Les régimes pauvres en protéines supplémentés par les céto-analogues des Acides Aminés: Pour qui ? Comment?

Low protein diet supplemented with ketoanalogues of amino acids in patients with CKD

Philippe Chauveau
Aurad-Aquitaine et CHU de Bordeaux

FIGURE 1: The extensive literature on the low-protein diet issue in chronic kidney disease.

Kidney Int Suppl. 1987 Oct;22:S67-71.

Comparison of ketoacids and low protein diet on advanced chronic renal failure progression.

Jungers P[1], Chauveau P, Ployard F, Lebkiri B, Ciancioni C, Man NK.

Blood Purification 6: 299–314 (1988)

© 198
0253–5068/8

Amino Acids and Keto Acids in the Treatment of Chronic Renal Failure[1]

Paul Jungers, Philippe Chauveau

Department of Nephrology and INSERM U. 90, Necker Hospital, Paris, France

ACADÉMIE DE PARIS
UNIVERSITÉ RENÉ DESCARTES

FACULTÉ DE MÉDECINE
NECKER-ENFANTS-MALADES

N°

MEMOIRE
pour le
Certificat d'Etudes Spéciales
de
NEPHROLOGIE
par

PHILIPPE CHAUVEAU
NÉ LE 29 NOVEMBRE 1954 À VANVES (92)

PRÉSENTÉ LE 30 SEPTEMBRE 1985

UTILISATION DES CÉTO-ANALOGUES DES ACIDES AMINÉS ESSENTIELS DANS LE TRAITEMENT DE L'INSUFFISANCE RÉNALE CHRONIQUE DE DEGRÉ MAJEUR. ESSAI PROSPECTIF CONTROLÉ CHEZ 19 PATIENTS.

TRAVAIL DU DÉPARTEMENT DE NÉPHROLOGIE
SOUS LA DIRECTION DU PROFESSEUR P. JUNGERS

1985 – 2018: retour vers le futur
« back to the future »

Protein intake: Quality and/or quantity Evidences

- Protein intake tend to increase in general population
- High protein intake is associated with decline in renal function
- Quality of protein influence this relationship
- Recommended DPI for general population is 0.8 g/kg/d
- Mean DPI is higher than 0.8 even in stage 4+

Associations of processed meat and unprocessed red meat intake with incident diabetes: the Strong Heart Family Study[1–4]

Amanda M Fretts, Barbara V Howard, Barbara McKnight, Glen E Duncan, Shirley AA Beresford, Mihriye Mete, Sigal Eilat-Adar, Ying Zhang, and David S Siscovick

CLINICAL EPIDEMIOLOGY www.jasn.org

Red Meat Intake and Risk of ESRD

Quan-Lan Jasmine Lew,* Tazeen Hasan Jafar,†‡ Hiromi Wai Ling Koh,§ Aizhen Jin,‖ Khuan Yew Chow,‖ Jian-Min Yuan,¶** and Woon-Puay Koh†§

*Singhealth Polyclinics, Singapore; †Duke-NUS Medical School, Singapore; ‡Department of Renal Medicine, Singapore General Hospital, Singapore; §Saw Swee Hock School of Public Health, National University of Singapore, Singapore; ‖National Registry of Diseases Office, Health Promotion Board, Singapore; ¶Division of Cancer Control and Population Sciences, University of Pittsburgh Cancer Institute, Pittsburgh, Pennsylvania; and **Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pennsylvania

CLINICAL EPIDEMIOLOGY www.jasn.org

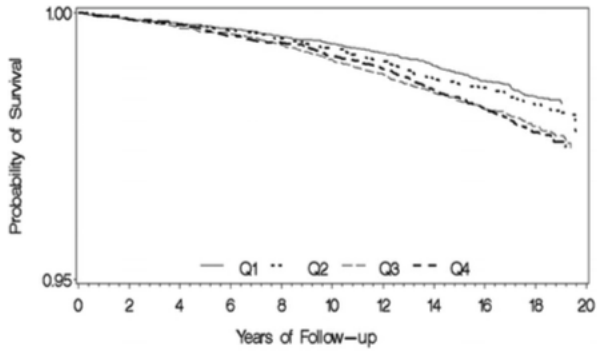
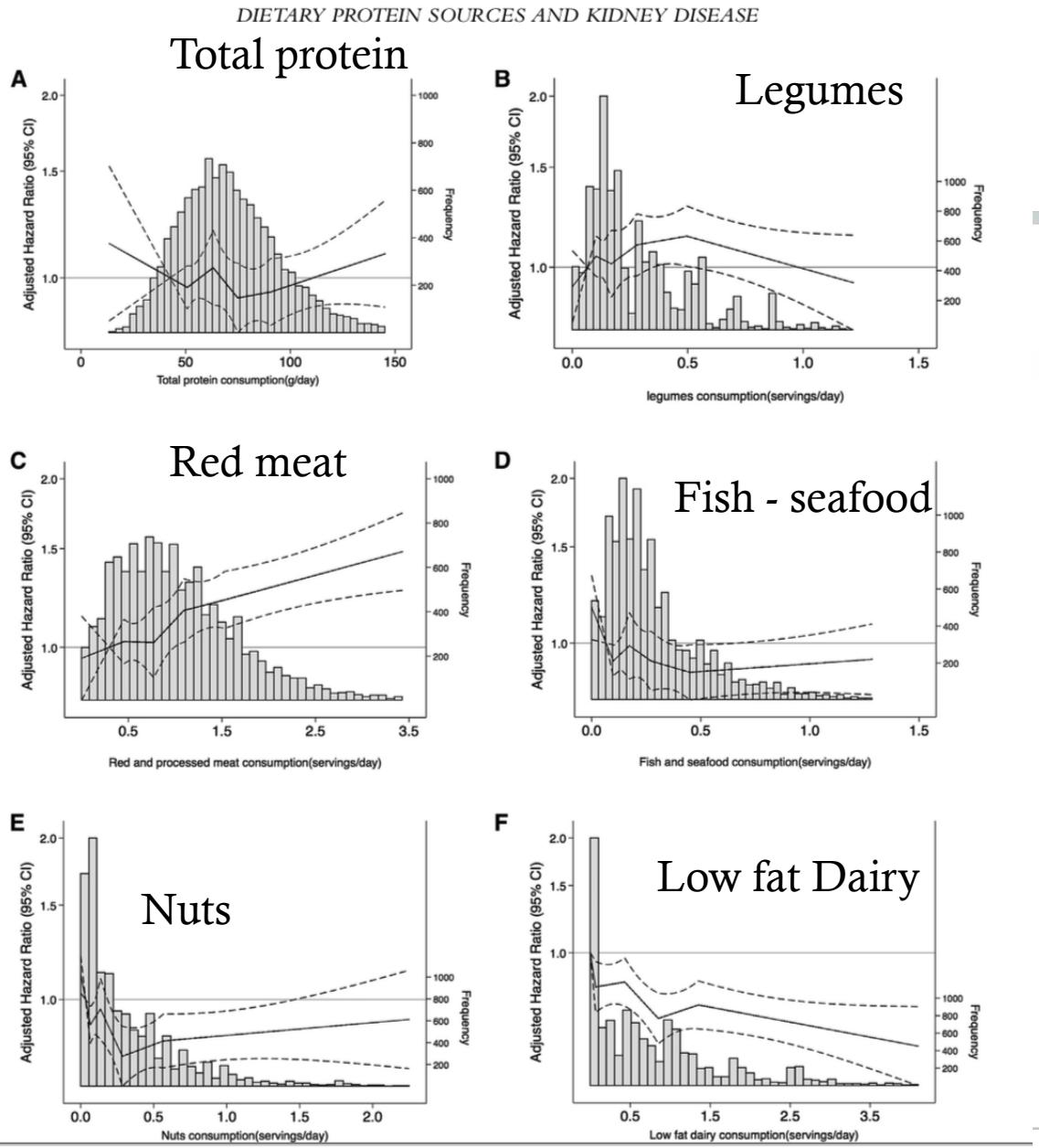


Figure 1. Kaplan–Meier survival curves show probability of ESRD-free survival according to the quartile (Q) intake of red meat. Log-rank *P* value for difference in survival was <0.001.

Dietary Protein Sources and Risk for In Chronic Kidney Disease: Results From the Atherosclerosis Risk in Communities (ARIC) Study

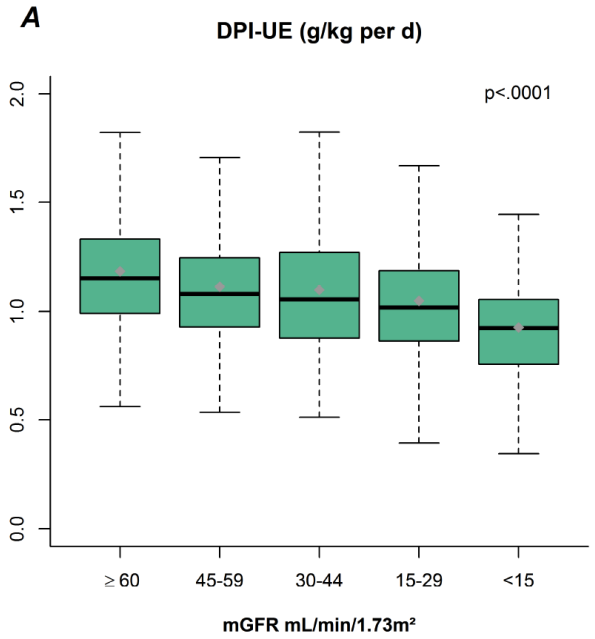
Bernhard Haring, MD, MPH,★ Elizabeth Selvin, PhD, MPH,†'‡ Menglu Liang, ScM, MPH,†'‡ Josef Coresh, MD, PhD, MHS,†'‡ Morgan E. Grams, MD, PhD, MHS,†'§ Natalia Petruski-Ivleva, MS,¶ Lyn M. Steffen, PhD, MPH, RD,★★ and Casey M. Rebholz, PhD, MS, MPH†'‡

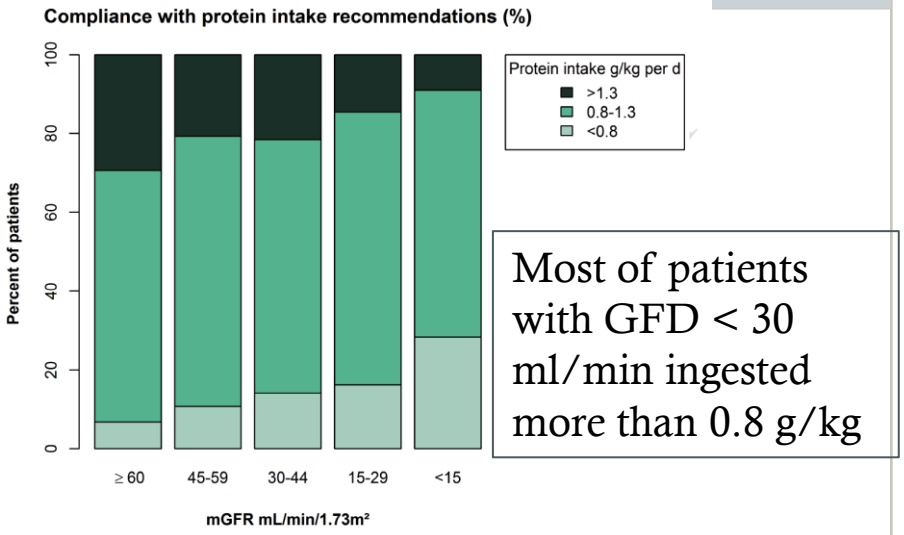


- ARIC study
- 11 952 subjects
- 44-66 years old
- GFR > 60 ml/min
- F-Up 23 yrs
- HR
  - Decrease in GFR > 25%
  - Stage 3 +

Cohorte Nephrotest

Metzger et al. KI report Jan 2018

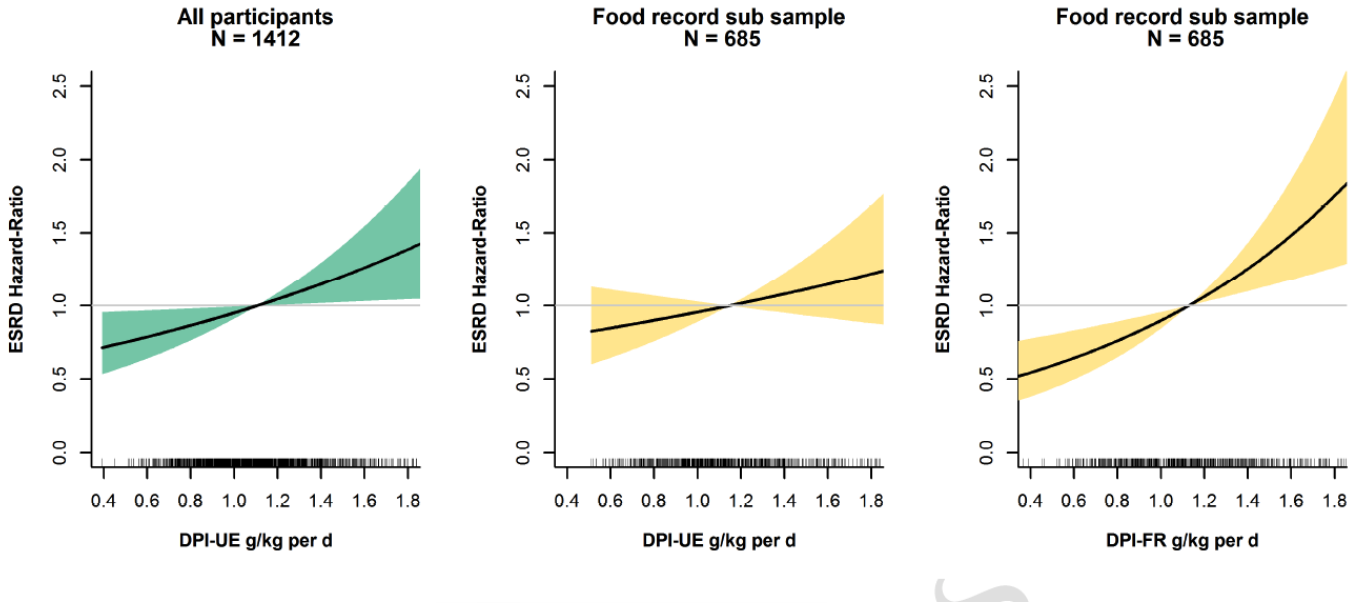




Most of patients with GFD < 30 ml/min ingested more than 0.8 g/kg

- 1595 patients,cohorte 2000-2010
  - DPI using Maroni formula, 24 hurinary urea (+ 784 7-days dietary recall)
  - ^{51}Cr-EDTA (mGFR)
  - Endpoint: ESRD dialysis or transplantation

Cohorte Nephrotest

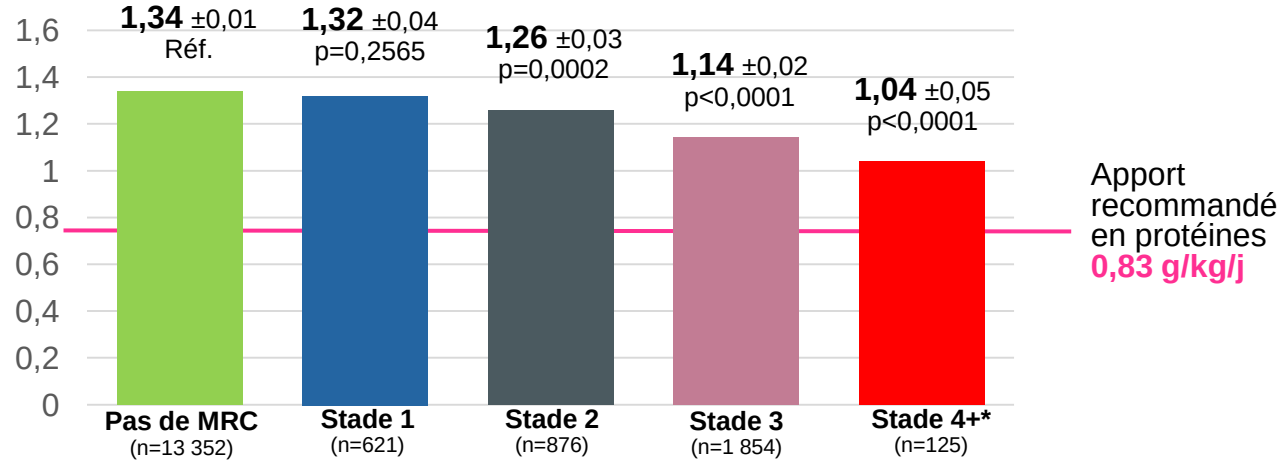


- -> ESRD hazart ratio and protein intake

NHANES study
Mean protein intake according to GFR stage

- **Cohorte NHANES [National Health and Nutrition Examination Survey] USA**
  - **n=21 456 adultes américains ≥ 20 ans**, **suivis de 2001 à 2008**, avec enquête diététique, entretiens, informations socio-économiques, examen médical et examens biologiques…
  - **Analyse transversale** réalisée sur **n=16 872 adultes non-dialysés** disposant des données nécessaires à l'analyse
  - **Objectif** : Evaluer s'il existe une association entre apports protéiques alimentaires et MRC ou non, et si le stade de l'IRC est associé à des différences dans les apports protéiques

Apport journalier en protéines selon enquête - rapporté au poids idéal (g/kg poids idéal/j)
En fonction de l'existence ou non d'une MRC, et le cas échéant du stade de l'IRC



*Inclut des patients avec un stade 5 non dialysé

➢ **Protein intake is higher than recommendation at all stage of CKD**

Moore LW, *et al.* The mean dietary protein intake at different stages of chronic kidney disease is higher than current guidelines. Kidney Int 2013;83:724-32.

Protein intake and CKD

- Recommendations
- LPD or supplemented very low protein diet
  - Meta-analysis
- Why and when should we prescribe amino-acids supplementation?
- Nutritional safety

Need for an AA supplementation

Population générale

Apport en protéines recommandé

0,8 g/kg/j

Stades 4 et 5 de l'IRC, en pré-dialyse

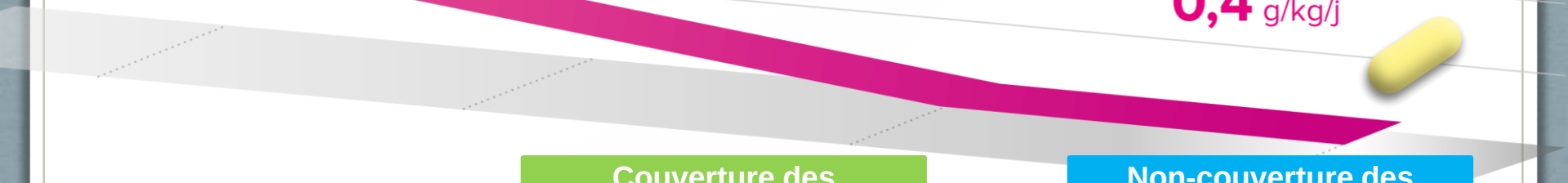
Apports réduits en protéines recommandés

Alimentation hypoprotidique

0,6 g/kg/j

Alimentation hypoprotidique « stricte »

0,4 g/kg/j



Couverture des besoins en AA essentiels par l'apport en protéines

- **Si 50% des protéines apportées sont de haute valeur biologique**
- **Sinon, nécessité d'une supplémentation en AA essentiels**

Non-couverture des besoins en AA essentiels par l'apport en protéines

- **Nécessité d'une supplémentation en AA essentiels**

Fouque M, Aparicio M. Eleven reasons to control the protein intake of patients with chronic kidney disease. Nat Clin Pract Nephrol 2007; 3:383-92.

Protein intake and CKD (g/kg/j) Recommendations

	ESPEN *Clinical Nutrition* 2000	K/DOQI *American Journal of Kidney Diseases* 2000	KDIGO *Kidney International Supplements* 2012	Consensus Experts *Journal of Renal Nutrition* 2012	ISRNM *Kidney International* 2013	Review 2016 *The Lancet Diabetes & Endocrinology* 2016
Stade 3	**0,55 - 0,6**		**< 1,3**	3a : **0,8** 3b : **0,6-0,7**	**0,6 - 0,8**	**0,6 - 0,8**
Stade 4	**0,55 - 0,6** *ou* **0,28** **+ Céto-A***	**0,6 - 0,75**	**0,8**	**0,6** *ou* **0,3 - 0,4** **+ Céto-A***	**0,6 - 0,8**	**0,6 - 0,8** *ou* **< 0,6** **+ Céto-A***
Stade 5	**0,55 - 0,6** *ou* **0,28** **+ Céto-A***	**0,6 - 0,75**	**0,8**	**0,6** *ou* **0,3 - 0,4** **+ Céto-A***	**0,6 - 0,8**	**0,6 - 0,8** *ou* **< 0,6** **+ Céto-A***
Dialyse		**1,2**			**> 1,2**	**> 1** (HémoD) **> 1,2** (DPéri)

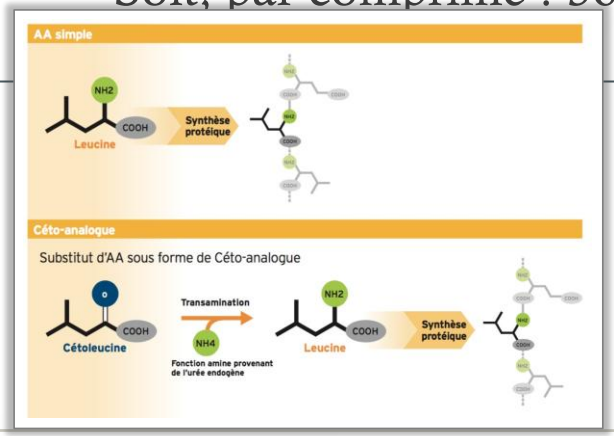
*** Supplémentation en céto-analogues (0,1 g/kg/j)**

Ketosteril®

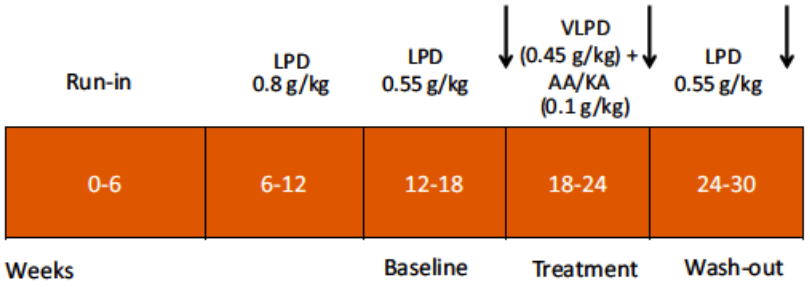
- Chaque comprimé contient :
  - 67 mg d'un sel calcique du cétoanalogue de l'isoleucine
  - 101 mg d'un sel calcique du cétoanalogue de la leucine
  - 86 mg d'un sel calcique du cétoanalogue de la valine
  - 68 mg d'un sel calcique du cétoanalogue de la phénylalanine
  - 59 mg d'un sel calcique de l'hydroxyanalogue de la méthionine

  + lysine (105 mg), thréonine (53 mg), tryptophane (23 mg), histidine (38 mg), tyrosine (30 mg) + AA non essentiels.

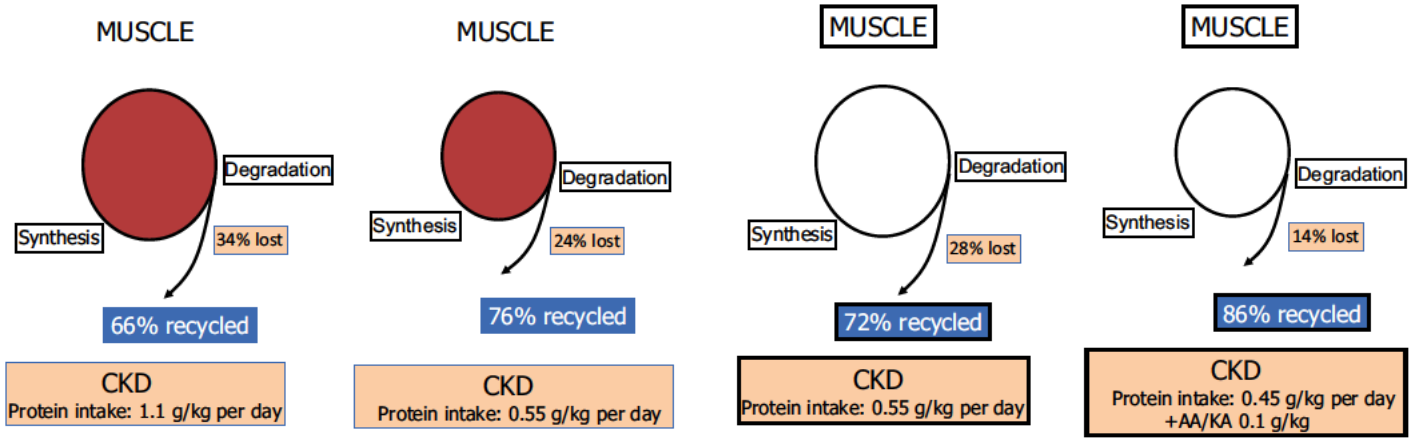
  Soit, par comprimé : 36mg d'azote et 50 mg de calcium



Keto analogs are simple carbon chains, without amine group
They are regenerated in AA by transamination, from NH4 from endogenous urea



TRANSLATIONAL RESEARCH

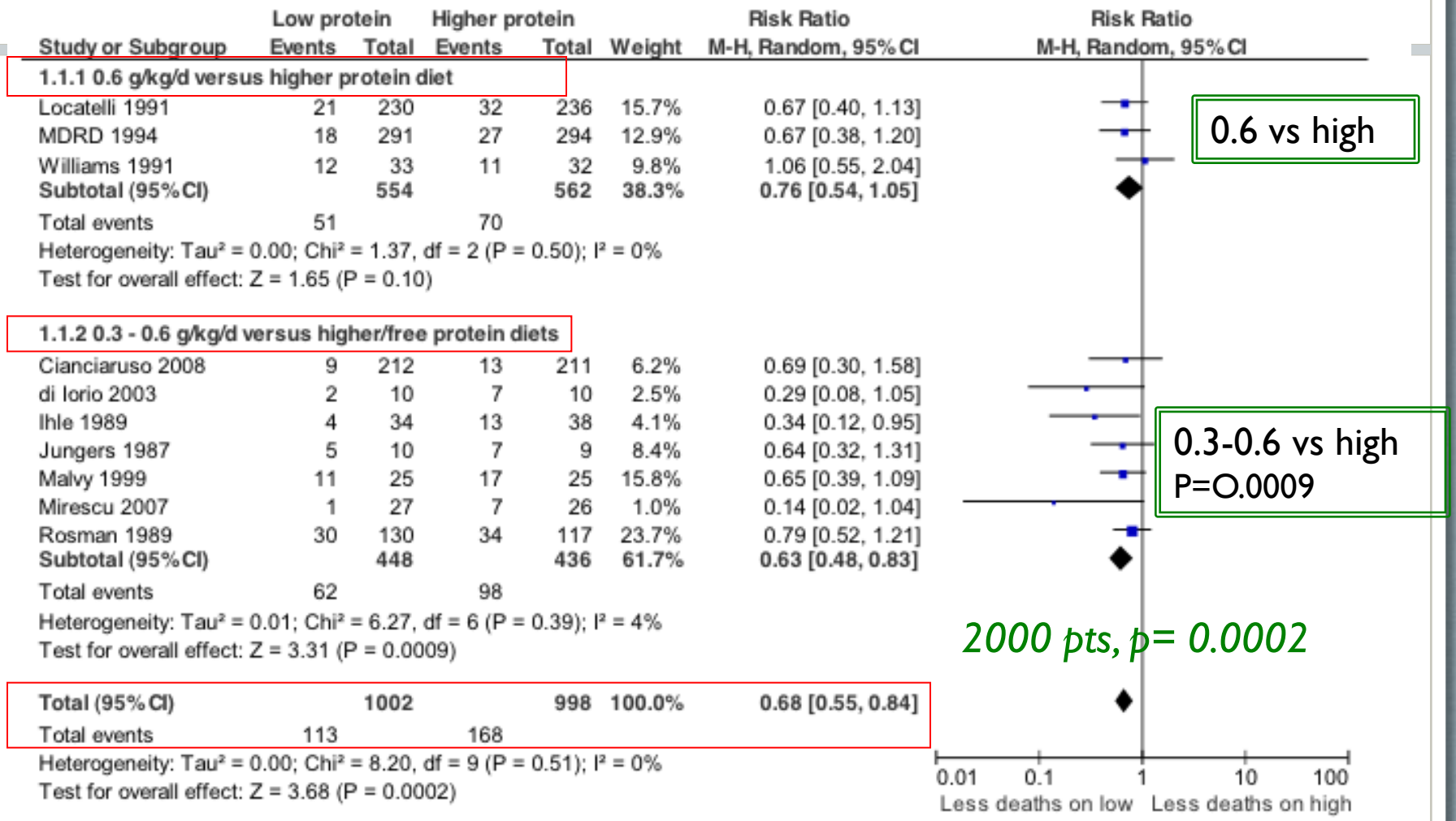


Forearm perfusion method associated with the kinetics of 2H-phenylalanine in patients with CKD
6 Patients. Age: 64 years, DFG: 17 (protocol 1) to 12 (Protocol 2)

During sVLPD 86% of amino-acids are recycled even in CKD stage 5

Garibotto et al. KI report 2018

Cochrane systematic review and meta-analysis



Study or Subgroup	Low protein Events	Total	Higher protein Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
1.1.1 0.6 g/kg/d versus higher protein diet						
Locatelli 1991	21	230	32	236	15.7%	0.67 [0.40, 1.13]
MDRD 1994	18	291	27	294	12.9%	0.67 [0.38, 1.20]
Williams 1991	12	33	11	32	9.8%	1.06 [0.55, 2.04]
Subtotal (95% CI)		**554**		**562**	**38.3%**	**0.76 [0.54, 1.05]**
Total events	51		70			
Heterogeneity: Tau² = 0.00; Chi² = 1.37, df = 2 (P = 0.50); I² = 0%						
Test for overall effect: Z = 1.65 (P = 0.10)						
1.1.2 0.3 - 0.6 g/kg/d versus higher/free protein diets						
Cianciaruso 2008	9	212	13	211	6.2%	0.69 [0.30, 1.58]
di Iorio 2003	2	10	7	10	2.5%	0.29 [0.08, 1.05]
Ihle 1989	4	34	13	38	4.1%	0.34 [0.12, 0.95]
Jungers 1987	5	10	7	9	8.4%	0.64 [0.32, 1.31]
Malvy 1999	11	25	17	25	15.8%	0.65 [0.39, 1.09]
Mirescu 2007	1	27	7	26	1.0%	0.14 [0.02, 1.04]
Rosman 1989	30	130	34	117	23.7%	0.79 [0.52, 1.21]
Subtotal (95% CI)		**448**		**436**	**61.7%**	**0.63 [0.48, 0.83]**
Total events	62		98			
Heterogeneity: Tau² = 0.01; Chi² = 6.27, df = 6 (P = 0.39); I² = 4%						
Test for overall effect: Z = 3.31 (P = 0.0009)						
Total (95% CI)		**1002**		**998**	**100.0%**	**0.68 [0.55, 0.84]**
Total events	113		168			
Heterogeneity: Tau² = 0.00; Chi² = 8.20, df = 9 (P = 0.51); I² = 0%						
Test for overall effect: Z = 3.68 (P = 0.0002)						

Fuque et al, Cochrane Database Syst Rev, Issue 3, 2009

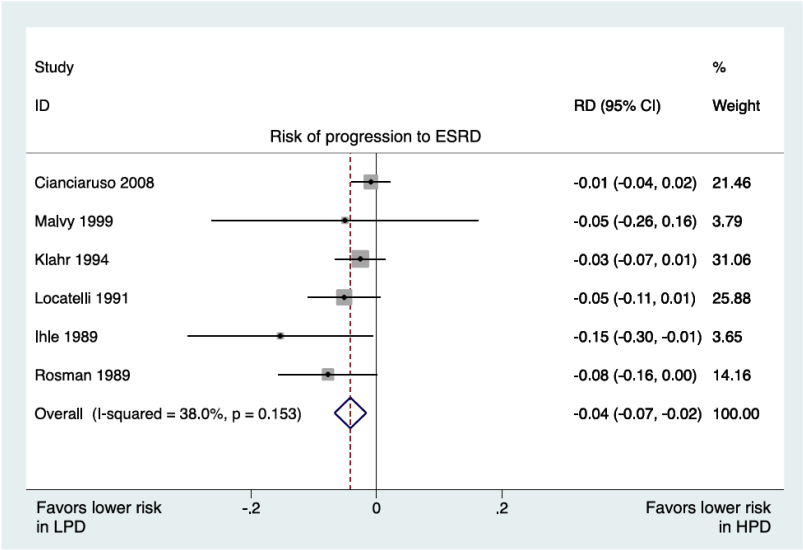
ORIGINAL ARTICLE

Journal of Cachexia, Sarcopenia and Muscle (2017)
Published online in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/jcsm.12264

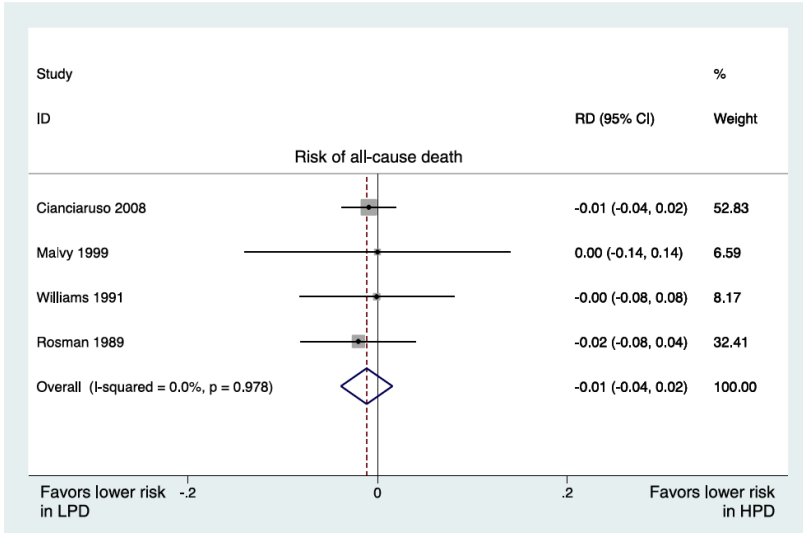
Low-protein diet for conservative management of chronic kidney disease: a systematic review and meta-analysis of controlled trials

Connie M. Rhee[1†], Seyed-Foad Ahmadi[1,2†], Csaba P. Kovesdy[3,4] & Kamyar Kalantar-Zadeh[1,2,5,6,7*]

A



B



Long-term outcome on renal replacement therapy in patients who previously received a keto acid–supplemented very-low-protein diet[1,2]

Philippe Chauveau, Lionel Couzi, Benoit Vendrely, Valérie de Précigout, Christian Combe, Denis Fouque, and Michel Aparicio

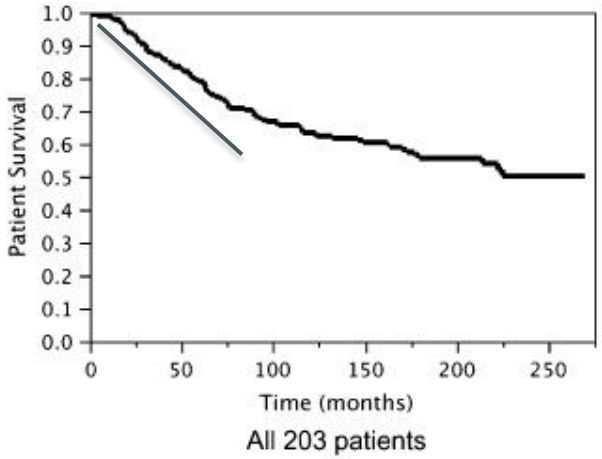


FIGURE 2. Survival curve of the whole study population treated with hemodialysis and/or transplantation after stopping treatment with a supplemented very-low-protein diet.

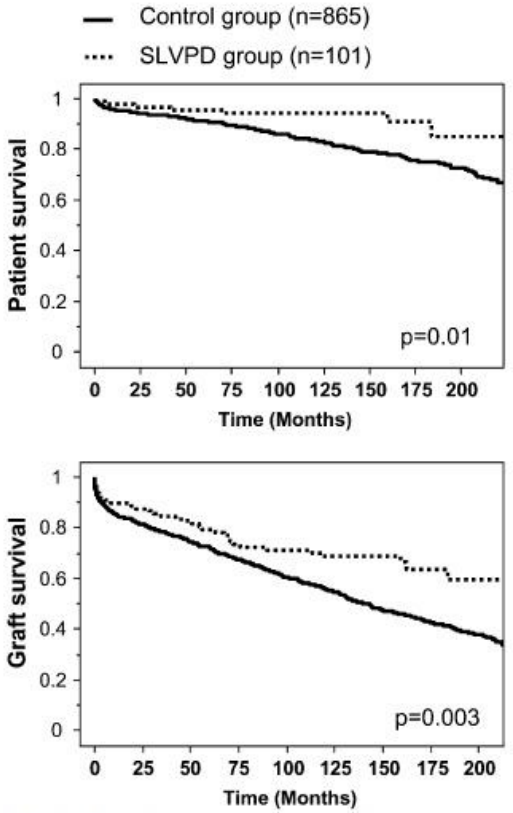


FIGURE 4. Patient survival and graft survival in patients in the supplemented very-low-protein diet (SVLPD; n = 101) and control (n = 865) groups. The survival rate was higher in the SLVPD group than in the control group (P = 0.01, log-rank test).

Safety: when a sufficient energy intake is provided malnutrition is rarely observed in most of the published study

12 major fields impact by a low protein diet

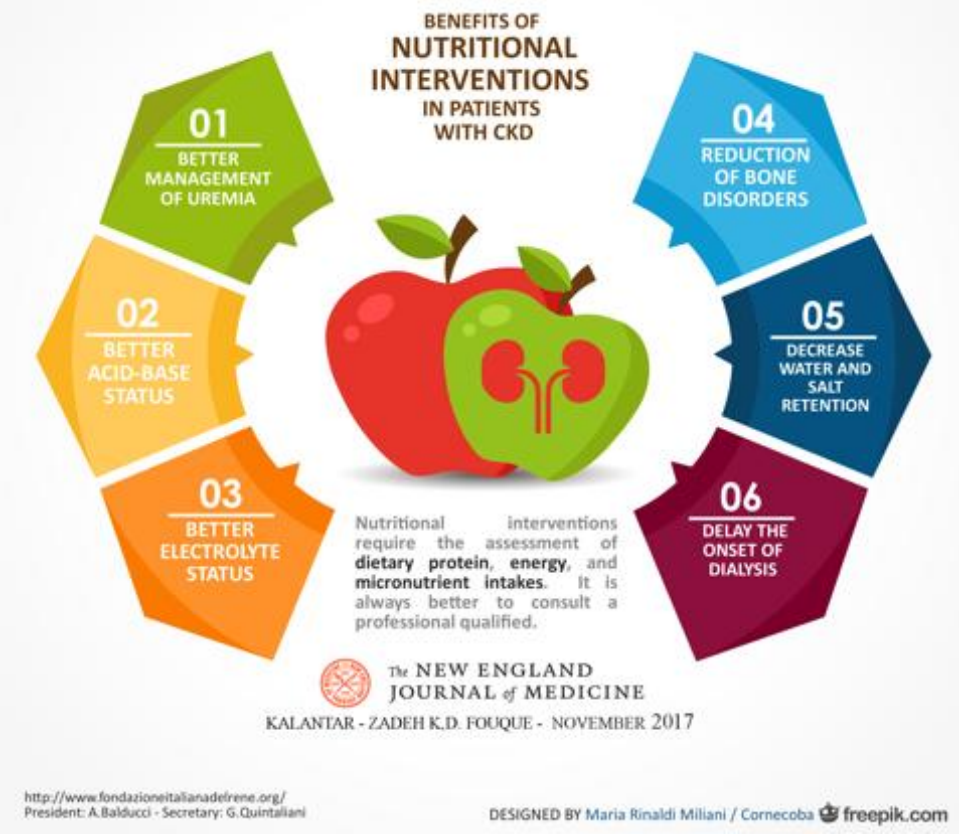


Table 1. Potential Benefits and Challenges of a Low-Protein Diet (LPD) in the Nutritional Management of Chronic Kidney Disease (CKD).*

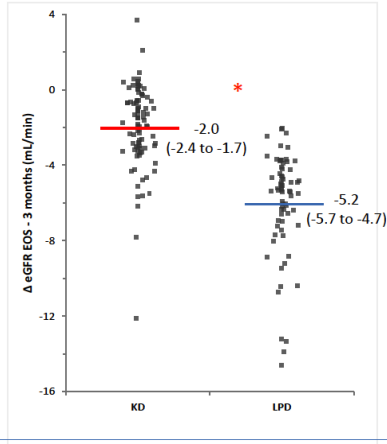
Measure	Potential Benefits of LPD	Challenges and Risks of LPD	Comments
CKD progression	Synergistic effect with angiotensin-pathway modulators to lower intraglomerular pressure†	In first several months, slight drop in GFR may be observed, as shown in MDRD study‡	Inconclusive results in MDRD study, but small effect size in meta-analyses§
Proteinuria	Consistent antiproteinuric effect, which may mitigate hypo-albuminemia	LPD is contrary to notion that DPI must be increased to replace urinary protein loss	Some data suggest that even larger effect may be achieved with DPI of <0.6 g/kg/day
Uremia management and deferral of dialysis	Supported by consistent and biologically plausible data for almost a century	Unlikely to worsen uremia but potential risk of resurfacing or exacerbating PEW	Patients at increased risk for PEW may benefit from supplements (e.g., EAA or KA)
Metabolic acidosis	H^+ generation decreased in proportion to reduction in DPI, especially with larger proportion of plant-based food	The need for >50% HBV protein may prompt higher intake of non–plant-based foods that are more acidogenic	Although >50% HBV protein is recommended, the remainder can be from plant-based foods
Mineral and bone disease	The lower phosphorus content of LPD improves measures of mineral bone disease, including sHPT and high FGF-23	Higher calcium content in some KA preparations may increase calcium load	Additional improvements in bone health are possible by alleviating acidosis
PEW	Ameliorating hypoalbuminemia in patients with proteinuria may help neutralize circulating inflammatory compounds	Weight loss may occur; the habit of LPD intake may continue after starting thrice-weekly hemodialysis, when higher protein intake is recommended	Half of dietary protein source should be HBV protein; liberalize diet during correction of PEW
Cardiovascular and metabolic health	Lower protein intake is associated with lower dietary salt and saturated fat intake and may be less atherogenic, given higher proportion of plant-based food	Higher dietary fat intake (to achieve DEI of 30–35 kcal/kg/day) may confound the goal of achieving a heart-healthy diet	Higher proportions of unsaturated fat and complex carbohydrates recommended
Glycemic control and insulin response	Improvement in insulin resistance is likely	With LPD or VLPD, higher carbohydrate and fat intake (to achieve DEI 30–35 kcal/kg/day) may worsen glycemic control	Given increased insulin half-life and "burnt-out diabetes" with CKD progression, preventing hypoglycemic episodes is prudent
Quality of life and adherence to LPD	Enhanced patient-centeredness, given that many patients seek nutritional therapies and dietary advice	Challenges with adherence; diet fatigue, poor palatability, and cravings reported	Recommend creative recipes and strategies to engage patients
Mortality	There are no convincing data to suggest reduced mortality, although dialysis deferral is a potential mechanism, given high mortality during early dialysis	Increased mortality highly unlikely with DPI of 0.6–0.8 g/kg/day unless severe PEW emerges and is uncorrected	Consider supplements or other corrective strategies whenever PEW is suspected or diagnosed
Hypertension management	MDRD and other data suggest improved BP control	Reduction in BP is more likely a result of concomitant lower salt intake than of LPD itself	Higher potassium intake from more plant-based foods may be a potential mechanism
Microbiome modulation	Improved microbiome profile may be achieved through reduced uremic toxin generation	Possibility of promoting unfavorable microbiome milieu cannot be excluded	Uremia itself can lead to unfavorable microbiome

* A diet that provides 0.6 to 0.8 g of protein per kilogram of body weight per day is recommended most frequently. BP denotes blood pressure, DEI dietary energy intake, DPI dietary protein intake, EAA essential amino acids, FGF-23 fibroblast growth factor 23, GFR glomerular filtration rate, HBV high biologic value, KA ketoacids, MDRD Modification of Diet in Renal Disease, PEW protein–energy wasting, sHPT secondary hyperparathyroidism, and VLPD very-low-protein diet.
† Angiotensin-pathway modulators include angiotensin-converting–enzyme inhibitors and angiotensin-receptor blockers. See Tables S4 and S5 and Box S2 in the Supplementary Appendix for additional data and examples of meals.
‡ The MDRD study data were originally presented by Klahr et al.,[12] with an additional analysis by Levey et al.[13]
§ A meta-analysis by Kasiske et al. showed a significant but rather small effect size of an LPD in slowing the CKD progression rate.[14]

Table 1. Potential Benefits and Challenges of a Low-Protein Diet (LPD) in the Nutritional Management of Chronic Kidney Disease (CKD).*

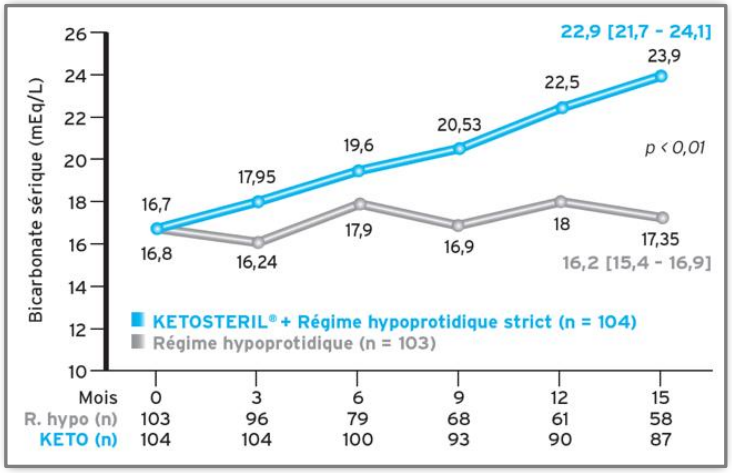
Measure	Potential Benefits of LPD	Challenges and Risks of LPD	Comments
CKD progression	Synergistic effect with angiotensin-pathway modulators to lower intraglomerular pressure†	In first several months, slight drop in GFR may be observed, as shown in MDRD study‡	Inconclusive results in MDRD study, but small effect size in meta-analyses§
Proteinuria	Consistent antiproteinuric effect, which may mitigate hypo-albuminemia	LPD is contrary to notion that DPI must be increased to replace urinary protein loss	Some data suggest that even larger effect may be achieved with DPI of <0.6 g/kg/day
Uremia management and deferral of dialysis	Supported by consistent and biologically plausible data for almost a century	Unlikely to worsen uremia but potential risk of resurfacing or exacerbating PEW	Patients at increased risk for PEW may benefit from supplements (e.g., EAA or KA)
Metabolic acidosis	H^+ generation decreased in proportion to reduction in DPI, especially with larger proportion of plant-based food	The need for >50% HBV protein may prompt higher intake of non–plant-based foods that are more acidogenic	Although >50% HBV protein is recommended, the remainder can be from plant-based foods
Mineral and bone disease	The lower phosphorus content of LPD improves measures of mineral bone disease, including sHPT and high FGF-23	Higher calcium content in some KA preparations may increase calcium load	Additional improvements in bone health are possible by alleviating acidosis
PEW	Ameliorating hypoalbuminemia in patients with proteinuria may help neutralize circulating inflammatory compounds	Weight loss may occur; the habit of LPD intake may continue after starting thrice-weekly hemodialysis, when higher protein intake is recommended	Half of dietary protein source should be HBV protein; liberalize diet during correction of PEW

Median changes in eGFR (95% CI) during the study

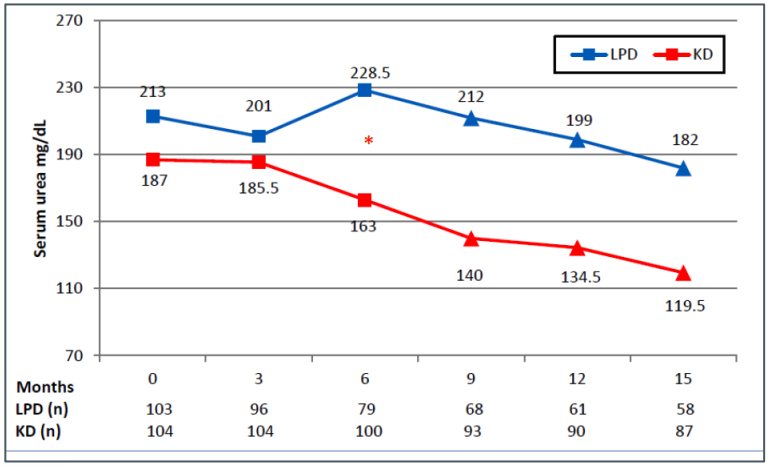


Metabolic effects during sVLPD: Garneata study. JASN 2016

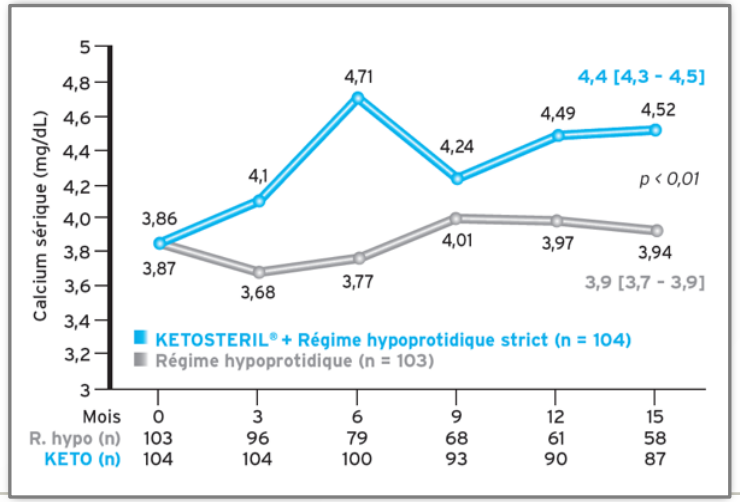
Bicarbonate



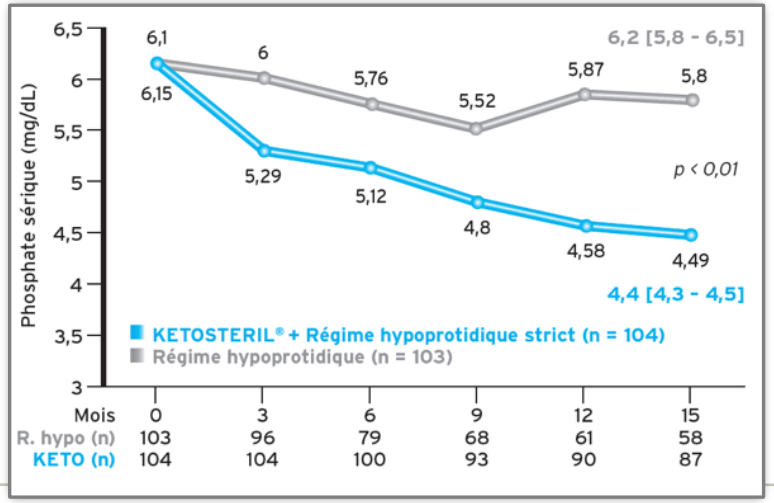
Urea



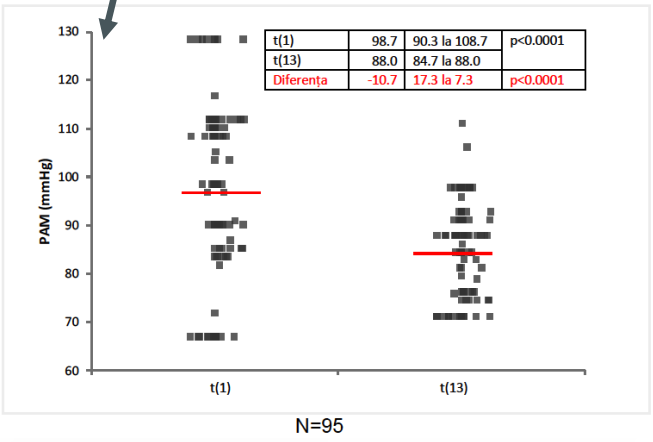
Calcium



Phosphate



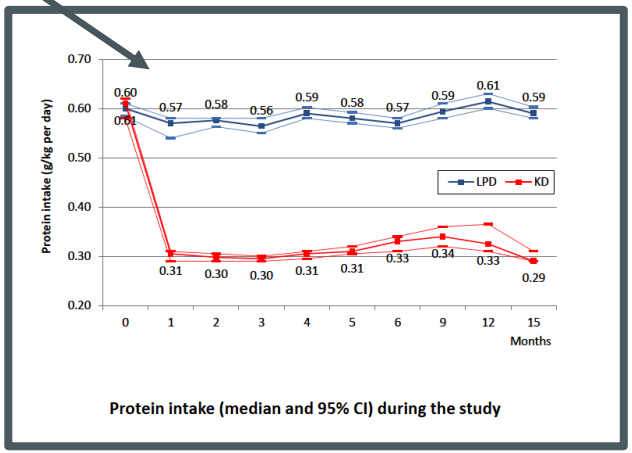
Cardiovascular and metabolic health	Lower protein intake is associated with lower dietary salt and saturated fat intake and may be less atherogenic, given higher proportion of plant-based food	Higher dietary fat intake (to achieve DEI of 30–35 kcal/kg/day) may confound the goal of achieving a heart-healthy diet	Higher proportions of unsaturated fat and complex carbohydrates recommended
Glycemic control and insulin response	Improvement in insulin resistance is likely	With LPD or VLPD, higher carbohydrate and fat intake (to achieve DEI 30–35 kcal/kg/day) may worsen glycemic control	Given increased insulin half-life and "burnt-out diabetes" with CKD progression, preventing hypoglycemic episodes is prudent
Quality of life and adherence to LPD	Enhanced patient-centeredness, given that many patients seek nutritional therapies and dietary advice	Challenges with adherence; diet fatigue, poor palatability, and cravings reported	Recommend creative recipes and strategies to engage patients
Mortality	There are no convincing data to suggest reduced mortality, although dialysis deferral is a potential mechanism, given high mortality during early dialysis	Increased mortality highly unlikely with DPI of 0.6–0.8 g/kg/day unless severe PEW emerges and is uncorrected	Consider supplements or other corrective strategies whenever PEW is suspected or diagnosed
Hypertension management	MDRD and other data suggest improved BP control	Reduction in BP is more likely a result of concomitant lower salt intake than of LPD itself	Higher potassium intake from more plant-based foods may be a potential mechanism
Microbiome modulation	Improved microbiome profile may be achieved through reduced uremic toxin generation	Possibility of promoting unfavorable microbiome milieu cannot be excluded	Uremia itself can lead to unfavorable microbiome



N=95

Diabetes & proteinuria

ASN 2017



Protein intake (median and 95% CI) during the study

Etude Garneata 2016

Sélection des patients

Point surement le plus important
« La sélection des patients »

1413 patients inclus

782 patients « adaptés » à un régime hypoprotidique

207 patients sélectionnés dans l'étude (14%)

KETOSTERIL® + Régime Hypoprotidique strict
(n = 104)

Régime Hypoprotidique
(n = 103)

Proposition de suivre un régime végétarien si nécessaire

Régime Hypoprotidique (0,6 g/kg/j - 30 kcal/kg/j)

Phase d'inclusion (33 mois) → **Phase de sélection** (3 mois) → **Phase d'étude** (3 + 12 mois)

EXCLUS

- **Ne correspondant pas aux critères d'éligibilité**
  n = 393
- **Refus de participer**
  - Dont ***Refus de suivre un régime végétarien***
  n = 191
- **Engagement dans une autre étude**
  n = 47

EXCLUS

- **Ne correspondant pas aux critères**
  - Critères d'éligibilité précédents
  - ***Non observance au régime prescrit (± 10%)***
  n = 483
- **Refus de participer**
  n = 92

SORTIS

- **Atteinte du critère principal**
  Progression de la MRC
- **Perdus de vue**
  NB : n = 7

Garneata L, *el al*. Ketoanalogue-supplemented vegetarian very low-protein diet and CKD progression. J Am Soc Nephrol 2016;27:2164-76.

Management of clinical survey
Dietitian and nephrologist cooperation

Consultation d'initiation

Néphrologue A → **Diététicien** B

Consultations de suivi

Néphrologue C → **Diététicien** D

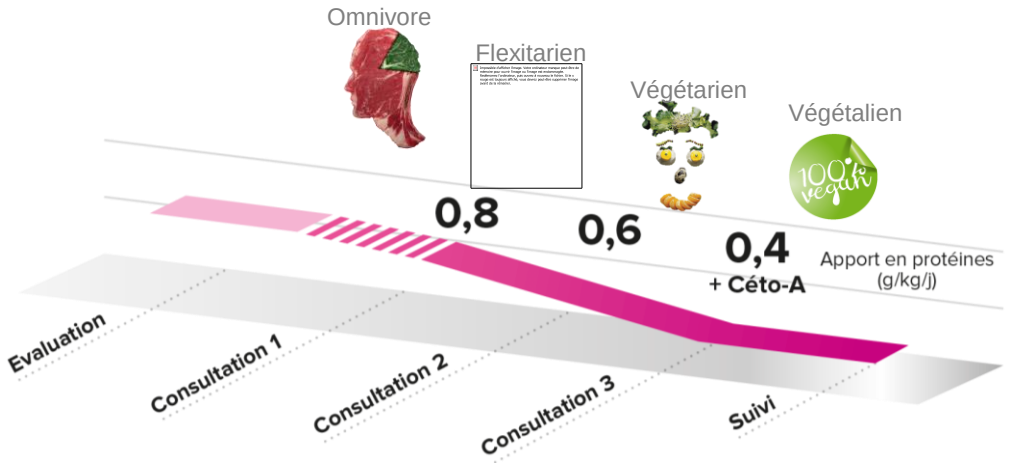
Un travail en « tandem », Néphrologue – Diététicien

- **Il n'existe pas de profil type de patient à qui proposer une alimentation hypoprotidique**
- **Les facteurs prédictifs identifiés, d'acceptation ou d'observance au régime, varient en fonction des études**

En pratique *Comment la mettre en place ?*

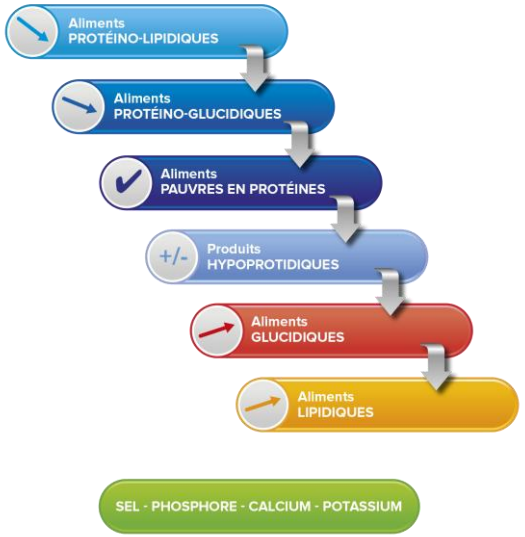
➢ Diminuer progressivement l'apport en protéines par paliers de 0,2 g/kg => *Jusqu'à 0,4 g/kg/j*

- Prévoir **2 à 3** consultations diététiques pour atteindre cet objectif
- Etablir un programme personnalisé & détaillé
- Accompagner le patient et évaluer régulièrement son observance



➢ Adapter progressivement les différents types d'aliments

1. Diminuer l'apport en protéines
2. Augmenter simultanément les autres sources énergétiques



➢ **L'apport calorique doit être maintenu à 30 – 35 Kcal/kg / jour +++**

Surveillance clinique & biologique

Examen clinique

- Poids
- IMC
- Tension Artérielle
- Symptômes

Bilan biologique

- **Bilan sanguin**
  - Ionogramme, kaliémie, acidose
  - Urée, créatinine
  - Paramètres phospho-calcique
  - Paramètres nutritionnels

- **Bilan urinaire des 24h**
  - Protéinurie
  - Urée, créatinine
  - Sodium, potassium

		T0	T + 1 mois		
Biochimie du sang	**Sodium**	140	**139**	mmol/l	*136-146*
	Potassium sérique	5,1	**4,7**	mmol/l	*3,5-5,1*
	Réserve alcaline	27	**25**	mmol/l	*22-29*
	Urée	22	**20**	mmol/l	*2,5-7,5*
	Créatinine	299	**310**	µmol/l	*62-115*
	Clairance de la créatinine				
	DFG calculé selon MDRD	20	**18**	ml/min/ 1,73 m²	
	HbA1c	7,9	**7,5**	%	
	Calcium	2,3	**2,3**	mmol/l	*2,2-2,7*
	Phosphore	1,2	**1,2**	mmol/l	*0,86 -1,50*
	Albumine	41	**40**	g/l	
	Préalbumine	0,39	**0,32**	g/l	
	CRP	1	**1**	mg//ml	
Biochimie des urines (des 24h)	**Protéinurie**	1	**0,6**	g/24h	***< 0,15***
	Urée U.	560	**380**	mmol/24h	*300-500*
	Créatinine U.	14	**13**	mmol/24h	*8,9-17,7*
	Sodium U.	179	**119**	mmol/24h	*50-220*
	Potassium U.	28	**25**	mmol/24h	

La bonne coordination du tandem Néphrologue – Diététicien dans le suivi régulier du patient

Consultation d'initiation

A B

- Identification des patients
- Présentation de la stratégie diététique
- Bilan clinique & biologique
- Prescription de la consultation diététique & Information du patient

- Evaluation des apports et des habitudes alimentaires
- Mesure de la composition corporelle
- Présentation des caractéristiques d'une alimentation réduite en protéines
- Elaboration d'un protocole d'alimentation personnalisé

Consultations de suivi

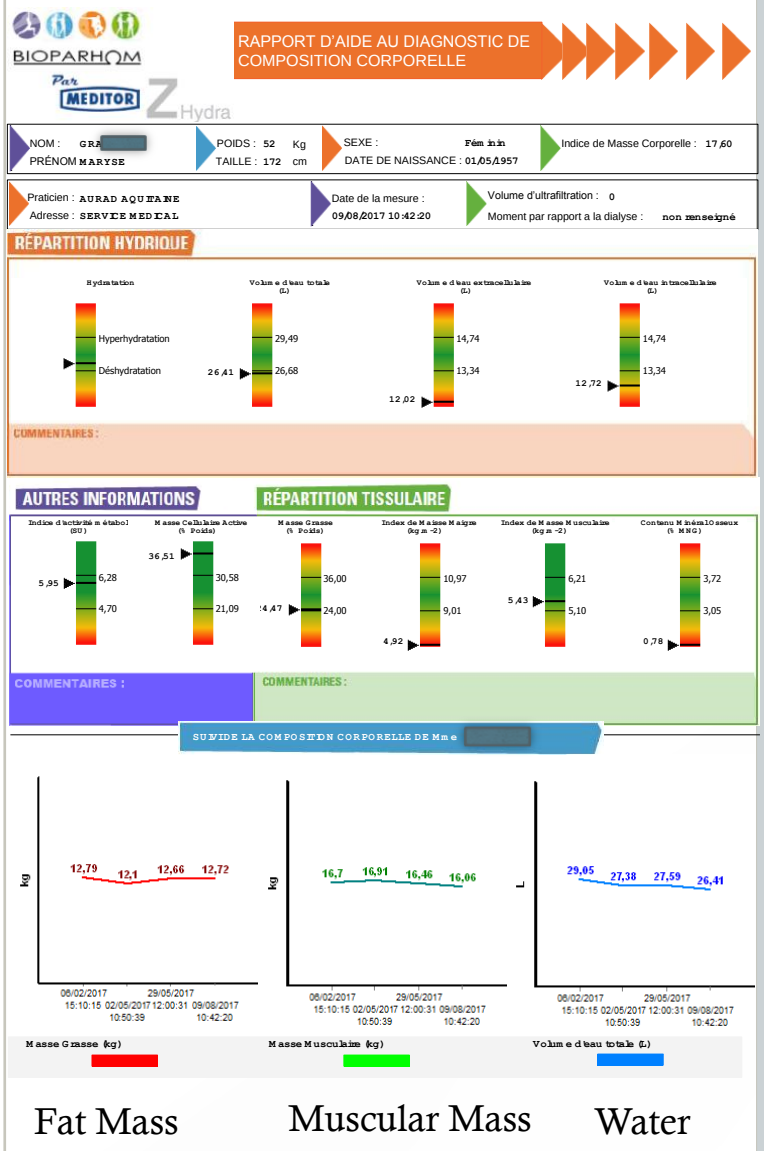
1 fois par mois les 6 premiers mois

C D

- Surveillance clinique & biologique
- Suivi de l'urée urinaire
- Suivi du sodium urinaire
- Suivi de la protéinurie
- Suivi de l'albuminémie
- Prescription de céto-analogues

- Suivi de l'état nutritionnel & Adaptation de l'apport énergétique
- Suivi de l'observance & Adaptation des habitudes alimentaires

Un travail en « tandem »… est un « tandem » gagnant !



Fat Mass Muscular Mass Water

- Mme GRA, 1 year survey
- 60 years
  - Transplanted in 2001
  - After 3 years of sVLPD !
- Waiting for 2nd RT
- GFR 15 ml/min, stable
  - 20 to 16 ml/min the year before
- BMI 18
- O.6 then 0.4 g/kg + 10 cp
  - nPNA 0.46
  - Cal: 1800

CONCLUSIONS
Low protein diet and CKD

- Strong evidences exist of effectiveness in reducing renal death and delay start of renal replacement therapy
- As with any change in eating habits, the selection of patients, information, education and monitoring during the diet are very important and help prevent undernutrition
- Need for more renal dietitian in nephrology unit
- Need for better reimbursement for prevention and dietitian consultation