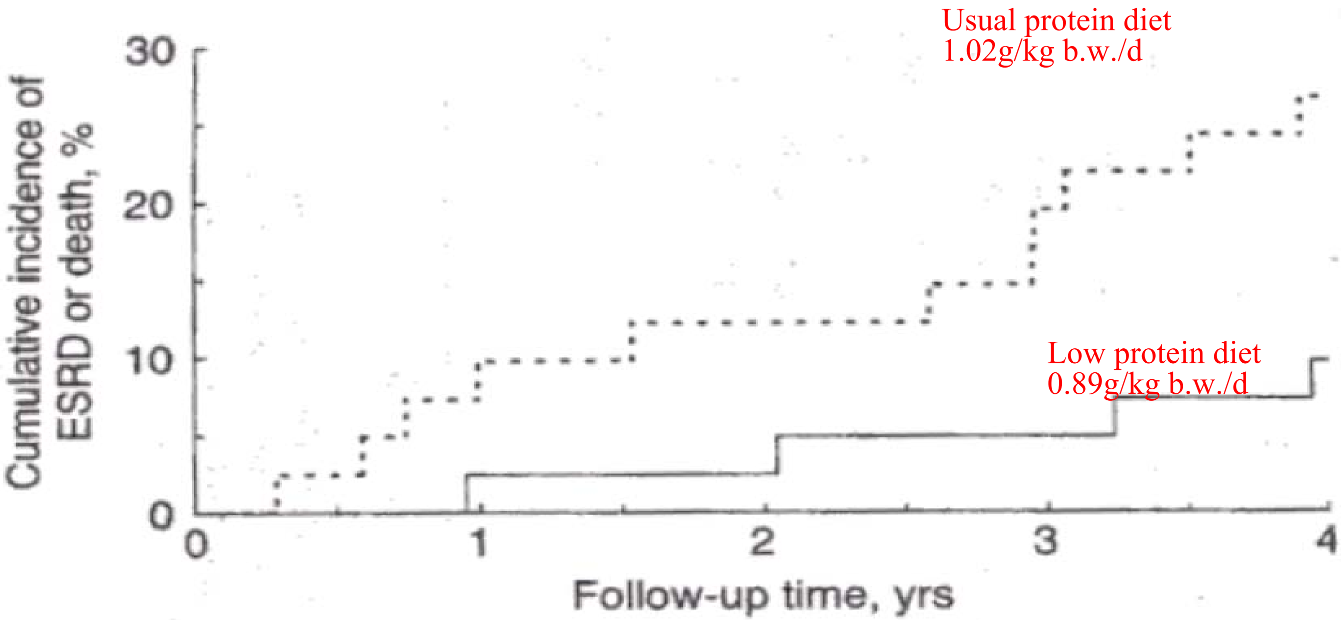


Figure 9-4. Cumulative incidence of end-stage renal disease or death in patients with type 1 diabetes receiving usual protein diet (1.02 g/kg b.w./d, dashed line) or low protein diet (0.89 g/kg b.w./d, solid line) during 4 years; $p = 0.042$, log rank [From Hansen HP, Tauber–Lassen E, Jensen BR, et al. Effect of dietary protein restriction on prognosis in patients with diabetic nephropathy. *Kidney Int* 2002;62(1):220–228, with permission.]

Inadequate Energy and Excess Protein Intakes May Be Associated With Worsening Renal Function in Chronic Kidney Disease

Objectives: Dietary energy and protein play important roles in chronic kidney disease (CKD). This study investigates the relationship between energy/protein intake status and renal function in CKD.

Design and Study Population: This cross-sectional study included 599 adult patients diagnosed with stage 3 to 5 CKD in nephrology and nutrition outpatient clinics in Taiwan.

Main Outcome Measure: Energy and protein intakes were assessed using 24-h dietary recall. We recorded recommended calorie/protein amounts and renal function indices, glomerular filtration rate (GFR), creatinine, and blood urea nitrogen (BUN). Patients were categorized into three intake calorie/protein groups by a ratio of actual intake vs. recommended intake. High intake was defined as a ratio of actual intake/recommended intake ≧110%, moderate intake as ≧90% to <110%, and low intake as <90%. Data were analyzed by paired *t* test, one-way analysis of variance, least significant differences, and multiple linear regression.

Results: The energy and protein intakes in CKD patients were significantly higher and lower than recommended levels ($P < .001$). Low energy intake was significantly related to worsening GFR at increments of −4.41 mL/min/1.73 m^2, compared with moderate and high energy intake ($P = .008$); high protein intake was also associated with worsening GFR at increments of −3.50 mL/min/1.73m^2, compared with moderate and low protein intake ($P < .001$). Low energy intake and high protein intake were significantly positively correlated with elevations in creatinine and BUN.

Conclusion: Lower energy and higher protein intakes than recommended may be associated with deteriorating renal function.

Journal of Renal Nutrition, Vol 18, No 2 (March), 2008: pp 187–194

Inadequate Energy and Excess Protein Intakes May Be Associated With Worsening Renal Function in Chronic Kidney Disease

Table 3. Renal Function Status Among Chronic Kidney Disease Patients (n = 599) by Different Levels of Energy and Protein Intake*†

	N (%)	GFR (mL/min/1.73 m^2)	*P*	Creatinine (mg/dL)	*P*	BUN (mg/dL)	*P*
Total calories							
Low	339 (56.6)	19.1 ± 12.1[a]	<.001	4.8 ± 2.9[b]	<.001	54.8 ± 27.8[b]	<.001
Moderate	161 (26.9)	24.7 ± 13.8[b]		3.8 ± 2.5[a]		45.8 ± 26.6[a]	
High	99 (16.5)	24.4 ± 13.4[b]		3.5 ± 2.1[a]		46.5 ± 25.7[a]	
Protein							
Low	40 (6.7)	24.0 ± 14.8[c,d]	.019	3.8 ± 2.7[c,d]	<.001	42.7 ± 22.0[c]	<.001
Moderate	76 (12.7)	24.8 ± 13.6[d]		3.1 ± 1.7[c]		40.3 ± 20.5[c]	
High	483 (80.6)	20.7 ± 12.7[c]		4.6 ± 2.9[d]		53.4 ± 28.3[d]	

BUN, blood urea nitrogen; GFR, glomerular filtration rate, using the equation of Cockcroft and Gault.

*Data are expressed as mean ± SD. Different superscripts ([a, b, c]) indicate significant differences from each other, using least significant differences tests. $P < .05$ was considered statistically different.

†For intake status of dietary energy and protein, patients were categorized into high, moderate, and low intake groups. High intake was defined as the ratio of actual intake/recommended intake (by dietitians) ≥110%; those <90% were defined as having low caloric intake. Those ratios within ranges ≥90% and <110% of recommended levels were considered to be the moderate intake group.

Journal of Renal Nutrition, Vol 18, No 2 (March), 2008: pp 187–194

Protein Restriction and Body Composition in Renal Disease

Objective: To study the effect of low-protein diet (LPD) on body composition (BC).

Study Design: A systematic review of the literature investigating BC during LPD treatment using total body potassium, dual energy X-ray absorptiometry, bioelectrical impedance analysis, and anthropometry.

Patients: Studies reporting data of patients treated with LPD 0.3–0.75 g/kg/day and a renal function of glomerular filtration rate (GFR) ≤20 mL/min, creatinine clearance ≤25 mL/min, on serum creatinine ≥500 μmol/L were included in the review. Fourteen studies with a total number of 666 subjects were found eligible.

Results: All studies except two concluded that treatment with LPD does not affect BC negatively. However, LPD should not be introduced in patients with a complicating disease, e.g., acidosis, septicaemia, and surgical treatment; neither should it be continued in patients who are unable to adhere to a diet prescription. Furthermore, LPD should be introduced with great caution in patients with an expected time to dialysis of ≤4 months due to an initial reduction of body weight and/or fat-free mass. Monitoring of treatment with LPD must be emphasized, including BC measurements and evaluation of protein and energy intake. These conclusions do not apply to patients with diabetes mellitus, because this diagnosis was excluded in a majority of reviewed studies.

Conclusion: There is no strong evidence that LPD impairs BC in patients with GFR ≤20 mL/min.

Journal of Renal Nutrition, Vol 18, No 2 (March), 2008: pp 167–186

Diabetic kidney disease(DKD)--2007K/DOQI

American Journal of Kidney Diseases, Vol 49, No 2, Suppl 2 (February), 2007: pp S95-S107

Recommendation 4: Behavioral self-management in diabetes and CKD --2007K/DOQI

- Behavioral self-management in diabetes and CKD is particularly challenging because of the intensive nature of the diabetes regimen.
- Education alone is not sufficient to promote and sustain healthy behavior change, particularly with such a complex regimen.
- 4.1 Self-management strategies should be key components of a multifaceted treatment plan with attention to multiple behaviors: (C)
  - Monitoring and treatment of glycemia,
  - Blood pressure,
  - Nutrition,
  - Smoking cessation,
  - Exercise, and
  - Adherence to medicines.

Guideline 5: Nutritional Management in Diabetes and Chronic Kidney Disease -- 2007K/DOQI

- Management of diabetes and CKD should include nutritional intervention. Dietary modifications may reduce progression of CKD.
- 5.1 Target dietary protein intake for people with diabetes and CKD stages 1-4 should be the RDA of 0.8 g/kg body weight per day. (B)

American Journal of Kidney Diseases, Vol 49, No 2, Suppl 2 (February), 2007: pp S95-S107

- RATIONALE
  - A dietary protein intake of 0.8 g/kg body weight per day, the RDA for this macronutrient, is a level that has been achieved in studies of diabetes and CKD. Reduction in albuminuria and stabilization of kidney function have been reported with dietary protein intake at the RDA level. Nutrition surveys indicate that most people eat in excess of the RDA for dietary protein. (Moderate)
  - If dietary protein intake is limited, an increase in carbohydrates and/or fats is required for adequate caloric intake. Increasing intake of *omega-3 and monounsaturated fats* may confer benefits on CKD. (Weak/Opinion)
  - People with diabetes and CKD should receive intervention from a specialty-trained registered dietitian that includes individualized management of multiple nutritional aspects. (Moderate)

American Journal of Kidney Diseases, Vol 49, No 2, Suppl 2 (February), 2007: pp S95-S107

Protein in diabetes management--ADA

- NUTRITION RECOMMENDATIONS FOR CONTROLLING DIABETES COMPLICATIONS
  - Reduction of protein intake to 0.8 –1.0 g / kg/body wt/ day−1 in individuals with diabetes and the earlier stages of chronic kidney disease (CKD) and to 0.8 g/kg body wt/ day in the later stages of CKD may improve measures of renal function (urine albumin excretion rate, glomerular filtration rate) and is recommended. (B)
  - MNT that favorably affects cardiovascular risk factors may also have a favorable effect on microvascular complications such as retinopathy and nephropathy. (C)

DIABETES CARE, VOLUME 31, SUPPLEMENT 1, JANUARY 2008 S66

Protein in diabetes management--ADA

- NUTRITION RECOMMENDATIONS FOR CONTROLLING DIABETES COMPLICATIONS
  - Progression of diabetes complications may be modified by improving glycemic control, lowering blood pressure, and, potentially, reducing protein intake. Normal protein intake (15–20% of energy) does not appear to be associated with risk of developing diabetic nephropathy, but the long-term effect on development of nephropathy of dietary protein intake ┐20% of energy has not been determined.
  - In several studies of subjects with diabetes and microalbuminuria, urinary albumin excretion rate and decline in glomerular filtration were favorably influenced by reduction of protein intake to 0.8 –1.0 g/kg body wt/day⁻.
  - In individuals with diabetes and macroalbuminuria, reducing protein from all sources to 0.8 g/kg body wt/day⁻ has been associated with slowing the decline in renal function; however, such reductions in protein need to maintain good nutritional status in patients with chronic renal failure.

DIABETES CARE, VOLUME 31, SUPPLEMENT 1, JANUARY 2008 S66

Types of dietary protein and the progression of CKD

- withdraw red meat?
- soy protein?

Withdrawal of red meat from the usual diet reduces albuminuria and improves serum fatty acid profile in type 2 diabetes patients with macroalbuminuria[1–3]

TABLE 1

Daily dietary intake during the usual diet, the chicken diet, and the low-protein diet, according to weighed diet records in type 2 diabetes patients with macroalbuminuria[1]

	Usual diet	Chicken diet	Low-protein diet	P^2
Energy (kcal)	1901 ± 480[a]	1870 ± 452[a]	1634 ± 451[b]	< 0.001
Carbohydrate (% of energy)	46.9 ± 6.7[a]	50.0 ± 7.0[a]	58.7 ± 6.8[b]	< 0.001
Protein (% of energy)	21.9 ± 3.4[a]	21.2 ± 3.9[a]	11.6 ± 1.5[b]	< 0.001
Total lipids (% of energy)	30.8 ± 6.3	28.9 ± 5.5	29.5 ± 6.8	0.451
Fatty acids				
Saturated (% of energy)	9.1 ± 2.5	7.8 ± 2.0	8.8 ± 2.1	0.103
Monounsaturated (% of energy)	−10.2 ± 2.3	9.4 ± 2.1	9.6 ± 3.2	0.484
Polyunsaturated (% of energy)	8.1 ± 2.6[a]	9.3 ± 2.5[b]	9.5 ± 2.6[b]	0.022
PUFA:SFA	0.96 ± 0.44[a]	1.24 ± 0.32[b]	1.13 ± 0.43[a,b]	0.039
Cholesterol (mg)	236 ± 72[a]	250 ± 86[a]	59 ± 35[b]	< 0.001
Total fiber (g)	20.0 ± 7.5[a]	22.8 ± 9.3[a]	27.0 ± 8.1[b]	< 0.001
Iron (mg)	15.6 ± 4.3[a]	13.4 ± 3.6[b]	12.3 ± 4.0[b]	< 0.001
Phosphorus (mg)	1358 ± 348[a]	1288 ± 299[a]	963 ± 286[b]	< 0.001
Zinc (mg)	15.9 ± 5.1[a]	10.7 ± 2.5[b]	5.5 ± 2.0[c]	< 0.001
Potassium (mg)	2873 ± 962	2780 ± 886	2788 ± 986	0.685
Calcium (mg)	807 ± 239	813 ± 223	805 ± 294	0.984

[1] All values are $\bar{x}$ ± SD; $n = 17$. SFA, saturated fatty acid; PUFA, polyunsaturated fatty acid. Values in a row with different superscript letters are significantly different, $P < 0.05$ (Bonferroni adjustment test for multiple comparisons).

[2] Repeated-measures ANOVA.

Withdrawal of red meat from the usual diet reduces albuminuria and improves serum fatty acid profile in type 2 diabetes patients with macroalbuminuria[1–3]

TABLE 2

Renal function and serum lipid profile after test diets in type 2 diabetes patients with macroalbuminuria[1]

	Usual diet	Chicken diet	Low-protein diet	P^2
UAER (μg/min)[3]	312.8[a] (223.7–1223.7)[4]	269.4[b] (111–1128)	229.3[b] (76.6–999.3)	< 0.001
GFR ($mL \cdot min^{-1} \cdot 1.73\ m^{-2}$)	81.8 ± 22.2[5]	83.3 ± 26.1	81.9 ± 25.3	0.860
Total cholesterol (mmol/L)	5.37 ± 1.18	5.08 ± 0.96	5.06 ± 0.91	0.069
HDL cholesterol (mmol/L)	1.14 ± 0.26	1.14 ± 0.23	1.14 ± 0.21	0.989
LDL cholesterol (mmol/L)	3.89 ± 0.99	3.64 ± 0.91	3.55 ± 0.84	0.123
Non-HDL cholesterol (mmol/L)[3]	4.23 ± 1.06[a]	3.92 ± 0.99[b]	3.92 ± 0.93[b]	0.042
Triacylglycerols (mmol/L)[3]	1.46 (0.60–4.73)[a]	1.22 (0.50–3.88)[b]	1.51 (0.62–7.35)[a]	0.012

[1] $n = 17$. UAER, urinary albumin excretion rate; GFR, glomerular filtration rate. Values in a row with different superscript letters are significantly different, $P < 0.05$ (Bonferroni adjustment test for multiple comparisons).

[2] Repeated-measures ANOVA.

[3] Tested with log-transformed values.

[4] Median; range in parentheses (all such values) for variables with non-Gaussian distribution.

[5] $\bar{x}$ ± SD (all such values).

Am J Clin Nutr 2006;83:1032–8.

Withdrawal of red meat from the usual diet reduces albuminuria and improves serum fatty acid profile in type 2 diabetes patients with macroalbuminuria[1–3]

TABLE 4

Glycemic control, blood pressure measurements, and nutritional indexes after diets[1]

	Usual diet	Chicken diet	Low-protein diet	P^2
Fasting plasma glucose (mmol/L)	6.33 ± 2.31	6.66 ± 1.93	5.89 ± 2.51	0.467
Fructosamine (mmol/L)	3.7 ± 0.7	3.4 ± 0.6	3.6 ± 0.7	0.119
Mean blood pressure (mm Hg)	86.9 ± 10.1	87.3 ± 11.2	88.3 ± 9.5	0.726
BMI (kg/m^2)	26.1 ± 2.5[a]	26.0 ± 2.6[a]	25.7 ± 2.7[b]	0.007
Triceps skinfold thickness (mm)	16.4 ± 8.7	16.9 ± 2.8	15.4 ± 7.9	0.100
Midupper arm muscle area (mm^2)	56.2 ± 9.7	56.8 ± 8.3	54.7 ± 8.3	0.315
Waist circumference (cm)	97.4 ± 6.7	97.4 ± 7.7	96.9 ± 6.7	0.504
Waist-to-hip ratio	1.00 ± 0.05	0.99 ± 0.05	1.00 ± 0.06	0.725

[1] All values are $\bar{x}$ ± SD; $n = 17$. Values in a row with different superscript letters are significantly different, $P < 0.05$ (Bonferroni adjustment test for multiple comparisons).

[2] Repeated-measures ANOVA.

Comparison of a Vegetable-Based (Soya) and an Animal-Based Low-Protein Diet in Predialysis Chronic Renal Failure Patients

- 15 patients with CRF(GFR 15~50 ml/min/1.73m^2); 9 位完成試驗
- Dietary components
- prescribed :

Table 1. Makeup of VPD and APD

Kilocalories/kg/day	32
Protein, g/kg/day	0.75
Phosphorus mg/kg/day	11
Calcium, mg/day	1,000
Cholesterol, mg/day	280
% of total calories	
Carbohydrates	60
Fat	30
Protein	10

- The study was a randomized crossover design. The patients were randomly assigned to either VPD or APD. They stayed on one diet for 6 months and then switched to the other diet for a second 6-month period.

Nephron 1998;79:173–180

Comparison of a Vegetable-Based (Soya) and an Animal-Based Low-Protein Diet in Predialysis Chronic Renal Failure Patients

Table 2. Constituents of prestudy diet, VPD, and APD

Daily intake	Prestudy diet	VPD	APD
Kilocalories/day	1,822±152	2,049±88*,#	1,838±78
Kilocalories/kg/day	27.7±0.6	31.6±0.8#	28.8±1.3
Protein			
g/day	62.2±8.7	48.9±2.9*,#	52.5±2.3#
g/kg/day	0.93±0.13	0.71±0.05*,#	0.85±0.04
% calories from			
Carbohydrates	55.9±1.9	54.5±1.3	55.5±1.5
Fat	30.2±2.0	35.8±0.9*,#	33.3±1.3#
Proteins	13.5±1.9	9.45±0.6*,#	11.13±0.7
Cholesterol, mg/day	223±32	236±12	235±22
Calcium, mg/day	623±30	525±29#	647±37
Phosphate, mg/day	871±51	789±52*,#	894±49
Iron, mg/day	11.3±0.9	13.2±0.3*,#	10.5±0.3
SFA, % of total calories	6.9±0.3	6.0±0.3*,#	7.3±0.2
MUFA, % of total calories	11.4±1.1	11.1±0.6*	12.3±0.5#
PUFA, % of total calories	11.7±1.0	18.9±0.5*,#	13.4±0.6#

SFA = Saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.

* $p < 0.05$ (VPD compared to APD); # $p < 0.05$ (VPD or APD compared to prestudy diet).

Dietary compliance: VPD better than APD (higher calorie intake, lower protein and phosphate intakes)

Nephron 1998;79:173–180

Comparison of a Vegetable-Based (Soya) and an Animal-Based Low-Protein Diet in Predialysis Chronic Renal Failure Patients

Table 4. Comparison of the renal function in the three diet groups as measured by ^{51}Cr-EDTA, C_{Cr}, and serum creatinine at the end of 6 months of treatment with VPD and APD

	Prestudy diet	VPD	APD
^{51}Cr-EDTA-clearance, ml/min/1.73 m^2	28.81±3.3	28.11±3.4	29.56±3.8
C_{Cr}, ml/min/1.73 m^2	30.5±3.6	25.09±2.9	28.62±4.0
Serum creatinine, mg%	3.11±0.2	3.2±0.32	3.2±0.26

There were no significant differences between the groups.

- Deterioration in nutritional status: no (VPD better)
- Dietary compliance: VPD better than APD (higher calorie intake , lower protein and phosphate intakes)

Nephron 1998;79:173–180

Positive effect of dietary soy in ESRD patients with systemic inflammation—correlation between blood levels of the soy isoflavones and the acute-phase reactants

Methods. End-stage renal disease patients on chronic HD, with elevated CRP (>10.0 mg/l) were enrolled in this pilot study. The subjects were double-blind randomly distributed with 2:1 ratio to receive isoflavone-containing soy-based nutritional supplements (soy group) or isoflavone-free milk protein (control group) for 8 weeks. Serum isoflavone, inflammatory markers and nutrition markers were assessed at baseline and at the end of the treatment.
Results. Thirty-two subjects were enrolled. Fifteen subjects in the soy group and 10 in the control group completed the study; five dropouts were due to acute illness and two due to food intolerance. After intervention, blood isoflavone levels were 5- to 10-fold higher in the soy group than in the control group [e.g. median genistein (25–75th percentile): 337.9 (175.5–1007) nM in the soy group *vs* 41.4 (22.9–100.4) nM in the control group; $P < 0.001$]. However, the isoflavone levels ranged widely in the soy group (e.g. genistein: 33–1868 nM) and, depending on the individual compound, four to seven subjects had end-of-treatment levels that were not different from baseline. Variation from baseline of the individual serum isoflavone levels (Δ-isoflavone) and CRP displayed a strong inverse correlation in the soy group ($R = -0.599$, $P < 0.02$). In addition, Δ-isoflavone correlated positively with the variation of albumin ($R = 0.522$, $P = 0.05$) and insulin-like growth factor-1 ($R = 0.518$, $P < 0.05$). Group levels of CRP were not statistically different after intervention although a trend towards lower levels was noted in the soy group [18.2 (12.7–29.1) mg/l at baseline *vs* 9.7 (5.2–20.7) mg/l at week 8; NS] but not in the control group [20.6 (9.2–38.5) *vs* 17.6 (9.1–40.7) mg/l].
Conclusion. These data suggest the possibility of beneficial effects of isoflavone-rich soy foods on the inflammatory and nutritional status of HD patients with underlying systemic inflammation.

Nephrol Dial Transplant (2006) 21: 2239–2246

Positive effect of dietary soy in ESRD patients with systemic inflammation—correlation between blood levels of the soy isoflavones and the acute-phase reactants

Table 3. Dietary intake (A) and blood concentration of isoflavones, inflammation markers and nutrition markers (B) at baseline and at the end of 8 week intake of soy-based or milk-based supplements

	Control group (10)		Soy group (15)	
	Week 0	Week 8	Week 0	Week 8
(A)				
Protein intake (g/kg/day)[a]	0.90 ± 0.16	0.96 ± 0.16	0.81 ± 0.11	0.85 ± 0.14
Energy intake (kcal/kg/day)[a]	26.9 ± 5.3	25.1 ± 4.1	24.8 ± 2.8	28.5 ± 4.4
Normalized protein catabolic rate (g/kg/day)[a]	0.95 ± 0.08	1.04 ± 0.1	0.99 ± 0.05	1.03 ± 0.07
(B)				
Genistein (nM)[b]	64.6* (17.3–150.7)	41.4* (22.9–100.4)	40.6* (17.4–72.5)	337.9** (175.5–1007.0)
Daidzein (nM)[b]	135.5* (104.9–297.9)	146.2* (68.1–189.7)	127.9* (77.0–202.6)	1038.5** (563.6–1829.8)
Total Isoflavones (nM)[b]	300.6* (185.6–415.3)	226.2* (116.7–358.0)	189.6* (129.6–399.5)	2094.0** (1379.9–3656.6)
CRP (mg/l)[b]	20.6 (9.2–38.5)	17.5 (9.1–40.7)	18.2 (12.7–29.1)	9.7 (5.2–20.7)
IL-6, unstim. (pg/ml)[b]	22.2 (14.2–48.9)	32.7 (14.5–86.4)	14.0 (6.4–23.2)	10.3 (8.0–14.0)
IL-6, with LPS (pg/ml)[b]	2720.8 (890.6–6924.7)	2076.3 (524.9–3268.2)	2314.3 (1108.9–5985.7)	3249.0 (1288.5–4024.4)
TNF-α, unstim. (pg/ml)[b]	12.5 (7.1–23.5)	9.0 (7.3–17.6)	7.6 (6.5–13.7)	7.5 (6.6–8.1)
TNF-α, with LPS (pg/ml)[b]	726.9 (312.5–1540.5)	504.6 (257.1–975.3)	598.6 (302.8–1582.6)	790.9 (445.4–1120.1)
Pre-Albumin (mg/dl)[b]	25.2 ± 2.9	25.7 ± 3.6	31.2 ± 1.9	32.6 ± 1.9
IGF-1 (ng/dl)[b]	87.2 ± 10.7	88.2 ± 15.1	91.3 ± 8.2	93.6 ± 9.1
Albumin (g/dl)[b]	3.77 ± 0.16	3.62 ± 0.17	3.84 ± 0.07	3.81 ± 0.07

[a]Variable presented as mean ± SEM; [b]variable presented as median (25–75th percentile).
*,**Different symbols within each variable indicate significant difference ($P < 0.001$; ANOVA, *post hoc* Bonferroni's analysis).

Nephrol Dial Transplant (2006) 21: 2239–2246

K/DOQI Clinical Practice Guidelines for Nutrition in Chronic Renal Failure (CRF), Guidelines 24 and 25 (2000)

- **Guideline 24.** 慢性腎衰竭(GFR <25 mL/min.)非透析病患的飲食蛋白質攝取建議量，應考慮低蛋白質飲食，每天每公斤體重0.6公克的蛋白質(0.6 g protein/kg/day). 對於無法接受此種飲食或無法維持足夠熱量攝取之病患，應予調整至每天每公斤體重0.75公克的蛋白質(0.75 g protein/kg/day). (Evidence and Opinion).”

- **Guideline 25.** 慢性腎衰竭(GFR <25 mL/min.)非透析病患的飲食熱量攝取建議量，60歲以下應予以每天每公斤體重35(?)大卡的熱量；60歲或60歲以上應予以每天每公斤體重30~35(?)大卡的熱量. (Evidence and Opinion).”

American Journal of Kidney Diseases,Vol 35, No 6, Suppl 2 (June), 2000: pp S17-S104

慢性腎衰竭的營養治療

- **CKD Stages 1 ~ 3**
  - 對於CKD Stages 1 ~ 3的病患，每日蛋白質建議攝取量為 0.75 g/kg/d ，避免營養不良，個別化的飲食指導.
  - 對於每日蛋白質建議攝取量少於0.75 g/kg/d 之病患應密切監測營養狀況，避免營養不良.
- **CKD Stage 4 ~ 5**
  - 對於CKD Stage 4 ~ 5 (GFR <25 mL/min)的病患考慮低蛋白飲食 (0.6 g/kg/d)時 ；最重要的是維持病患良好的營養狀況並定期由營養師監測與評估.

Am J Kidney Dis 2002; 39:S1.

Nutrition, inflammation and chronic kidney disease

- Chronic inflammation as a catabolic stimulus in advanced chronic kisney disease

- Treatment options for protein-energy wasting and chronic inflammation in advanced kidney disease
  - Nutritional interventions
  - Antiinflammatory interventions

T. Alp Ikizler **Current Opinion in Nephrology and Hypertension** 2008, 17:162–167

Body mass index and fat mass are the primary correlates of insulin resistance in nondiabetic stage 3–4 chronic kidney disease patients[1–3]

ABSTRACT

Background: Insulin resistance has been noted in patients with chronic kidney disease (CKD). The determinants of insulin resistance have not been well-studied in CKD patients.

Objective: The objective of this study was to examine the degree and determinants of insulin resistance in persons without diabetes but with stage 3–4 CKD.

Design: Demographic characteristics, metabolic hormones, and inflammatory markers were measured in 95 nonobese stage 3–4 CKD patients without prior diagnosis of diabetes mellitus and 36 control subjects without CKD. The estimated glomerular filtration rate (eGFR) was measured by using the Modification of Diet in Renal Disease study equation. Insulin resistance was measured with the use of the homeostasis model assessment of insulin resistance (HOMA-IR).

Results: After age and sex adjustments, HOMA-IR scores were significantly and positively correlated with body mass index (BMI) and percentage body fat. After control for age, race, adiponectin concentrations, sex, and eGFR in a multivariate regression model, BMI remained as the only significant predictor of insulin resistance (standardized regression coefficient = 0.55; $P < 0.001$). When substituted for BMI, percentage body fat also was an independent predictor of insulin resistance. The prevalence of abnormal HOMA did not differ significantly between CKD patients (98%) and BMI-matched control subjects (94%).

Conclusion: Whereas insulin resistance is highly prevalent in stage 3–4 CKD, the primary determinant of insulin resistance in this population is BMI, specifically, fat mass. *Am J Clin Nutr* 2007; 86:1642–8.

CKD3~4 -- 95人
Control -- 36人
BS、insulin、HOMA-IR 、cytokines conc. 、CRP 、adiponectin、resistin、BIA(fat%)

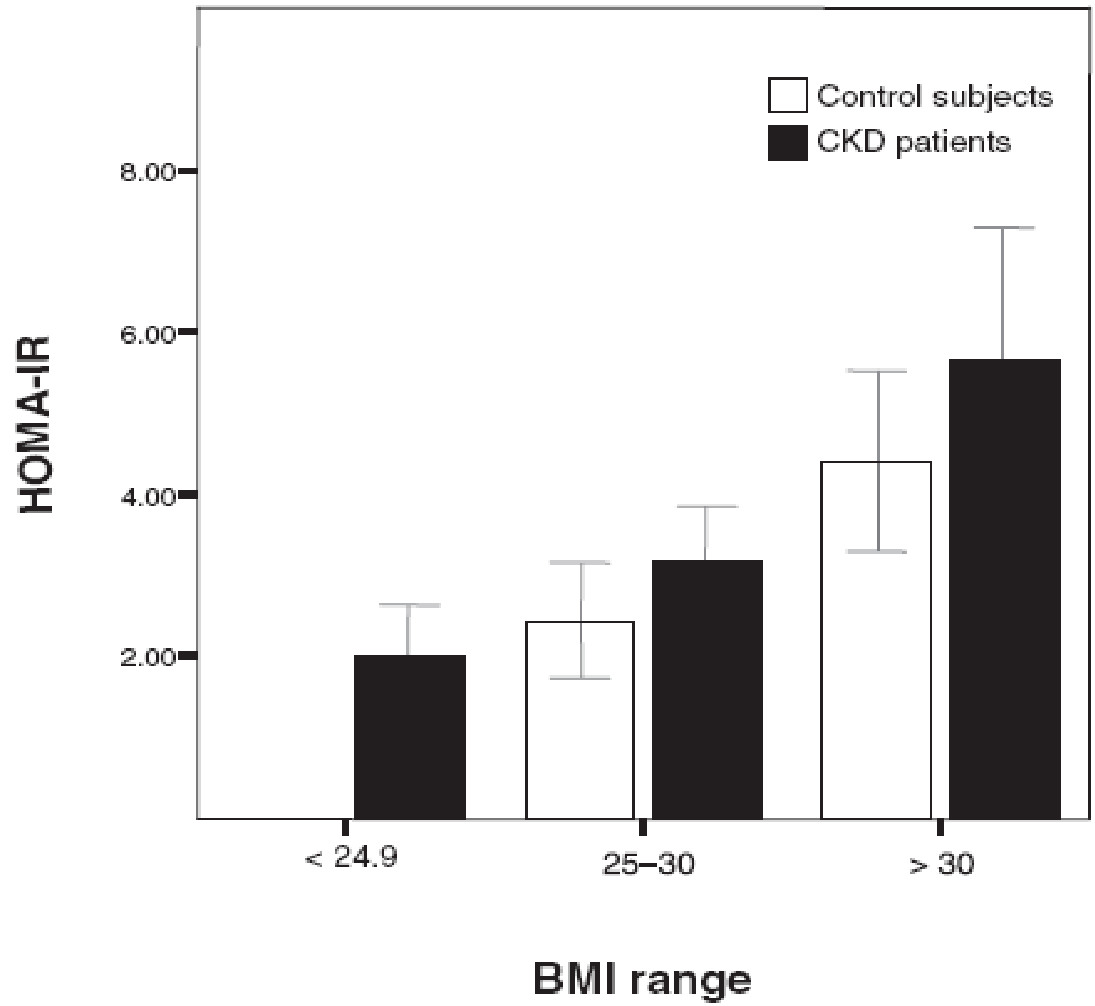


FIGURE 1. Homeostasis model assessment of insulin resistance (HOMA-IR) versus BMI ranges in patients with chronic kidney disease (CKD) and control subjects. Mean HOMA-IR scores in CKD patients and control subjects by BMI ranges of normal (<24.9), overweight (25–30), and obese (>30) differed significantly between the 3 ranges ($P < 0.001$, linear trend test). Error bars represent 95% CIs. BMI <24.9 data from control subjects were omitted because $n = 3$.

Am J Clin Nutr 2007;86:1642–8.

Insulin resistance

- Insulin resistance has been shown to be an independent predictor of survival in dialysis patients.
- Siew et al.(2007) have demonstrated a direct association between a marker of insulin resistance — the homeostasis model assessment (HOMA) index — and increased protein catabolism in skeletal muscle(18 non-diabetic chronic hemodialysis patients.). BMI $25.4 \pm 4.4\ kg/m^2$

  It is unclear why insulin resistance would have a significant effect on skeletal muscle balance and not whole-body protein balance, and this observation requires further research.

  Kidney International (2007) **71**, 146–152
- Y taketani et al.(2007)--
  - A low glycemic index diet and low phosphate diet would be a useful treatment for CKD patients, especially those with DM, to reduce their risk of CVD and mortality.
  - This diet may be beneficial in the prevention of several aging-related diseases such as CVD even in healthy individuals. ***J. Med. Invest. 54:359-365, August, 2007***

營養需求量－熱量

- 維持(理想)體重為原則
- 提供熱量的食物：

五大營養素

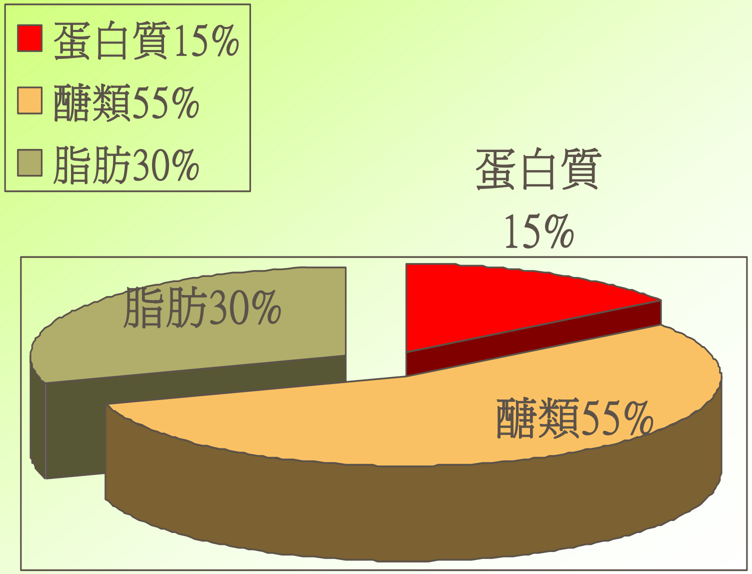
- 醣類
- 蛋白質
- 脂肪

} 提供熱量

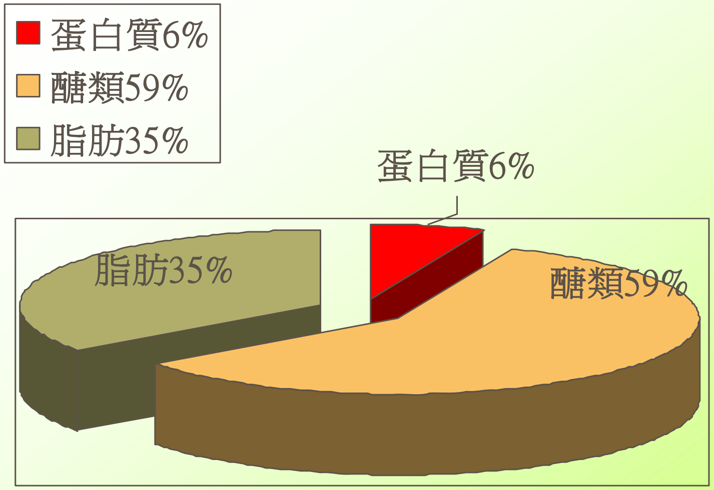
醣　類--每公克產生4大卡
蛋白質--每公克產生4大卡
脂　肪--每公克產生9大卡

- 維生素
- 礦物質

} 不提供熱量


蛋白質15%
醣類55%
脂肪30%
蛋白質
15%
脂肪30%
醣類55%

100%


蛋白質6%
醣類59%
脂肪35%
蛋白質6%
脂肪35%
醣類59%

Dietary protein and the progression of CKD

- Guideline 5: Nutritional management in Diabetes and CKD
  - If dietary protein intake is limited, an increase in carbohydrates and/or fats is required for adequate caloric intake.
  - Increasing intake of omega-3 and monounsaturated fats may confer benefits on CKD. (weak/opinion)

American Journal of Kidney Diseases, Vol 49, No 2, Suppl 2 (February), 2007: pp S21-S41

Fat

Table 14-3. American Heart Association guidelines for ω-3 fatty acids

Patients without documented CHD:
Eat a variety of (preferably oily) fish at least twice per week. Include oils and foods rich in α-linolenic acid (flaxseed, canola, and soybean oils; flaxseed and walnuts).

Patients with documented CHD:
Consume approximately 1 g EPA + DHA/d, preferably from oily fish. EPA + DHA supplements could be considered in consultation with the physician.

Patients who need triglyceride lowering: 2–4 g of EPA + DHA/d provided as capsules under a physicians care.

CHD, coronary heart disease; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid.

From Kris-Etherton P, William H, Appel L. Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. *Circulation* 2002;106: 2747–2757, with permission.

Handbook of nutrition and the kidney 2005

Plasma Polyunsaturated Fatty Acids and the Decline of Renal Function

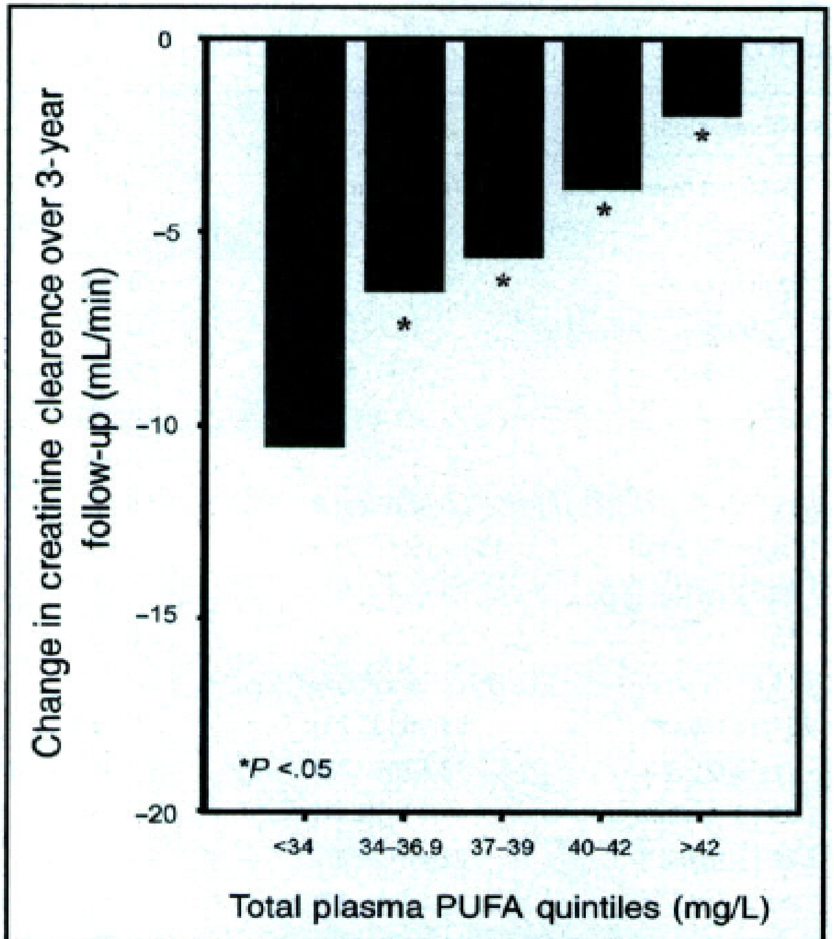


Fig. 1. Relationship of total plasma PUFAs (in quintiles; mg/L) at enrollment with change in creatinine clearance between enrollment and 3-year follow-up visit after adjusting for baseline creatinine clearance (* $P < 0.05$ in relation to the lowest quintile).
P for linear trend across quintiles <0.0001.

METHODS: We performed a cross-sectional and prospective analysis of 931 adults, ≥65 years old, enrolled in the InCHIANTI study, a population-based cohort in Tuscany, Italy. Plasma PUFAs were measured at enrollment, and creatinine clearance was estimated by the Cockcroft-Gault equation at baseline and after 3-year follow-up.

CONCLUSION: High PUFA concentrations, both n-3 FA and n-6 FA, may attenuate the age-associated decline in renal function among older community-dwelling women and men.

Clinical Chemistry; Mar 2008; 54, 3;475-481

反式脂肪

- 可能含有反式脂肪的食品
  速食類：炸薯條、炸雞、炸鹽酥雞
  點心類：甜甜圈、麵包、酥皮點心、糕餅類
  零食類：洋芋片、零食餅乾、油炸速食麵、微波加熱爆米花
- 其他：奶精／奶油球、植物性奶油、乳瑪琳、烤酥油
- 可能產生反式脂肪的烹調方法
  - 油炸、酥製、烘烤...

Advanced Glycoxidation End Products in Commonly Consumed Foods

ABSTRACT

Objective Advanced glycoxidation end products (AGEs), the derivatives of glucose-protein or glucose-lipid interactions, are implicated in the complications of diabetes and aging. The objective of this article was to determine the AGE content of commonly consumed foods and to evaluate the effects of various methods of food preparation on AGE production.

Design Two-hundred fifty foods were tested for their content in a common AGE marker $^{\epsilon}$N-carboxymethyllysine (CML), using an enzyme-linked immunosorbent assay based on an anti-CML monoclonal antibody. Lipid and protein AGEs were represented in units of AGEs per gram of food.

Results Foods of the fat group showed the highest amount of AGE content with a mean of 100±19 kU/g. High values were also observed for the meat and meat-substitute group, 43±7 kU/g. The carbohydrate group contained the lowest values of AGEs, 3.4±1.8 kU/g. The amount of AGEs present in all food categories was related to cooking temperature, length of cooking time, and presence of moisture. Broiling (225°C) and frying (177°C) resulted in the highest levels of AGEs, followed by roasting (177°C) and boiling (100°C).

Conclusions The results indicate that diet can be a significant environmental source of AGEs, which may constitute a chronic risk factor for cardiovascular and kidney damage.

J Am Diet Assoc. 2004;104:1287-1291.

Table 1. Advanced glycoxidation end products (AGE) content of selected foods prepared by standard cooking methods

Food item	AGE[a] (kU/g or /mL of food)
Fats	
Almonds, roasted	66.5 kU/g
Oil, olive	120 kU/mL
Butter	265 kU/g
Mayonnaise	94 kU/g
Proteins	
Chicken breast, broiled×15 min	58 kU/g
Chicken breast, fried×15 min	61 kU/g
Beef, boiled×1 h	22 kU/g
Beef, broiled×15 min	60 kU/g
Tuna, roasted×40 min	6 kU/g
Tuna, broiled×10 min	51 kU/g
Cheese, American	87 kU/g
Cheese, Brie	56 kU/g
Egg, fried	27 kU/g
Egg yolk, boiled	12 kU/g
Tofu, raw	8 kU/g
Tofu, broiled	41 kU/g
Carbohydrates	
Bread, whole-wheat center	0.54 kU/g
Pancake, homemade	10 kU/g
Milk, cow, whole	0.05 kU/mL
Milk, human, whole	0.05 kU/mL
Enfamil (infant formula)	4.86 kU/mL
Apple	0.13 kU/g
Banana	0.01 kU/g
Carrots	0.1 kU/g
Green beans	0.18 kU/g

[a]AGE denotes $^{\epsilon}$N-carboxymethyl-lysine (CML)-like immunoreactivity, assessed by enzyme-linked immunosorbent assay based on monoclonal antibody (4G9) (30,31).

Restricted intake of dietary advanced glycation end products retards renal progression in the remnant kidney model

Diet-derived advanced glycation end products (AGEs) contribute significantly to accumulation of AGEs in renal insufficiency. To test whether modulation of dietary AGEs would impact on progression of chronic renal disease, 5/6 nephrectomy rats were randomly placed on three diets that differed only in AGEs content (low AGEs diet (LAD), high AGEs diet (HAD), and standard rodent diet (SRD)) for 5–13 weeks. Compared with SRD- or HAD-fed rats, LAD-treated animals showed decreased proteinuria and retarded decline of creatinine clearance without alteration of blood pressure. Glomerular volume was reduced by 23% compared with HAD-fed rats at week 13 ($P<0.001$). Renal fibrosis progressed with time in the remnant kidneys from HAD-fed rats. However, LAD-fed animals presented a better-preserved structure of the kidneys. LAD-fed rats demonstrated significantly decreased serum and renal AGEs concentration ($P<0.01$ and $P<0.01$). This was associated with marked decrease of intrarenal advanced oxidation protein products and thiobarbituric acid reactive substances, as well as increase of glutathione peroxidase activity. LAD treatment also downregulated expression of monocyte chemoattractant protein-1 and transforming growth factor-β1 and ameliorated macrophage infiltration in the remnant kidney. These results demonstrated that restriction of dietary AGEs intake retards progression of renal fibrosis and dysfunction in the remnant kidney model.

Kidney International (2007) **71,** 901–911. doi:10.1038/sj.ki.5002162;

Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease[1–3]

- fructose intake will be a risk factor for hypertension, insulin resistance, hypertriacylglycerolemia, obesity, type 2 diabetes, preeclampsia, chronic kidney disease, stroke, cardiovascular disease, and mortality.
- reducing uric acid in patients with uric acid concentrations └6.0 mg/dL will improve endothelial dysfunction, decrease systemic vascular resistance, lower blood pressure, lower triacylglycerol concentrations, improve body weight, lower the risk of the progression of renal disease, and reduce cardiovascular disease risk.
- low-fructose diets coupled with mild purine restriction will improve weight and reduce cardiovascular disease risk.
- fructokinase will be identified as a key enzyme mediating the cardiorenal disease syndrome; genetic polymorphisms will be associated with cardiovascular disease risk, and blocking the enzyme will provide a novel way to prevent cardiorenal disease.
- Clearly, much more work needs to be done to prove or disprove this hypothesis.

Am J Clin Nutr 2007;86:899–906.

維生素*Vitamins*

- 較容易缺乏
  - Vit.B6、folic acid、 Vit.C
- 不需要額外補充
  - Vit.A、Vit.K、Vit.E
- 醫師處方– 避免自行補充
- 適量的蔬果

腎臟專科營養師基礎訓練班, 2006

鈉 Sodium

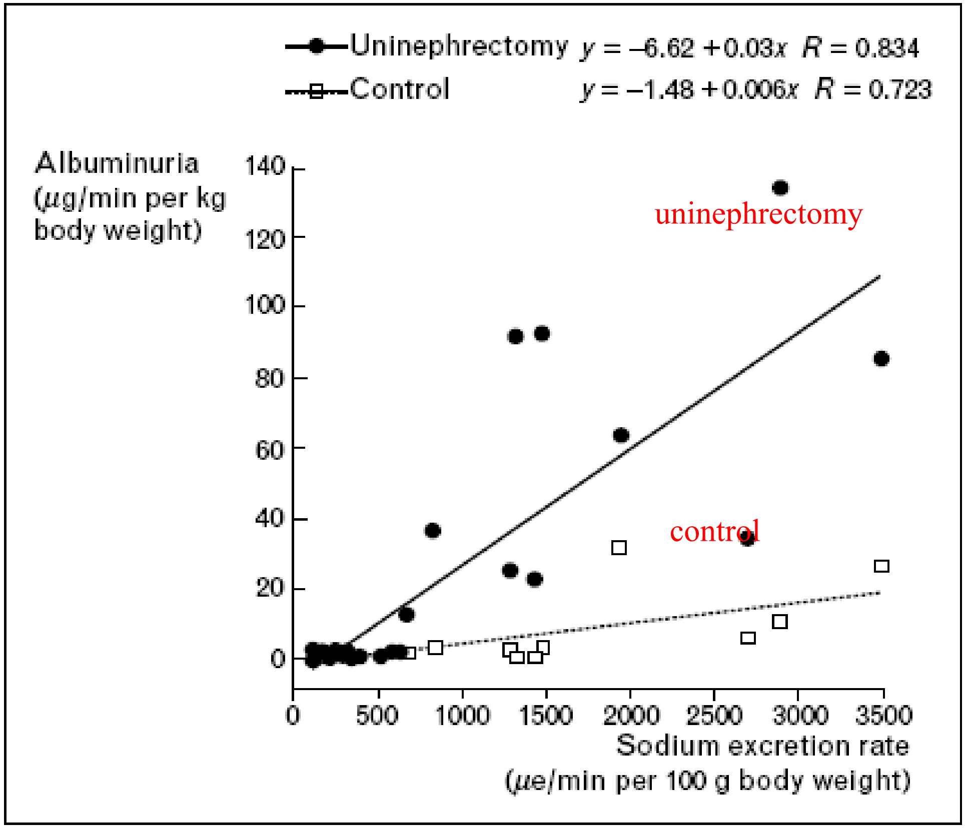
- **Because of the complex interactions between dietary sodium, hypertension, CVD, and CKD, the**
- **NKF-K/DOQI Clinical Practice Guidelines on Hypertension and Antihypertension Agents(2004) 建議 CKD 病患每日飲食鈉的攝取量≦ 2.4g.**

- **如果病患有鈉流失疾病或藥物影響，依據血液檢查值再個別化調整.**

Handbook of nutrition and the kidney 2005

Effect of salt intake on progression of chronic kidney disease

Figure 3 Relationship between dietary salt intake and albuminuria in normal (control) rats and uninephrectomized Lewis rats



A direct correlation between degree of salt intake and albumin excretion rate was identified in both groups, although the slope of the line was increased in those rats that had undergone unilateral nephrectomy. Data were obtained from Sanders *et al.* [41].

- study showed： control rats and uninephrectomized rats manifested salt-dependent increases in albuminuria without overt renal pathology when fed 0.3%, 1.0% and 8.0% NaCl diets (fig 3);
- administration of 8.0% NaCl diet to Fisher/Lewis rats following renal transplantation accelerated the development of chronic allograft nephropathy. An increase in tubulointerstitial fibrosis and glomerular sclerosis was demonstrated in these transplanted organs (Sanders et al., 2001).

Current Opinion in Nephrology and Hypertension 2006, 15:54–60

Effect of salt intake on progression of chronic kidney disease - human studies

- Proteinuria increased in the high-salt group and fell in the low-salt group (Cianciaruso et al., 1998), that support salt restriction in this population of patients with advanced CKD.

- Dietary salt restriction should be considered within the context of primary prevention of progressive renal failure in at-risk populations.

- Patients with diseases whose pathogenesis is related to excess production of TGF-β1 , such as chronic allograft nephropathy and overt diabetic nephropathy , may have accelerated loss of function when dietary salt intake is increased.

Current Opinion in Nephrology and Hypertension 2006, 15:54–60

磷 ：*Phosphorus*

- 建議及早介入處理維生素D代謝異常與副甲狀腺機能亢進.
- 飲食中的磷與鈣應於CKD 早期評估.
- The NKF-K/DOQI Clinical Practice Guidelines for Bone Metabolism and Disease (2003) 建議：當血磷上升大於4.6 mg/dL (1.49 mmol/L), 或血漿中副甲狀腺荷爾蒙會上升或兩者皆上升時；限制每日飲食中的磷至 800 ~ 1000 mg (調整飲食蛋白質的需要).
- 血中磷的值由飲食修正與磷結合劑藥物介入控制.
- 磷結合劑藥物應與餐中的高磷食物一起使用.

Handbook of nutrition and the kidney 2005

Phosphorus

- 高血磷是透析病患不良預後的主要因子, 易導致心血管疾病等死亡率的增加. [Block GA, et al., 1998, Rodriguez-Benot A, et al.,2005]
- 過多的磷與鈣會沉積在動脈和其它軟骨組織, 導致動脈硬化等心臟疾病的發生. [Ribeiro S, et al., 1998]
- 血磷升高也刺激副甲狀腺荷爾蒙導致續發性副甲狀腺機能亢進與腎性骨病變.[Block GA, et al., 1998, Goodman WG, 2004,Malluche HH, et al., 2002]
- 美國飲食中的磷含量逐漸增加, 主要來自於便利商店(超商)食品與速食(快餐)食品(convenience and fast foods)中的含磷食品添加物(phosphorus containing additives). [Calvo MS, et al., 1996]

J Ren Nutr. 2007 September ; 17(5): 350–354.

Phosphorus

- 據估計(依個人的食品選擇), 飲食中添加物的磷可多達1000 mg/day. [Bell RR, et al., 1977]
- 而且, 食品添加物中的磷幾乎完全被吸收(almost entirely absorbed),而天然食物中的磷約只吸收60% .[Uribarri J, et al., 2003]
- 衛教病患有關高磷食物是控制高血磷症的重要關鍵,[Ford JC, et al., 2004; Cupisti A, et al., 2004] 但是含磷添加物的使用使得病患與營養師很難準確估計食物中的磷含量.

J Ren Nutr. 2007 September ; 17(5): 350–354.

四、食品添加物之分類---依據衛生署公告之「食品添加物使用範圍及用量標準」，食品添加物依其用途區分為下列17類：(部分)

品質改良劑	Quality improvement, distillery and foodstuff processing agent為改良加工食品品質、釀造或食品製造加工必需時使用之物質	三偏磷酸鈉、硫酸鈣、食用石膏等77種
營養添加劑	Nutritional enriching agent強化食品營養之物質	維生素礦物質胺基酸等122種
著色劑	Coloring agent對食品產生著色作用之物質	食用紅色六號等27種
香 料	Flavoring agent增強食品香味之物質	香莢蘭醛等90種
調味劑	Seasoning agent賦予食品酸味甘味甜味之物質	L-麩酸鈉(味精)、檸檬酸、糖精等53種
粘稠劑(糊料)	Pasting (Binding) agent賦予食品滑溜感與粘性之物質	鹿角菜膠、CMC等21種
結著劑	Coagulating agent增強肉類魚肉類黏性之物質	磷酸鹽類等16種
食品工業用化學藥品	Chemicals for food industry提供食品加工上所需之酸及鹼	鹽酸、氫氧化鈉等10種
溶 劑	Dissolving agent (Solvent)食用油脂、香辛料精油之萃取月溶劑	己烷、丙二醇等6種
乳化劑	Emulsifier讓水與油等無法相互均一混合之原料乳化之物質	脂肪酸甘油酯、脂肪酸蔗糖酯、Polysorbate等12種
其 他	Others分別具有消泡、過濾、防蟲、被膜等之物質	矽樹脂、矽藻土、胡椒基丁醚、蟲膠等13種

PHOSPHORUS CONTAINING FOOD ADDITIVES AND THE ACCURACY OF NUTRIENT DATABASES: IMPLICATIONS FOR RENAL PATIENTS

Catherine M. Sullivan, MS, RD, LD[1,2], Janeen B. Leon, MS, RD, LD[1,2], and Ashwini R. Sehgal, MD[1,2,3,4,5]

Abstract

Objective—Phosphorus containing additives are increasingly added to food products. We sought to determine the potential impact of these additives. We focused on chicken products as an example.

Methods—We purchased a variety of chicken products, prepared them according to package directions, and performed laboratory analyses to determine their actual phosphorus content. We used ESHA Food Processor SQL Software to determine the expected phosphorus content of each product.

Results—Of 38 chicken products, 35 (92%) had phosphorus containing additives listed among their ingredients. For every category of chicken products containing additives, the actual phosphorus content was greater than the content expected from nutrient database. For example, actual phosphorus content exceeded expected phosphorus content by an average of 84 mg/100g for breaded breast strips. There was also a great deal of variation within each category. For example, the difference between actual and expected phosphorus content ranged from 59 to 165 mg/100g for breast patties. Two 100 g servings of additive containing products contain an average of 440 mg of phosphorus, or about half the total daily recommended intake for dialysis patients.

Conclusion—Phosphorus containing additives significantly increase the amount of phosphorus in chicken products. Available nutrient databases do not reflect this higher phosphorus content, and the variation between similar products makes it impossible for patients and dietitians to accurately estimate phosphorus content. We recommend that dialysis patients limit their intake of additive containing products and that the phosphorus content of food products be included on nutrition facts labels.

J Ren Nutr. 2007 September ; 17(5): 350–354.

Phosphorus

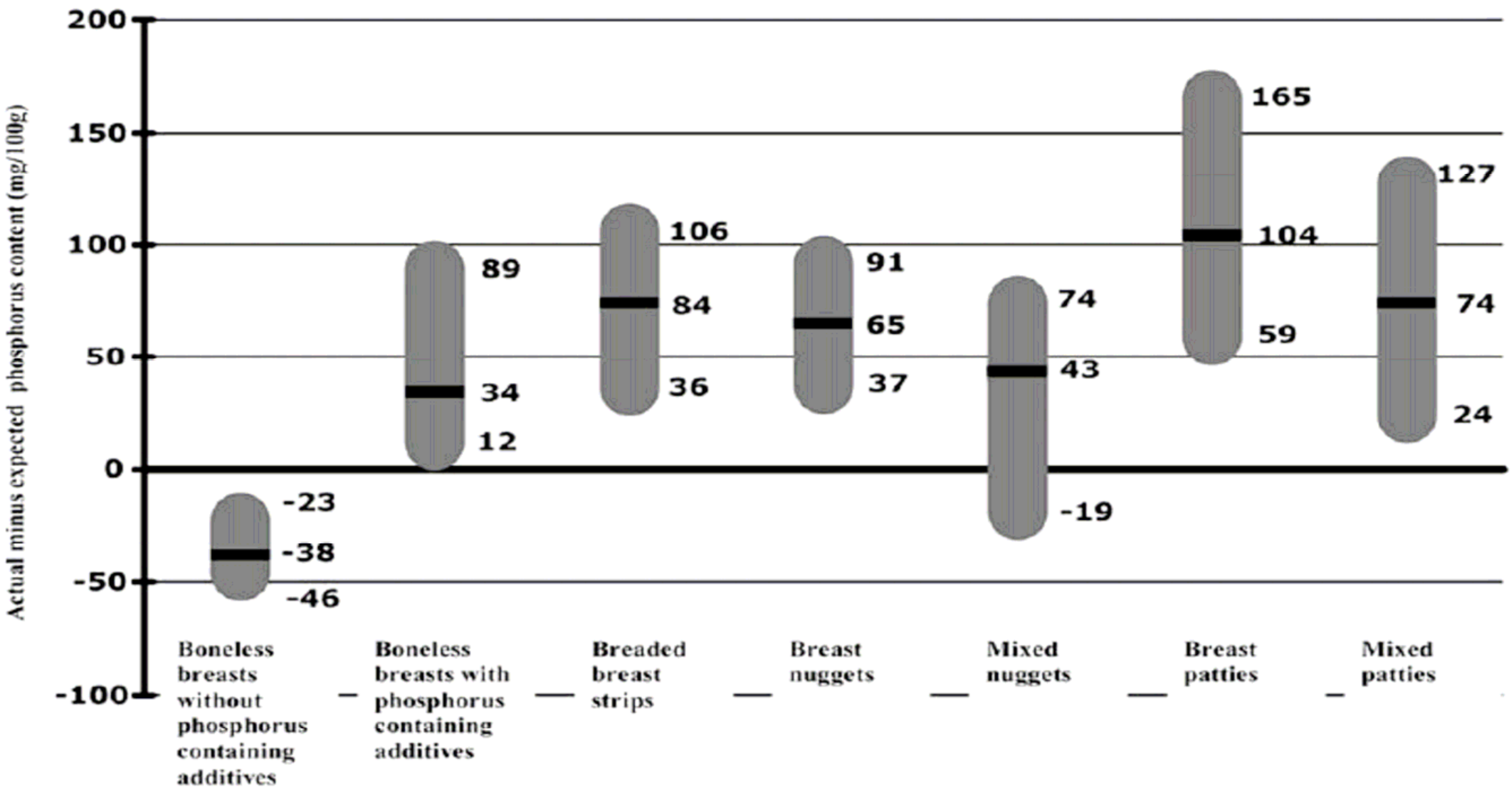


Figure 1.
Actual minus expected content of 38 chicken products. For each category of chicken products, the black line represents the mean value while the top and bottom of the gray bar represent the maximum and minimum values.

J Ren Nutr. 2007 September ; 17(5): 350–354.

Phosphorus

- 本研究發現：含磷食品添加物出現在多種雞肉產品中，顯著增加這些產品的磷含量. 而參考資料庫並不能反應此高磷含量，而且相似產品之間差異性也大，使得病患與營養師不能正確估計食物的磷含量.
- 便利商店(超商)食品(convenience foods)與速食(快餐)食品(fast foods)傾向於比較美味而且製備容易，但是通常含有磷的添加.
- 作者建議：透析病患應限制食用添加了含磷食品添加物的產品；製造商發展低磷產品；執政者需要要求廠商將食品磷含量列入食品標示之中.
- 衛教透析病患限制攝取含添加物的產品是合理的建議，但有以下挑戰：
  - 研究分析：市售不含添加物的產品可能種類有限或不易購得.
  - 成份標示部份產品並未列出，很難辨識是否添加含磷添加物.
  - 雖然知道有含磷添加物若未標示含量，仍然不能準確估計磷含量.
  - 不含添加物的產品，如：生雞胸肉，可能需要較複雜的製備方式才能較美味. 這對於體能不佳或社交有限的透析病患是個負擔

J Ren Nutr. 2007 September ; 17(5): 350–354.

一般標示

- 品名: 蒜味香腸
- 淨重：270 公克 ±5%
- 主要原料：豬 肉、天然豬腸衣
- 副原料：食鹽、糖、香辛料
- 食品添加物：磷酸鹽(結著劑)、異抗壞血酸鈉 (抗氧化劑)、味精 (調味劑)、硝酸鹽(保色劑)、亞硝酸鹽(保色劑)
- 保存條件期限：充氮包冷藏 70天 (5℃以下)
- 有效日期：請見包裝標示 (西元年月日)
- 製造廠商：滋味珍食品股份有限公司
- 地址:台南市中華西路一段103號
- 服務電話:0800-011112 http://www.blackbridge.com.tw

國人磷攝取量

- 依照國民營養調查之估計，台灣地區 19 - 64歲成人來自膳食的磷攝取量平均為男性1087 mg/d，女性858 mg/d， 中位數為男性800 mg 以上，女性600 mg 以上，均達到建議攝取量之平均值； 主要的食物來源為五穀根莖類、乳類、蛋、豆、魚、肉類、蔬菜類。若以現行國人之飲食型態，遵循國人每日飲食指南之建議，膳食可供應的磷量為1305 mg/d。營養師設計之普通飲食經定量分析之含磷量平均為1037 mg/d 。這些攝取量均未計入來自食品添加物的含磷量，因此實際攝取量有低估之可能性。

(資料摘自行政院衛生署出版之「國人膳食營養素參考攝取量及其 說明修訂第六版」)
營養素辭典- 磷/蕭寧馨著

國人磷攝取量

- 隨著加工食品磷酸鹽類添加物的使用量增多，以及含磷碳酸飲料的消耗量增加，磷攝取量有日漸增加的趨勢，美國估計過去二十年內磷攝取量增加了10 – 15 %。已有研究指出含磷酸之碳酸飲料與低鈣血症有顯著關聯　。此外，腎功能減退者必須限制磷攝取量，但是腎衰竭初期通常不易發覺。當我國衛生署放寬飲料添加磷酸之使用限制時，國人磷攝取量之變化趨勢與對骨骼與腎臟健康之影響必需密切注意。
- 國人磷的飲食建議攝取量– 800mg (>19 years)

(資料摘自行政院衛生署出版之「國人膳食營養素參考攝取量及其 說明修訂第六版」)
營養素辭典- 磷/蕭寧馨著

Phosphorus Content of Foods

- 每日磷的攝取量多寡依賴所攝食的方式.
- 典型美式飲食, 年輕與中老年男性約消耗約1600 mg/day, 老年女性約1000 mg/day (19).這些膳食磷攝取量大部份來自"天然"食品的磷含量,並未涵蓋食品添加物與補充劑之磷.
- 真正估計飲食中的磷含量需要考慮3類主要來源：
  - natural phosphorus contained in raw or unprocessed food as cellular and protein constituents;
  - phosphorus added to foods during processing; and
  - phosphorus contained in dietary supplements.
- 用於肉品製造可使瘦肉較濕潤，改善風味與濕度，強化肉品過程之一 (22).
- 磷酸鹽也使用在ham和bacon以降低氧化,穩定蛋白質,改善色澤與風味.
- 加入5% sodium hydrogen phosphate 可以預防ham水分流失,使產品更柔軟多汁.

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Phosphorus Content of Foods

- 高蛋白質食物如: meat, milk, dairy, eggs, 及cereals天然含有高量的磷,佔每日飲食總磷攝取的最大部分. Table 1 提供一部分不同類別食物的磷含量.
- 近來, 隨著加工食品的使用量增加(包括磷的添加), 已顯著增加此礦物質的每日攝取量.
- In 1990, 美國含磷食品添加物估計約提供成人每日470 mg的磷攝取,此量將隨著人們對即食食品與速食食品的攝取增加而增加.
- 事實上,依據個別食物的選擇, 簡單的增加飲食中加工食品的攝食比率即可能增加磷攝取多達 1000 mg/day.

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TABLE 1. Phosphorus content of selected foods*

Food	Portion size	P (mg)	Protein (g)	P/prot
Meats				
Veal	3 oz	212	31	6.8
Chicken	140 g	259	35	7.4
Lamb	3 oz	162	22	7.4
Beef	3 oz	200	26.4	7.6
Turkey	3 oz	208	24	8.7
Fish	3 oz	221	19.4	11.4
Pork	3 oz	224	18	12.4
Crab	3 oz	238	16.5	14.4
Breads, cereals, and nuts				
Bread	1 slice	25	3.4	7.3
Bagel	3.5"	68	7.5	9
Almonds	1 oz	134	6	22.3
Pistachio	1 oz	137	6	22.8
Walnuts	1 oz	98	4.3	22.8
Biscuits	2.5"	98	4.2	23.3
Cereals	1 cup	259	5.2	49.8
Milk, dairy, and eggs				
Egg white	1 large	5	3.6	1.4
Egg whole	1 large	96	6.3	15.3
Cheese	1 oz	133	6.6	20
Yogurt	8 fl oz	327	11.9	27.5
Milk	1 cup	222	7.9	28
Legumes and rice				
Peas	1 cup	114	7.5	15
Beans	1 cup	183	12	15
Lentils	1 cup	356	17.9	19.9
Rice, white	1 cup	74	4	18.4
Rice, brown	1 cup	162	5	32

*Individual values taken from the USDA National Nutrient Database. P/prot = ratio of phosphorus to protein content.

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Phosphorus Bioavailability

- 各種食物所獲得的磷之生物利用率是分析飲食中磷攝取的重要考慮因素.
- 植物中大部分的磷(約75%)是以phytate型式存在. 人類的小腸不能分泌分解此物質的酵素phytase(分解飲食中的phytate釋放出磷),因此phytate phosphorus不被人類利用,除非食物被phytase加工處理過,如麵包發酵劑(phytase-containing yeast).
- 肉類中的磷大部分是細胞內的有機磷,很容易被腸胃道分解釋放出無機磷而被吸收.
- 牛奶中的磷屬於另一種生物利用率.
- 食品加工所添加的磷幾乎以無機鹽的形式存在,因此,幾乎完全被吸收(20).
- 通常混合飲食(typical mixed diet)中的磷約有60%被吸收，此百分比將依所選擇的食物類別與加工食品的多寡而定.
- 飲食中若以fast food和processed meats作為蛋白質來源將有較高比率的磷吸收.

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Food Demineralization

- A method has been described to extract minerals from foods by soaking them in water . Mineral content was measured before and after water treatment.
- The phosphorus reduction was 51% for vegetables, 48% for legumes, 38% for meats, 70% for flours, and 19% for cheddar cheese.
- Boiling foods can also significantly reduce dietary phosphate while preserving protein intake, namely reducing the effective phosphate intake per gram of dietary protein. This can represent additional advice to the patient for limiting the dietary phosphorus load at the same protein intake.

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What Should Be the Daily Phosphorus Intake in CKD Patients?

- Based on this background the working group from K⁄DOQI concluded that: ‘‘Given the lack of evidence of adverse effects, and the evidence of positive benefit of dietary phosphate restriction, it is the consensus of the Work Group that dietary phosphate restriction be initiated in patients with CKD when PTH levels are elevated (GFR<60 ml ⁄ minute/1.73 m2, stage 3) or with elevated blood levels of serum phosphorus (stages 4 and 5)’’. The actual dietary recommendation is for less than 1000 mg phosphorus per day.

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What Should Be the Daily Phosphorus Intake inCKD Patients?

- In practical terms, however, it is very difficult to provide a dietary phosphorus intake of less than 1000 mg/day because of the high levels of dietary protein intake advised as well as the large amount of unaccounted phosphorus in processed foods as discussed extensively above.
- The close association between phosphorus and protein content in food makes it very difficult to restrict dietary phosphorus content without simultaneously restricting dietary protein intake, especially protein of animal origin.

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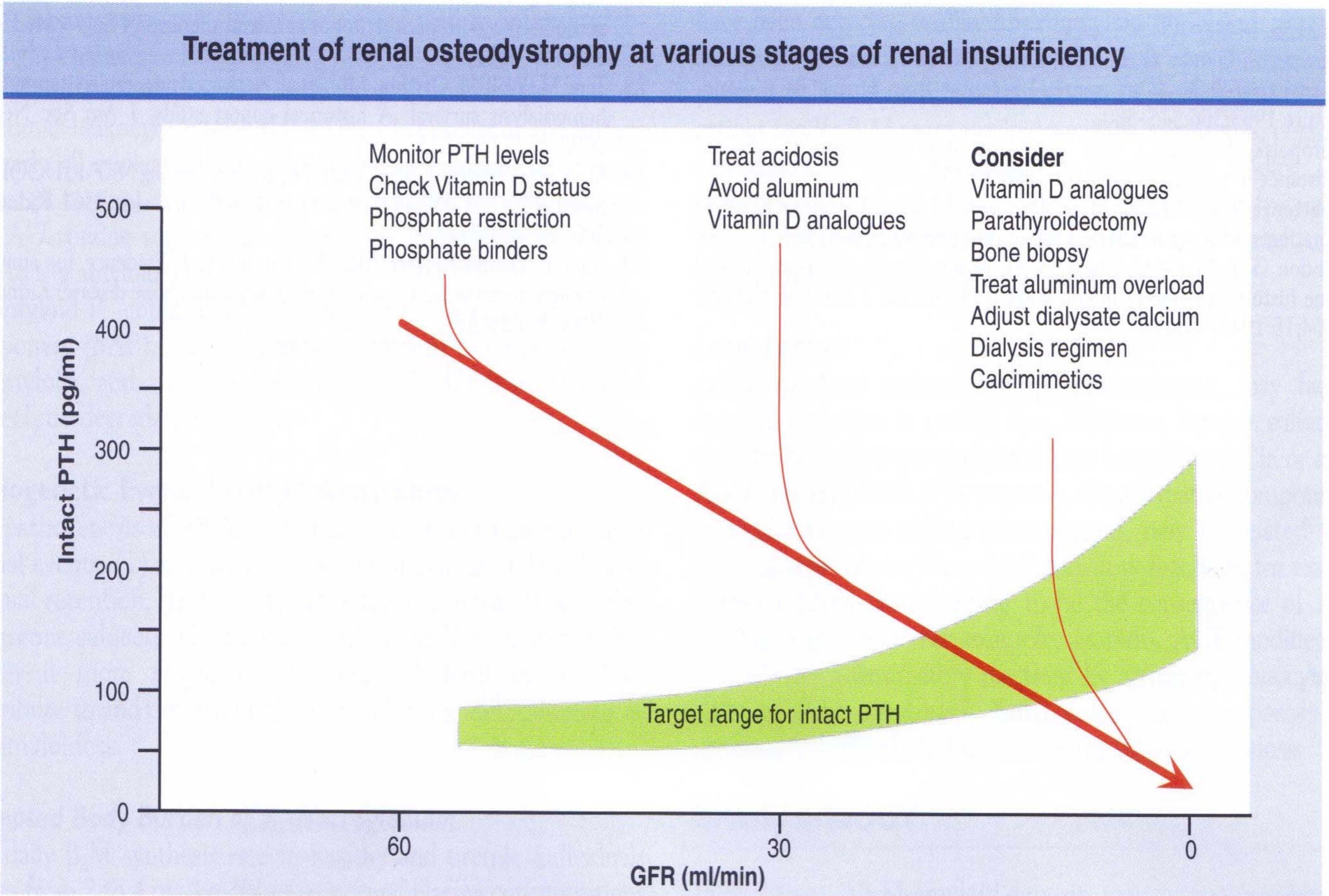

Figure 74.15 Treatment of renal osteodystrophy at various stages of renal insufficiency. GFR, glomerular filtration rate; PTH, parathyroid hormone.

Notes

- Phosphate binders, in general, bind only up to 300 to 400 mg phosphorus per day, even when given in maximum doses.
- Thus, the use of phosphate binders does not substitute for the need to restrict dietary phosphorus.

(Kopple JD, Diseases of the kidney& urinary tract. Chap103, 2007)

- HD removal 800–1000 mg/ session or about 300 mg/day; PD removal about 423 mg/day

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Calcium

- Total dietary intake of calcium has become a concern because of the evidence that an excessive calcium intake can exacerbate vascular and other extra skeletal calcifications.
- The amount of dietary calcium that can be labeled as excessive in the population of patients with CKD has not to date been defined by any research.
- Maintaining total calcium intake within the RDA of calcium for adults is a reasonable goal until new evidence suggests otherwise.
- The RDA for calcium is 1200mg (for both men and women aged 19 to 75 years).

Handbook of nutrition and the kidney 2005

慢性腎臟病的貧血飲食

- Conclusion: Small amounts of meat (≥ 50 g) significantly increase nonheme-iron absorption from a phytate-rich meal low in vitamin C.
- 國民營養調查
  - 男性: 14.2mg total iron
  - 女性: 11.4mg total iron
- 60gm low protein diet
  - 含11 mg total iron
    - 0.4 mg absorbable if protein from beef
    - 0.2 mg absorbable if protein from chicken , fish
- 若使用EPO需要supplement iron
  - 靜脈
  - 口服

腎臟專科營養師基礎訓練班, 2006

貧血飲食

1.造血所需的營養素包含：鐵質、蛋白質、維生素C 及維生素B12 、葉酸等，必須各種營養素相互配合，才能達成最佳造血功能。（某些貧血，如再生不良性貧血等，飲食無法改善。）

2.富含營養素的食物來源

－鐵質：內臟、瘦肉、貝類、蛋黃、乾豆類（紅豆、黃豆等）、乾果類（紅棗、黑棗、葡萄乾等）、深綠色蔬菜、全穀類等。

－蛋白質：瘦肉、豆類及豆製品、蛋、奶類等。

－維生素C：枸櫞類水果（柳橙、檸檬、蕃石榴、蕃茄等）、深綠色蔬菜等。

－維生素B12：內臟、各類瘦肉、牛奶等。

－葉酸：內臟、核果類、深綠色蔬菜、健素粉等

與貧血有關的營養素

- 蛋白質
- 鐵質
- 維生素C
- 維生素B6
- 維生素B12
- 葉酸
- 銅、鋅、鈷
- 維生素E

鐵

- 功能

  血色素、肌紅素及細胞色素的主要成分；幫助能量的產生

- 缺乏

  缺鐵性貧血(小球性貧血)

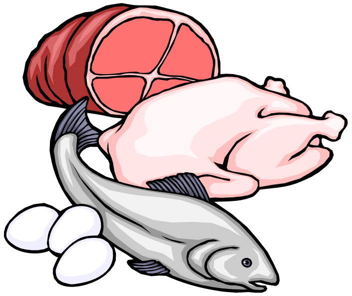
- 來源

  肝臟、紅色肉類、魚類、蛋黃、豆類、深色蔬菜

- 吸收率

  血紅素鐵的吸收率約為20%(23~25%)

  非血紅素鐵的吸收率不到10% (≈3~5%)



Iron (Nonheme-iron absorption from a phytate-rich meal is increased by the addition of small amounts of pork meat)

- Forty-five healthy women with a mean (± SD) age of 24 ± 3 y,mean weight of 64 ± 8 kg, and mean body mass index (in kg/m2)of 22.6 ± 2.7 volunteered for the study.
- Each subject was given 2 test meals: a basic meal (A) and the basic meal with added meat (B). The meat meals contained either 25, 50 or 75 g pork. The subjects were randomly assigned to receive 1 of the 3 meat meals.

TABLE 2
Composition of the test meals[1]

	Basic meal	Meat meals		
		25 g meat	50 g meat	75 g meat
Energy (MJ)	2.2	2.3	2.4	2.5
Total protein (g)	10.6	18.4	22.2	27.3
Nonheme iron (mg)	2.3	2.5	2.5	2.3
Heme iron (mg)	—	0.06	0.12	0.19
Phytic acid				
(mg)	220	220	220	220
(μmol)	358	358	358	358
Ascorbic acid (mg)	7.4	7.4	7.4	7.4
Calcium (mg)	50.5	58.4	58.8	64.3
Zinc (mg)	2.6	2.9	3.0	3.2

[1] The basic meal contained 0 g meat.

Am J Clin Nutr 2003;77:173–9

Iron (Nonheme-iron absorption from a phytate-rich meal is increased by the addition of small amounts of pork meat)

TABLE 1

Iron status and nonheme-iron absorption data[1]

	Meat content of meal			
	0 g (*n* = 45)	25 g (*n* = 15)	50 g (*n* = 15)	75 g (*n* = 15)
Serum ferritin (μg/L)	—	16.9 (14.4, 19.7)	20.7 (17.8, 24.2)	21.0 (18.0, 24.5)
Hemoglobin (g/L)	—	125.1 (121.7, 128.6)	126.8 (123.3, 130.2)	125.9 (122.4, 129.4)
Absorption from reference dose[2] (%)	—	36.4[a] (29.2, 45.4)	27.1[a,b] (21.8, 33.8)	25.0[b] (20.0, 31.1)
Absorption from meals (%)				
Unadjusted data	4.3[c] (3.5, 5.4)	5.1[c,d] (4.0, 6.5)	6.3[e] (4.9, 8.0)	6.7[e,f] (5.3, 8.5)
Data adjusted to 40 μg serum ferritin/L	2.1[c] (1.7, 2.6)	2.4[c,d] (1.9, 3.1)	3.1[e] (2.4, 3.8)	3.3[e,f] (2.6, 4.1)
Data adjusted to 40% absorption from reference dose	5.9[c] (4.9, 7.2)	7.1[c,d] (5.7, 8.8)	8.6[e] (6.9, 10.7)	9.2[e,f] (7.4, 11.5)
Meat meal:basic meal	—	1.15[g] (1.02, 1.34)	1.44[g,h] (1.26, 1.65)	1.57[h] (1.34, 1.77)

[1] Least-squares means; 95% CI in parentheses. Values in a row with different superscript letters are significantly different (linear mixed models with post hoc Tukey-Kramer tests): [ab] $P < 0.05$, [ce] $P < 0.001$, [cf] $P < 0.001$, [df] $P < 0.04$, [gh] $P < 0.05$.

[2] Three milligrams Fe as ferrous sulfate and 30 mg ascorbic acid in 10 mL of a 0.01-mol HCl/L solution.

Am J Clin Nutr 2003;77:173–9

Iron (Nonheme-iron absorption from a phytate-rich meal is increased by the addition of small amounts of pork meat)

- Results: Twenty-five grams meat did not increase nonheme-iron absorption significantly ($P = 0.13$), whereas absorption increased 44% ($P < 0.001$) and 57% ($P < 0.001$), respectively, when 50 and 75 g meat were added to the basic meal. In absolute values, this corresponds to an absorption that was 2.6% and 3.4% higher, respectively, than that with the basic meal after adjustment of the data to a level of 40% absorption from a reference dose.
- Conclusion: Small amounts of meat (≥ 50 g) significantly increase nonheme-iron absorption from a phytate-rich meal low in vitamin C.

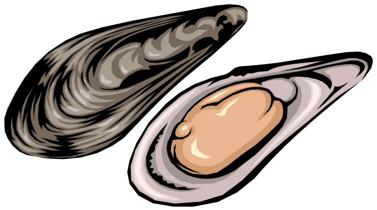
Am J Clin Nutr 2003;77:173–9

鉀 ; Potassium; K

- 飲食中的鉀離子只有在尿量少於1000c.c.或血鉀上升時才需要限制.
- 預防心血管疾病飲食(TLC)或糖尿病飲食鼓勵多吃蔬果及全穀類食物時，可能導致鉀攝取過量(>6g) ，血鉀上升.
- The NKF-K/DOQI Guidelines for Hypertension (2004) suggest:
  - potassium intake greater than 4g per day for stage 1 to 2
  - potassium intake between 2~4g per day for stage 3 to 4.

Handbook of nutrition and the kidney 2005

鋅



功能

參與核酸及蛋白質的合成之酵素作用、如:新細胞的合成(傷口癒合、腦部發育…)、體內抗氧化酵素的重要成分、提升免疫力、促進生育力、保護血管防止動脈硬化…

缺乏

生長遲緩、傷口癒合不佳、貧血、食慾不振等

鋅在攝護腺中含量高，攝護腺發炎時，鋅含量明顯降低

來源

海鮮類食物如:牡蠣、南瓜子等種子類及核仁食物、其他如:肉類、肝、蛋…

Beverages & Others

1. cola
2. red grape juice
3. wine
4. green tea extract
5. mangosteen fruit (山竹)

Evaluation of the Effect of Cola Drinks on Bone Mineral Density and Associated Factors

- 研究用的實驗動物來自於Gulhane Medical Academy (Ankara, Turkey). 研究一開始, 30隻10週大的 Sprague-Dawley rats, 重約140–160 g, 分成四組.
  - group 1 of 10 male rats + water & cola
  - group 2 of 10 female rats+ water & cola
  - group 3 of 5 male rats + water (control)
  - group 4 of 5 female rats+ water(control)
- 每隔幾天變換液體容器的位置確保動物不會在習慣位置攝取. 動物依自己的意願攝取水或可樂飲料,模擬人類自由攝取型態.
- 每天監測四組之食物量與飲水或可樂量,紀錄之後換新
- 大鼠每週一與每週五秤重並紀錄.每日監測實驗動物的情況. 研究持續30天.

Evaluation of the Effect of Cola Drinks on Bone Mineral Density and Associated Factors

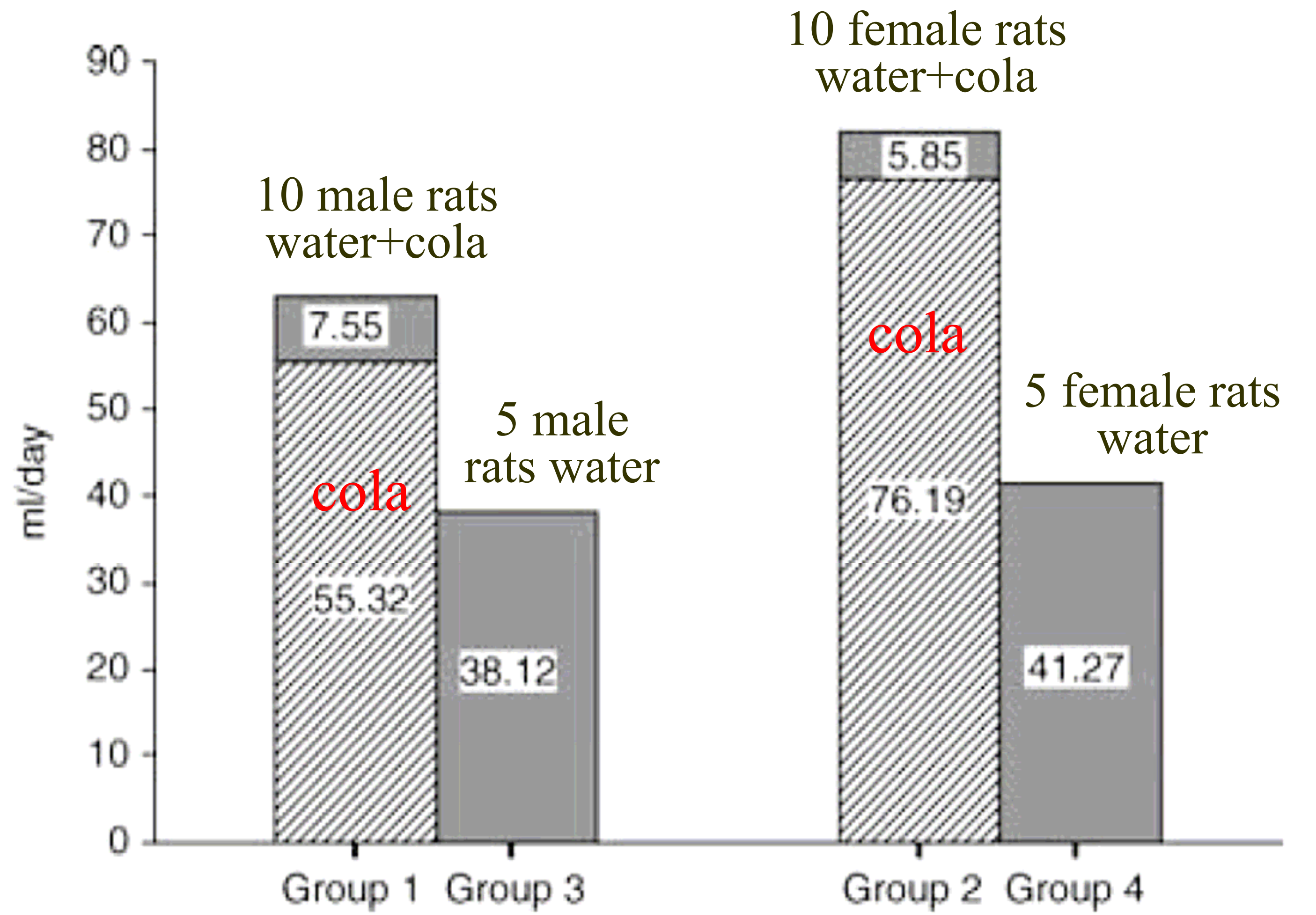


Fig. 1. Water (grey area), cola (scattered area) and total liquid consumption of groups.

Basic & Clinical Pharmacology & Toxicology,2007,100, 334–338

Evaluation of the Effect of Cola Drinks on Bone Mineral Density and Associated Factors

- 食道末端的樣本檢測組織病理學顯示：雖然攝取低pH值的飲料,並無食道炎情形.
- 腎臟的組織病理研究顯示：廣泛的腎小管內出血(extensive intertubular haemorrhage)與絲球充血(glomerular congestion)　(fig. 2).

- **可樂飲料之pH值變化為1.38 ~ 1.72. 在一開罐之pH值為1.38, 1 hr後增至1.72 ,之後在 1.44 ~ 1.64變動. 樣本之磷酸含量為700 mg/l以下.**

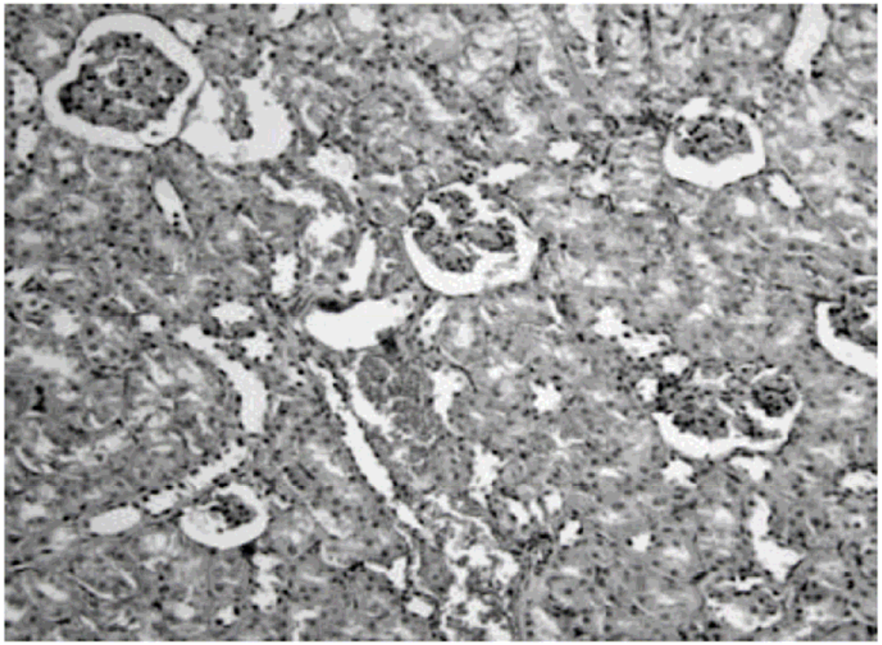


Fig. 2. Foci of intertubular bleeding and extensive glomerular congestion in kidney.

Basic & Clinical Pharmacology & Toxicology,2007,100, 334–338

Evaluation of the Effect of Cola Drinks on Bone Mineral Density and Associated Factors

Table 2.

Biochemical values of the blood samples (mean ± S.D.).

Biochemical parameters	Group 1 (n = 10) male	Control (n = 5) male	Group 2 (n = 10) female	Control (n = 5) female
Calcium (mg/dl)	10.99 ± 0.45	11.10 ± 0.90	11.21 ± 1.02	11.70 ± 0.74
Ionized calcium (mg/dl)	6.15 ± 0.22	6.30 ± 0.32	6.27 ± 0.42	6.60 ± 0.30
Phosphorus (mg/dl)	10.30 ± 1.44	9.30 ± 0.98	8.73 ± 1.04	9.10 ± 1.10
Magnesium (mg/dl)	3.40 ± 0.23	2.90 ± 0.27	3.33 ± 0.56	3.20 ± 0.41
1-α,25-hydroxyvitamin D (ng/ml)	28.82 ± 5.44	23.30 ± 4.29	33.90 ± 7.59	33.80 ± 6.23
Uric acid (mg/dl)	1.31 ± 0.22	0.80 ± 0.17	1.30 ± 0.64	1.30 ± 0.18
Triiodothyronine (T_3) (pg/ml) ↓	2.45 ± 0.28	2.69 ± 0.22	2.60 ± 0.35	2.81 ± 0.38
Thyroxine (T_4) (ng/dl) ↓	2.53 ± 0.42*	3.33 ± 0.48	2.27 ± 0.67	2.05 ± 0.58
Progesterone (ng/ml)	0.62 ± 0.27*	0.21 ± 0.08	11.89 ± 1.53*	6.27 ± 0.93
Cholesterol (mg/dl)	64.80 ± 6.52*	47.60 ± 5.01	67.20 ± 6.57*	55.00 ± 5.82
VLDL (mg/dl)	26.22 ± 7.18*	13.23 ± 5.24	18.66 ± 8.14	22.50 ± 6.12
Triglyceride (mg/dl)	131.22 ± 36.58*	67.09 ± 21.24	93.00 ± 40.41	59.30 ± 18.43
Iron (μg/dl) ↓	183.77 ± 32.71*	269.16 ± 21.92	284.40 ± 31.52*	334.00 ± 27.35

*Statistically significant when compared to the control group of the same sex ($P < 0.05$). VLDL, very low-density lipoprotein.

Basic & Clinical Pharmacology & Toxicology,2007,100, 334–338

Evaluation of the Effect of Cola Drinks on Bone Mineral Density and Associated Factors

Table 3.

Bone mineral density values of the groups (normal ± S.D.).

股骨	Subject groups		Control groups	
	Group 1 (n = 10) male	Group 2 (n = 10) female	Group 3 (n = 5) male	Group 4 (n = 5) female
BMD (total femur) (g/cm^2)	0.121 ± 0.009*†	0.133 ± 0.021*	0.150 ± 0.004	0.158 ± 0.005
BMC (total femur) (g)	0.139 ± 0.016*	0.138 ± 0.025	0.190 ± 0.010	0.165 ± 0.035
BMD (0.07 cm^2) (g/cm^2)	0.185 ± 0.023*†	0.202 ± 0.032*	0.234 ± 0.033	0.227 ± 0.021

*Statistically significant when compared to the control group of the same sex ($P < 0.05$); †Statistically significant when compared to group 2 ($P < 0.01$). BMC, bone mineral concentration; BMD, bone mineral density.

Basic & Clinical Pharmacology & Toxicology,2007,100, 334–338

Comparative effects of dietary supplementation with red grape juice and vitamin E on production of superoxide by circulating neutrophil NADPH oxidase in hemodialysis patients[1–3]

ABSTRACT

Background: Atherosclerotic cardiovascular disease is the most common cause of death among hemodialysis patients; it has been attributed to increased oxidative stress, dyslipidemia, malnutrition, and chronic inflammation. Activation of neutrophils is a well-recognized feature in dialysis patients, and superoxide-anion production by neutrophil NADPH oxidase may contribute significantly to oxidative stress.

Objective: The aim of the study was to compare the effects of dietary supplementation with concentrated red grape juice (RGJ), a source of polyphenols, and vitamin E on neutrophil NADPH oxidase activity and other cardiovascular risk factors in hemodialysis patients.

Design: Thirty-two patients undergoing hemodialysis were recruited and randomly assigned to groups to receive dietary supplementation with RGJ, vitamin E, or both or a control condition without supplementation or placebo. Blood was obtained at baseline and on days 7 and 14 of treatment.

50 mL concentrated RGJ twice a day, at lunch and at dinner, for 2 wk.

Results: RGJ consumption but not vitamin E consumption reduced plasma concentrations of total cholesterol and apolipoprotein B and increased those of HDL cholesterol. Both RGJ and vitamin E reduced plasma concentrations of oxidized LDL and ex vivo neutrophil NADPH oxidase activity. These effects were intensified when the supplements were used in combination; in that case, reductions in the inflammatory biomarkers intercellular adhesion molecule 1 and monocyte chemoattractant protein 1 also were observed.

Conclusions: Regular ingestion of concentrated RGJ by hemodialysis patients reduces neutrophil NADPH-oxidase activity and plasma concentrations of oxidized LDL and inflammatory biomarkers to a greater extent than does that of vitamin E. This effect of RGJ consumption may favor a reduction in cardiovascular risk. *Am J Clin Nutr* 2008;87:1053–61.

Spain

Concentrated red grape juice exerts antioxidant, hypolipidemic, and antiinflammatory effects in both hemodialysis patients and healthy subjects[1–3]

ABSTRACT

Background: Patients treated with hemodialysis frequently experience cardiovascular complications attributed, among other causes, to dyslipidemia, increased oxidative stress, and inflammation.

Objective: The aim of the study was to study the effects of dietary supplementation with concentrated red grape juice (RGJ), a source of polyphenols, on lipoprotein profile, antioxidant capacity, LDL oxidation, and inflammatory biomarkers.

Design: Twenty-six patients receiving hemodialysis and 15 healthy subjects were instructed to drink 100 mL RGJ/d for 14 d. Blood was drawn at baseline, twice during RGJ supplementation, and twice during the 6-mo follow-up period. As a control, 12 other randomly recruited hemodialysis patients not receiving RGJ were studied. Lipids, apolipoproteins, oxidized LDL, and antioxidant vitamins were measured in plasma. The bioavailability of RGJ polyphenols was assessed in healthy subjects.

Results: The maximum plasma concentration of quercetin was achieved 3 h after RGJ ingestion, which indicates that supplement-derived polyphenols are rapidly absorbed. In both healthy subjects and hemodialysis patients, RGJ consumption increased the antioxidant capacity of plasma without affecting concentrations of uric acid or ascorbic acid; reduced the concentration of oxidized LDL; and increased the concentration of cholesterol-standardized α-tocopherol. RGJ supplementation also caused a significant decrease in LDL-cholesterol and apolipoprotein B-100 concentrations, while increasing the concentrations of HDL cholesterol and apolipoprotein A-I. In a further study in hemodialysis patients, RGJ supplementation for 3 wk significantly reduced plasma monocyte chemoattractant protein 1, an inflammatory biomarker associated with cardiovascular disease risk.

Conclusion: Dietary supplementation with concentrated RGJ improves the lipoprotein profile, reduces plasma concentrations of inflammatory biomarkers and oxidized LDL, and may favor a reduction in cardiovascular disease risk. *Am J Clin Nutr* 2006;84:252–62.

Spain

Wine consumption and renal diseases: new perspectives

Investigations into the relation between wine consumption and kidney disease have been limited. Patients with chronic renal failure show accelerated atherosclerotic damage and, considering the well-known protective effect of wine on the cardiovascular system, moderate wine consumption might be advantageous. Oxidative stress and endothelial dysfunction, which are inter-related, play a role in the pathophysiology of many renal diseases, including acute and chronic renal failure. Ethanol and non-alcoholic wine components, especially polyphenols, influence oxidative balance and endothelial function. Although long-term alcohol abuse has been associated with many renal alterations in humans, in experimental studies wine polyphenols enhanced kidney antioxidant defenses, exerted protective effects against renal ischemia/reperfusion injury, and inhibited apoptosis of mesangial cells. Moreover, in diabetic patients the administration of moderate amounts of red wine and a polyphenol-enriched diet slowed the progression of diabetic nephropathy. Moreover, the unfavorable effect of ethanol on blood pressure control seems to be counterbalanced by polyphenol protective effects. There is convincing evidence of a beneficial effect of controlled wine consumption patients with renal disease, but controlled clinical trials are needed to confirm this hypothesis.

Nutrition 23 (2007) 598–602

Assessment of protein-energy malnutrition

- Visceral protein
  - Serum albumin > 4.0 g/dL
- Somatic protein and fat
  - Anthropometry：edema-free weight ; body mass index (BMI)
  - Subjective global assessment (SGA)
- Dietary protein intake
  - nPNA = urinary nitrogen excretion
  - Serum cholesterol
  - Dietary interview or diaries
- Serum bicarbonate concentration >22 mol/L
- CRP

Guideline 9. Association of level of GFR with nutritional status (2002) (non-dialyzed)

- The nutritional status of patients with chronic kidney disease should be *monitored* at regular intervals:
  - every 1 to 3 months for patients with GFR <30 mL/min/1.73 m^2 (CKD Stages 4 and 5) and
  - every 6 to 12 months for patients with GFR 30 to 59 mL/min/1.73 m^2 (CKD Stage 3).

K/DOQI Clinical Practice Guidelines for Chronic Kidney Disease. Am J Kidney Dis 2002; 39:S1.

慢性腎臟病的飲食技巧~

怎麼吃?

吃什麼?

吃多少?

蛋白質熱量營養不良–攝取量不足原因與對策

- 噁心、食慾不振
- 味覺改變
- 食物不好吃
- 不當飲食限制
- 牙齒不良(咀嚼功能不良)
- 腸胃障礙
- 採購製備困難
- 情緒問題
- 特殊飲食文化偏好
- 透析不全
- 住院率高

- 腸胃障礙
  - 採用質軟、好咀嚼、不塞牙縫的食物
  - 選用不油膩、易消化的食物
  - 增加餐次
  - 適當的鹹度
  - 正確飲食觀念

蛋白質熱量營養不良– 攝取量不足對策

- 噁心、食慾不振、味覺改變、食物不好吃
  - 適當的調味
  - 選體積小、高營養密度的食物
  - 少量多餐可增加進食量
  - 挑不噁心的時候進餐
  - 適量補充含鋅高的食物
  - 選想吃的食物
  - 進餐是一種任務，進食時保持愉快的之情及環境
  - 適當而有規律的運動，可增加食慾
  - 必要時應告知醫師開增加食慾的藥物幫助您。

六大類食物

- 奶類– 1 杯；含豐富的鈣質及蛋白質
  - 低蛋白質奶粉
- 五穀根莖類– 1.5~2 碗；提供糖類和少量蛋白質
  - 低氮澱粉 (提供糖類)
- 蛋豆魚肉類 2~3 份；含豐富蛋白質、部分脂肪
  - 磷離子、鉀離子
- 蔬菜類 – 3碟；含維生素、礦物質與纖維
  - 鉀離子
- 水果類 – 2個；含維生素、礦物質與纖維
  - 鉀離子
- 油脂類 – 4~6 湯匙；含脂肪
  - **Omega-3 ；monounsaturated fatty acid**

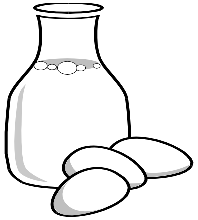
營養需求量－蛋白質(由氨基酸組成)

蛋白質的食物含量

食物來源	份量	蛋白質量
飯、麵	1碗飯 = 2碗麵=1個饅頭(中)	8 公克
牛奶	1杯= 240c.c.	8 公克
雞蛋	1個	7 公克
肉、魚類	1兩肉(約30公克)	7 公克
豆類	1杯豆漿(240c.c.) = 豆腐1塊(約80公克)=豆乾2塊	7 公克

高生理價蛋白質

- 定義: 含有足量『必需氨基酸』（共八種）且可提供良好的生長及氮平衡者
- 食物來源:
  - 雞蛋
  - 牛奶
  - 肉類
  - 魚類



- 若是純素者，最好能改為蛋奶素，蛋白質的品質會較完整
  - 素食者的蛋白質食物來源，包括：豆腐、豆乾、豆皮、豆包、素雞等

低生理價蛋白質

- 避免 “低生理價蛋白質”含量高之食品
- 豆類： 綠豆、紅豆、毛豆、蠶豆、豌豆仁
- 麵筋製品：麵筋、麵腸、烤麩
- 種子、堅果類： 花生、核桃、腰果、杏仁

低蛋白質飲食之熱量需求

蛋白質	主食類	豆蛋魚肉類	蔬菜類	水果類	油脂類	蛋白質	脂肪	醣類	熱量
40g	6	4	2	2	8	40	60	120	1100(1000)
35g	4	4	2	2	6	36	50	90	1000(800)
	6	3.5				37	45	120	950(850)
30g	4	3	2	2	5	29	40	90	800(700)

- low protein diet risk
  - 降低蛋白質容易;攝取足夠熱量較難
  - K/DOQI (stage 4~5)
    - <60 歲：35kcal/kg/d
    - ≥ 60 歲：30~35kcal/kg/d
  - 25~35Kcal

Wt.	50kg	60kg	70kg
protein	30	36	42
HBV prot.	15~22	19~27	21~31
熱量需求Kcal	1500	1800	2100

腎臟專科營養師基礎訓練班, 2006

低蛋白質飲食之熱量來源

- 米食 vs. 麵食
- 商業配方：
  - 單品補充
    - 油脂：高熱能(83.5%fat; 708kcal/100g)、
      益補(63.5%fat; 576kcal/100g; 40kcal/匙)、
    - 醣類：多卡(38kcal/匙)、糖飴(含Vit.&Min; .38kcal/匙)、
      粉飴(378kcal/100g; 34kcal/匙)
  - 完整營養
    - 三多低蛋白配方LPF
    - 亞培腎補納SUPLENA

		energy	Protein/g	Fat/g	CHO/g	Na	K	Ca	P
LPF	Can(8oz)	475	7.1(5.9%)	22.7(43%)	60.6(51%)	186	265	331	175
SUPLENA	100g	484	7.8(6.4%)	22.2(41.3%)	62.6(51.7%)	250	375	125	120

低蛋白質飲食之熱量來源

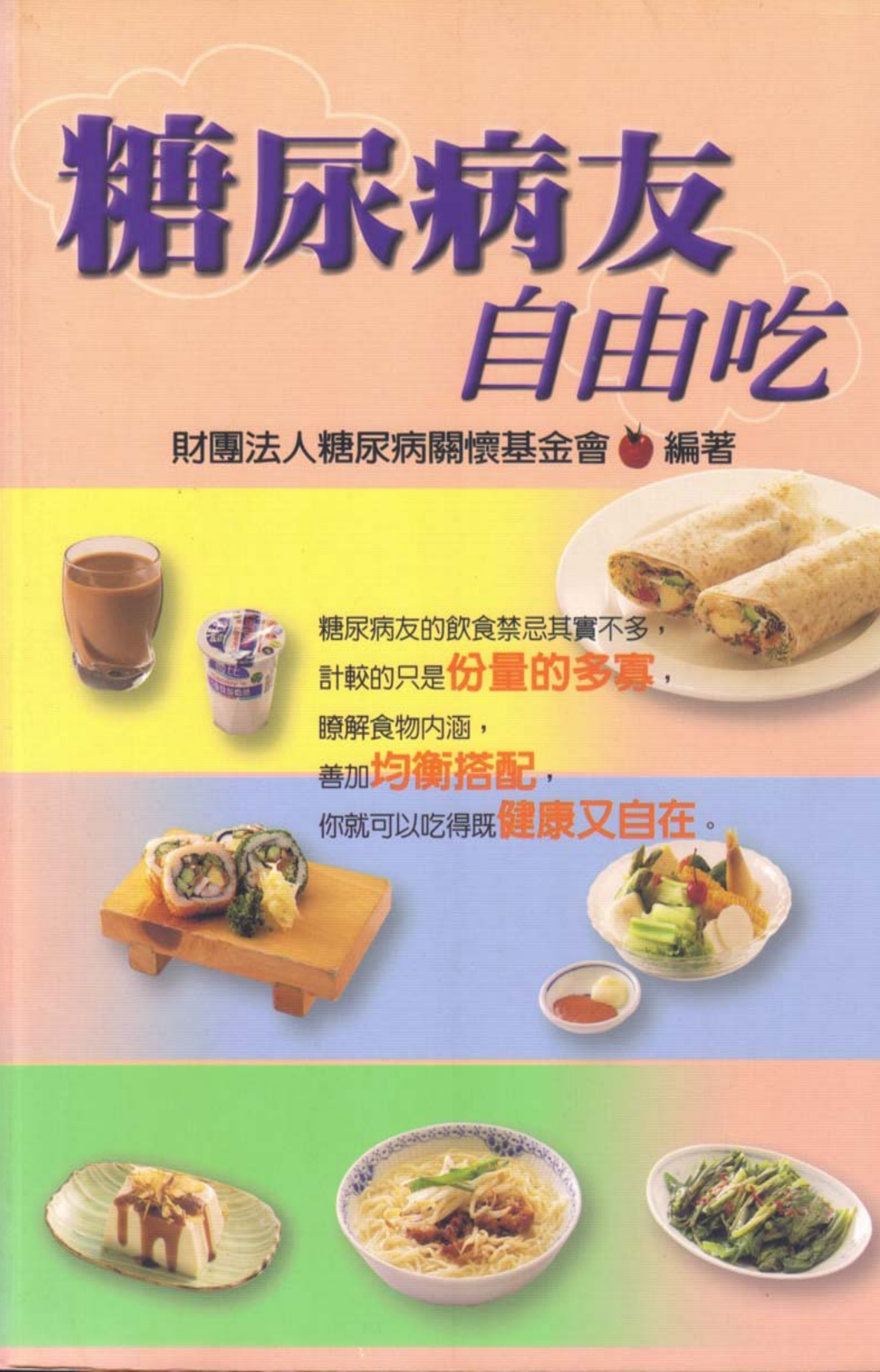
- 天然食物來源：
  - 植物油
  - 低蛋白質食物
  - 低氮澱粉：澄粉、蕃薯粉、太白粉、玉米粉、藕粉、西谷米、粉圓、粉皮

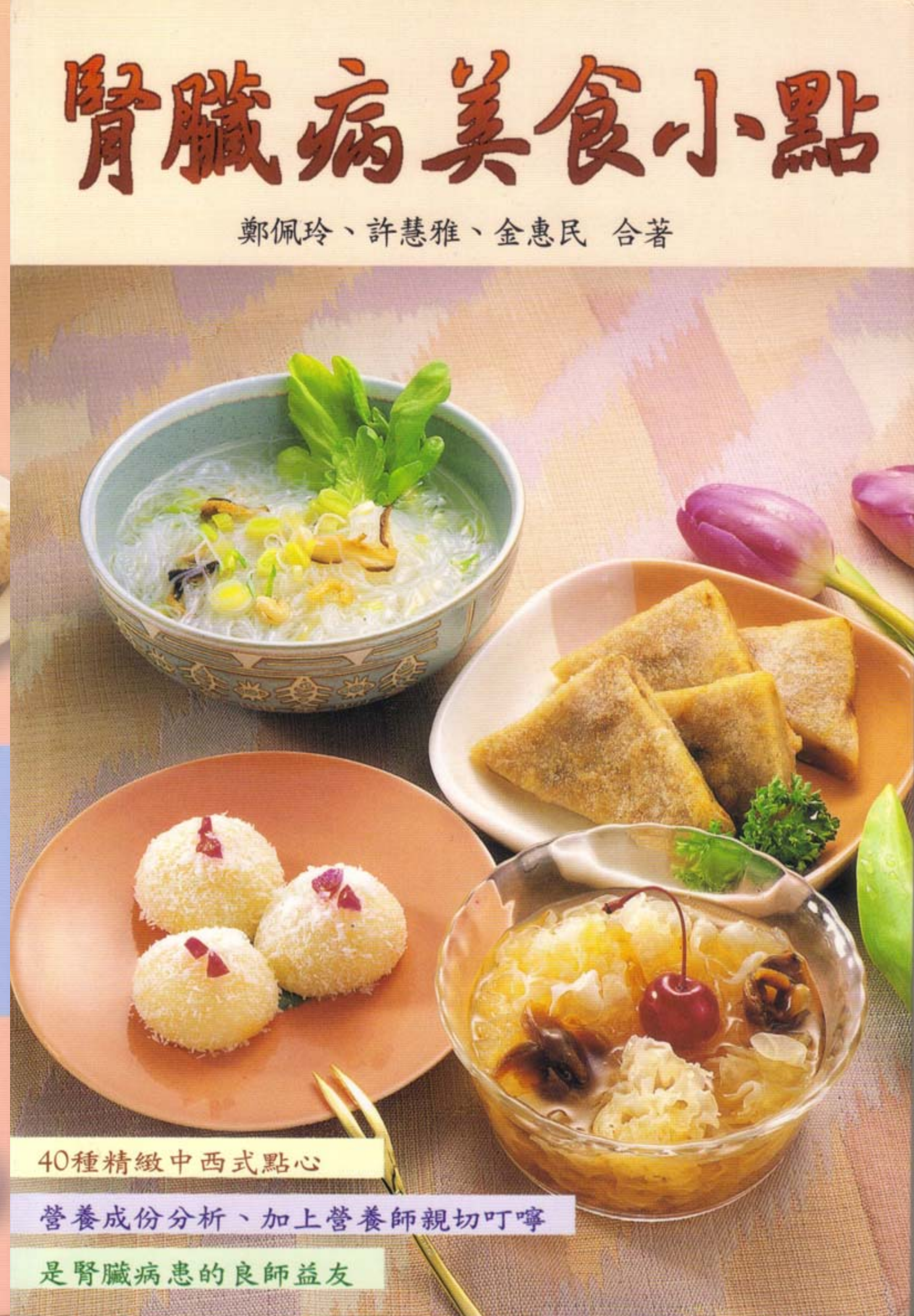
腎臟專科營養師基礎訓練班, 2006

慢性腎臟病友的外食飲食技巧

慢性腎臟病外食注意事項

- 熟記每份蛋、豆、魚、肉類及主食類之份量(米製品蛋白質低於麵製品)
- 選擇適合的用餐場所
- 依據自己的蛋白質攝取量取選用食物
- 避免磷、鉀、鈉含量高的食物與湯品(若需使用磷結合劑, 不要忘記)
- 攝取足夠的熱量

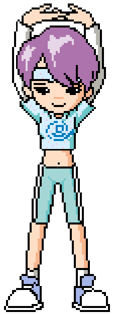

糖尿病友
自由吃
財團法人糖尿病關懷基金會 編著
糖尿病友的飲食禁忌其實不多，
計較的只是份量的多寡，
瞭解食物內涵，
善加均衡搭配，
你就可以吃得既健康又自在。

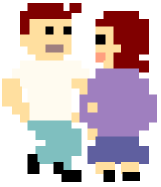

腎臟病美食小點
鄭佩玲、許慧雅、金惠民 合著
40種精緻中西式點心
營養成份分析、加上營養師親切叮嚀
是腎臟病患的良師益友

良好生活習慣~健康的條件

- 均衡的飲食
- 適度的運動
- 充足的睡眠
- 足夠的水分
- 正當的休閒
- ……

慢性腎臟病友還需配合醫囑
控制病情(藥物、檢查...)





健康的條件-健康的生活型態

- 均衡的飲食
- 適度的運動
- 充足的睡眠
- 足夠的水分
- 正當的休閒
- ……

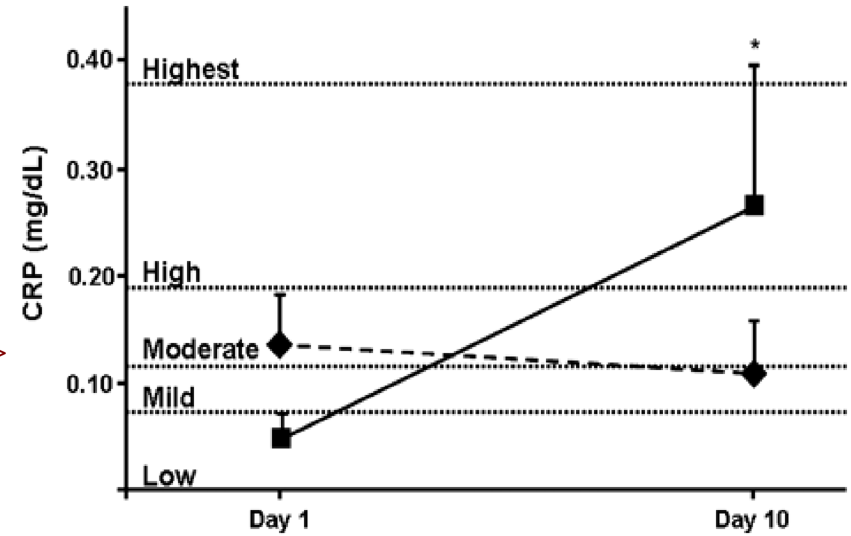


Figure 3. Mean (± SEM) changes in plasma C-reactive protein concentration (CRP) in four subjects undergoing 10 days of partial sleep deprivation (■; 4.2 h of sleep a night) and five control subjects (◆; 8.2 h of sleep a night). * Denotes significant (*P*<0.05) difference between day 1 and day 10 values. Dotted lines represents the boundaries for cardiovascular risk quintiles of CRP concentration derived from analysis of >5,000 apparently healthy Americans (from Ridker[48] Circulation. 2001;103:1813–1818). Data from Meier-Ewert et al.[53] (J Am Coll Cardiol. 2004;43:678–683).


Thanks for your attention!