Genômica Nutricional e Doenças Cardiovasculares

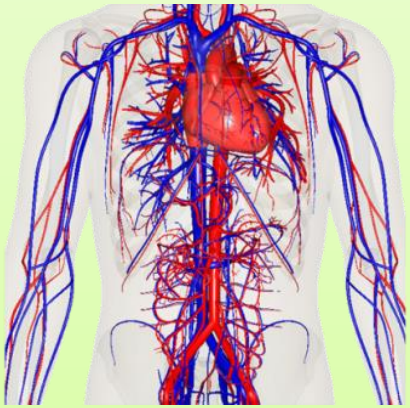
Ms. Mychelle Kytchia Rodrigues Nunes Duarte

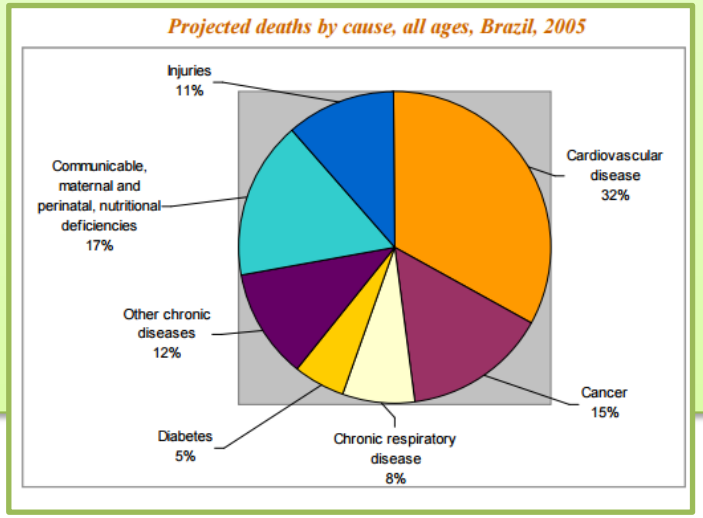
A Doença Cardiovascular (DCV) - 30% das mortes globais (OMS).

No Brasil - 340.284 óbitos no ano de 2014,

12.411 referente a doenças das artérias, das arteríolas e capilares segundo o o Código Internacional de Doenças (CID-10)

No Rio Grande do Norte no ano de 2014 - foram 5.164 óbitos devido as DCV.

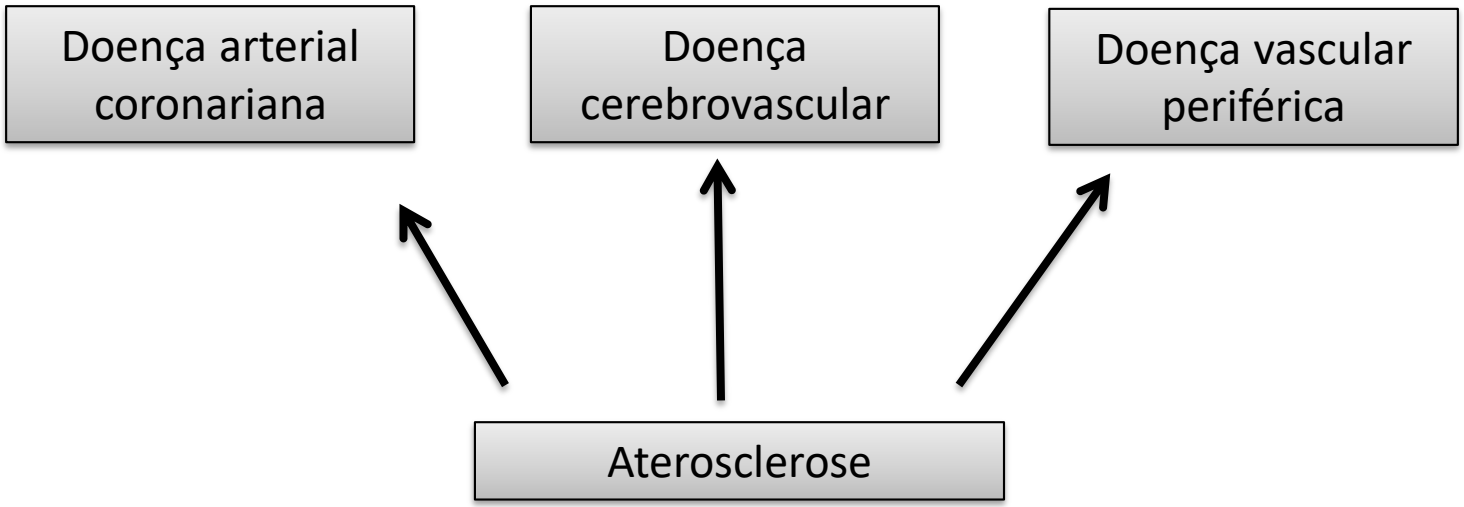




I Diretriz sobre o consumo de Gorduras e Saúde Cardiovascular. 2013.
DATASUS, 2016.

As DCV alteram o funcionamento do sistema circulatório, formado por coração, vasos sanguíneos e linfáticos.

Podem ser classificadas em:



Feron IM, Faux SP. Oxidative stress and cardiovascular disease: novel tools give (free) radical insight. J Mol Cell Cardiol 2009;47(3):372-81.

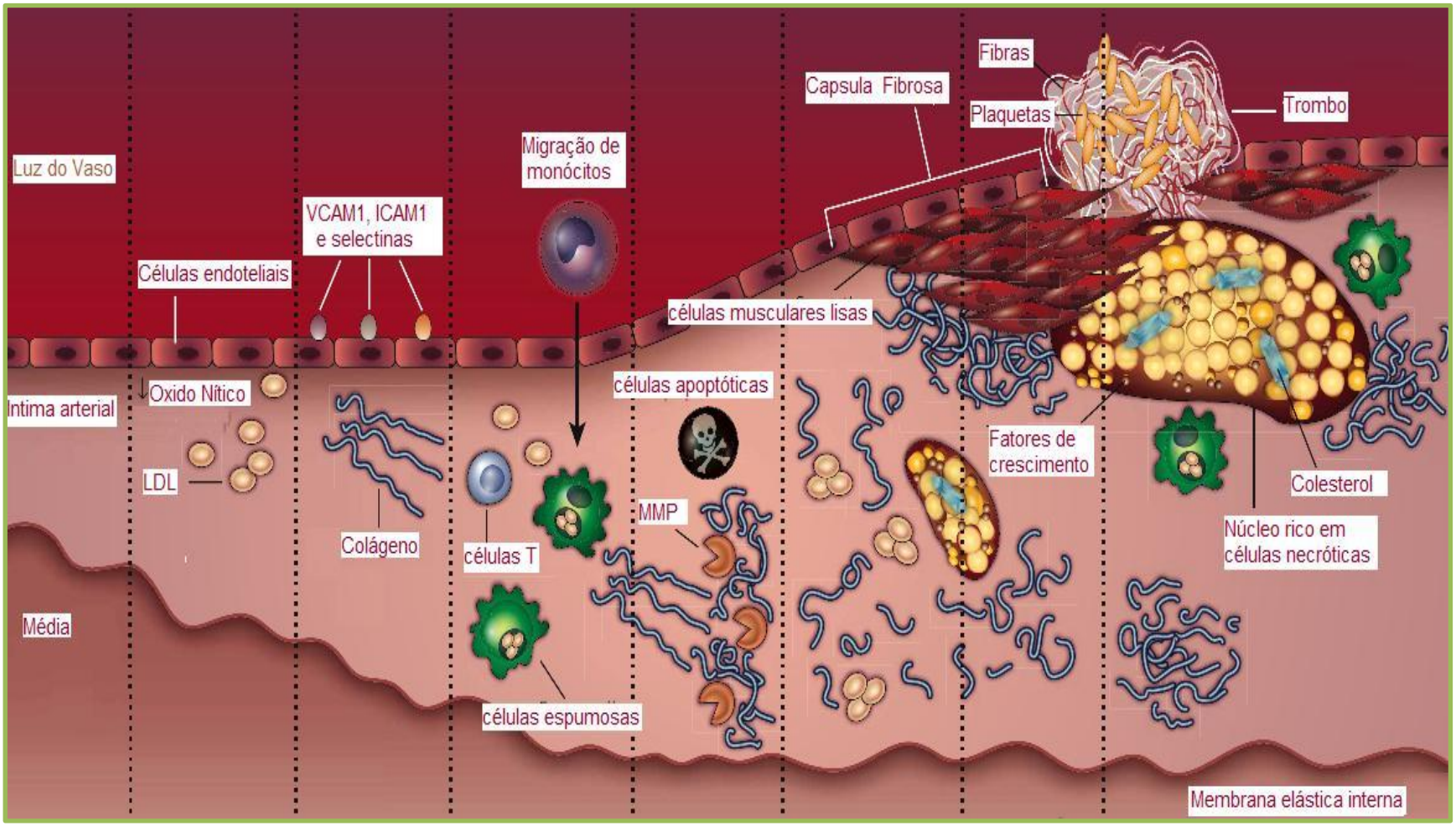


Figura 1. Representação da progressão do processo de aterosclerose. Adaptado de Sanz & Fayad, Nature, 2008.

BioMed Central The Open Access Publisher | BMC Medicine
this article | search | submit a manuscript | register

BMC Med. 2014; 12: 158. PMCID: PMC4199062
Published online 2014 Sep 25. doi: 10.1186/s12916-014-0158-6

Dietary calcium intake and mortality risk from cardiovascular disease and all causes: a meta-analysis of prospective cohort studies

Xia Wang, Hongxia Chen, Yingying Ouyang, Jun Liu, Gang Zhao, Wei Bao, and Maosheng Yan

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Conclusions: This meta-analysis of prospective cohort studies suggests that dietary calcium intake is associated with cardiovascular mortality in a U-shaped manner and that high dietary calcium intake (>900 mg/day) is not associated with a decreased risk of all-cause mortality.

Eur J Nutr. 2012 December ; 51(8): 917–926. doi:10.1007/s00394-011-0268-2.

Nutrient and food intakes of middle-aged adults at low risk of cardiovascular disease: the international study of macro-/ micronutrients and blood pressure (INTERMAP)

BMC Medicine, 2014.
Eur J Nutr, 2012.

Food and food subgroup intake (g/1,000 kcal) by low cardiovascular disease (CVD) risk status[a], all participants (Japan, People's Republic of China, United Kingdom, United States), 1996–1999 the INTERMAP Study ($n = 4,680$)

Food subgroup[b]	Not low CVD risk ($n = 3,923$)	Low CVD risk ($n = 757$)	*P* value[c]
Total fruits	76.6 (28.0–145.5)	99.1 (48.9–168.6)	<0.001
Total vegetables	152.2 (104.4–212.1)	177.0 (124.0–244.0)	<0.001
Total grains	147.4 (91.2–283.9)	209.6 (120.4–332.7)	0.085
Pasta, rice, including recipes	54.7 (14.7–191.0)	128.8 (34.2–247.8)	<0.001
Nuts and legumes	3.9 (0.2–15.2)	3.0 (0.1–11.4)	0.024
Non-high-fat dairy (milk, yogurt, frozen yogurt, etc.)	40.5 (2.8–105.9)	49.5 (0.7v118.1)	0.020
High-fat dairy (cream, cheese, ice cream, milk, cheese, recipes, etc.)	5.4 (0.0–18.9)	3.3 (0.0–15.0)	<0.001
Fish, fish roe, shellfish	6.6 (0.0–30.6)	12.3 (0.0–35.4)	0.002
Poultry	9.0 (0.0–22.5)	6.9 (0.0–18.0)	<0.001
Beef, pork, veal, game meats	18.5 (7.4–34.6)	14.8 (4.9–27.9)	<0.001
Eggs	9.2 (2.3–19.0)	8.4 (2.0–17.5)	0.031
Processed meats	3.3 (0.0–10.9)	0.0 (0.0–7.4)	<0.001
Total visible fats (animal fats, margarines, table spreads, oils, shortenings, dressings)	13.1 (8.4–19.1)	13.3 (8.5–18.9)	0.874
Snacks, sweets	16.8 (5.6–30.7)	17.70 (6.2–33.4)	0.074
Alcoholic beverages	0.9 (0.0–86.5)	0.3 (0.0–18.4)	<0.001
Non-alcoholic beverages (excluding tea, coffee)	226.0 (0.0–576.0)	41.00 (0.0–485.7)	<0.001
Sugar-sweetened beverages[d] (UK and US samples only)	62.7 (0.0–188.7)	49.6 (0.0–142.8)	0.007
Total energy intake (kcal/day), mean (SD)	2,194.2 (633.2)	1,908.5 (514.7)	<0.001

CVD cardiovascular disease

Conclusions—Lower energy intake and differential intake of multiple specific nutrients and foods are characteristic of individuals at low risk for developing CVD. Identification of dietary habits associated with LR is important for further development of public health efforts aimed at reduction/prevention of CVD.

BMC Medicine, 2014.
Nutrients, 2015.

OPEN ACCESS Freely available online

PLOS ONE

Lower Zinc Bioavailability May Be Related to Higher Risk of Subclinical Atherosclerosis in Korean Adults

Su Kyoung Jung[1,2], Mi-Kyung Kim[1,2*], Young-Hoon Lee[3], Dong Hoon Shin[4], Min-Ho Shin[5], Byung-Yeol Chun[6], Bo Youl Choi[1,2]

J Am Soc Nephrol. 2009 Aug; 20(8): 1797–1804.
PMCID: PMC2723984
doi: 10.1681/ASN.2008060649

High Protein Intake Associates with Cardiovascular Events but not with Loss of Renal Function

Nynke Halbesma,[*] Stephan J.L. Bakker,[*] Desiree F. Jansen,[†] Ronald P. Stolk,[†] Dick De Zeeuw,[‡] Paul E. De Jong,[*] Ronald T. Gansevoort,[*] and for the PREVEND study group

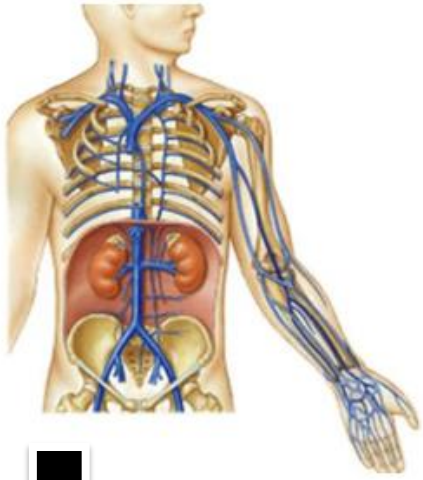
Europe PMC Funders Group
Author Manuscript
Cochrane Database Syst Rev. Author manuscript; available in PMC 2014 September 15.

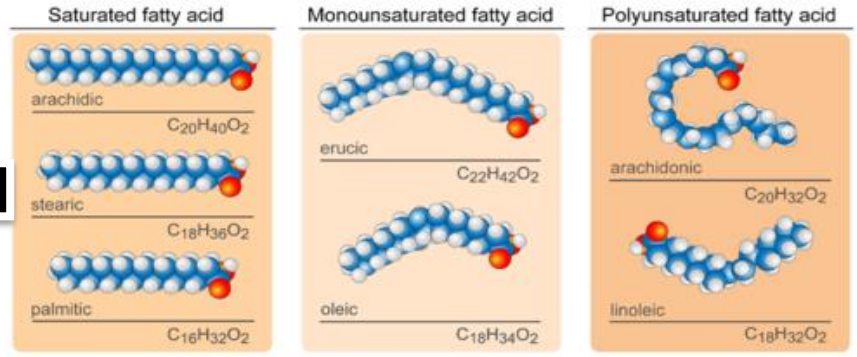
Published in final edited form as:
Cochrane Database Syst Rev. ; (7): CD002137. doi:10.1002/14651858.CD002137.pub2.

Reduced or modified dietary fat for preventing cardiovascular disease

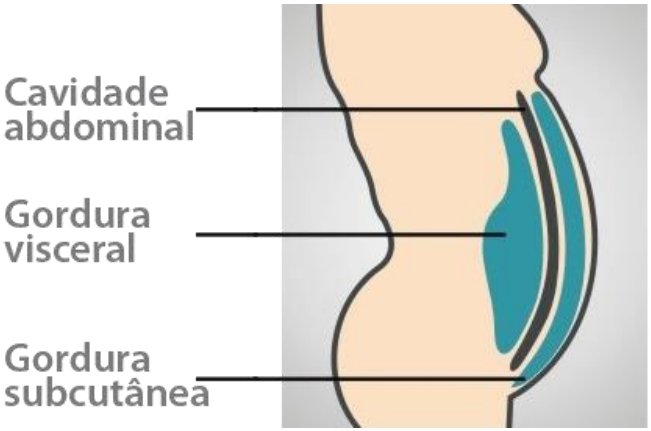
Lee Hooper[1], Carolyn D Summerbell[2], Rachel Thompson[3], Deirdre Sills[4], Felicia G Roberts[5], Helen Moore[2], and George Davey Smith[6]

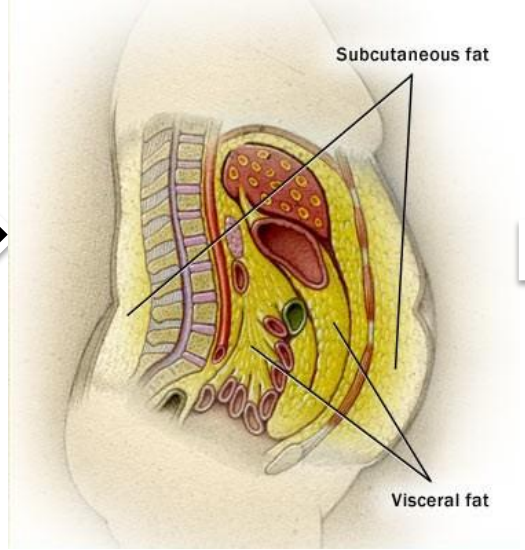
Plos One, 2013
J Am Nephrol, 2009
Cochrane Database Syst Ver, 2011

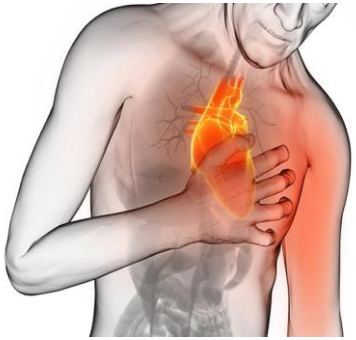




I Diretriz sobre o consumo de Gorduras e Saúde Cardiovascular. 2013.
I Diretriz Brasileira de prevenção Cardiovascular. 2013.







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PLOS ONE

A Pilot Investigation of Visceral Fat Adiposity and Gene Expression Profile in Peripheral Blood Cells

Masaya Yamaoka[1], Norikazu Maeda[1]*, Seiji Nakamura[2], Susumu Kashine[1], Yasuhiko Nakagawa[1], Aki Hiuge-Shimizu[1], Kohei Okita[1], Akihisa Imagawa[1], Yuji Matsuzawa[3], Ken-ichi Matsubara[2], Tohru Funahashi[1], Iichiro Shimomura[1]

PLoS One. 2012.

Clinica Chimica Acta 421 (2013) 184–190

Contents lists available at SciVerse ScienceDirect

Clinica Chimica Acta

journal homepage: www.elsevier.com/locate/clinchim

Novel genes detected by transcriptional profiling from whole-blood cells in patients with early onset of acute coronary syndrome

Vivian N. Silbiger [a,b,*], André D. Luchessi [a], Rosário D.C. Hirata [a], Lídio G. Lima-Neto [a], Débora Cavichioli [a], Angel Carracedo [c], Maria Brión [c,d], Joaquín Dopazo [e], Francisco García-García [e], Elizabete S. dos Santos [f], Rui F. Ramos [f], Marcelo F. Sampaio [f], Dikran Armaganijan [f], Amanda G.M.R. Sousa [f], Mario H. Hirata [a]

[a] Faculty of Pharmaceutical Sciences, University of Sao Paulo, Brazil
[b] Department of Clinical and Toxicologic Analyses, Federal University of Rio Grande do Norte, Brazil
[c] Group of Genomic Medicine, Galician Foundation of Genomic Medicine, CIBERER-University of Santiago, Spain
[d] Genetics of Cardiovascular and Ophthalmologic Diseases, Hospital-University Complex of Santiago (CHUS), Spain
[e] Functional Genomics Node National Institute for Bioinformatics (INB), Centro de Investigación Príncipe Felipe, Spain
[f] Institute Dante Pazzanese of Cardiology, Brazil

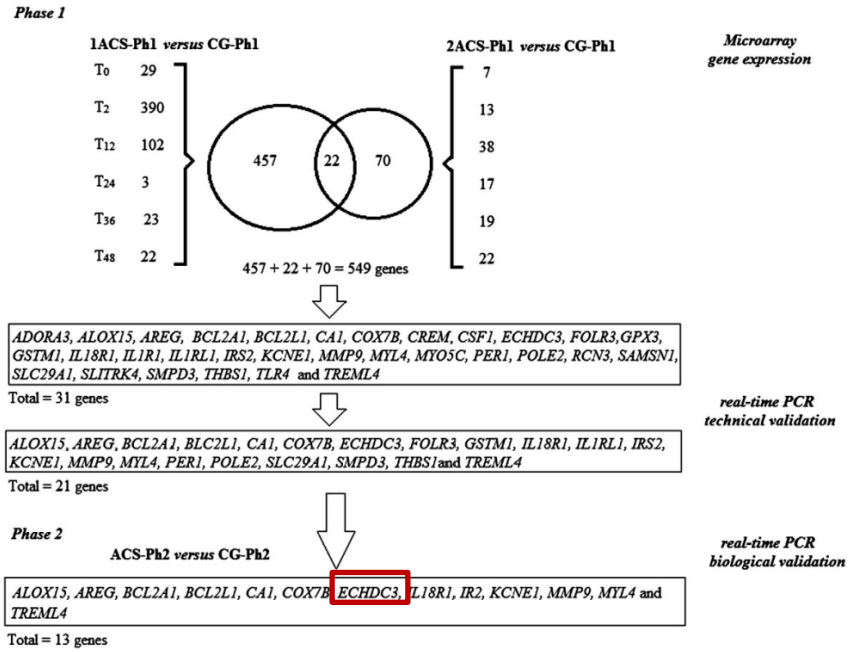
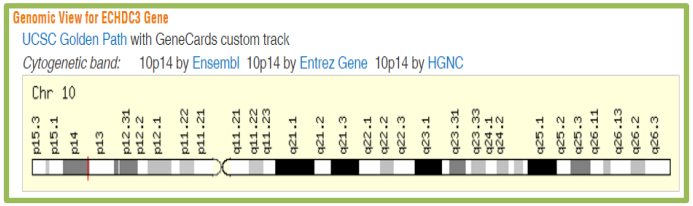
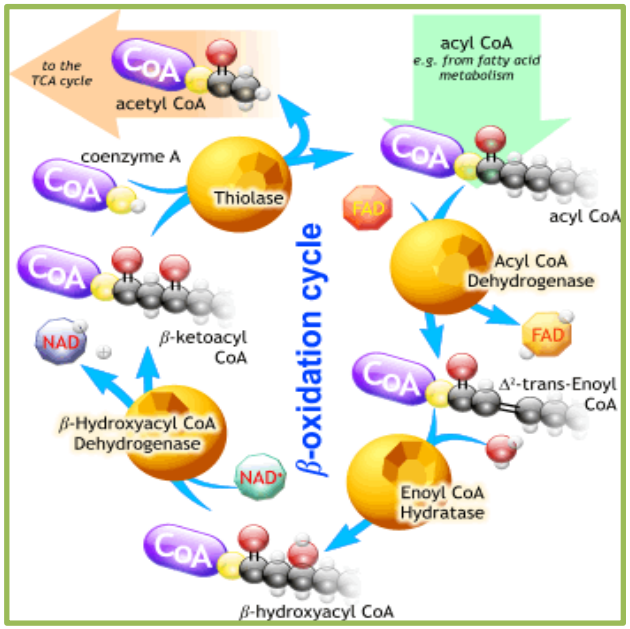
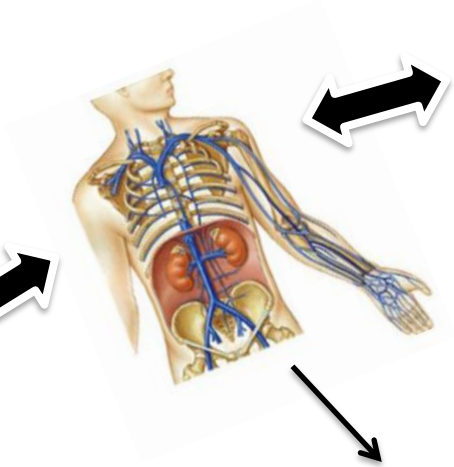
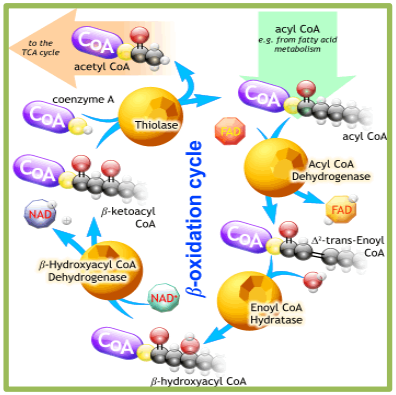


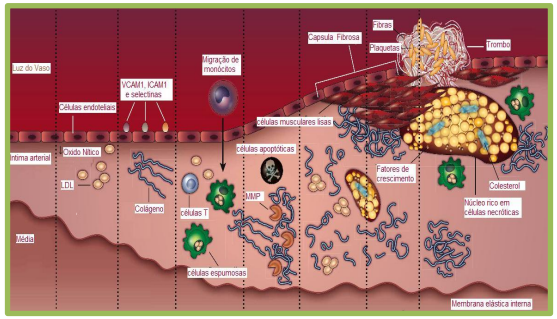
Fig. 1. Candidate gene expression biomarkers history of heart ischemia CG-Ph1: control group from phase 1; 1ACS-Ph1: patient with acute coronary syndrome without previously historic of heart ischemia from phase 1; 2ACS-Ph1: patient with acute coronary syndrome with previously history of heart ischemia from phase 1; CG-Ph2: control group from phase 2;ACS-Ph2: patient with acute coronary syndrome from phase 2; T_0: first stage; T_2: second stage; T_{12}: third stage; T_{24}: fourth stage; T_{36}: fifth stage; T_{48}: sixth stage.

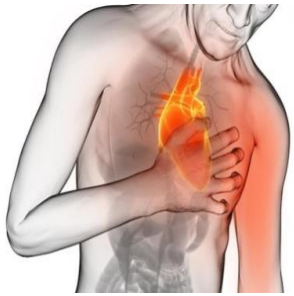







to the TCA cycle
acetyl CoA
acyl CoA
e.g. from fatty acid metabolism
coenzyme A
Thiolase
acyl CoA
Acyl CoA Dehydrogenase
β-ketoacyl CoA
β-oxidation cycle
Δ²-trans-Enoyl CoA
β-Hydroxyacyl CoA Dehydrogenase
Enoyl CoA Hydratase
β-hydroxyacyl CoA


Luz do Vaso
Células endoteliais
VCAM1, ICAM1 e selectinas
Migração de monócitos
Cápsula Fibrosa
Fibras
Plaquetas
Trombo
Íntima arterial
Óxido Nítrico
LDL
Colagenol
células T
células apoptóticas
MMP
Fatores de crescimento
Colesterol
Núcleo rico em células necróticas
Média
células espumosas
Membrana elástica interna



O artigo intitulado *“* ***The relationship of the oleic acid level and ECHDC3 mRNA expression with the extent of coronary lesion”*** foi publicado no periódico “ Lipids in Health and Disease”.

Duarte *et al. Lipids in Health and Disease* (2016) 15:144
DOI 10.1186/s12944-016-0312-6

Lipids in Health and Disease

RESEARCH Open Access

The relationship of the oleic acid level and *ECHDC3* mRNA expression with the extent of coronary lesion

Mychelle Kytchia Rodrigues Nunes Duarte[1], Jéssica Nayara Góes de Araújo[2], Victor Hugo Rezende Duarte[2], Katiene Macêdo de Oliveira[2], Juliana Marinho de Oliveira[3], Antonio Augusto Ferreira Carioca[4], Raul Hernandes Bortolin[2], Adriana Augusto Rezende[2], Mario Hiroyuki Hirata[5], Rosário Domingues Hirata[5], Dan Linetzky Waitzberg[6], Severina Carla Vieira Cunha Lima[7], André Ducati Luchessi[2] and Vivian Nogueira Silbiger[2*]

Identificar biomarcadores para o diagnóstico precoce das doenças cardiovasculares por meio da relação de biomoléculas e do estado nutricional com a extensão da lesão aterosclerótica.

Objetivos Específicos:

- Selecionar pacientes com risco cardiovascular, que tinham sido encaminhados para realização da cinecoronariografia pela primeira vez;
- Avaliar o perfil bioquímico, extensão da lesão aterosclerótica, concentração sérica de ácidos graxos, expressão do RNAm do *ECHDC3,* consumo alimentar e dietético em pacientes sem lesão e diferentes extensões de lesão aterosclerótica;
- Correlacionar a concentração sérica de ácidos graxos, o consumo alimentar e dietético, dados antropométricos, expressão do *ECHDC3* em pacientes sem lesão e diferentes extensões de lesão aterosclerótica;

Metodologia

HUOL
Hospital Universitário
Onofre Lopes

Aprovado pelo Comitê de Ética
Protocolo 520/11
CAAE: 0001.0.051.294-11

HEMODINÂMICA DO HUOL

↓

Recrutamento

Inclusão

Pacientes
30-74 anos
Ambos os sexos

Realizar a
cinecoronariografia
pela primeira vez

Exclusão

Doença pré-estabelecida

- Cardiomiopatia
- Doença Hepática
- Doença Renal
- IAM
- Distúrbios endócrinos, com exceção de diabetes

Metodologia

Primeira Casuística

Triagem de pacientes - 59

Aprovado pelo Comitê de Ética
Protocolo 520/11
CAAE: 0001.0.051.294-11

Mensuração da pressão arterial

coleta de sangue periférico

Cinecoronariografia

Friesinger

Antropometria

Peso e altura

Exames laboratoriais

- Glicose
- Colesterol
- Triglicérides
- HDLc
- LDL
- Creatinina
- Uréia
- Ácido úrico
- AST
- ALT

Concentração sérica de ácidos graxos

Cromatografia gasosa

Expressão gênica

Células mononucleares
Extração de RNA
cDNA
PCR real time

Metodologia

Segunda Casuística

Triagem de pacientes - 41

Aprovado pelo Comitê de Ética
Protocolo 520/11
CAAE: 0001.0.051.294-11

- Coleta de sangue periférico
  - Exames laboratoriais
    - Glicose
    - Colesterol
    - Triglicérides
    - HDLc
    - LDL
    - Creatinina
    - Uréia
    - Ácido úrico
    - AST
    - ALT
  - Expressão gênica
    - Células mononucleares
    - Extração de RNA
    - cDNA
    - PCR real time
- Cinecoro-nariografia
  - Friesinger
- Consumo Alimentar e Dietético
  - 2 –R24h
  - Virtual Nutri
- Antropometria
  - Peso e altura
  - Perimetros
  - Composição corpórea
    - Percentual de gordura
    - Gordura Visceral
- Aferição de pressão arterial

Metodologia

Índice de Friesinger

Quantifica as lesões das artérias coronárias, utilizando uma variação de 0 a 15 pontos. Cada um das três artérias coronárias principais e seus ramos (descendente anterior, circunflexa e coronária direita) foi marcada separadamente, recebendo uma pontuação de zero a cinco:

0) nenhuma anormalidade arteriográfica

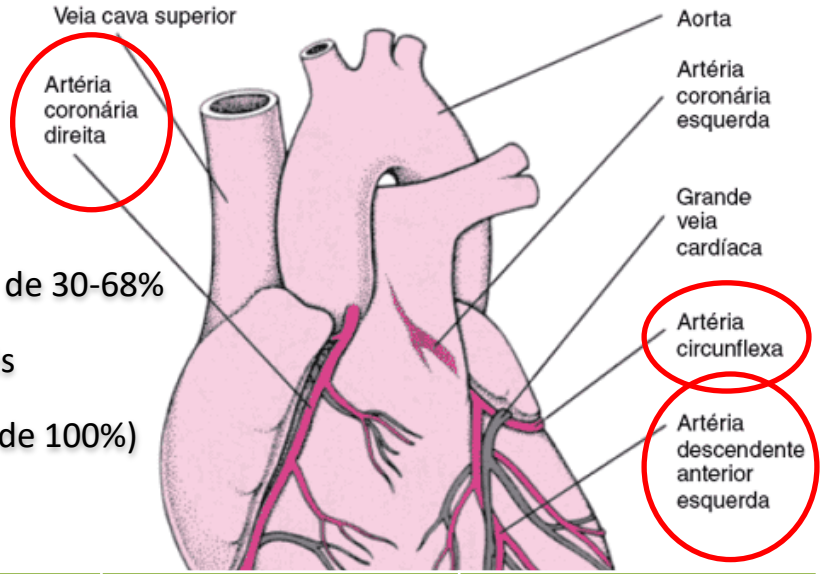
1) irregularidades da artéria ou estenose menor que 30%

2) estenose de 30-68%

3) múltiplas lesões ou pelo menos dois segmentos com estenose de 30-68%

4) estenose de 69-100%, exceto oclusão dos segmentos proximais

5) obstrução total (ou oclusão) de um segmento proximal (lesão de 100%)



Categorização	Sem lesão	Poucas lesões	Intermediárias lesões	Graves lesões
Pontuação	0	1-4	5-9	10-15

Bampi ABA, Rochitte CE, Favarato D, Lemos PA, da Luz PL. Comparison of non-invasive methods for the detection of coronary atherosclerosis. Clinics (Sao Paulo). 2009;64(7):675–82.
Chagas P, Caramori P, Galdino TP, Barcellos CDS De, Gomes I, Schwanke CHA. Egg consumption and coronary atherosclerotic burden. Atherosclerosis [Internet]. Elsevier Ltd; 2013;229(2):381–4.

Metodologia

MEDICINA
USP

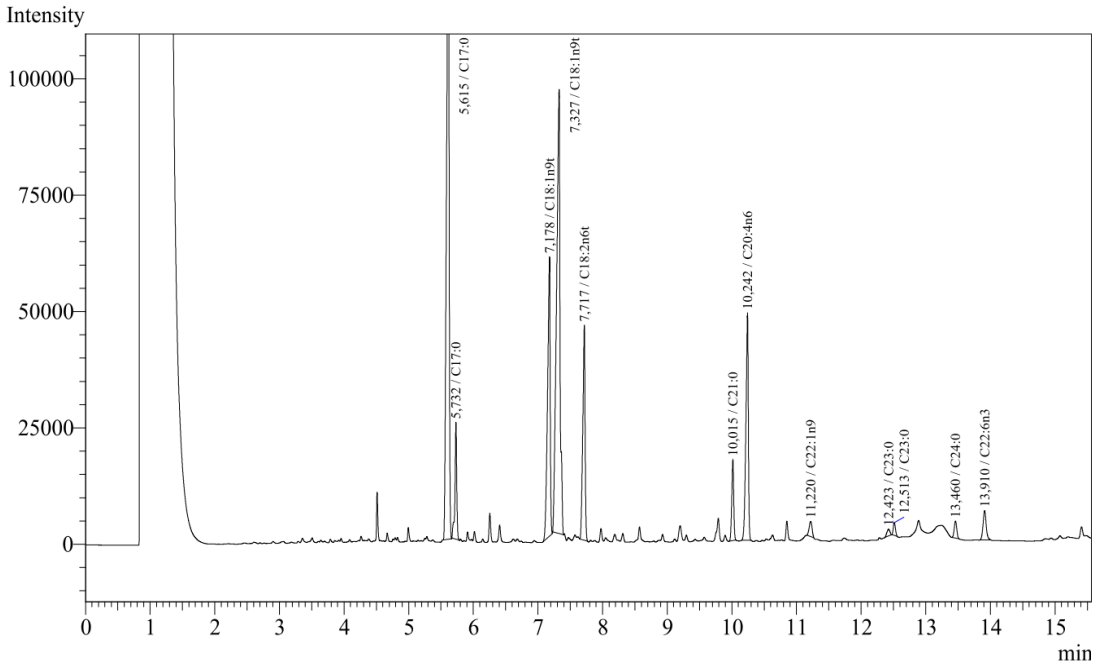
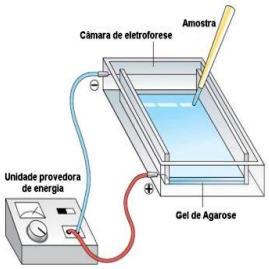


Figura 2. Exemplo do perfil sérico de ácidos graxos obtido através do CG-2010, Shimadzu (Paciente 37).

Metodologia

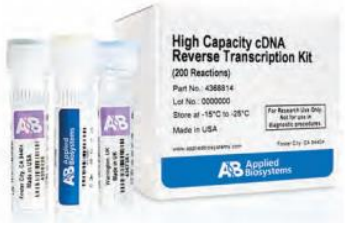
Extração do RNA TOTAL – células mononucleares

RiboPure™ – Blood kit



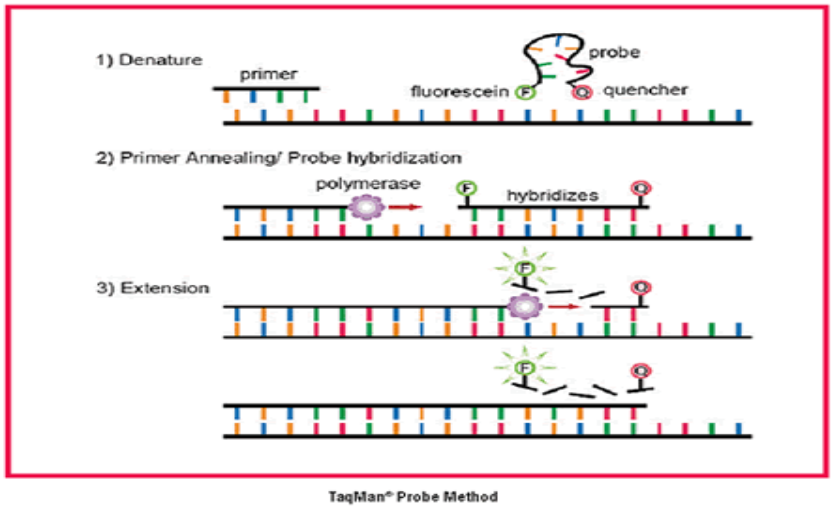
Agarose Gel Eletro. 1.0%
MOPS Buffer

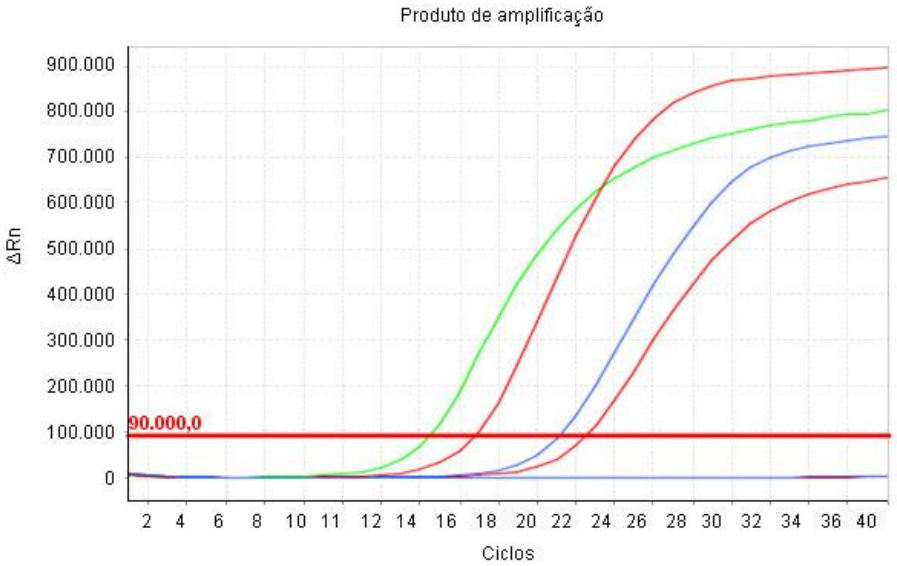
Qubit® 2.0
Fluorometer



Metodologia

Sistema TaqMan – Quantificação relativa





$$\Delta Ct = (Ct_{\text{gene alvo}} - Ct_{\text{controle endógeno}})$$

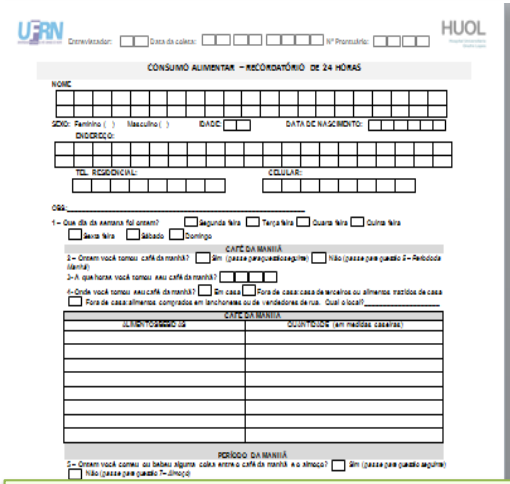
$2^{-\Delta CT}$

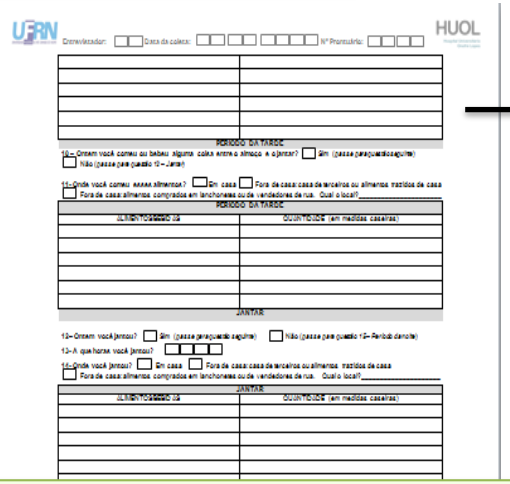
ACTB

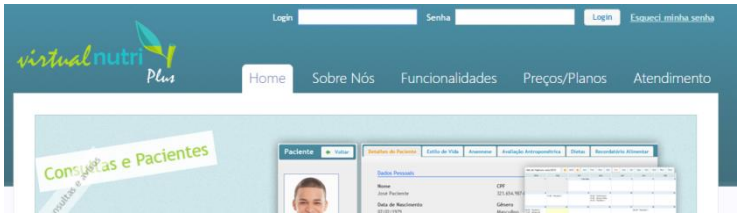
Fonte: http://www.gibthai.com/services/technical_detail.php?ID=12

Metodologia

Consumo Alimentar e Dietético







Os dados dietéticos da variabilidade, variabilidade intrapessoal foram ajustados pelo metodo de Nusser et al. 1996;

Em seguida, os resultados foram ajustados pela energia;

Foi utilizado a análise pela ANOVA para determinar a variabilidade intrapessoal e para estimar a variância intra e interpessoal foi utilizado a média quadrática;

Para controlar os fatores de confundimento inerentes a ingestão de energia total e remover as variáveis externas, os dados foram ajustados pela energia usando o método do resíduo.

J Am Stat Assoc., 1996.

Am J Epidemiol. 1986

Metodologia

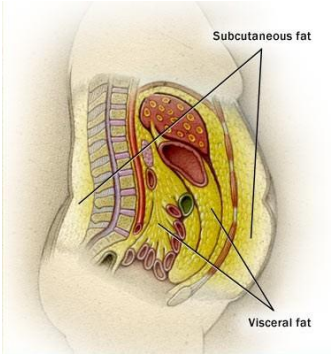
Avaliação Antropométrica

Balança de Bioimpedância Elétrica (BIA) tetrapolar da marca Omron HBF-514C (Omron Healthcare, Japão)

Percentual de gordura

Percentual de gordura visceral

EUTRÓFICOS	ALTO PG%	MUITO ALTO PG%
<9%	10-14%	>15%



American Journal of Clinical Nutrition, 2000

Metodologia

Avaliação Antropométrica

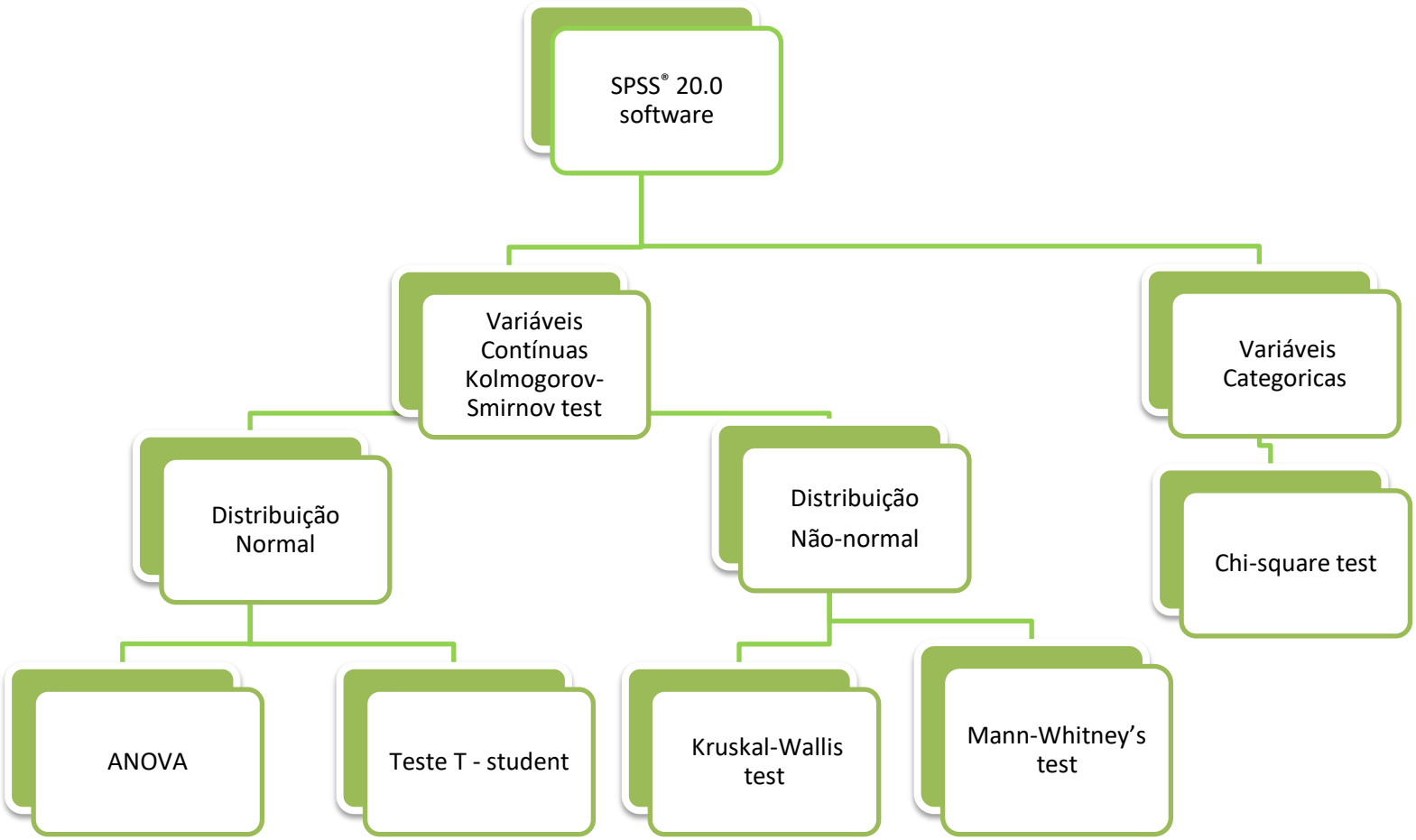
Perímetros

Perímetro da Cintura

Perímetro do Quadril

Wang J, Et al. 2003

Metodologia



O nível de significância estatística aceitável foi $p < 0.05$.

Artigo – Resultados e Discussão

Table 1. Demographic, anthropometric and clinical data of patients classified according to the extent of coronary lesion

Variables	Total (n=59)	Without lesion (n=18)	Low lesion (n=17)	Intermediate lesion (n=17)	Major lesion (n=7)	*p-value*
Age, years	60.0 ± 9.0	55.0 ± 9.0	62.0 ± 9.0	63.0 ± 10	60.0 ± 7.0	0.080
Sex male, %	54.2	44.4	58.8	64.7	42.9	0.582
BMI, kg/m^2	26.82±5.65	27.86 ± 4.70	25.27 ± 8.52	26.92 ± 4.29	27.43 ± 1.13	0.630
Obesity, %	20.3	33.3	17.6	17.6	0	0.133
Dyslipidemia, %	91.5	77.8	100.0	94.1	100.0	0.080
Diabetes mellitus, %	30,5	33.3	23.5	35.3	28.6	0.884
Hypertension, %	79.7	77.8	76.5	82.4	85.7	0.944
Diastolic pressure, mmHg	84.0±20.0	81.0±8.0	80.0±24.0	90.0±18.0	90.0±32.0	0.355
Systolic pressure, mmHg	143.0±26.0	137.0±24.0	139.0±22.0	155.0±32.0	141.0±21.0	0.187
Alcoholism, %	18.6	27.8	17.6	5.9	28.6	0.350
Smoking, %	22.0	27.8	17.6	17.6	28.6	0.825
Physical activity, %	47.5	50.0	58.8	35.3	42.9	0.573

BMI, Body Mass Index; Data are presented as mean ± standard deviation for parametric samples and as median (interquartile range) for non-parametric samples. Categorical variables were compared by Chi-square test. Parametric analysis was performed by ANOVA way followed by test T. Non-parametric samples were performed by Kruskal-Wallis test followed by Mann Whitney test. P-values < 0.05 were considered statistically significant.

Artigo – Resultados e Discussão

Cont. Table 1. Demographic, anthropometric and clinical data of patients classified according to the extent of coronary lesion

Biochemical Analyses						
Glucose, mmol/l	93,0 mg/dL	4.75(3.89-14.26)	4.88(3.75-11.04)	5.55(3.72-21.87)	5.94(4.44-13.04)	0.443
Total cholesterol, mmol/l	178 mg/dL	4.36(3.08-6.50)	4.51(3.19-6.89)	5.05(2.95-7.85)	4.61(3.65-7.02)	0.555
HDL-cholesterol, mmol/l	35,5 mg/dL	0.95(0.57-1.45)	0.93(0.54-1.30)	0.91(0.41-1.32)	0.83(0.62-1.40)	0.669
LDL-cholesterol, mmol/l	103,3 mg/dL	2.38(1.49-4.57)	2.63(1.21-5.00)	3.26(1.21-5.74)	2.89(1.93-5.27)	0.419
Triglycerides, mmol/l	143 mg/dL	1.63(0.79-6.94)	2.25(0.86-6.96)	1.62(0.75-9.47)	1.45(0.85-3.24)	0.612
ALT, µKat/l	26 U/L	0.38 (0.17-1.57)	0.43 (0.25-1.04)	0.33 (0.17-2.19)	0.60 (0.25-1.45)	0.723
AST, µKat/l	31 U/L	0.52 (0.25-1.49)	0.43 (0.25-0.95)	0.52 (0.25-1.57)	0.60 (0.25 -1.57)	0.546
Ureia, mmol/l	35 mg/dL	5.93(3.34-9.52)	5.68(4.31-9.85)	5.93(3.37-10.35)	5.85(4.84-6.35)	0.693
Creatinine, µmol/l	0,90 mg/dL	79.56(53.04-141.44)	79.56(53.04-123.76)	88.40(53.04-106.08)	70.72(17.68-106.08)	0.323
Uric acid, mmol/l	4,9 mg/dL	0.27(0.16-0.52)	0.28(0.11-0.43)	0.29(0.11-0.43)	0.33(0.14-0.37)	0.965

BMI, Body Mass Index; HDL-cholesterol, High density lipoprotein; LDL-cholesterol, Low density lipoprotein; AST, aspartate aminotransferase; ALT, Alanine transaminase. Data are presented as mean ± standard deviation for parametric samples and as median (interquartile range) for non-parametric samples. Categorical variables were compared by Chi-square test. Parametric analysis was performed by ANOVA way followed by test T. Non-parametric samples were performed by Kruskal-Wallis test followed by Mann Whitney test. P-values < 0.05 were considered statistically significant.

Artigo – Resultados e Discussão

Table 2 –Serum fatty acid concentration according to the extent of coronary lesion

Fatty acids (%)	Total (n=59)	Without lesion (n=18)	Low lesion (n=17)	Intermediate lesion (n=17)	Major lesion (n=7)	*p-value*
SFA	44.12±3.85	44.70±4.18	43.79±4.46	43.93±2.74	43.93±4.40	0.904
Myristic (C14:0)	0.89±0.30	0.94±0.36	0.91±0.26	0.87±0.31	0.80±0.19	0.756
Palmitic (C16:0)	30.83±3.07	31.40±2.95	30.61±3.15	30.52±2.80	30.63±4.22	0.830
Stearic (C18:0)	12.40±1.89	12.36±2.15	12.27±2.10	12.54±1.70	12.50±1.30	0.979
MUFA	24.87±3.10	23.66±2.88	25.97±2.84a	25.77±2.96a	23.14±3.35	0.032
Palmitoleic (C16:1)	2.55±0.75	2.41±0.62	2.73±.63	2.56±0.89	2.47±0.98	0.658
Oleic (C18:1)	22.32±2.77	21.25±2.54	23.24±2.48a	23.22±2.73a	20.66±2.89	0.027
PUFA	31.01±4.78	31.65±4.46	30.24±5.37	30.30±3.99	32.94±6.01	0.529
n-6	26.77±4.29	27.26±3.66	25.96±5.13	26.46±3.54	28.26±5.53	0.628
Linoleic (C18:2)	18.06±3.84	18.64±3.29	17.23±4.35	18.10±3.30	18.47±5.39	0.742
Arachidonic (C20:4)	8.71±2.37	8.62±1.73	8.73±2.47	8.36±2.20	9.78±3.89	0.620
n-3	4.23±1.17	4.38±1.56	4.29±1.02	3.84±0.86	4.68±0.91	0.363
a-linolenic (C18:3)	0.54±0.20	0.56±0.20	0.53±0.21	0.52±0.16	0.57±0.31	0.916
EPA (C20:5)	0.46±0.18	0.53±0.24	0.42±0.15	0.43±0.16	0.52±0.11	0.223
DPA (C22:5)	0.83±0.23	0.83±0.21	0.86±0.17	0.80±0.28	0.85±0.33	0.891
DHA (C22:6)	2.39±0.93	2.46±1.26	2.48±0.82	2.10±0.67	2.74±0.73	0.410
n-6/n-3	6.77±2.12	6.80±1.93	6.61±2.96	7.14±1.63	6.16±1.29	0.765
SCD16	0.08±0.02	0.08±0.02	0.09±0.02	0.08±0.02	0.08±0.03	0.398
SCD18	1.85±0.40	1.77±0.38	1.95±0.38	1.90±0.45	1.68±0.36	0.376

EPA, Eicosapentaenoic; DHA, Docosahexaenoic; DPA, Docosapentaenoic SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid, n6, omega-6; n3, omega-3. Data are presented as mean ± standard deviation. Comparisons were performed by ANOVA way test followed by test t between pars. P-values < 0.05 were considered statistically significant. a, compared to Without lesion group.

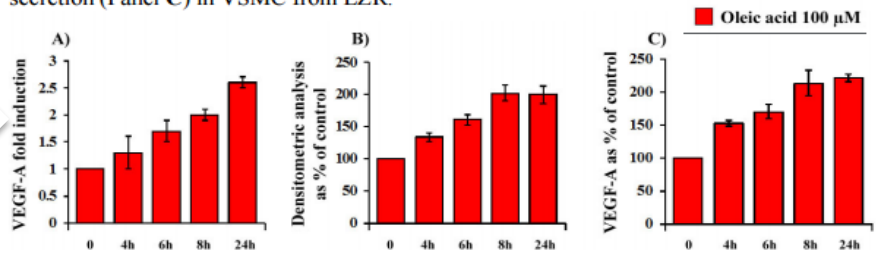
Artigo – Resultados e Discussão

Int. J. Mol. Sci. **2013**, *14*, 18861-18880; doi:10.3390/ijms140918861

Oleic Acid Increases Synthesis and Secretion of VEGF in Rat Vascular Smooth Muscle Cells: Role of Oxidative Stress and Impairment in Obesity

Gabriella Doronzo, Michela Viretto, Cristina Barale, Isabella Russo, Luigi Mattiello, Giovanni Anfossi † and Mariella Trovati *

Figure 1. Time-dependent (4–24 h of incubation with 100 µM oleic acid) influence of oleic acid on VEGF-A mRNA transcription (Panel **A**); protein synthesis (Panel **B**) and secretion (Panel **C**) in VSMC from LZR.



Ma *et al. Lipids in Health and Disease* 2011, **10**:53
http://www.lipidworld.com/content/10/1/53

LIPIDS IN HEALTH AND DISEASE

RESEARCH Open Access

Oleic acid induces smooth muscle foam cell formation and enhances atherosclerotic lesion development via CD36

Shuangtao Ma†, Dachun Yang†, De Li, Bing Tang and Yongjian Yang*

Camargo *et al. BMC Genomics* 2010, **11**:253
http://www.biomedcentral.com/1471-2164/11/253

RESEARCH ARTICLE Open Access

Gene expression changes in mononuclear cells in patients with metabolic syndrome after acute intake of phenol-rich virgin olive oil

Antonio Camargo†1, Juan Ruano†1, Juan M Fernandez1, Laurence D Parnell2, Anabel Jimenez1, Monica Santos-Gonzalez3, Carmen Marin1, Pablo Perez-Martinez1, Marino Uceda4, Jose Lopez-Miranda1 and Francisco Perez-Jimenez*1

Artigo – Resultados e Discussão

Pearson analysis showed a positive correlation between oleic acid with triglyceride levels (r = 0.397, p = 0.002), SCD16 (r = 0.352, p = 0.006) and SCD18 (**r = 0.751**, p = 0.027).

Nutrition 32 (2016) 88–94

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Applied nutritional investigation

Interaction of SNP in the CRP gene and plasma fatty acid profile in inflammatory pattern: A cross-sectional population-based study

Erica Oki M.Sc. [a], Marina M. Norde M.Sc. [a], Antônio A.F. Carioca M.Sc. [a], Renata E. Ikeda B.Sc. [a], José M.P. Souza Ph.D. [b], Inar A. Castro Ph.D. [c], Dirce M.L. Marchioni Ph.D. [a], Regina M. Fisberg Ph.D. [a], Marcelo M. Rogero Ph.D. [a,*]

[a] Nutrition Department, School of Public Health, University of São Paulo, Brazil
[b] Department of Epidemiology, School of Public Health, University of São Paulo, Brazil
[c] Department of Food and Experimental Nutrition, Faculty of Pharmaceutical Sciences, University of São Paulo, Brazil

Table 2
Plasma fatty acid (%) by inflammatory status

Plasma fatty acid (%)	Non-inflammatory cluster (n = 169)	Inflammatory Cluster (n = 93)	P-value*
SFA	39.44 (2.64)	39.89 (2.88)	0.209
Myristic (C14:0)	0.77 (0.56–1.06)	0.86 (0.54–1.21)	0.165
Palmitic (C16:0)	27.44 (2.55)	28.14 (2.68)	**0.039**
Stearic (C18:0)	11.15 (1.49)	10.81 (1.73)	0.098
MUFA	19.23 (16.37–21.72)	20.57 (17.48–23.76)	0.051
Palmitoleic (C16:1)	1.77 (1.35–2.24)	2.07 (1.05)	0.164
Oleic (C18:1)	17.38 (15.11–19.61)	18.68 (16.14–21.05)	0.058
PUFA	40.93 (4.81)	39.32 (4.85)	**0.011**
n-6	36.71 (4.58)	35.29 (4.63)	**0.018**
Linoleic (C18:2)	25.26 (3.89)	24.68 (4.20)	0.266
DGLA (C20:3)	2.45 (0.56)	2.43 (0.55)	0.761
Arachidonic (C20:4)	9.01 (2.19)	8.19 (1.75)	**0.002**
n-3	3.96 (3.61–4.64)	3.76 (3.45–4.43)	0.173
α-linolenic (C18:3)	0.61 (0.49–0.75)	0.65 (0.49–0.83)	0.247
Eicosapentaenoic (C20:5)	0.49 (0.36–0.63)	0.47 (0.35–0.60)	0.382
Docosapentaenoic C22:5	0.80 (0.65–0.99)	0.78 (0.70–0.87)	0.253
Docosahexaenoic (C22:6)	2.06 (1.70–2.46)	1.89 (1.54–2.42)	0.081
n3 HUFA	3.39 (2.92–4.01)	3.15 (2.68–3.77)	0.080
SCD-16 (C16:1/16:0)	0.06 (7.85–10.48)	0.07 (0.05–0.09)	0.299
SCD-18 (C18:1/C18:0)	1.55 (1.29–1.89)	1.74 (1.37–2.16)	**0.045**
D6D (C20:3/18:2 n-6)	0.07 (0.06–0.08)	0.07 (0.06–0.08)	0.343
D5D (C20:4/20:3)	3.76 (2.96–4.49)	3.32 (2.89–4.12)	**0.047**
n-6/n-3	9.22 (2.27)	9.20 (2.05)	0.953

DGLA, dihomo-γ-linoleic; HUFA, highly unsaturated fatty acid; MUFA, monounsaturated fatty acid; n3, omega-3; n6, omega-6; PUFA, polyunsaturated fatty acid; SCD, delta-9-desaturase; SFA, saturated fatty acid D6D, delta-6-desaturase; D5D, delta-5-desaturase
Values expressed as mean (standard deviation) or median (interquartile range)
* P-value for Student's *t* test. Values in bold are statistically significant ($P < 0.05$).

Artigo – Resultados e Discussão

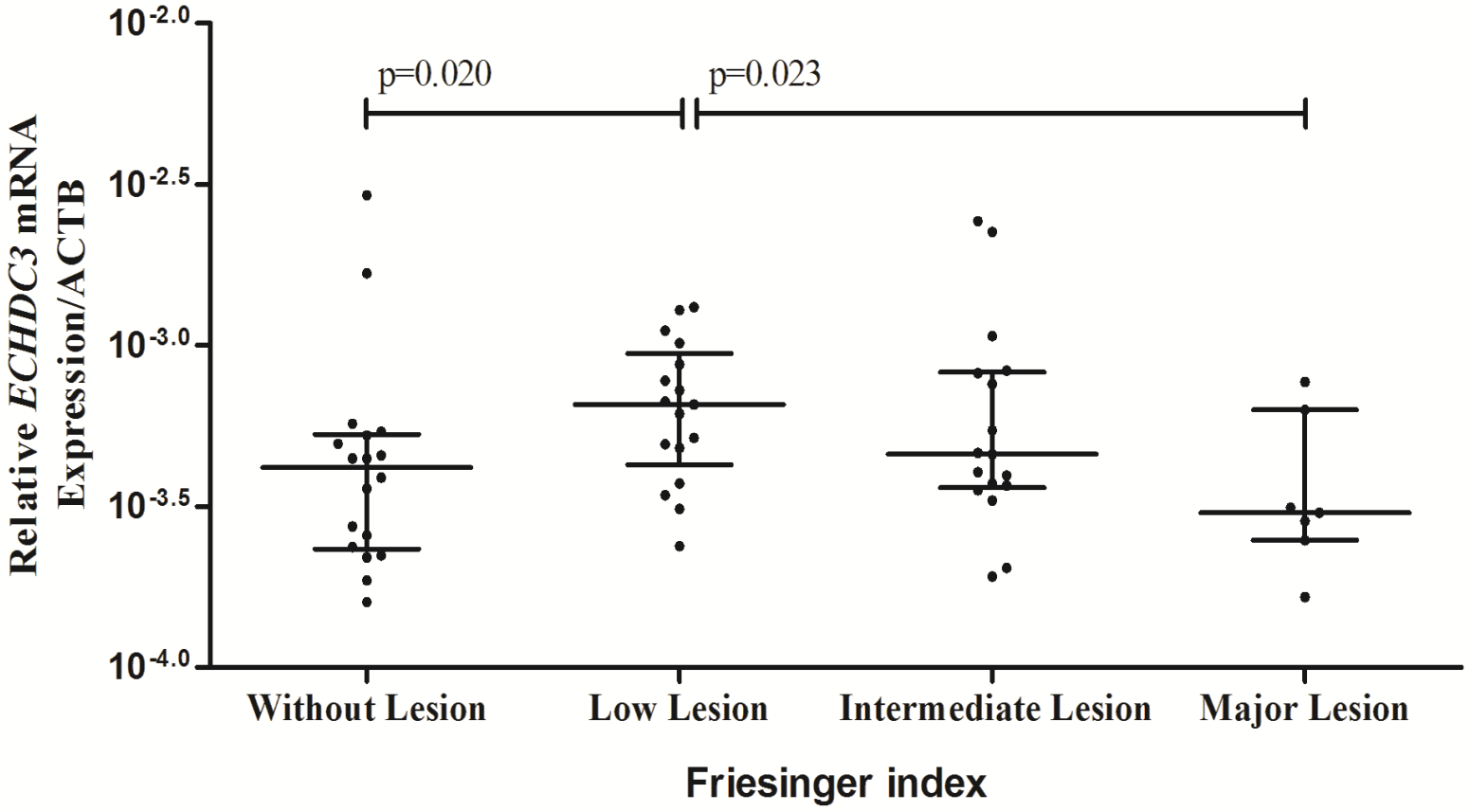


Figure 3. *ECHDC3* mRNA expression relative to ACTB expression according to the extent of coronary lesion. Data are presented as median and interquartile range. Relative expression of *ECHDC3* was calculated using the 2-deltaCT method. Statically analysis was performed by Kruskal-Wallis test followed by Mann Whitney test.

Artigo – Resultados e Discussão

ECHDC3 gene expression positively correlated with SFA (r = 0.259, p = 0.048).

ORIGINAL RESEARCH

American Heart Association | American Stroke Association

Enoyl Coenzyme A Hydratase Domain–Containing 2, a Potential Novel Regulator of Myocardial Ischemia Injury

Jianhai Du, PhD; Zhixin Li, PhD; Quan-Zhen Li, PhD; Tongju Guan, MSc; Qiuhui Yang, PhD; Hao Xu, PhD; Kirkwood A. Pritchard, Jr, PhD; Amadou K. S. Camara, PhD; Yang Shi, PhD

```
ECHDC2_RAT      MLRALPRALRLRRPWMSPGARGCAAHASTRTPEIQVQALTGPNQGITEILMNRPHARNAL 60
ECHDC2_MOUSE    MLRVLPRALRLPCSWRFSGARDCASHATTRTPEIQVQALTGPNQGITEILMNRPNARNAL 60
ECHDC2_HUMAN    MLRVL----CLLRPWRPLRARGCASDGAAGGSEIQVRALAGPDQGITEILMNRPSARNAL 56
                ***.*      *  .*    **.**:..::  .****:**:**:*********** *****
ECHDC2_RAT      GNVFVSELLEALAQLREDQQVRVLLFRSAVKGVFCAGADLKERERMSAAEVGTFVQRLRG 120
ECHDC2_MOUSE    GNVFVSELLEALAQLREDQQVRVLLFRSAVKGVFCAGADLKEREQMSDVEVGTFVQRLRG 120
ECHDC2_HUMAN    GNVFVSELLETLAQLREDRQVRVLLFRSGVKGVFCAGADLKEREQMSEAEVGVFVQRLRG 116
                **********:*******:*********.***************:** .***.*******
ECHDC2_RAT      LMSEIAAFPAPTIAAMDGFALGGGLELALACDLRIAASSAVMGLIETTRGLLPGAGGTQR 180
ECHDC2_MOUSE    LMSEIAAFPVPTIAAMDGFALGGGLELALACDLRIAASSAVMGLIETTRGLLPGAGGTQR 180
ECHDC2_HUMAN    LMNDIAAFPAPTIAAMDGFALGGGLELALACDLRVAASSAVMGLIETTRGLLPGAGGTQR 176
                **.:*****.************************:************************
ECHDC2_RAT      LPRCLGVALAKELIFTGRRLNGVQAHELGLVNHAVAQNEEGDAAYHRALALAQEILPQAP 240
ECHDC2_MOUSE    LPRCLGVALAKELIFTGRRLNGAQARELGLVNHAVAQNEEGNAAYHRALALAQEILPQAP 240
ECHDC2_HUMAN    LPRCLGVALAKELIFTGRRLSGTEAHVLGLVNHAVAQNEEGDAAYQRARALAQEILPQAP 236
                ********************.*.:*: ***************:***:** ***********
ECHDC2_RAT      IAVRLGKVAIDRGMEVDIASGMAIEHMCYAQNIPTQDRLEGMAAFREKRPPKFVGK 296
ECHDC2_MOUSE    IAVRLGKVAIDRGMEVDIASGMAIEQMCYAQNIPTQDRLEGMAAFREKRAPKFVGK 296
ECHDC2_HUMAN    IAVRLGKVAIDRGTEVDIASGMAIEGMCYAQNIPTRDRLEGMAAFREKRTPKFVGK 292
                ************ *********** *********:**************.******
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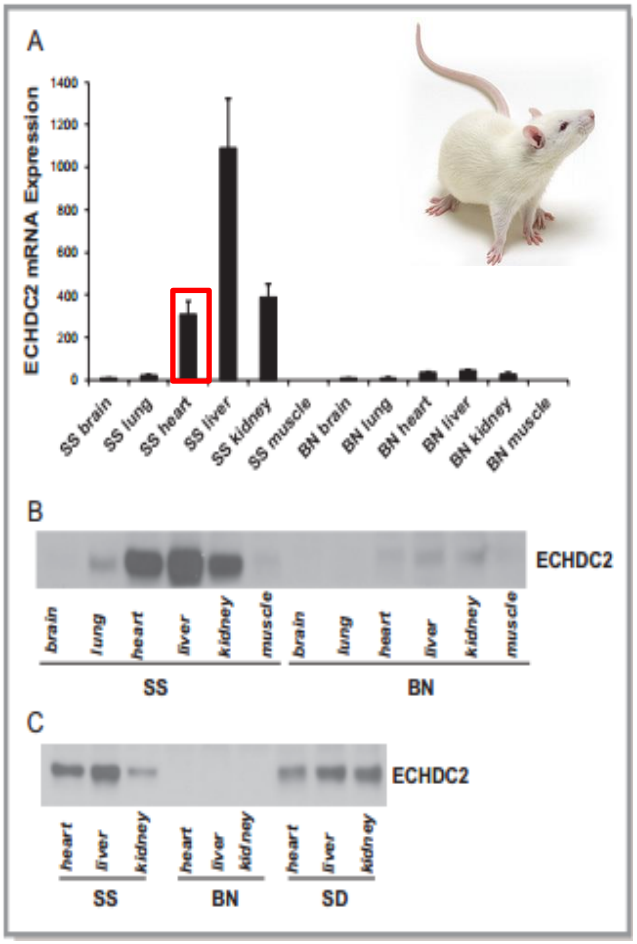
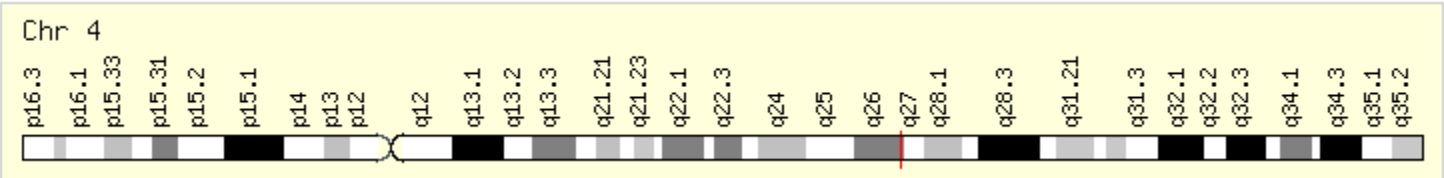


Doenças Cardiovasculares e Nutrigenética

Doenças Cardiovasculares e Nutrigenética

Gene
FABP2
4q28-q31



Polimorfismo na Ala54Thr (rs1799883)

Parâmetros antropométricos, perfil lipídico, resistência insulínica

PLOS ONE | A Peer-Reviewed, Open Access Journal

PLoS One. 2016; 11(9): e0163421.
Published online 2016 Sep 29. doi: 10.1371/journal.pone.0163421

Gene Polymorphisms of *FABP2*, *ADIPOQ* and *ANP* and Risk of Hypertriglyceridemia and Metabolic Syndrome in Afro-Caribbeans

Laurent Larifla,[1,2,*] Christine Rambhojan,[1] Marie-Odile Joannes,[1] Suliya Maimaitiming-Madani,[3] Jean-Paul Donnet,[1] Thérèse Marianne-Pépin,[1] Roger Chout,[1] Ronan Roussel,[4] and Lydia Foucan[1]

Larifla L, Rambhojan C, Joannes M-O, et al. Gene Polymorphisms of *FABP2*, *ADIPOQ* and *ANP* and Risk of Hypertriglyceridemia and Metabolic Syndrome in Afro-Caribbeans. Palmer ND, ed. *PLoS ONE*. 2016;11(9):e0163421. doi:10.1371/journal.pone.0163421.

Doenças Cardiovasculares e Nutrigenética

PLOS ONE | A Peer-Reviewed, Open Access Journal

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462 indivíduos afro-caribenhos

sem complicações cardiovasculares ou utilização de medicamentos

⇓

rs1799883- FABP2 , rs1501299- ADIPOQ and rs5065- ANP
três genotipagem

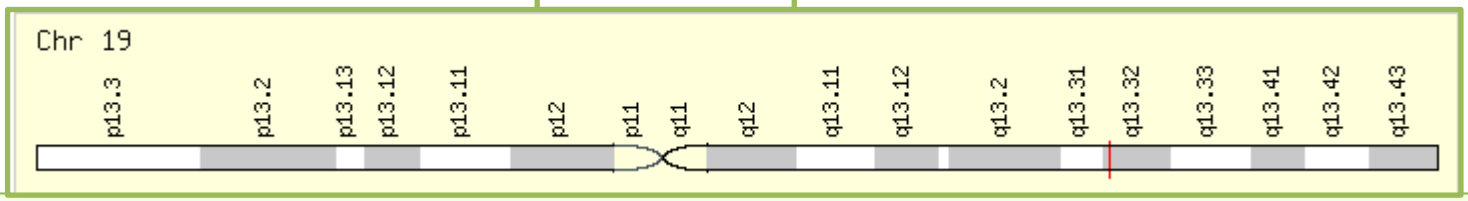
⇓

Associação de variantes de genes *FABP2 , ANP e ADIPOQ* com MetS
Efeito potencial sobre o metabolismo lipídico.

Larifla L, Rambhojan C, Joannes M-O, et al. Gene Polymorphisms of *FABP2*, *ADIPOQ* and *ANP* and Risk of Hypertriglyceridemia and Metabolic Syndrome in Afro-Caribbeans. Palmer ND, ed. *PLoS ONE*. 2016;11(9):e0163421. doi:10.1371/journal.pone.0163421.

Doenças Cardiovasculares e Nutrigenética

Gene
APOE
19q13.32



Uma glicoproteína do soro que se encontra **associada aos quilomícrons circulantes, aos quilomícrons remanescentes e as VLDL**

A **contribuição** da APOE para a variabilidade nas **concentrações do colesterol** é importante.

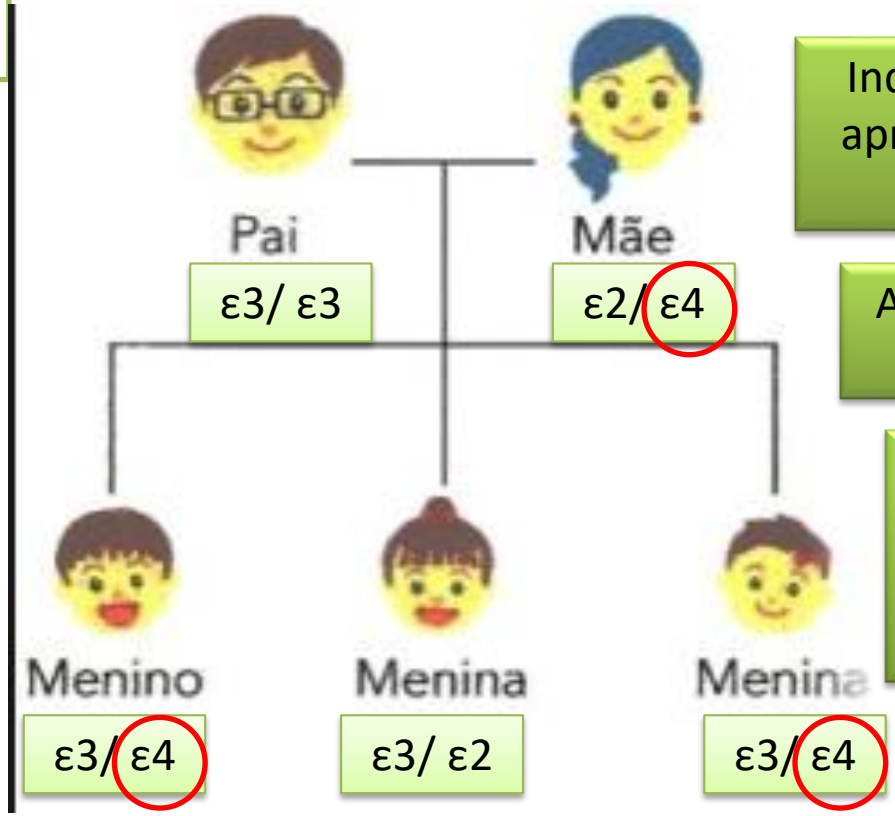
rs429358 e rs7412 – mais estudados

ε2 (10%) ε3 (60%) ε4 (30%)

-Minihane, AM; Jofre-Monseny, L.; Olano-Martin, E.; Rimbach, G. ApoE genotype, cardiovascular risk and resposiveness to dietary fat manipulation. Proceedings of the Nutrition Society. 2007; 66:183-97.
-Agrawl, S.; Mastana, S. Genetics of coronary heart disease with reference to APOAI-CII-AIV gene region. World Journal of Cardiology. 2014; 6(8): 755-763.

Doenças Cardiovasculares e Nutrigenética

Gene
APOE
19q13.32



Indivíduos que carreiam o alelo ε4 apresentam 40 a 50% de risco para DCV do que o ε3/ ε3

Apresentam maiores quantidades de TG e LDL-c

Maior redução nas concentrações de TG ao consumirem altas doses de DHA (3,7g/dia)

-Minihane, AM; Jofre-Monseny, L.; Olano-Martin, E.; Rimbach, G. ApoE genotype, cardiovascular risk and resposiveness to dietary fat manipulation. Proceedings of the Nutrition Society. 2007; 66:183-97.
-Agrawl, S.; Mastana, S. Genetics of coronary heart disease with reference to APOAI-CII-AIV gene region. World Journal of Cardiology. 2014; 6(8): 755-763.

Doenças Cardiovasculares e Nutrigenética

Gene
APOE
19q13.32

Am J Clin Nutr. 2012 Dec;96(6):1447-53. doi: 10.3945/ajcn.112.043240. Epub 2012 Nov 7.

APOE genotype influences triglyceride and C-reactive protein responses to altered dietary fat intake in UK adults.

Carvalho-Wells AL[1], Jackson KG, Lockyer S, Lovegrove JA, Minihane AM.

Indivíduos adultos do Reino Unido
carreadores do alelo ε4
apresentam maior redução nas concentrações de TG
ao consumirem altas doses de **DHA (3,7g/dia)**
que o ε3/ ε3

Os autores sugerem que isso ocorra em razão da **maior atuação da isoforma APOE4 na remoção de remanescentes de VLDL.**

As **isoformas ε2 e ε3** - metabolismo **HDL**

-Carvalho-Wells AL, Jackson KG, Lockyer S, Lovegrove JA, Minihane AM. Am J Clin Nutr. 2012 Dec;96(6):1447-53. doi: 10.3945/ajcn.112.043240.

Doenças Cardiovasculares e Nutrigenética

Gene
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APOE genotype influences triglyceride and C-reactive protein responses to altered dietary fat intake in UK adults.

Carvalho-Wells AL[1], Jackson KG, Lockyer S, Lovegrove JA, Minihane AM.

indivíduos **ε4/ ε4**
Aumento das concentrações séricas **de PCR**
consumo de gorduras saturadas

indivíduos **ε4/ ε4**
alta sensibilidade no consumo de lipídios.

-Carvalho-Wells AL, Jackson KG, Lockyer S, Lovegrove JA, Minihane AM. Am J Clin Nutr. 2012 Dec;96(6):1447-53. doi: 10.3945/ajcn.112.043240.

Doenças Cardiovasculares e Nutrigenética

Gene
APOE
19q13.32

Biochem Genet. 2010 Apr;48(3-4):342-55. doi: 10.1007/s10528-010-9331-6. Epub 2010 Jan 21.

The influence of nutrigenetics on the lipid profile: interaction between genes and dietary habits.

de Andrade FM[1], Bulhões AC, Maluf SW, Schuch JB, Voigt F, Lucatelli JF, Barros AC, Hutz MH.

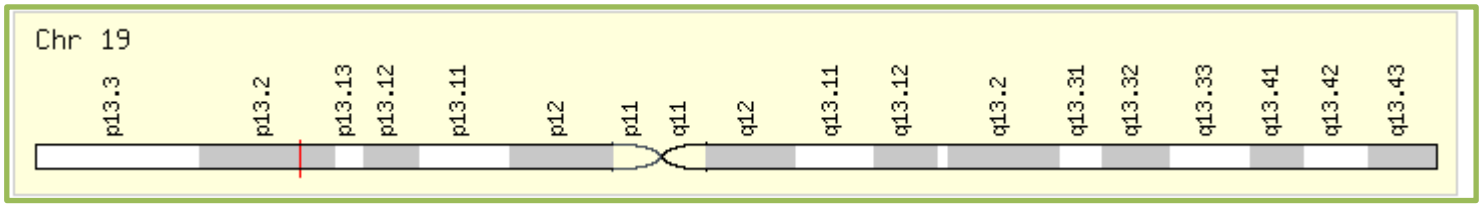
Carreadores do alelo **ε4**
São mais responsivos a mudanças no consumo de lipídios

Carreadores do alelo **ε2**
São mais responsivos a diminuição de carboidrato

-de Andrade FM, Bulhões AC, Maluf SW, Schuch JB, Voigt F, Lucatelli JF, Barros AC, Hutz MH. Biochem Genet. 2010 Apr;48(3-4):342-55. doi: 10.1007/s10528-010-9331-6.

Doenças Cardiovasculares e Nutrigenética

Gene
LDLr
19p13.2



Codifica a proteína LDLr – receptor de LDL

A expressão irá aumentar → Concentração intracelular de colesterol diminui

HHS Public Access
Author manuscript
Peer-reviewed and accepted for publication

About author manuscripts | Submit a manuscript

J Thromb Haemost. Author manuscript; available in PMC 2009 Sep 1.

LDL Receptor Polymorphisms and the Risk of Coronary Heart Disease: the Atherosclerosis Risk in Communities Study

N. Franceschini,[1] H. Muallem,[2] K.M. Rose,[1] E. Boerwinkle,[3] and N. Maeda[2]

-Franceschini N, Muallem H, Rose KM, Boerwinkle E, Maeda N. LDL Receptor Polymorphisms and the Risk of Coronary Heart Disease: the Atherosclerosis Risk in Communities Study. *Journal of thrombosis and haemostasis : JTH*. 2009;7(3):496-498. doi:10.1111/j.1538-7836.2008.03262.x.

Doenças Cardiovasculares e Nutrigenética

Gene
LDLr
19p13.2

SNPs avaliados – rs 1433099 (C>T) / rs 2738466 (A>G)

Frequência dos alelos T e G
27% (brancos), 54% (afro-americanos) – T
26% (brancos), 17% (afro-americanos) – G

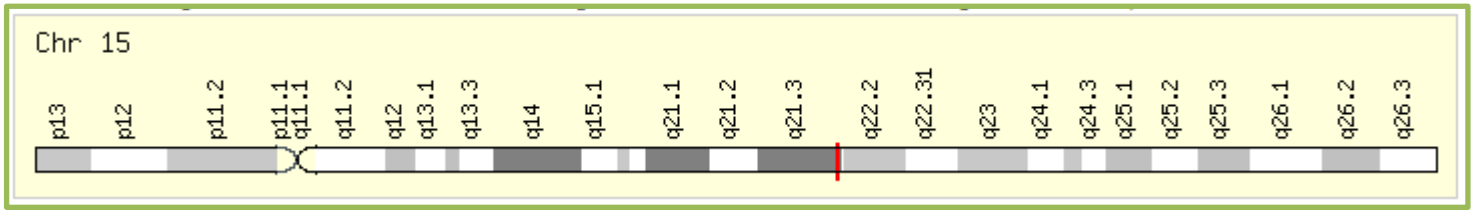
Afro-americanos – T (rs 1433099) – risco 23% DAC
TT (rs 1433099) – risco 47% DAC

SNP rs 2738466 – não foi associado a DAC

-Franceschini N, Muallem H, Rose KM, Boerwinkle E, Maeda N. LDL Receptor Polymorphisms and the Risk of Coronary Heart Disease: the Atherosclerosis Risk in Communities Study. *Journal of thrombosis and haemostasis : JTH*. 2009;7(3):496-498. doi:10.1111/j.1538-7836.2008.03262.x.

Doenças Cardiovasculares e Nutrigenética

Gene
LIPC
15q21-q23



É uma enzima sintetizada e secretada pelo fígado – metabolismo lipídico

A LIPC transforma a LDL em uma forma mais densa, menor → + aterogênica

Estudos contraditórios quanto ao papel aterogênico

- Nettleton JA, Steffen LM, Ballantyne CM, Boerwinkle E, Folsom AR. Associations between HDL-cholesterol and polymorphisms in hepatic lipase and lipoprotein lipase genes are modified by dietary fat intake in African American and White Adults . *Atherosclerosis*. 2007;194(2):e131-e140. doi:10.1016/j.atherosclerosis.2006.11.025.

Doenças Cardiovasculares e Nutrigenética

Gene
LIPC
15q21-q23

HHS Public Access
Author manuscript
Peer-reviewed and accepted for publication

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Atherosclerosis. 2007 Oct; 194(2): e131–e140.
Published online 2006 Dec 8. doi: 10.1016/j.atherosclerosis.2006.11.025

Associations between HDL-cholesterol and polymorphisms in hepatic lipase and lipoprotein lipase genes are modified by dietary fat intake in African American and White Adults [1,2,3]

Jennifer A. Nettleton, Lyn M. Steffen, Christie M. Ballantyne, Eric Boerwinkle, and Aaron R. Folsom

- Nettleton JA, Steffen LM, Ballantyne CM, Boerwinkle E, Folsom AR. Associations between HDL-cholesterol and polymorphisms in hepatic lipase and lipoprotein lipase genes are modified by dietary fat intake in African American and White Adults . *Atherosclerosis*. 2007;194(2):e131-e140. doi:10.1016/j.atherosclerosis.2006.11.025.

Doenças Cardiovasculares e Nutrigenética

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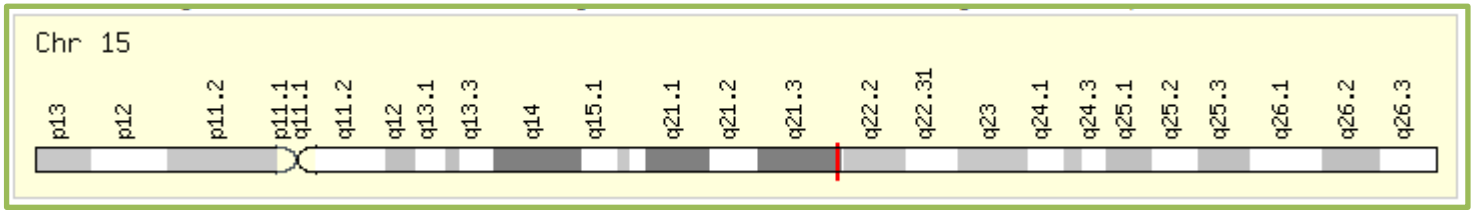
LIPC
15q21-q23



SNP -514 C>T (rs 1800588) – maiores concentrações HDL-c - TT

Foi avaliado no estudo a relação entre o SNP e o consumo alimentar

Indivíduos caucasianos – não houve interferência SNP sobre o perfil lipídico

Consumo de lipídios elevado:
HDL- c semelhante - Mulheres CC/Mulheres TT
HDL-c menor - Mulheres CT

- Nettleton JA, Steffen LM, Ballantyne CM, Boerwinkle E, Folsom AR. Associations between HDL-cholesterol and polymorphisms in hepatic lipase and lipoprotein lipase genes are modified by dietary fat intake in African American and White Adults . *Atherosclerosis*. 2007;194(2):e131-e140. doi:10.1016/j.atherosclerosis.2006.11.025.

Doenças Cardiovasculares e Nutrigenética

Gene	NCBI dbSNP	risco	resultado	análise
FABP2 (M)	rs1799883	A	GA	Alerta
FABP2 (F)	rs1799883	G	GA	Alerta
ApoE (SNPs)	rs429358/ rs7412	-	TT /CC	
ApoE genótipo		E4	E3/E3	Alerta
LDLR (C>T)	rs688	T	CT	Alerta
LDLR (G>T)	rs 6511720	T	GG	Ok
LDLR (C>T)	rs 5925	C	CT	Alerta
LIPC (A>G)	rs 2070895	A	GG	Ok
LIPC (C>T)	rs 1800588	C	CC	Cuidado

23 genes estudados

Doenças Cardiovasculares e Nutrigenética

- Dentre os SNPs avaliados a paciente **apresenta 6SNPs** que aumentam o risco para dislipidemia.
- Neste caso está presente **alto risco para hipercolesterolemia familiar (LDLR rs 688 e rs 5925**).
- **Baixas concentrações de HDL** (LIPC rs 1800588)
- A paciente pode não apresentar melhora da HDL com suplementação de ácidos graxos (APOA1).

- **Sugestões:** Controle rigoroso dos lipídios, visto que vários genes podem favorecer o aumento do colesterol total, aumento de HDL e redução de LDL-c.
- Consumir nozes e castanha de forma moderada.
- Monitorar exames.

Doenças Cardiovasculares e Nutrigenética

"Os avanços em **genômica nutricional** têm favorecido a elaboração de **recomendações nutricionais mais específicas**, visando a redução do risco e o tratamento das DCVs.

Os resultados de **grandes estudos não podem ser extrapolados**, uma vez que esta relação depende de **fatores ambientais, como a própria alimentação, das características de cada população**.

Muitos esforços ainda são necessários para que o **cuidado individual personalizado seja alcançado,** uma vez que a gama de **genes e SNPs envolvidos nas DCNT é extensa**."

Franco, LP; Morais, CC; Rogero, MM; Horst, MA; Cominetti, C. Doenças Cardiovasculares: enfoque nas dislipidemias. In: Cominetti, C; Rogero, MM; Horst, MA. Genômica Nutricional: dos fundamentos à nutrição molecular. Manole: 2016.

Obrigada!

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Especialista em Nutrição Clínica Funcional – UNICSUL - SC
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Pós-graduanda em Nutrição Esportiva Funcional – UNICSUL - SC
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