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Gesättigte Fettsäuren in der Ernährung

Ist ihr schlechtes Image wissenschaftlich begründet?

Fette und Öle: Wie wirken sie auf unsere Gesundheit und unser Wohlbefinden?

Bern, 23. September 2008

P. Colombani
Departement Agrar- und Lebensmittelwissenschaften ETH Zürich

Neue Zürcher Zeitung FORSCHUNG UND TECHNIK Mittwoch, 26. September 2007 · Nr. 223 B 3

Gesättigte Fette – besser als ihr Ruf

Fehlende wissenschaftliche Evidenz für eine Zunahme von Herz-Kreislauf-Erkrankungen

Von Paolo C. Colombani*

Seit rund 50 Jahren gelten Nahrungsfette im Allgemeinen und gesättigte Fettsäuren im Besonderen als Risikofaktoren für Herz-Kreislauf-Erkrankungen. Tatsächlich gibt es aber nur wenige Studien, die diese sogenannte Lipid-Hypothese untermauern; die allermeisten Arbeiten hingegen fanden keinen Einfluss auf das Erkrankungsrisiko.

Fette – sowohl pflanzlichen als auch tierischen Ursprungs – standen schon vor Jahrmillionen bei unseren Urahnen auf dem Speiseplan. So lässt sich unter anderem anhand der Struktur und Abnutzung fossiler Zähne schliessen, dass die Australopithecinen vor rund 3 Millionen Jahren ebenso wie vor rund 1,5 Millionen Jahren *Homo habilis* und *Homo erectus*, die ältesten Vertreter der Gattung *Homo*, sowohl härtere Nahrung wie Nüsse als auch zähere Nahrung wie Fleisch assen. Der Stoffwechsel des Menschen hatte also genügend Zeit, sich an das Vorhandensein von Fetten zu gewöhnen. Ein eindrückliches Beispiel hierfür ist unser Herz, das 60 bis 90 Prozent seiner Energie aus Fetten bezieht.

Hoher Anteil in der Muttermilch

Die offiziellen Empfehlungen zum Fettanteil in der Nahrung pendeln jedoch um einen engen und niedrigen Bereich von 30 Prozent der gesamten Energiezufuhr; für die gesättigten Fettsäuren, die zum Beispiel in Kokosfett, Butter, Palmfett und Schweineschmalz in grösseren Mengen vorkommen, liegt dieser Grenzwert gar bei 10 Prozent

Die Lebensmittelpyramide wurde bereits an die neuen – und alten – Erkenntnisse angepasst: Der Anteil der Kohlenhydrate an der Energiezufuhr wurde gesenkt, der Protein- und der Fettanteil erhöht. sge

Cholesterinsenkung beziehen und folglich auch nicht a priori schliessen, dass andere potenziell cholesterinsenkende Massnahmen wie die Reduktion der gesättigten Fettsäuren in der Nahrung das Risiko ähnlich beeinflussen wie die Medikamente. Beim Versuch, den Gesamtfettgehalt der Nahrung sowie deren Gehalt an gesättigten Fettsäuren von üblichen Werten auf die empfohlenen sowie weiter darunter zu senken (bei gleichzeitiger Erhöhung des Kohlenhydratanteils), werden nämlich nicht nur die erwünschten Veränderungen erzielt; das hat unter anderem eine 2005 im führenden Fachjournal der Ernährungswissenschaften, dem «American Journal of Clinical Nutrition», veröffentlichte Studie gezeigt. In dieser Arbeit sank das «schlechte» LDL-Cholesterin bei einer entsprechenden Diät zwar tatsächlich um 7 bis 12 Prozent, doch auch das «gute» HDL-Cholesterin nahm um 8 bis 12 Prozent ab, und die gesamte Menge an Fett im Blut stieg um 14 bis 16 Prozent.

Darüber hinaus führt die durch die restriktive Empfehlung bei den Fetten bedingte hohe Kohlenhydratzufuhr praktisch immer zu einer hohen glykämischen Belastung des Körpers. Der Stoffwechsel wird dabei für lange Zeit mit hohen Blutzuckerwerten konfrontiert und bedarf für die Normalisierung dieser Werte vermehrt des Hormons Insulin. Insulin regelt aber nicht nur den Blutzuckergehalt, sondern hemmt auch die Verstoffwechselung von Fetten in beträchtlicher Weise, was zu einer Anlagerung von Fett in der Muskulatur führt; diese intramuskuläre Fettanlagerung wird zurzeit als wichtige Ursache des Diabetes Typ 2 diskutiert. Die offiziellen Empfehlungen, wonach die Kohlenhydrate leicht über 60

Generelle Empfehlung zur möglichst niedrigen Zufuhr an gesättigten Fettsäuren (SFA)

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Dietary Reference Intakes for Energy … Fatty acids …

Saturated fatty acids are synthesized by the body to provide an adequate level needed for their physiological and structural functions; they have no known role in preventing chronic diseases. Therefore, neither an AI nor RDA is set for saturated fatty acids. There is a positive linear trend between total saturated fatty acid intake and total and low density lipoprotein (LDL) cholesterol concentration **and** increased risk of coronary heart disease (CHD). A UL is not set for saturated fatty acids because any incremental increase in saturated fatty acid intake increases CHD risk. It is neither possible nor advisable to achieve 0 percent of energy from saturated fatty acids in typical whole-food diets. This is because all fat and oil sources are mixtures of fatty acids, and consuming 0 percent of energy would require extraordinary changes in patterns of dietary intake.

Food and Nutrition Board and Institute of Medicine. Washington, DC. National Academy Press, 2005

Koronare Herzkrankheit

"Koronare Herzkrankheit" ist der Oberbegriff für Krankheitsbilder, die durch eine Mangeldurchblutung (Ischämie) der Herzkranzarterien hervorgerufen werden und deren Ursache meist eine **Arteriosklerose** ist.

Die koronare Herzkrankheit ist einer der Hauptgründe für vorzeitige Todesfälle, Invalidität und die Einbusse von Lebensqualität der Schweizer Bevölkerung.

Schweizerische Herzstiftung. http://www.swissheart.ch/d/herz/krankheiten/koronareherzkrankheit.htm, Zugriff 11.9.2008

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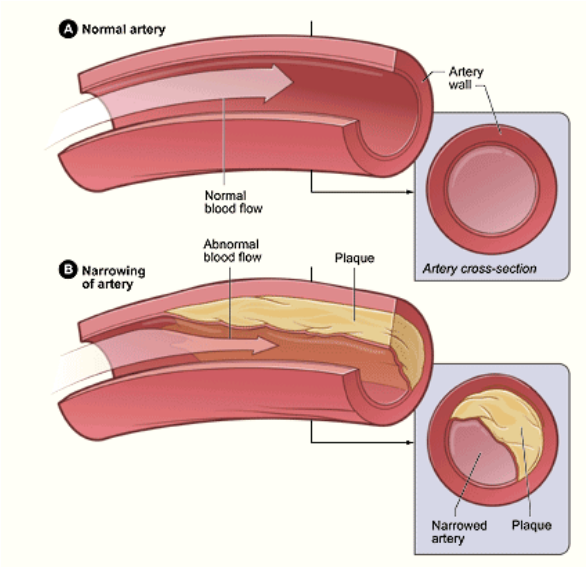
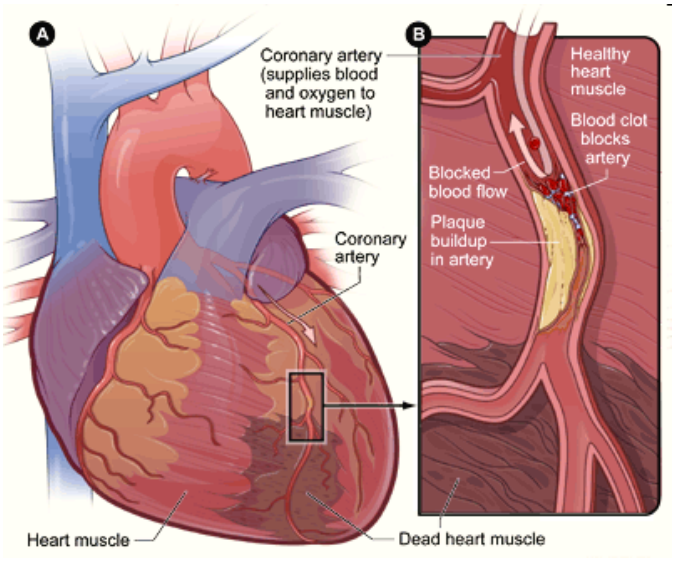
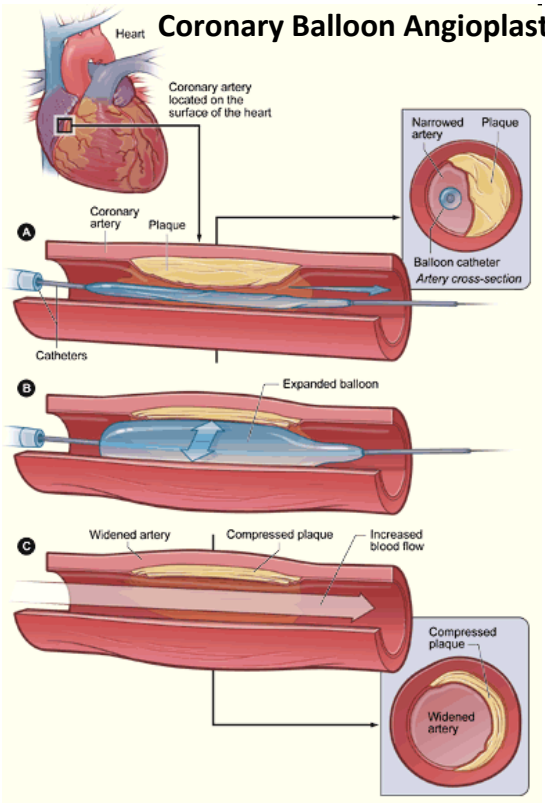


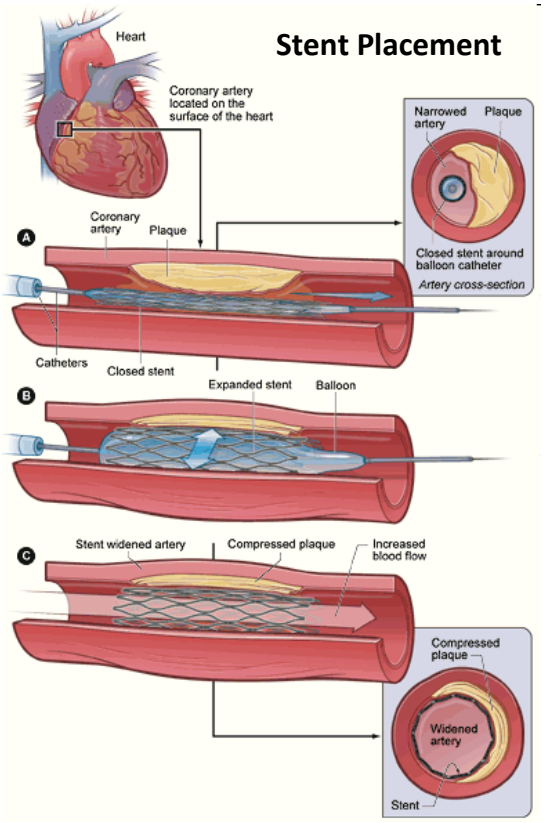


http://www.nhlbi.nih.gov/health/dci/Diseases/Cad/CAD_All.html, Zugriff 11.9.2008

Coronary Balloon Angioplasty

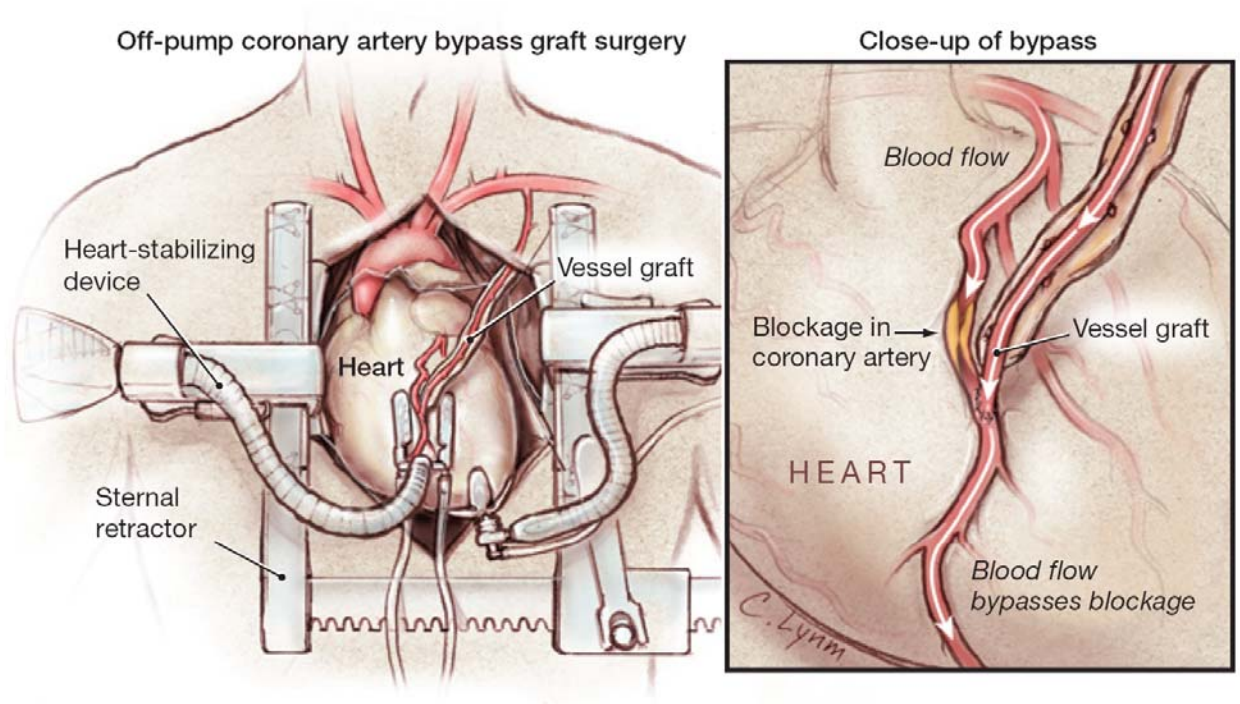


Stent Placement



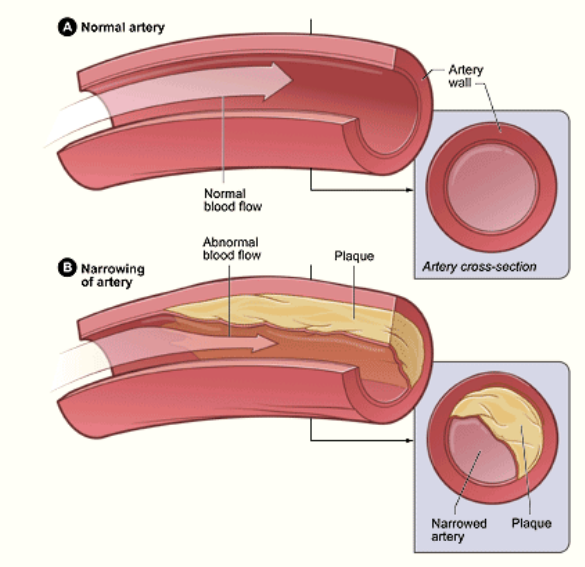
http://www.nhlbi.nih.gov/health/dci/Diseases/Cad/CAD_All.html, Zugriff 11.9.2008

Coronary Artery Bypass Grafting



Parmet et al. JAMA 299:1856, 2008

Wie kommt es zur Atherosklerose?



There is a positive linear trend between total saturated fatty acid intake and total and LDL cholesterol concentration **and** increased risk of coronary heart disease.

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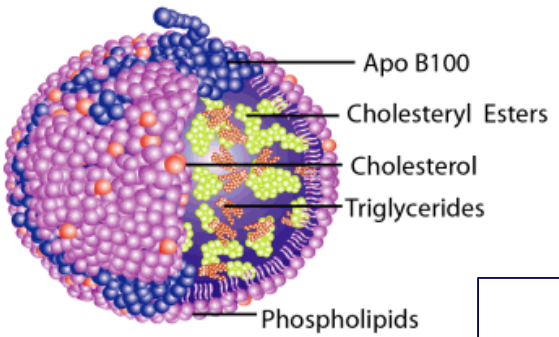
Wie kommt es zur Atherosklerose?



LDL Cholesterol

+ Oxidation = oxidiertes LDL Chol

Physiol Rev
84: 1381–1478, 2004; 10.1152/physrev.00047.2003.

Role of Oxidative Modifications in Atherosclerosis

ROLAND STOCKER AND JOHN F. KEANEY, JR.

B. Evidence in Support of the LDL Oxidation Hypothesis

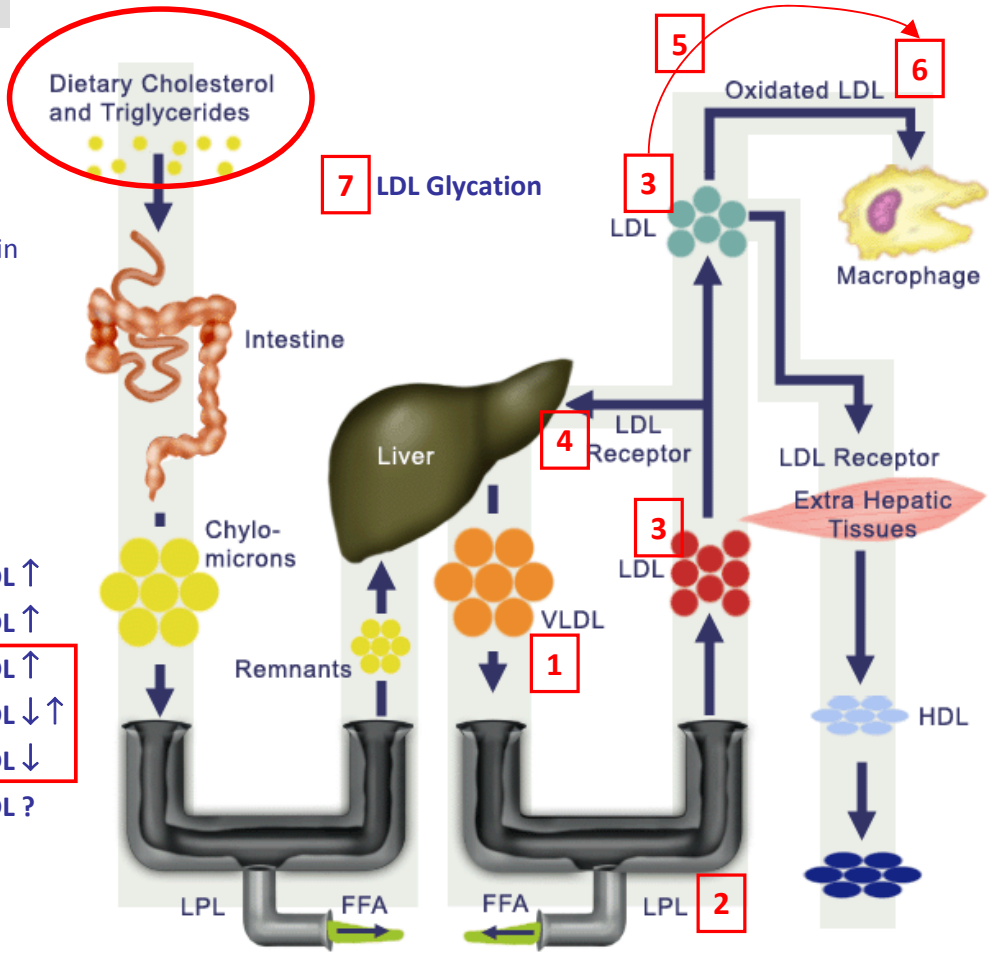
1. LDL does not support foam cell formation

Lipoprotein metabolism

VLDL Very Low Density Lipoprotein
LDL Low Density Lipoprotein
HDL High Density Lipoprotein

LPL Lipoprotein Lipase

1: Dietary glycemic load ↑ ⇒VLDL ↑
Glucose/Fructose ↑ ⇒VLDL ↑
SFA ↑ ⇒VLDL ↑
MUFA ↑ ⇒VLDL ↓↑
PUFA ↑ ⇒VLDL ↓
Cholesterol ↑ ⇒VLDL ?



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TECHNICAL REPORT SERIES

No. 117

STUDY GROUP ON ATHEROSCLEROSIS AND ISCHAEMIC HEART DISEASE

WORLD HEALTH ORGANIZATION

PALAIS DES NATIONS

GENEVA

1957

It is now known that various animal species differ markedly in their response to exogenous cholesterol, i.e., cholesterol contained in natural foods or added in pure form to the diet. Man is greatly different in this respect from the rabbit and the chick, the two favourite species for experimentation. It now appears that, provided other conditions are constant, **the blood cholesterol level in man usually responds little, or not at al, to variations in cholesterol intake corresponding to the range represented by the great majority of human diets**.

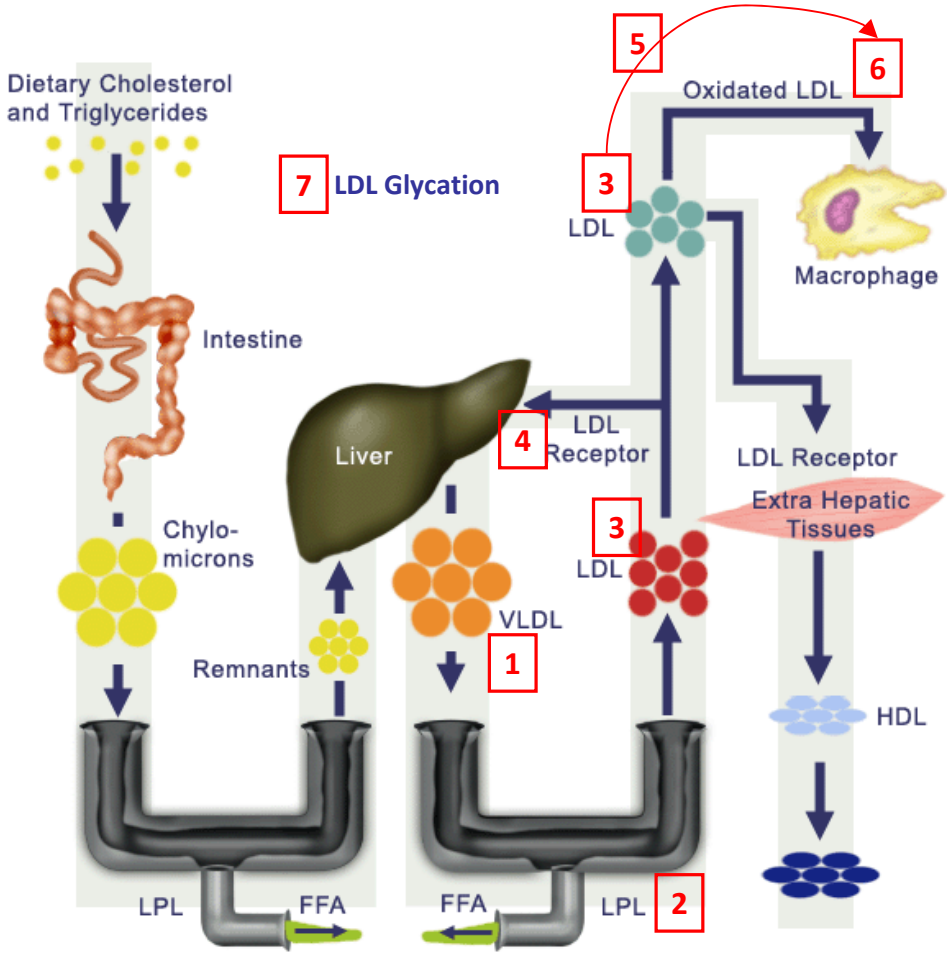
Lipoprotein metabolism

VLDL **V**ery **L**ow **D**ensity **L**ipoprotein
LDL **L**ow **D**ensity **L**ipoprotein
HDL **H**igh **D**ensity **L**ipoprotein

LPL **L**ipo**p**rotein **L**ipase

2: Problem
Endothelial LPL present in different locations: muscle, adipocytes, macrophages, …

LPL activation differs depending on location…



Lipoprotein lipase: the regulation of tissue specific expression and its role in lipid and energy metabolism

Karina Preiss-Landl, Robert Zimmermann, Günter Hämmerle and Rudolf Zechner

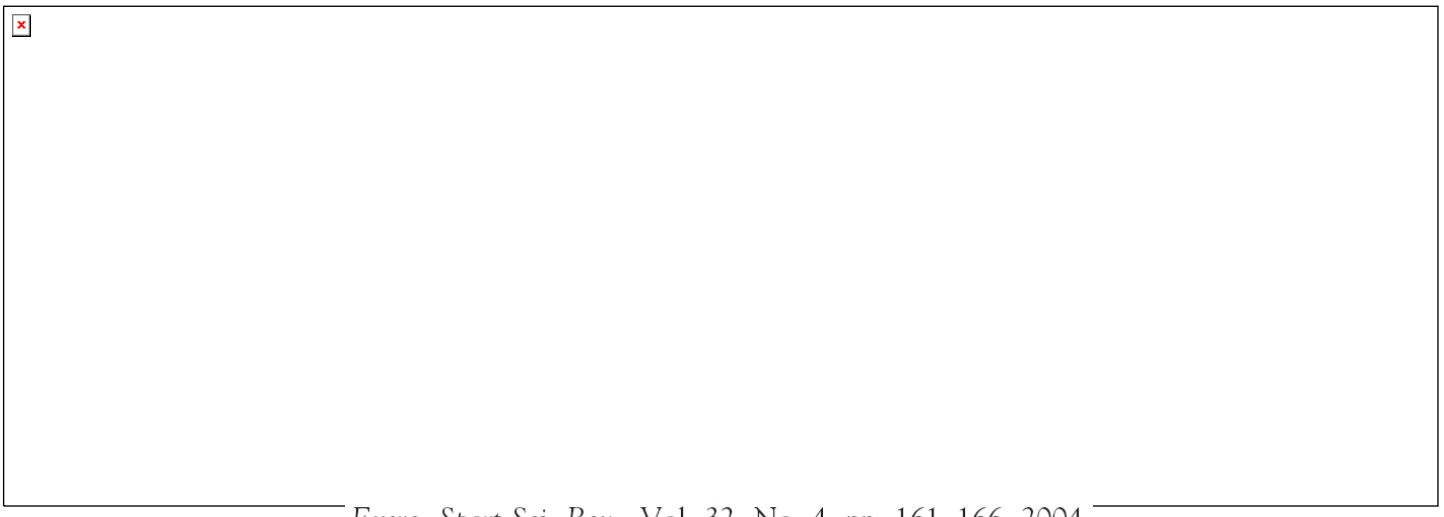
Curr Opin Lipidol 13:471–481. © 2002

…the tissue specific messenger RNA, translational, and post-translational regulation of LPL expression is poorly understood.

On one hand, high levels of LPL, especially in cardiac and skeletal muscle, are strongly associated with the anti-atherogenic lipoprotein profile of low plasma triglycerides and high HDL-cholesterol levels. On the other hand, high LPL expression levels in macrophages are associated with an increased risk to develop atherosclerotic lesions.

Exercise Physiology versus Inactivity Physiology: An Essential Concept for Understanding Lipoprotein Lipase Regulation

Marc T. Hamilton,[1,2] Deborah G. Hamilton,[1] and Theodore W. Zderic[1]



Exerc. Sport Sci. Rev., Vol. 32, No. 4, pp. 161–166, 2004.

Lipoprotein metabolism

VLDL Very Low Density Lipoprotein
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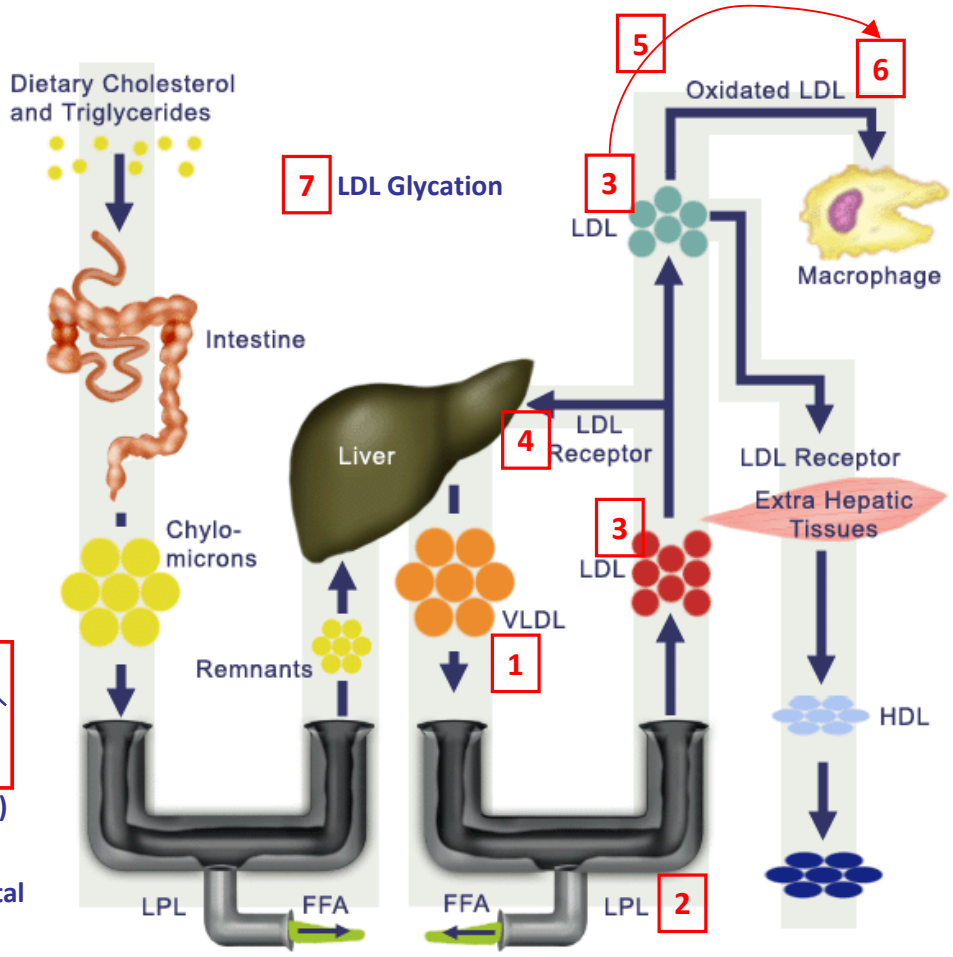

3: General belief

Dietary glycemic load ↑ ⇒LDL ↑

SFA ↑ ⇒LDL ↑
MUFA ↑ ⇒LDL ↓↑
PUFA ↑ ⇒LDL ↓

Cholesterol ↑ ⇒ LDL (↑)

Problem

In most studies only effects on total LDL investigated…

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2005

Journal of Atherosclerosis and Thrombosis Vol. 12, No. 5

Review

Lipid Triad or Atherogenic Lipoprotein Phenotype: A Role in Cardiovascular Prevention?

Manfredi Rizzo[1] and Kaspar Berneis[2]

[1] *Department of Clinical Medicine and Emerging Diseases, University of Palermo, Italy.*
[2] *Department of Internal Medicine, University Hospital Bruderholz, Switzerland.*

The term "lipid triad" or "atherogenic lipoprotein phenotype" has been introduced to describe a common form of dyslipidemia, characterized by three lipid abnormalities: increased plasma triglyceride levels, decreased HDL-cholesterol concentrations and the presence of **small, dense LDL particles**.

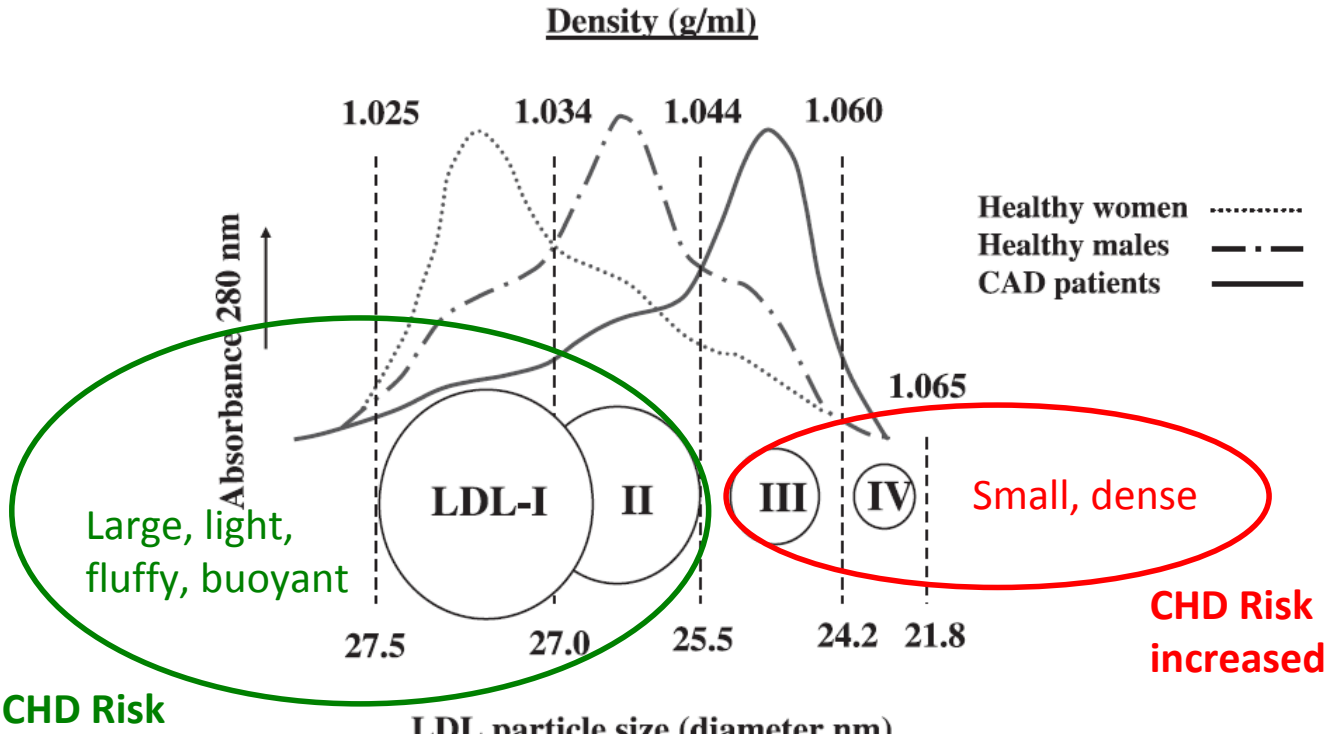
REVIEW ARTICLE

Who needs to care about small, dense low-density lipoproteins?

M. Rizzo,[1] K. Berneis[2]

Increasing evidence suggest that the 'quality' rather than only the 'quantity' of low-density lipoprotein (LDL) exerts a great influence on the cardiovascular risk. Small, dense LDL seem to be an important predictor of cardiovascular events and progression of coronary artery disease…

Int J Clin Pract, November 2007, **61**, 11, 1949–1956



Int J Clin Pract, November 2007, **61**, 11, 1949–1956

Lipoprotein metabolism

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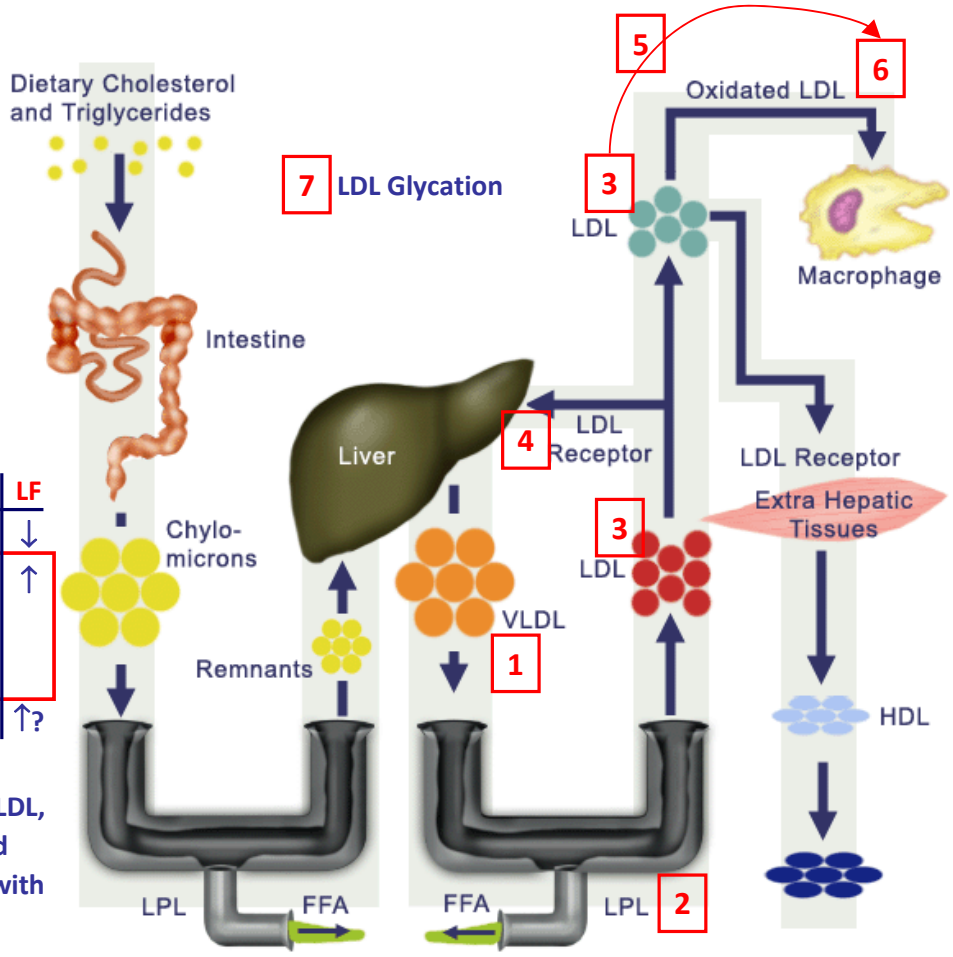

	LDL	SD	LF
3: Dietary glycemic load ↑ ⇒	↑		↓
SFA ↑ ⇒			↑
MUFA ↑ ⇒		↓	
*PUFA ↑ ⇒		↓	
*Cholesterol ↑ ⇒			↑?

*<u>Generally</u>
Small, dense LDL correlate with VLDL, so unless otherwise demonstrated (like with SFA), same risk factors with VLDL and LDL SD to be expected.

Lipoprotein metabolism

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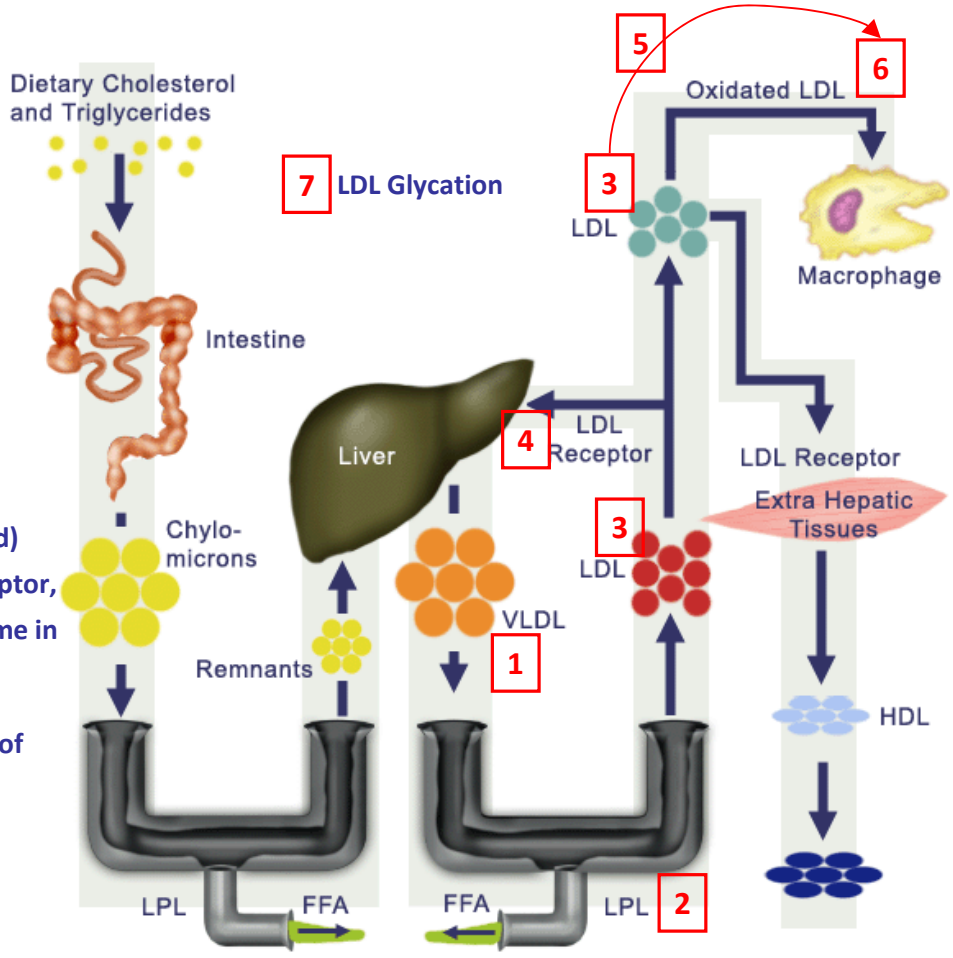
LDL Low Density Lipoprotein
HDL High Density Lipoprotein

LPL Lipoprotein Lipase



4: Modified LDL (oxidized or glycated) has decreased affinity to LDL receptor, leading to prolonged residence time in blood circulation.

LPL, in contrast, mediates uptake of modified LDL into macrophages.

Lipoprotein metabolism

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LDL Low Density Lipoprotein
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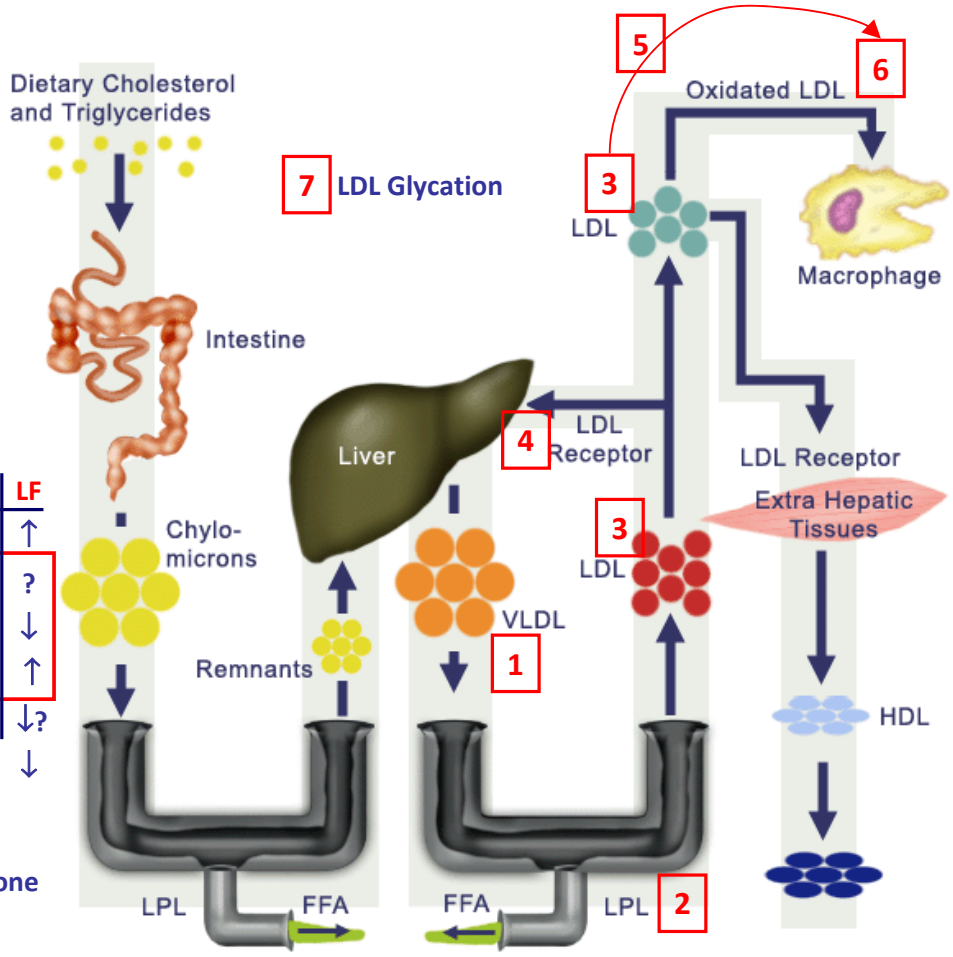

Oxidation of LDL	SD	LF
5: Dietary glycemic load ↑ ⇒	↑	↑
SFA ↑ ⇒	?	?
MUFA ↑ ⇒	↓	↓
n-6 PUFA ↑ ⇒	↑	↑
n-3 PUFA ↑ ⇒	↓?	↓?
Mediterranean diet ↑ ⇒	↓	↓

Generally
Small, dense LDL is likely more prone to oxidation than large, fluffy LDL

Lipoprotein metabolism

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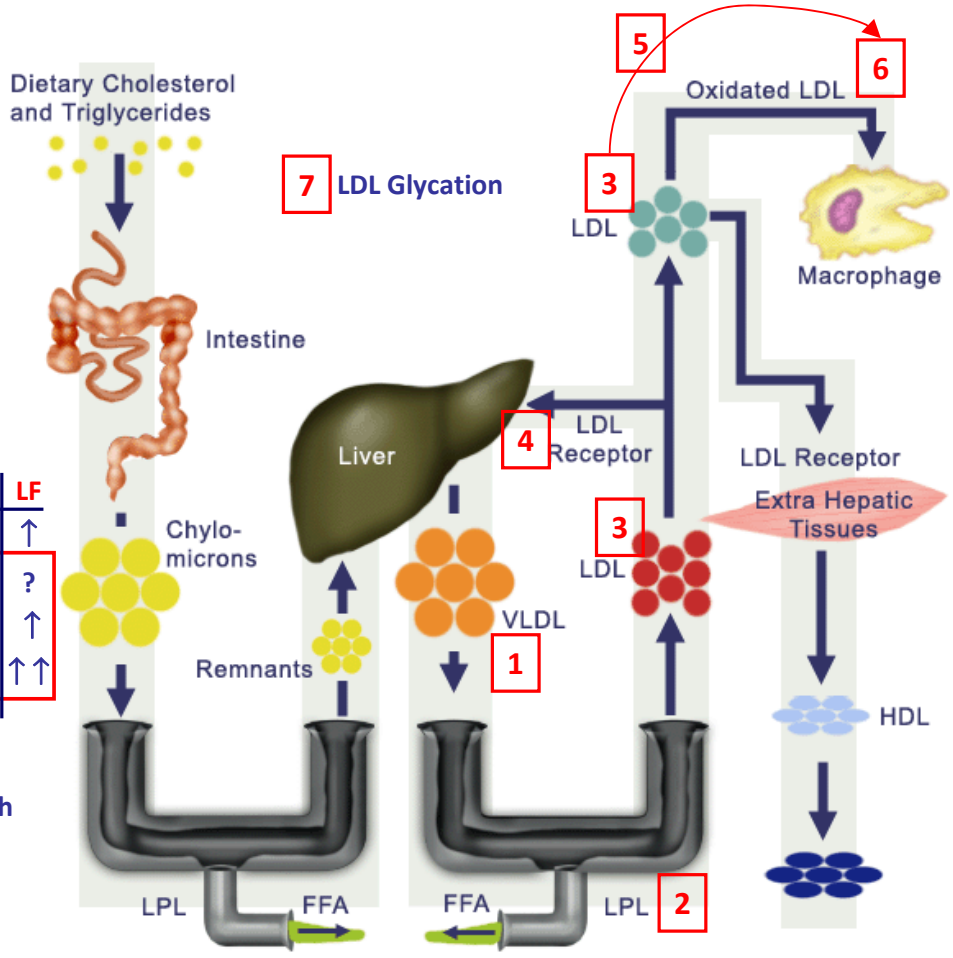
LDL Low Density Lipoprotein
HDL High Density Lipoprotein

LPL Lipoprotein Lipase

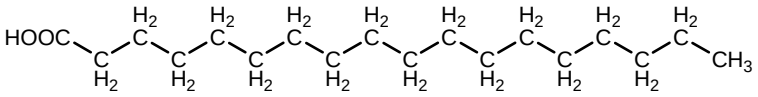


oxidized LDL	SD	LF
6: Dietary glycemic load ↑ ⇒	↑	↑
*SFA ↑ ⇒	?	?
*MUFA ↑ ⇒	↑	↑
*PUFA ↑ ⇒	↑↑	↑↑

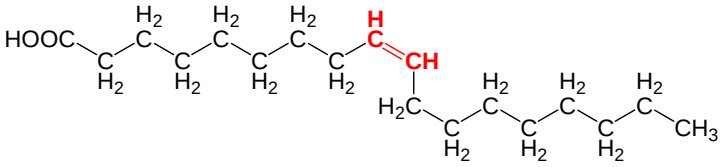
*Generally
Oxidative instability increases with number of double bounds.

Oxidationsstabilität von Fettsäuren

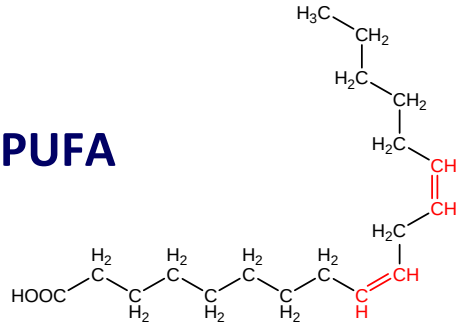
SFA

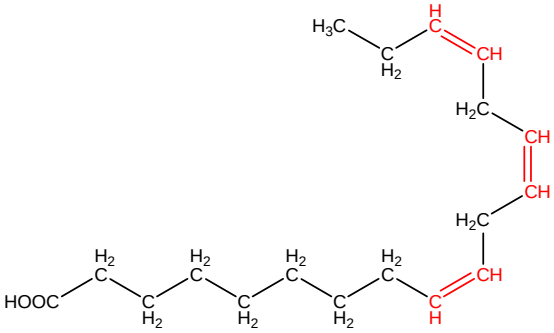


>> MUFA



>>>> PUFA





Nutritional, Dietary and Postprandial Oxidative Stress[1,2]

Helmut Sies,*†[3] Wilhelm Stahl,* and Alex Sevanian†[4]

Postprandial oxidative stress, as a subform of nutritional oxidative stress, ensues from **sustained postprandial hyperlipidemia and/or hyperglycemia** and is associated with a higher risk for atherosclerosis, diabetes, and obesity.

Unsaturated fatty acids incorporated into LDL and oxidized LDL are an atherogenic factor.

Postprandial oxidative stress is attenuated when dietary antioxidants are supplied together with a meal rich in oxidized or oxidizable lipids.

J. Nutr. 135: 969–972, 2005.

Glycation as an atherogenic modification of LDL

Nahla Younis[a], Reena Sharma[b], Handrean Soran[b], Valentine Charlton-Menys[b], Mohamed Elseweidy[a] and Paul N. Durrington[b]

[a]Department of Biochemistry, Faculty of Pharmacy, Zagazig University, Zagazig, Egypt and [b]Cardiovascular Research Group, School of Clinical and Laboratory Sciences, University of Manchester, Manchester, UK

Correspondence to Professor Paul N. Durrington, Cardiovascular Research Group, School of Clinical & Laboratory Sciences, Core Technology Facility (3rd Floor), University of Manchester, 46 Grafton Street, Manchester M13 9NT, UK
Tel: +44 161 275 1200/1; fax: +44 161 275 1183; e-mail: pdurrington@manchester.ac.uk

Current Opinion in Lipidology 2008, 19:378–384

Purpose of review
To highlight the potential importance of glycation as an atherogenic modification of LDL in people with diabetes and those without.
Recent findings
Small dense LDL which is known to be most closely associated with atherogenesis is more susceptible to glycation than more buoyant LDL. Glycation and oxidation of LDL appear to be intimately associated.
Summary
Glycation of LDL occurs chiefly due to the nonenzymatic reaction of glucose and its metabolites with the free amino groups of lysine in which LDL is rich. Higher concentrations of glycated LDL are present in individuals with diabetes than in those without, but, even in the latter, there is generally more circulating glycated LDL than oxidatively modified LDL. Probably, oxidation and glycation of LDL are at least partially interdependent, but both prevent LDL receptor-mediated uptake and promote macrophage scavenger receptor uptake. The recognition that LDL glycation is at least as important as oxidation in atherogenesis may lead to improvements in our understanding of its mechanism and how to prevent it.

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Biochemical Society Transactions (2007) Volume 35, part 3

The induction of macrophage foam cell formation by chylomicron remnants

K.M. Botham[1], E.H. Moore, C. De Pascale and F. Bejta

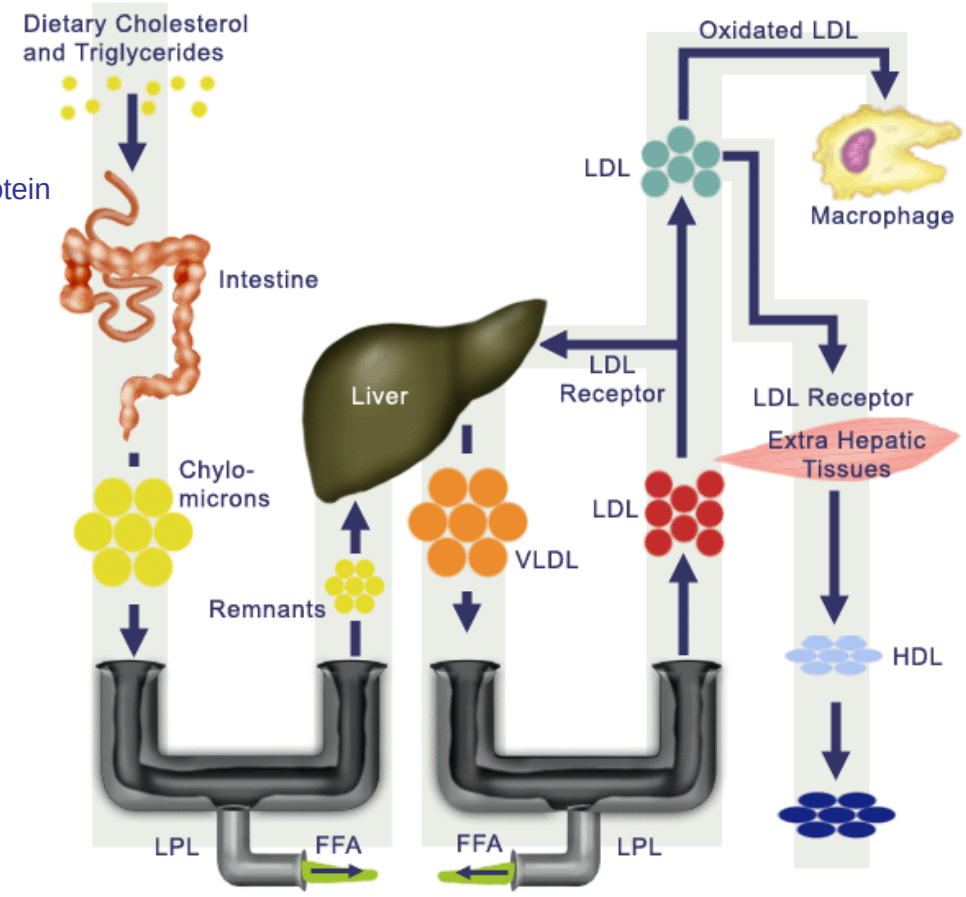
Recent studies, however, have provided considerable evidence to indicate that chylomicron remnants, which carry dietary lipids in the blood, induce foam cell formation without oxidation.

Furthermore, oxidation of chylomicron remnants, in striking contrast with LDL, inhibits, rather than enhances, their uptake and induction of lipid accumulation.

Lipoprotein metabolism

VLDL **V**ery **L**ow **D**ensity **L**ipoprotein
LDL **L**ow **D**ensity **L**ipoprotein
HDL **H**igh **D**ensity **L**ipoprotein

LPL **L**ipo**p**rotein **L**ipase



Evidenz-basierte Medizin und (in Anlehnung Ernährung)

…ist jede Form von medizinischer Behandlung, bei der patientenorientierte Entscheidungen ausdrücklich auf der Grundlage von nachgewiesener Wirksamkeit getroffen werden. Der Wirksamkeitsnachweis erfolgt dabei durch statistische Verfahren.

http://de.wikipedia.org/wiki/Evidenzbasierte_Medizin, Zugriff 16.9.2008

Evidenz-basierte Medizin und (in Anlehnung Ernährung)

- Level 1: Es gibt ausreichende Nachweise für die Wirksamkeit aus systematischen Überblicksarbeiten (Meta-Analysen) über zahlreiche randomisiert-kontrollierte Studien.
- Level 2: Es gibt Nachweise für die Wirksamkeit aus zumindest einer randomisierten, kontrollierten Studie.
- Level 3: Es gibt Nachweise für die Wirksamkeit aus methodisch gut konzipierten Studien, ohne randomisierte Gruppenzuweisung.
- Level 4a: Es gibt Nachweis für die Wirksamkeit aus klinischen Berichten.
- Level 4b: Stellt die Meinung respektierter Experten dar, basierend auf klinischen Erfahrungswerten bzw. Berichten von Experten-Komitees.

http://de.wikipedia.org/wiki/Evidenzbasierte_Medizin, Zugriff 16.9.2008

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Level	Therapy/Prevention, Aetiology/Harm	Prognosis	Diagnosis	Differential diagnosis/symptom prevalence study
1a	SR (with homogeneity*) of RCTs	SR (with homogeneity*) of inception cohort studies; CDR† validated in different populations	SR (with homogeneity*) of Level 1 diagnostic studies; CDR† with 1b studies from different clinical centres	SR (with homogeneity*) of prospective cohort studies
1b	Individual RCT (with narrow Confidence Interval‡)	Individual inception cohort study with ≥ 80% follow-up; CDR† validated in a single population	Validating** cohort study with good††† reference standards; or CDR† tested within one clinical centre	Prospective cohort study with good follow-up****
1c	All or none§	All or none case-series	Absolute SpPins and SnNouts††	All or none case-series
2a	SR (with homogeneity*) of cohort studies	SR (with homogeneity*) of either retrospective cohort studies or untreated control groups in RCTs	SR (with homogeneity*) of Level >2 diagnostic studies	SR (with homogeneity*) of 2b and better studies
2b	Individual cohort study (including low quality RCT; e.g., <80% follow-up)	Retrospective cohort study or follow-up of untreated control patients in an RCT; Derivation of CDR† or validated on split-sample§§§ only	Exploratory** cohort study with good†††reference standards; CDR† after derivation, or validated only on split-sample§§§ or databases	Retrospective cohort study, or poor follow-up
2c	"Outcomes" Research; Ecological studies	"Outcomes" Research		Ecological studies
3a	SR (with homogeneity*) of case-control studies		SR (with homogeneity*) of 3b and better studies	SR (with homogeneity*) of 3b and better studies
3b	Individual Case-Control Study		Non-consecutive study; or without consistently applied reference standards	Non-consecutive cohort study, or very limited population
4	Case-series (and poor quality cohort and case-control studies§§)	Case-series (and poor quality prognostic cohort studies***)	Case-control study, poor or non-independent reference standard	Case-series or superseded reference standards
5	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"

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Dietary Fat Intake and Risk of Coronary Heart Disease in Women: 20 Years of Follow-up of the Nurses' Health Study

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TABLE 2. Relative risks of coronary heart disease according to intake of specific types of dietary fat, Nurses' Health Study, United States, 1980–2000

	Quintile					P_{trend}
	1 (lowest)	2	3	4	5 (highest)	
Total fat						
Median (% of energy)	28.3	32.6	35.6	38.7	44.0	
Age-adjusted RR*	1	0.97	1.02	1.17	1.26	0.001
95% CI*		0.84, 1.12	0.88, 1.18	1.01, 1.35	1.07, 1.47	
Multivariate† RR	1	0.94	0.91	0.98	0.92	0.49
95% CI		0.81, 1.08	0.79, 1.06	0.84, 1.15	0.77, 1.09	
Saturated fat						
Median (% of energy)	10.1	11.9	13.3	14.8	17.6	
Age-adjusted RR	1	1.05	1.16	1.35	1.52	<0.0001
95% CI		0.91, 1.21	1.00, 1.34	1.16, 1.56	1.30, 1.79	
Multivariate‡ RR	1	0.94	0.96	1.01	0.97	0.93
95% CI		0.80, 1.11	0.79, 1.16	0.81, 1.26	0.73, 1.27	

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Low-Carbohydrate-Diet Score and the Risk of Coronary Heart Disease in Women

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N Engl J Med 2006;355:1991-2002.

Variable	Decile 1	Decile 10	P Value for Trend
Glycemic load	Relative Risk of Coronary Heart Disease in Women		
Age- and smoking-adjusted	1.0	1.13 (0.90–1.43)	0.10
Multivariate‡	1.0	1.90 (1.15–3.15)	0.003
Total fat			
Age- and smoking-adjusted	1.0	1.18 (0.95–1.46)	0.05
Multivariate**	1.0	0.99 (0.79–1.23)	0.86
Animal fat			
Age- and smoking-adjusted	1.0	1.36 (1.08–1.72)	0.003
Multivariate††	1.0	0.98 (0.75–1.28)	0.66
Vegetable fat			
Age- and smoking-adjusted	1.0	0.86 (0.69–1.09)	0.09
Multivariate‡‡	1.0	0.75 (0.57–0.98)	0.006

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Dietary fat intake and early mortality patterns – data from The Malmö Diet and Cancer Study

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Table 5 Relative risks (95% CI) for total mortality by quartiles of relative fat intake for women and men. Adjusted for age, alcohol consumption, smoking, social class, marital status, physical activity, BMI and fibre intake. Saturated, monounsaturated and polyunsaturated fats were included simultaneously in the multivariate analysis. Adjustments were made for total fat intake for the ratio between unsaturated and saturated fats. Also shown is the percentage of daily energy intake (EI) that the relevant fat contributes

	Quartiles				
	1	2	3	4	*P* for trend
Women					
Total fat					
RR (95% CI)	1.00 (ref)	1.08 (0.84–1.40)	0.93 (0.71–1.22)	1.22 (0.94–1.58)	0.26
% of EI	30.8	36.5	40.3	46.1	
Saturated fat					
RR (95% CI)	1.00 (ref)	0.96 (0.73–1.26)	0.82 (0.60–1.10)	0.89 (0.64–1.23)	0.39
% of EI	12.2	15.2	17.5	21.8	
Men					
Total fat					
RR (95% CI)	1.00 (ref)	0.92 (0.75–1.13)	**0.77 (0.62–0.95)**[b]	0.89 (0.72–1.10)	0.14
% of EI	31.7	37.8	41.7	47.7	
Saturated fat					
RR (95% CI)	1.00 (ref)	0.85 (0.67–1.07)	1.04 (0.81–1.32)	0.91 (0.69–1.19)	0.72
% of EI	12.3	15.3	17.6	22.3	

Table 6 Relative risks (95% CI) for cardiovascular mortality by quartiles of relative fat intake for women and men. Adjusted for age, alcohol consumption, smoking, social class, marital status, physical activity, BMI and fibre intake. Saturated, monounsaturated and polyunsaturated fats were included simultaneously in the multivariate analysis. Adjustments were made for total fat intake for the ratio between unsaturated and saturated fats. Also shown is the percentage of daily energy intake (EI) that the relevant fat contributes

	Quartiles				
	1	2	3	4	*P* for trend
Women					
Total fat					
RR (95% CI)	1.00 (ref)	0.99 (0.57–1.72)	0.80 (0.45–1.43)	0.74 (0.40–1.36)	0.25
% of EI	30.8	36.5	40.3	46.1	
Saturated fat					
RR (95% CI)	1.00 (ref)	0.89 (0.49–1.62)	0.76 (0.39–1.45)	0.55 (0.26–1.17)	0.16
% of EI	12.2	15.2	17.5	21.8	
Men					
Total fat					
RR (95% CI)	1.00 (ref)	0.76 (0.53–1.09)	0.74 (0.52–1.07)	**0.65 (0.45–0.94)**[b]	**0.03**
% of EI	31.7	37.8	41.7	47.7	
Saturated fat					
RR (95% CI)	1.00 (ref)	1.03 (0.69–1.53)	1.24 (0.82–1.89)	0.94 (0.58–1.53)	0.98
% of EI	12.3	15.3	17.6	22.3	

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Generelle Empfehlung zur möglichst niedrigen Zufuhr an gesättigten Fettsäuren (SFA)

Wirklich begründet??