

Dr. Scott M. Grundy -- Responsible For The Diet Which Is Killing America!

Here is one of the hidden architects of the massive billions suckered out of victims who fall for the lies put out by the [American Heart Association](#), and the Government. He has participated in more than 250 "research studies" where his name is shown as one of the authors. Perhaps more than any other individual he is responsible for the lies about cholesterol and the sale of billions of dollars of harmful "cholesterol lowering drugs."

[I have listed about 80 of the studies he has done, below. There are many more like these.](#)

I wrote about Dr. Grundy many years ago, in my Book, *Life Flow One, The Solution For Heart Disease*. Dr. Grundy was the man who introduced the terrible diet into the American Heart Association, which then released it to the public, and got all the traditional doctors to recommend it. It actually causes heart disease -- rather than prevent it. Avoid that diet at all costs -- remember Dr. Grundy as the author of death!

Read, below, some of the text from that book, about Dr. Grundy. Anything authored by him you must suspect of exactly opposite of the truth. You will often find him NOT listed as the primary author, so that some younger person can begin to build up a (false) reputation for this mythical science called "high cholesterol disease." But, you'll find, hidden, influences and money from the drug industries which control our current health care system.

Here is an "official" biography:

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Our research at the Center for Human Nutrition is concerned with the causes of hypercholesterolemia in humans. We have been able to identify several major causes of hypercholesterolemia beyond those resulting from genetic absence of the LDL receptors. We have found that some of these disorders are due to a reduced expression of LDL receptors, defective LDL particles that bind poorly to LDL receptors, excess formation of LDL particles and LDL that are overloaded with cholesterol. We are examining which of these abnormalities are due to genetics and which are due to dietary influence.

In addition we are studying an abnormality in cholesterol transport that we have named "atherogenic dyslipidemia", a syndrome characterized by a complex of lipoprotein disorders. Three important factors related to this syndrome are under investigation: an enzyme-lipoprotein lipase, an hepatic triglyceride lipase and one transport protein called cholesterol ester transport protein.

Additionally we are carrying out studies on the role of antioxidant vitamins in the prevention of coronary heart disease. We are studying the dietary fatty acids and their role in suppression of the immune system as well as their role in coronary heart disease.

We are continuing our studies in Diabetes Mellitus and we currently are studying the effects of obesity on insulin resistance.

Jialal I and Grundy SM (1992) Effect of dietary supplementation with alpha-tachopherol on the oxidative modification of low density lipoprotein . *J Lipid Res* 33:899-906

Vega GL and Grundy SM (1993) Occurrence of species of low-density lipoprotein with defective clearance in patients with primary moderate hypercholesterolemia. *Atherosclerosis and Thrombosis* 13:579-589

Denke MA and Grundy SM (1994) Individual responses to a cholesterol-lowering diet in fifty men with moderate hypercholesterolemia. *Arch Int Med* 154:317-325

Denke MA (1994) Individual responsiveness to a cholesterol-lowering diet in postmenopausal women with moderate hypercholesterolemia. *Arch Int Med* 154:1977-1982

Cohen JC, Wang Z, Grundy SM, Stoesz MR and Guerra R (1994) Variation at the hepatic lipase and apolipoprotein AI/CIII/AIV loci is a major cause of genetically determined variation in plasma HDL cholesterol levels. *J Clin Invest* 94:2377-2384

Garg A, Bantle JP, Henry RR, Coulston AM, Griver KA, Raatz SK, Brinkley L, Chen Y-Di, Grundy SM, Huet BA and Reaven GM (1994) Effects of varying carbohydrate content of diet in patients with non-insulin-dependent diabetes mellitus. *JAMA* 271:1421-1428

The War On America

From Page 90+ of Life Flow One, The Solution For Heart Disease

By Karl Loren

There are all sorts of wars. The most insidious are those where the enemy is completely hidden and the victim thinks he is receiving help. In fact, this was the final chapter in the intended betrayal of America into the hands of the legalized drug cartels.

The planning for this final attack started in 1971, immediately after the Framingham Study was released. Remember that the Framingham Study, itself, made no finding that dietary cholesterol caused heart disease -- in fact it found exactly the opposite.

But, the master planners simply took the color "white" and called it "black!"

In 1971 these master planners formed the Task Force on Arteriosclerosis. The terrible story behind this corrupt group is very well told by Thomas Moore in his Book, heart Failure.

Over many pages, in great detail, Mr. Moore reports on individual master planners. There was a peak in their activities in December 1984 when they formed the Consensus Development Conference.

One of these master planners was Dr. Daniel Steinbert, a physician who worked on cholesterol drug research at the University of California at San Diego. he was the Chairman of the conference and cut off any speaker who criticized the lie that was being launched. he is, perhaps, the father of death by cholesterol lies!

Another master planner was Dr. Robert I. Levy of Columbia University.

Another master planner was Dr. Richard Peto, an Oxford University epidemiologist.

Technically, the head master planner was Dr. Basil M. Rifkind. He presented false data to the Conference and Dr. Steinberg cut off any debate on it. Dr. Rifkind. had taken over from Dr. Steinberg, in some flawed research referred to as the Coronary Primary Prevention Trial, CPPT, of which more later. (Dr. Rifkind has been active spreading lies and preventing truth even a few years ago. he has not disappeared!)

Dr. Rifkind said, at the time: It is thought to be the first study in man to establish conclusively that lowering cholesterol reduces heart attacks and heart attack deaths."

Dr. William E. Conner, another master planner, from the Oregon Health Sciences University, told the Conference that the best method to lower cholesterol in the blood was a stringent diet, with lower cholesterol. This is false, but was presented with a straight face and fancy slides.

Another master planner was Scott M. Grundy, author of the American Heart Association diet which warned against butter and eggs. This may well have been the first point where the hidden puppeteers emerged into some public view. From the viewpoint of the hidden master planners, the American heart Association would be the best agency to take over since it could then give its blessing to all the research and promotion of the drug cure of the cholesterol disease!

You find it hard to believe that these lies were hatched in so blatant a fashion?

As powerful as Dr. Steinberg was, controlling the out-come of this Conference, the real masters were still behind the scenes -- the puppeteers pulling the strings.

There were prominent scientists who objected to this steam-roller being pushed along by Dr. Steinberg.

Ad. Edward H. Ahrens, Jr., from Rockefeller University said:

"I think the public is being hosed by the a NIH and the American heart Association.

They desire to do something good. They're hoping to God that this is the right thing to do. But they are not acting on the basis of scientific evidence, but on the basis of a plausible but untested idea."

Another objector was Dr. Thomas Chalmers, of Mt. Sinai Medical School and the Harvard School of Public Health:

"They have made an unconscionable exaggeration of all the data."

Another objector was David Kritchevsky who announced that his research did NOT show that dietary cholesterol caused blood cholesterol.

Another objector, simply ignored by Steinberg, was Robert E. Olson, a physician and specialist on nutrition at the State University of new York who said:

"We have to keep an open mind on whether we understand this disease or not. My view is that we do not."

Two objectors (Ahrens and Corday) felt so strongly about the conclusions being rammed down their throats by Steinberg that they wanted to issue a minority report. They were simply manipulated out of the way. There WAS no minority report.

Remember, this was all happening around Christmas time, in an academic setting, in 1984. This made no news for your local paper!

The conclusions of the Conference had already been prepared before the participants met. The concept of the findings appeared much earlier, in 1982, written by the American Heart Association.

By 1985, with all the powerful lies placed in the media and corrupt medical journals, the final plan for the attack on America was given a name:

National Cholesterol Education Program

You won't hear much about this attack today because, fortunately, it failed -- mostly.

It was launched in 1987.

In 1987 I had already, for more than two years, been heavily involved in giving lectures on heart disease. One of the first persons who ever heard me was Bob Hutton, in 1985. he tried the vitamin formula I recommended, and some months latter wrote this letter to me:

Dear Karl,

You asked me to report on my feelings after taking your vitamin formula.

May I go back in history?

Nine years ago I had colon cancer which required two operations in 8 days. I am completely cured -- only a little scar and 3 feet less of large colon.

Three years ago I had a double bypass and was told that I already was living on borrowed time.

Three weeks later the colon operation developed adhesions and I had another major operation.

Before that I had had 16 intravenous chelation treatments.

This leads me to always believe that I was kidnapped and the whole thing was created by a spasm of the heart! This fact was verified by my regular doctor - the one who gave me the chelation. Before Christmas I had 4 chelations -- one a month.

From that time on I had no treatments until I began taking your formula.

I was dizzy at times; my fingers turned gray and numb upon exposure to cold; and I was getting short of breath.

After taking Life Glow for only two weeks my dizziness disappeared. My fingers no longer responded negatively to cold. And I have a lot of energy. I was walking at the rate of 15 minutes per mile for three or four miles almost every day.

Now, after two months of your vitamin formula I find even bigger improvements. I am a member of Golden K (Kiwanis for old retired men). Yesterday we planted flowers around a retirement home.

I am not the youngest member but I did more work than any six of the others. I pushed a rototiller, then spaded more ground and planted flowers while on my knees without a single pain getting up.

If you have ever gardened you'll realize that work that I did. Today, I remembered that I had a knee that used to lock and pain when I arose from kneeling.

I feel truly great, ready for many more years to add to my 71. there seems to be one flaw in your formula; physically I feel great but I see no improvement in my mind. Of course, if you have nothing to build upon you can produce no results.

I know this is too long for your wants, so feel free to shorten and paraphrase. I would say that after taking your formula for only two weeks my dizziness disappeared and my circulation improved greatly. After 2 months I have much more energy and I found my carotid artery had opened up.

My long ago injured knee is responding in a wonderful manner. I have the energy of a fifty year old.

We leave for Canada the last of this month, where I will be chopping wood, moving boats, lifting logs and walking over hills -- and fishing.

As ever,m

Robert L. Hutton.

Read more of this expose of the cholesterol fraud in my Book, Life Flow One, The Solution For Heart Disease.

Lovastatin

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Mevacor

Here is one of the most evil drugs in traditional medicine. There are far worse drugs used in psychiatry, but for traditional medicine the false disease of "high cholesterol" has long been the target of the harmful drug with the generic name "Lovastatin." It is better known as the Merck drug which makes BILLIONS of dollars for Merck:

Official From Merck:

ADVERSE REACTIONS

MEVACOR is generally well tolerated; adverse reactions usually have been mild and transient. Less than 1% of patients were discontinued from controlled clinical studies of up to 14 weeks due to adverse experiences attributable to MEVACOR. About 3% of patients were discontinued from extensions of these studies due to adverse experiences attributable to MEVACOR; about half of these patients were discontinued due to increases in serum transaminases.

The median duration of therapy in these extensions was 5.2 years.

In the EXCEL study (see CLINICAL PHARMACOLOGY, *Clinical Studies*), 4.6% of the patients treated up to 48 weeks were discontinued due to clinical or laboratory adverse experiences which were rated by the investigator as possibly, probably or definitely related to therapy with MEVACOR. The value for the placebo group was 2.5%.

Clinical Averse Experiences

Adverse experiences reported in patients treated with MEVACOR in controlled clinical studies are shown in the table below.

MEVACOR (N=613) %	Placebo (N=82) %	Cholestyramine (N=88) %	Probucol (N=97) %
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<http://www.oralchelation.com/technical/grundy1.htm>

System	MEVACOR	Placebo	MEVACOR	Placebo
Gastrointestinal				
Constipation	4.9	--	34.1	2.1
Diarrhea	5.5	4.9	8.0	10.3
Dyspepsia	3.9	--	13.6	3.1
Flatulence	6.4	2.4	21.6	2.1
Abdominal pain/cramps	5.7	2.4	5.7	5.2
Heartburn	1.6	--	8.0	--
Nausea	4.7	3.7	9.1	6.2
Musculoskeletal				
Muscle cramps	1.1	--	1.1	--
Myalgia	2.4	1.2	--	--
Nervous System/Psychiatric				
Dizziness	2.0	1.2	--	1.0
Headache	9.3	4.9	4.5	8.2
Skin				
Rash/pruritus	5.2	--	4.5	--
Special Senses				
Blurred vision	1.5	--	1.1	3.1
Dysgeusia	0.8	--	1.1	--

Laboratory

Tests

Marked persistent increases of serum transaminases have been noted (see WARNINGS).

About 11% of patients had elevations of creatine phosphokinase (CPK) levels of at least twice the normal value on one or more occasions. The corresponding values for the control agents were cholestyramine, 9 percent and probucol, 2 percent. This was attributable to the noncardiac fraction of CPK. Large increases in CPK have sometimes been reported (see WARNINGS, *Skeletal Muscle*).

Expanded Clinical Evaluation of Lovastatin (EXCEL) Study

Clinical Adverse Experiences

MEVACOR was compared to placebo in 8,245 patients with hypercholesterolemia (total cholesterol 240,300 mg/dL in the randomized, double-blind, parallel, 48-week EXCEL study. Clinical adverse experiences reported as possibly, probably or definitely drug-related in > or = to 1% in any treatment group are shown in the table below. For no event was the incidence on drug and placebo statistically different.

	Placebo (N=1663) %	MEVACOR 20mg q.p.m. (N=1642) %	MEVACOR 40mg q.p.m. (N=1645) %	MEVACOR 20mg b.i.d. (N=1646) %	MEVACOR 40mg b.i.d. (1649) %
Body As a Whole					
Asthenia	1.4	1.7	1.4	1.5	1.2
Gastrointestinal					
Abdominal pain	1.6	2.0	2.0	2.2	2.5
Constipation	1.9	2.0	3.2	3.2	3.5
Diarrhea	2.3	2.6	2.4	2.2	2.6
Dyspepsia	1.9	1.3	1.3	1.0	1.6
Flatulence	4.2	3.7	4.3	3.9	4.5
Nausea	2.5	1.9	2.5	2.2	2.2
Musculoskeletal					
Muscle Cramps	0.5	0.6	0.8	1.1	1.1
Myalgia	1.7	2.6	1.8	2.2	3.0
Nervous System/ Psychiatric					
Dizziness	0.7	0.7	1.2	0.5	0.5
Headache	2.7	2.6	2.8	2.1	3.2
Skin					
Rash	0.7	0.8	1.0	1.2	1.3
Special Senses					
Blurred vision	0.8	1.1	0.9	0.9	1.2

Other clinical adverse experiences reported as possibly, probably or definitely drug-related in 0.5 to 1.0 percent of patients in any drug-treated group are listed below. In all these cases the incidence on drug and placebo was not statistically different. *Body as a Whole*: chest pain; *Gastrointestinal*: acid regurgitation, dry mouth, vomiting; *Musculoskeletal*: leg pain, shoulder pain, arthralgia; *Nervous*

Concomitant Therapy

In controlled clinical studies in which lovastatin was administered concomitantly with cholestyramine, no adverse reactions peculiar to this concomitant treatment were observed. The adverse reactions that occurred were limited to those reported previously with lovastatin or cholestyramine. Other lipid-lowering agents were not administered concomitantly with lovastatin during controlled clinical studies. Preliminary data suggests that the addition of either probucol or gemfibrozil to therapy with lovastatin is not associated with greater reduction in LDL cholesterol than that achieved with lovastatin alone. In uncontrolled clinical studies, most of the patients who have developed myopathy were receiving concomitant therapy with immunosuppressive drugs, gemfibrozil or niacin (nicotinic acid) (see WARNINGS *Skeletal Muscle*).

The following effects have been reported with drugs in this class. Not all the effects listed below have necessarily been associated with lovastatin therapy.

Skeletal: muscle cramps, myalgia, myopathy, rhabdomyolysis, arthralgias.

Neurological: dysfunction of certain cranial nerves (including alteration of taste, impairment of extra-ocular movement, facial paresis), tremor, dizziness, vertigo, memory loss, paresthesia, peripheral neuropathy, peripheral nerve palsy, psychic disturbances, anxiety, insomnia, depression.

Hypersensitivity Reactions: An apparent hypersensitivity syndrome has been reported rarely which has included one or more of the following features: anaphylaxis, angioedema, lupus erythematosus-like syndrome, polymyalgia rheumatica, vasculitis, purpura, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, eosinophilia, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever, chills, flushing, malaise, dyspnea, toxic epidermal necrolysis, erythema multiforme, including Stevens-Johnson syndrome.

Gastrointestinal: pancreatitis, hepatitis, including chronic active hepatitis, cholestatic jaundice, fatty change in liver; and rarely, cirrhosis, fulminant hepatic necrosis, and hepatoma; anorexia, vomiting.

Skin: alopecia, pruritus. A variety of skin changes (e.g., nodules, discoloration, dryness of skin/mucous membranes, changes to hair/nails) have been reported.

Reproductive: gynecomastia, loss of libido, erectile dysfunction.

Eye: progression of cataracts (lens opacities), ophthalmoplegia.

Laboratory Abnormalities: elevated transaminases, alkaline phosphatase, gamma-glutamyl transpeptidase, and bilirubin; thyroid function abnormalities.

Studies Featuring Dr. Scott M. Grundy

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Section A

Record 1 from database: **MEDLINE**

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Title

Linkage between cholesterol 7alpha-hydroxylase and high plasma low-density lipoprotein cholesterol concentrations.

Author

Wang J; Freeman DJ; Grundy SM; Levine DM; Guerra R; Cohen JC

Address

The Center for Human Nutrition, Dallas, Texas 75235-9052, USA.

Source

J Clin Invest, 1998 Mar, 101:6, 1283-91

Abstract

Interindividual differences in plasma low-density lipoprotein cholesterol (LDL-C) levels reflect both environmental variation and genetic polymorphism, but the specific genes involved and their relative contributions to the variance in LDL-C are not known. In this study we investigated the relationship between plasma LDL-C concentrations and three genes with pivotal roles in LDL metabolism: the low-density lipoprotein receptor (LDLR), apolipoprotein B (APOB), and cholesterol 7alpha-hydroxylase (CYP7). Analysis of 150 nuclear families indicated statistically significant linkage between plasma LDL-C concentrations and CYP7, but not LDLR or APOB. Further sibling pair analyses using individuals with high plasma LDL-C concentrations as probands indicated that the CYP7 locus was linked to high plasma LDL-C, but not to low plasma LDL-C concentrations. This finding was replicated in an independent sample. DNA sequencing revealed two linked polymorphisms in the 5' flanking region of CYP7. The allele defined by these polymorphisms was associated with increased plasma LDL-C concentrations, both in sibling pairs and in unrelated individuals. Taken together, these findings indicate that polymorphism in CYP7 contributes to heritable variation in plasma LDL-C concentrations. Common polymorphisms in LDLR and APOB account for little of the heritable variation in plasma LDL-C concentrations in the general population.

Language of Publication

English

Unique Identifier

98171515

MeSH Heading (Major)

Apolipoproteins B|BL/*GE/ME; Cholesterol 7 alpha-Monooxygenase|*GE/ME; Lipoproteins, LDL
Cholesterol|BL/*GE/*ME; Receptors, LDL|*GE/ME

MeSH Heading

Adult; Alleles; Apolipoproteins E|BL/GE/ME; Cholesterol|BL; DNA|AN/GE; Female; Human; Linkage
(Genetics); Lipoproteins|BL; Male; Middle Age; Pedigree; Polymerase Chain Reaction; Polymorphism
(Genetics); Sequence Analysis, DNA; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0021-9738

Country of Publication

UNITED STATES

Section A

Record 2 from database: **MEDLINE**

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Title

Effects of crystalline nicotinic acid-induced hepatic dysfunction on serum low-density lipoprotein cholesterol and lecithin cholesteryl acyl transferase.

Author

Tatò F; Vega GL; Grundy SM

Address

**Department of Clinical Nutrition of the University of Texas Southwestern Medical Center and
The Veterans Affairs Medical Center at Dallas, 75235-9052, USA.**

Source

Am J Cardiol, 1998 Mar, 81:6, 805-7

Abstract

Marked lowering of plasma total and low-density lipoprotein cholesterol levels that occur during treatment of dyslipidemia with pharmacologic doses of nicotinic acid result from hepatotoxicity. Therefore, a marked reduction in low-density lipoprotein may suggest generalized liver toxicity and drug treatment should be discontinued.

The above section, in red, represents totally false and deceptive information about niacin. There is a tremendous and important role that niacin can play in your health. Rigged "tests" and results such as this are the basis, later, for the drug companies to attempt to get niacin banned from sale on the basis that it is dangerous. These false studies need to be identified as they pop up -- Dr. Scott M. Grundy has depended on false reports for the spurious claims he makes. Karl.

Language of Publication

English

Unique Identifier

98186331

MeSH Heading (Major)

Acyltransferases|*ME; Lipoproteins, LDL Cholesterol|*BL; Liver|*PP; Liver Diseases|*BL/*CI/PP; Niacin|*AE; Phosphatidylcholines|*ME

MeSH Heading

Case Report; Human; Liver Function Tests; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0002-9149

Country of Publication

UNITED STATES

Section A

Record 3 from database: **MEDLINE**

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Title

Effect of statins on metabolism of apo-B-containing lipoproteins in hypertriglyceridemic men.

Author

Vega GL; Grundy SM

Address

Center for Human Nutrition and Department of Clinical Nutrition, University of Texas Southwestern Medical Center at Dallas and the Veterans Affairs Medical Center at Dallas, 75235-9052, USA.

Source

Am J Cardiol, 1998 Feb, 81:4A, 36B-42B

Abstract

Our investigations indicate that most patients with moderate hypertriglyceridemia have marked defects in the metabolism of low-density lipoprotein (LDL) apolipoprotein B. Moreover, these patients have 2 major defects in the metabolism of triglyceride-rich lipoproteins, i.e., an accumulation of remnant lipoproteins (due in part to delayed hepatic clearance) and increased fractional conversion of very-low-density lipoprotein (VLDL) to LDL. Defective triglyceride-rich lipoprotein metabolism has been associated with insulin resistance. Statin therapy in hypertriglyceridemic patients improves the lipoprotein profile by decreasing both LDL cholesterol and remnant lipoproteins. However, statin therapy does not normalize LDL apolipoprotein B metabolism, and high-density lipoprotein (HDL) cholesterol levels remain low. Therefore, consideration may be given to combining a statin with a drug that alters triglyceride metabolism (e.g., fibrate or nicotinic acid) in high-risk patients with hypertriglyceridemia.

Language of Publication

English

Unique Identifier

98186007

MeSH Heading (Major)

Antilipemic Agents|*TU; Apolipoproteins B|*ME; Hydroxymethylglutaryl-CoA Reductase Inhibitors|*TU; Hypertriglyceridemia|DT/*ME; Lipoproteins, LDL|*ME; Lovastatin|*TU

MeSH Heading

Comparative Study; Gemfibrozil|TU; Human; Insulin Resistance; Lipoproteins|BL; Lipoproteins, LDL Cholesterol|BL; Lipoproteins, VLDL Cholesterol|BL; Male; Niacin|TU

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0002-9149

Country of Publication

UNITED STATES

Section A

Record 4 from database: **MEDLINE**

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Title

Hypertriglyceridemia, atherogenic dyslipidemia, and the metabolic syndrome.

Author

Grundy SM

Address

Department of Clinical Nutrition, Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas, 75235-9052, USA.

Source

Am J Cardiol, 1998 Feb, 81:4A, 18B-25B

Abstract

The importance of high serum cholesterol, especially a high level of low-density lipoprotein (LDL) cholesterol, as a risk factor for coronary artery disease is well established. Likewise, efficacy for decreasing risk for coronary artery disease by LDL-lowering therapy has recently been documented through clinical trials. However, many high-risk patients manifest elevated serum triglyceride levels, and the role of hypertriglyceridemia in causation of coronary artery disease remains to be elucidated. Nonetheless, there is growing evidence that hypertriglyceridemia is a marker for increased risk for coronary artery disease; in fact, it can serve as a marker for several atherogenic factors. These factors include increased concentrations of atherogenic triglyceride-rich lipoproteins; the atherogenic lipoprotein phenotype, or lipid triad; and the metabolic syndrome. The lipid triad consists of elevated serum triglycerides, small LDL particles, and low high-density lipoprotein (HDL) cholesterol. The metabolic syndrome includes the coexistence of the lipid triad, elevated blood pressure, insulin resistance (plus glucose intolerance), and a prothrombotic state. Many previous studies indicate that hypertriglyceridemia is strongly associated with all of these atherogenic factors. The clinical approach to treatment of patients with hypertriglyceridemia thus requires a broad-based strategy that includes reduction of atherogenic triglyceride-rich lipoproteins, reversal of the lipid triad, and favorable modification of the metabolic syndrome. The development of therapeutic regimens to effect these changes poses a challenge for future research on the problem of hypertriglyceridemia.

Language of Publication

English

Unique Identifier

98186004

MeSH Heading (Major)

Atherosclerosis|*ME; Hyperlipidemia|*ME; Hypertriglyceridemia|DT/EP/GE/*ME

MeSH Heading

Animal; Antilipemic Agents|TU; Coronary Disease|ET; Human; Hydroxymethylglutaryl-CoA Reductase Inhibitors|TU; Hypertension|CO; Insulin Resistance; Lipoproteins, HDL Cholesterol|BL; Lipoproteins, LDL|BL; Lipoproteins, LDL Cholesterol|BL; Lipoproteins, VLDL|BL; Multivariate Analysis; Risk Assessment; Risk Factors; Syndrome; Triglycerides|BL

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0002-9149

Country of Publication

UNITED STATES

Section A

Record 5 from database: **MEDLINE**

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Title

Hepatic lipase activity is lower in African American men than in white American men: effects of 5' flanking polymorphism in the hepatic lipase gene (LIPC).

Author

Vega GL; Clark LT; Tang A; Marcovina S; Grundy SM; Cohen JC

Address

The Center for Human Nutrition, Department of Clinical Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052, USA.

Source

J Lipid Res, 1998 Jan, 39:1, 228-32

Abstract

Plasma high density lipoprotein cholesterol (HDL-C) concentrations are higher in African American men than in white men, but the mechanism(s) responsible for this ethnic difference has not been elucidated. This study examined the relationship between hepatic lipase activity, plasma HDL-C concentrations, and a hepatic lipase polymorphism (-514T) in African American and white American men. Consistent with previous reports, plasma HDL-C concentrations were significantly higher in African American men than in white American men. Mean post-heparin plasma hepatic lipase activity was significantly lower in African American than in white American men (27 +/- 12 vs. 44 +/- 17 mmol x h(-1) x l(-1), P < 0.001). The -514T hepatic lipase allele was associated with low hepatic lipase activity in both populations, and was 3-fold more common among African Americans than white Americans. Taken together, these data suggest that genetic differences in hepatic lipase activity contribute to the differences in plasma HDL-C concentrations between African American men and white American men.

Language of Publication

English

Unique Identifier

98129503

MeSH Heading (Major)

Caucasoid Race|*; Lipase|*BL/*GE; Liver|*EN; Negroid Race|*; Polymorphism (Genetics)|*

MeSH Heading

Adult; Alleles; Genotype; Heparin|BL; Heterozygote; Homozygote; Human; Lipoproteins, HDL Cholesterol|BL;

Publication Type

JOURNAL ARTICLE

ISSN

0022-2275

Country of Publication

UNITED STATES

Section A

Record 6 from database: **MEDLINE**

[Go To The Top](#)

Title

Multifactorial causation of obesity: implications for prevention.

Author

Grundy SM

Address

Department of Clinical Nutrition, Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas, 75235-9052, USA. sgrund@mednet.swmed.edu

Source

Am J Clin Nutr, 1998 Mar, 67:3 Suppl, 563S-72S

Abstract

Obesity threatens to become the foremost cause of chronic disease in the world. Being obese can induce multiple metabolic abnormalities that contribute to cardiovascular disease, diabetes mellitus, and other chronic disorders. Unfortunately, prevalence of obesity is increasing both in the United States and worldwide. Reasons for the rising prevalence include urbanization of the world's population, increased availability of food supplies, and reduction of physical activity. Although severe obesity has received much attention in the clinical setting, most obesity in the general public is only moderate. Even so, moderate obesity can elicit several metabolic abnormalities that are precursors to chronic disease. Therefore, for the population as a whole, moderate obesity is responsible for most obesity-related disorders. Moderate obesity is undoubtedly multifactorial in origin, and acquired influences probably exceed genetic factors in its causation. These acquired causes thus deserve greater attention in the development of a public health strategy for the control of overweight in the general population. A major public health effort is urgently needed to counter the increasing frequency of moderate obesity in the United States and throughout the world.

Language of Publication

English

Unique Identifier

98156903

MeSH Heading (Major)

Obesity|*/PC

MeSH Heading

Aging|ME/PH; Basal Metabolism; Coronary Disease|BL/CO; Cultural Characteristics; Dietary Fats|AD; Disease Susceptibility|GE; Energy Intake; Exercise; Human; Risk Factors

Publication Type

ISSN

0002-9165

Country of Publication

UNITED STATES

Section A

Record 7 from database: **MEDLINE**

[Go To The Top](#)

Title

Influence of stearic acid on cholesterol metabolism relative to other long-chain fatty acids.

Author

Grundy SM

Address

Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas.

Source

Am J Clin Nutr, 1994 Dec, 60:6 Suppl, 986S-990S

Abstract

Stearic acid is a long-chain saturated fatty acid. However, in contrast with other saturated fatty acids, stearic acid apparently does not raise serum cholesterol concentrations. Studies carried out three decades ago provided strong suggestive evidence that this was the case. More recent investigations that specifically compared stearic acid with other fatty acids in human studies have confirmed that stearic acid is not hypercholesterolemic. Stearic acid was shown not to raise low-density-lipoprotein cholesterol relative to oleic acid, which is known to be neutral in its effects on cholesterol concentrations. In contrast, palmitic acid, another long-chain saturated fatty acid, definitely raises cholesterol concentrations. For this reason, fats rich in stearic acid might be used in place of those high in palmitic acid in cholesterol-lowering diets.

Language of Publication

English

Unique Identifier

95067751

MeSH Heading (Major)

Cholesterol*BL; Dietary Fats*ME; Fatty Acids*ME; Stearic Acids*ME

MeSH Heading

Animal; Dietary Carbohydrates|ME; Fatty Acids, Monounsaturated|ME; Fatty Acids, Unsaturated|ME; Human; Lauric Acids|ME; Myristic Acids|ME; Palmitic Acids|ME

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0002-9165

Country of Publication

UNITED STATES

Section A

Record 8 from database: **MEDLINE**

Title

Effects of varying carbohydrate content of diet in patients with non-insulin-dependent diabetes mellitus [see comments]

Author

Garg A; Bantle JP; Henry RR; Coulston AM; Griver KA; Raatz SK; Brinkley L; Chen YD; Grundy SM; Huet BA; et al

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052.

Source

JAMA, 1994 May, 271:18, 1421-8

Abstract

OBJECTIVE--To study effects of variation in carbohydrate content of diet on glycemia and plasma lipoproteins in patients with non-insulin-dependent diabetes mellitus (NIDDM). **DESIGN**--A four-center randomized crossover trial. **SETTING**--Outpatient and inpatient evaluation in metabolic units. **PATIENTS**--Forty-two NIDDM patients receiving glipizide therapy. **INTERVENTIONS**--A high-carbohydrate diet containing 55% of the total energy as carbohydrates and 30% as fats was compared with a high-monounsaturated-fat diet containing 40% carbohydrates and 45% fats. The amounts of saturated fats, polyunsaturated fats, cholesterol, sucrose, and protein were similar. The study diets, prepared in metabolic kitchens, were provided as the sole nutrients to subjects for 6 weeks each. To assess longer-term effects, a subgroup of 21 patients continued the diet they received second for an additional 8 weeks. **MAIN OUTCOME MEASURES**--Fasting plasma glucose, insulin, lipoproteins, and glycosylated hemoglobin concentrations. Twenty-four-hour profiles of glucose, insulin, and triglyceride levels. **RESULTS**--The site of study as well as the diet order did not affect the results. Compared with the high-monounsaturated-fat diet, the high-carbohydrate diet increased fasting plasma triglyceride levels and very low-density lipoprotein cholesterol levels by 24% ($P < .0001$) and 23% ($P = .0001$), respectively, and increased daylong plasma triglyceride, glucose, and insulin values by 10% ($P = .03$), 12% ($P < .0001$), and 9% ($P = .02$), respectively. Plasma total cholesterol, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol levels remained unchanged. The effects of both diets on plasma glucose, insulin, and triglyceride levels persisted for 14 weeks. **CONCLUSIONS**--In NIDDM patients, high-carbohydrate diets compared with high-monounsaturated-fat diets caused persistent deterioration of glycemic control and accentuation of hyperinsulinemia, as well as increased plasma triglyceride and very-low-density lipoprotein cholesterol levels, which may not be desirable.

Language of Publication

English

Unique Identifier

94231641

MeSH Heading (Major)

Diabetes Mellitus, Non-Insulin-Dependent|*BL/*DH/DT; Dietary Carbohydrates|*/AD/ME; Dietary Fats|*/AD/ME

MeSH Heading

Adult; Aged; Blood Glucose|ME; Comparative Study; Energy Intake; Fatty Acids, Monounsaturated|AD/ME; Female; Glipizide|TU; Human; Insulin|BL; Lipoproteins|BL; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; MULTICENTER STUDY; RANDOMIZED CONTROLLED TRIAL

ISSN

Country of Publication
UNITED STATES

Section A

Record 9 from database: **MEDLINE**
[Go To The Top](#)

Title

Prediction of total subcutaneous abdominal, intraperitoneal, and retroperitoneal adipose tissue masses in men by a single axial magnetic resonance imaging slice.

Author

Abate N; Garg A; Coleman R; Grundy SM; Peshock RM

Address

Department of Internal Medicine, University of Texas Southwestern Medical Center, USA.

Source

Am J Clin Nutr, 1997 Feb, 65:2, 403-8

Abstract

To develop a simplified but accurate method for determining the masses of various abdominal adipose tissue compartments, we studied the predictive value of masses of intraperitoneal, retroperitoneal, and subcutaneous abdominal adipose tissue determined on single axial abdominal magnetic resonance imaging (MRI) slices taken at various intervertebral levels from the 12th thoracic to 1st sacral vertebra (identified on a sagittal section) for the respective total masses of each compartment calculated from contiguous 10-mm thick MRI slices covering the entire abdomen in 49 men (26 without diabetes and 23 with non-insulin-dependent diabetes mellitus). The MRI slice at the intervertebral level between the lumbar (L) 2 and 3 vertebrae showed the highest and most consistent predictive value for all three compartments ($R^2 = 0.85$ for all). Furthermore, compared with other intervertebral levels, the L2-L3 level had a higher amount of intraperitoneal and retroperitoneal adipose tissue mass. We conclude that determining the masses of various abdominal adipose tissue compartments at the L2-L3 intervertebral level by MRI is an acceptably reliable and accurate method for studying abdominal adiposity in men.

Language of Publication

English

Unique Identifier

97174863

MeSH Heading (Major)

Abdomen[*]; Adipose Tissue[*]

MeSH Heading

Adult; Aged; Body Mass Index; Human; Magnetic Resonance Imaging; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Tomography, X-Ray Computed

Publication Type

JOURNAL ARTICLE

ISSN

0002-9165

Country of Publication

UNITED STATES

Record 10 from database: **MEDLINE**[Go To The Top](#)**Title**

Estimation of adipose tissue mass by magnetic resonance imaging: validation against dissection in human cadavers.

Author

Abate N; Burns D; Peshock RM; Garg A; Grundy SM

Address

Department of Clinical Nutrition, University of Texas Southwestern Medical Center at Dallas.

Source

J Lipid Res, 1994 Aug, 35:8, 1490-6

Abstract

The evaluation of adipose tissue distribution has become an essential component of investigations on the complications of obesity. However, a major limitation is lack of methodology for accurate estimation of adipose tissue mass in the different regions of the body. Therefore, we have tested the accuracy and precision of magnetic resonance imaging (MRI) as a method to measure adipose tissue mass in regions of the body not accessible with standard anthropometric methods. The mass of subcutaneous and intraabdominal adipose tissue estimated by MRI was compared with that obtained by direct weighing of the same adipose tissue compartments after dissection in human cadavers. MRI was performed on three unembalmed cadavers (two males, one female) who were subsequently dissected to isolate intraperitoneal, retroperitoneal, and subcutaneous adipose tissues. These same components were delineated by MRI. The results of the two methods were highly congruent. For the various compartments, the mean of the difference between the two methods was only 0.076 kg (95% confidence interval + 0.005 kg and + 0.147 kg). The "limits of agreement" between the two techniques were -0.066 kg and +0.218 kg. Multiple repeated estimates of mass of adipose tissue compartments were made to determine reproducibility of the MRI measurement; the coefficient of variation for repeated measures was below 14%. The results of this study show that MRI is an accurate and precise technique to evaluate adipose tissue mass in subcutaneous and intraabdominal compartments. Furthermore, MRI was found to be a valid method to separately evaluate the mass of intraabdominal subcompartments of intraperitoneal and retroperitoneal adipose tissue.

Language of Publication

English

Unique Identifier

95081728

MeSH Heading (Major)

Adipose Tissue|*AH; Magnetic Resonance Imaging|*MT; Organ Weight|*

MeSH Heading

Abdomen; Autopsy; Cadaver; Comparative Study; Female; Human; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0022-2275

Country of Publication

UNITED STATES

[Go To The Top](#)**Title**

Variation at the hepatic lipase and apolipoprotein AI/CIII/AIV loci is a major cause of genetically determined variation in plasma HDL cholesterol levels.

Author

Cohen JC; Wang Z; Grundy SM; Stoesz MR; Guerra R

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

J Clin Invest, 1994 Dec, 94:6, 2377-84

Abstract

Genetic factors have been shown to play an important role in determining interindividual variation in plasma HDL-C levels, but the specific genetic determinants of HDL cholesterol (HDL-C) levels have not been elucidated. In this study, the effects of variation in the genomic regions encoding hepatic lipase, apolipoprotein AI/CIII/AIV, and the cholesteryl ester transfer protein on plasma HDL-C levels were examined in 73 normotriglyceridemic, Caucasian nuclear families. Genetic factors accounted for 56.5 +/- 13% of the interindividual variation in plasma HDL-C levels. For each candidate gene, adjusted plasma HDL-C levels of sibling pairs who shared zero, one, or two parental alleles identical-by-descent were compared using sibling-pair linkage analysis. Allelic variation in the genes encoding hepatic lipase and apolipoprotein AI/CIII/AIV accounted for 25 and 22%, respectively, of the total interindividual variation in plasma HDL-C levels. In contrast, none of the variation in plasma HDL-C levels could be accounted for by allelic variation in the cholesteryl ester transfer protein. These findings indicate that a major fraction of the genetically determined variation in plasma HDL-C levels is conferred by allelic variation at the hepatic lipase and the apolipoprotein AI/CIII/AIV gene loci.

Language of Publication

English

Unique Identifier

95081423

MeSH Heading (Major)

Apolipoproteins*GE; Lipase*GE; Lipoproteins, HDL Cholesterol*BL; Liver*EN; Variation (Genetics)*

MeSH Heading

Apolipoprotein A-I|GE; Apolipoproteins A|GE; Apolipoproteins C|GE; Base Sequence; Causality; Human; Molecular Sequence Data; Nuclear Family; Statistics|MT; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0021-9738

Country of Publication

UNITED STATES

Section A

[Go To The Top](#)

Title Karl Loren <http://www.oralchelation.com/technical/grundy1.htm>
Effectiveness of low-dose crystalline nicotinic acid in men with low high-density lipoprotein cholesterol levels.

Author
Martin Jadraque R; Tato F; Mostaza JM; Vega GL; Grundy SM

Address
Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas, USA.

Source
Arch Intern Med, 1996 May, 156:10, 1081-8

Abstract
BACKGROUND: Hypoalphalipoproteinemia (low serum concentration of high-density lipoprotein cholesterol [HDL-C]) is a common pattern of dyslipidemia associated with coronary heart disease. High doses of nicotinic acid effectively raise HDL-C levels in this condition, but they are commonly accompanied by side effects. The efficacy of low doses of nicotinic acid that may produce fewer side effects has not been adequately studied. OBJECTIVE: To determine the effects of low-dose nicotinic acid on HDL-C levels in patients with hypoalphalipoproteinemia. METHODS: Forty-four men with low HDL-C levels (< 1.03 mmol/L [< 40 mg/dL]) entered the study. Twenty-four patients otherwise had normal lipid levels, and 20 were moderately hypertriglyceridemic (range of plasma triglyceride levels, 2.82 to 5.64 mmol/L 250 to 500 mg/dL). The trial consisted of 3 phases; each phase lasted 8 weeks. The first phase was diet only (30% fat diet); in the second phase, crystalline nicotinic acid was added at 1.5 g/d; and in the third phase, the dose was increased to 3 g/d. RESULTS: Of the 44 patients who entered the study, 37 completed the low-dose phase (1.5 g/d); the remaining patients were withdrawn because of side effects to nicotinic acid. Four other patients who completed the low-dose phase were excluded from the higher dose phase because of side effects that developed when they were receiving the low dose. Ten other patients withdrew during the high-dose phase because of side effects. In both groups, responses to nicotinic acid therapy tended to be dose-dependent. For both groups, the higher dose generally produced a greater reduction in apolipoprotein B-containing lipoproteins and a greater rise in HDL-C levels. However, for both groups, the low dose of nicotinic acid gave an average 20% increase in HDL-C levels. CONCLUSIONS: A low dose (1.5 g/d) of crystalline nicotinic acid causes an average 20% increase in HDL-C levels and significantly lowers triglyceride levels in both normolipidemic and hyperlipidemic patients with low HDL-C levels. Although the changes induced by this dose are less than those that can be achieved by a higher dose, the lower dose is better tolerated. Nicotinic acid may be useful in combined drug therapy for secondary prevention of coronary heart disease, and if higher doses cannot be tolerated, use of a lower dose should still be useful for producing a moderate rise in HDL-C levels in patients with hypoalphalipoproteinemia.

Language of Publication
English

Unique Identifier
96224805

MeSH Heading (Major)

Hypolipoproteinemia|BL/DH/*DT; Lipoproteins, HDL|*BL; Nicotinic Acids|*AD/AE/TU

MeSH Heading

Crystallization; Dose-Response Relationship, Drug; Human; Hypertriglyceridemia|BL/DH/DT; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Treatment Outcome

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE

ISSN

0003-9926

Country of Publication

UNITED STATES

Section A

Record 13 from database: **MEDLINE**[Go To The Top](#)**Title**

Hypercholesterolemia with cholesterol-enriched LDL and normal levels of LDL-apolipoprotein B. Effects of the step I diet and bile acid sequestrants on the cholesterol content of LDL.

Author

Vega GL; Grundy SM

Address

Department of Clinical Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052, USA.

Source

Arterioscler Thromb Vasc Biol, 1996 Apr, 16:4, 517-22

Abstract

One form of hypercholesterolemia is characterized by high levels of LDL cholesterol and normal levels of LDL-apolipoprotein (apo) B. The reason for hypercholesterolemia, therefore, is enrichment of LDL particles with cholesterol. We have reported previously that about one third of patients with primary moderate hypercholesterolemia have this lipoprotein pattern and have no apparent abnormality in LDL-apo B metabolism. The current study was designed to determine whether the combination of the Step I Diet (30% of total calories as fat, <10% saturated fatty acids, and <300 mg per day cholesterol) with or without cholestyramine therapy will correct the hypercholesterolemia in patients of this type. Ten hypercholesterolemic men of this type were identified and recruited into the study. Their LDL cholesterol levels were \geq 160 mg/dL and LDL-apo B levels were $<$ 120 mg/dL (LDL cholesterol/apo B ratio $>$ 1.60). For patient selection, subjects were challenged with a high fat diet (40% of total calories as fat, 18% saturated fatty acids, and 400 mg per day cholesterol) for 6 weeks to confirm persistence of a high LDL cholesterol/apo B ratio. Thereafter, they were started on a Step I Diet, and lipoprotein analyses were repeated. Finally, cholestyramine (16 g per day) was added to the Step I Diet. The Step I Diet alone significantly reduced the LDL cholesterol/apo B ratios and produced a trend toward lowering LDL cholesterol levels. Cholestyramine therapy further reduced LDL cholesterol levels and maintained a normal LDL cholesterol/apo B ratio. The present investigation thus confirms the existence of a form of moderate hypercholesterolemia that arises from a defect in LDL composition. In addition, it demonstrates that the combination of Step I Diet and cholestyramine therapy corrects this defect and normalizes LDL levels and LDL composition.

Language of Publication

English

Unique Identifier

96197199

MeSH Heading (Major)

Apolipoproteins B|*BL; Cholesterol|AN/*BL; Hypercholesterolemia|*BL/*TH; Lipoproteins, LDL|*BL/CH

MeSH Heading

Aged; Anticholesteremic Agents|TU; Base Sequence; Cholestyramine|TU; Diet, Fat-Restricted; Human; Male; Middle Age; Molecular Probes|GE; Molecular Sequence Data; Polymerase Chain Reaction; Reference Values; Single-Blind Method; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE

ISSN

1079-5642

Section A

Record 14 from database: **MEDLINE**

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Title

Genetic analysis of a polymorphism in the human apolipoprotein A-I gene promoter: effect on plasma HDL-cholesterol levels.

Author

Barre DE; Guerra R; Verstraete R; Wang Z; Grundy SM; Cohen JC

Address

Center for Human Nutrition, UT Southwestern Medical Center, Dallas 75235-9052.

Source

J Lipid Res, 1994 Jul, 35:7, 1292-6

Abstract

Previous studies have indicated that a G to A substitution at position -76 in the gene encoding apolipoprotein A-I (apoA-I) confers increased plasma high density lipoprotein cholesterol (HDL-C). Increased HDL-C may be a direct consequence of the A allele, or may reflect the action of a locus in linkage disequilibrium with the A allele. To elucidate this question, we examined the effect of this polymorphism in a large sample (n = 409) of unrelated Caucasians and their nuclear families (n = 22). To eliminate the confounding effects of hypertriglyceridemia, individuals with triglyceride levels greater than 150 mg/dl were excluded from the study. ApoA-I genotype was determined by polymerase chain reaction (PCR) amplification of genomic DNA and restriction digestion with Msp I. Individuals were grouped by genotype (GG, GA or AA) and mean adjusted HDL levels of the three groups were compared by one-way analysis of variance. Our analysis indicates that HDL-C levels do not vary by genotype, and no gene dosage effect is apparent in men or in women. Analysis of 22 informative Caucasian nuclear families showed no significant difference between individuals with the A allele and their GG siblings. These data suggest that polymorphism at -76 in the apoA-I gene does not directly affect HDL levels. Therefore, the increased HDL-C levels reported in other populations must reflect linkage disequilibrium between the A allele and a putative HDL-raising allele. As we find no evidence for association between the A allele and high HDL levels, this putative allele must occur at a low frequency in the population sampled in this study.

Language of Publication

English

Unique Identifier

95053412

MeSH Heading (Major)

Adenine|*CH; Apolipoprotein A-I|*GE; Guanine|*CH; Lipoproteins, HDL Cholesterol|*BL; Polymorphism (Genetics)|*; Promoter Regions (Genetics)|*

MeSH Heading

Adolescence; Adult; Aged; Aged, 80 and over; Alleles; Female; Homozygote; Human; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0022-2275

Section A

Record 15 from database: **MEDLINE**

[Go To The Top](#)

Title

RRR-alpha-tocopheryl acetate supplementation at pharmacologic doses decreases low-density-lipoprotein oxidative susceptibility but not protein glycation in patients with diabetes mellitus.

Author

Fuller CJ; Chandalia M; Garg A; Grundy SM; Jialal I

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052, USA.

Source

Am J Clin Nutr, 1996 May, 63:5, 753-9

Abstract

Patients with diabetes mellitus have an increased risk of premature atherosclerosis, which may be due in part to increased oxidizability of low-density lipoprotein (LDL). Numerous studies have shown that alpha-tocopherol can reduce the oxidative susceptibility of LDL in normoglycemic subjects; however, there are few studies in persons with diabetes. In addition, alpha-tocopherol may reduce the extent of protein glycation. Therefore, the objective of the present study was to assess the effect of RRR-alpha-tocopheryl acetate supplementation on LDL oxidizability and protein glycation in persons with diabetes without evidence of vascular disease. Twenty-eight persons with insulin-dependent diabetes mellitus (IDDM) and non-insulin-dependent diabetes mellitus (NIDDM) were randomly assigned to receive either placebo or 1632 mg (1200 IU) RRR-alpha-tocopherol/d, as tocopheryl acetate, for 8 wk. Plasma and LDL antioxidant concentrations and LDL oxidizability were assessed at both 0 and 8 wk. Plasma and LDL concentrations of alpha-tocopherol were significantly increased in the supplemented group only. Compared with the placebo group, the alpha-tocopherol-supplemented group had significant reductions in LDL oxidizability at 8 wk, as shown by the time-course curves of conjugated diene and lipid peroxide formation. Also, alpha-tocopherol supplementation produced a significant prolongation in the lag phases of both assays, which was evident in both the NIDDM and IDDM subgroups. However, there were no significant changes in glycated hemoglobin or in glycated plasma proteins after alpha-tocopherol supplementation. Thus, alpha-tocopherol supplementation may be beneficial in reducing LDL oxidizability in patients with diabetes.

Language of Publication

English

Unique Identifier

96204955

MeSH Heading (Major)

Antioxidants|AD/AN/*PD; Diabetes Mellitus, Insulin-Dependent|BL/*ME; Diabetes Mellitus, Non-Insulin-Dependent|BL/*ME; Lipoproteins, LDL|BL/*ME; Vitamin E|*AA/AD/BL/PD

MeSH Heading

Adult; Analysis of Variance; Blood Glucose|AN/ME; Dose-Response Relationship, Drug; Fatty Acids|BL; Food, Fortified; Hemoglobin A, Glycosylated|AN; Human; Lipid Peroxides|ME; Middle Age; Oxidation-Reduction; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Time Factors

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

Section A

Record 16 from database: **MEDLINE**

[Go To The Top](#)

Title

What is the desirable ratio of saturated, polyunsaturated, and monounsaturated fatty acids in the diet?

Author

Grundy SM

Address

University of Texas Southwestern Medical Center, Dallas, USA.

Source

Am J Clin Nutr, 1997 Oct, 66:4 Suppl, 988S-990S

Abstract

By reducing intakes of animal fats and gradually reducing intakes of trans fatty acids, a one-third reduction in cholesterol-raising fatty acids seems practical, from 12% to 7-8% of total energy intake. The intake of polyunsaturated fatty acids should not exceed current intakes, approximately 7% of total energy. Although further research is needed to determine a recommended ratio of oleic acid to carbohydrates, on the basis of the relatively low rates of coronary artery disease and cancer in both the Mediterranean region (where oleic acid intake is high at the expense of carbohydrates) and in populations consuming low-fat, high-carbohydrate diets, a reasonable compromise is a diet in which total fat is approximately 30% of energy, allowing for an intake of oleic acid of 15-16% of total energy.

Language of Publication

English

Unique Identifier

97463877

MeSH Heading (Major)

Dietary Fats|*AD/AN; Dietary Fats, Unsaturated|*AD/AN; Fatty Acids|*AD/AN; Fatty Acids, Monounsaturated|*AD/AN; Fatty Acids, Unsaturated|*AD/AN

MeSH Heading

Human

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0002-9165

Country of Publication

UNITED STATES

Section A

Record 17 from database: **MEDLINE**

[Go To The Top](#)

Title

Cholesterol management in patients with heart disease. Emphasizing secondary prevention to increase longevity.

Author

Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052, USA.

Source

Postgrad Med, 1997 Aug, 102:2, 81-4, 87-90

Abstract

Advances in treatment of elevated cholesterol levels and recent documentation of efficacy and safety in clinical trials justify expanded use of cholesterol-lowering therapy in clinical practice. Patients with CHD or other forms of clinical atherosclerotic disease can benefit from aggressive cholesterol management. Maximal dietary modification, weight control, and physical activity are valuable adjuncts to drug therapy in secondary prevention. Recent studies have shown that appropriate use of cholesterol-lowering drugs is cost-effective and efficacious in patients with CHD. Use of such drugs can increase patients' life expectancy. Primary care physicians have a key role in instituting intensive cholesterol management in patients with clinically manifest atherosclerotic disease. Furthermore, they should take the lead in coordinating with cardiovascular specialists to manage cholesterol levels in patients who have had a recent acute coronary syndrome or undergone a revascularization procedure.

Language of Publication

English

Unique Identifier

97416752

MeSH Heading (Major)

Anticholesteremic Agents|*TU; Coronary Disease|BL/CO/*PC; Hypercholesterolemia|CO/DH/*DT

MeSH Heading

Human; Longevity; Lovastatin|AA/TU; Niacin|TU; Support, Non-U.S. Gov't

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0032-5481

Country of Publication

UNITED STATES

Section A

Record 18 from database: **MEDLINE**

[Go To The Top](#)

Title

Determinants of plasma HDL-cholesterol in hypertriglyceridemic patients. Role of cholesterol-ester transfer protein and lecithin cholesteryl acyl transferase.

Author

Tato F; Vega GL; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas, USA.

Source

Arterioscler Thromb Vasc Biol, 1997 Jan, 17:1, 56-63

Hypertriglyceridemic patients commonly have low levels of HDL cholesterol. Elevated triglycerides per se may be one cause of low HDL levels, but other factors also may be involved. The current study was designed to define the role of cholesterol-ester transfer protein (CETP) in causation of a low HDL cholesterol in hypertriglyceridemic patients; in addition other factors-lecithin cholesterol acyl transferase (LCAT), hepatic triglyceride lipase (HTGL), and lipoprotein lipase (LPL)-were examined. Plasma activities of CETP and LCAT were measured in 137 male patients with moderate hypertriglyceridemia (plasma triglycerides [TGs] 200 to 500 mg/dL and LDL cholesterol < 160 mg/dL). Results were compared with those from 50 normolipidemic men of similar age and body habitus. In addition, lipase activities in postheparin plasma were measured in 118 of the subjects with hypertriglyceridemia. The activities of CETP and LCAT were 17% (P < .01) and 7% (P < .05), respectively, higher in the hypertriglyceridemic group than in control subjects. By stepwise regression analysis CETP appeared to contribute 15.2% and LCAT 9.8% to variation in HDL-cholesterol levels. Activities of LPL and HTGL together contributed an additional 14.1% to HDL-cholesterol variation. In contrast, levels of plasma TG accounted for only 5.4% of the variation. There were no differences in relative contributions of these parameters in patients with and those without coronary heart disease. This study indicates that several factors contribute to the variation in HDL-cholesterol levels in hypertriglyceridemic patients, and five factors-CETP, LCAT, HTGL, LPL, and triglyceride levels-account for almost half of this variation.

Language of Publication

English

Unique Identifier

97164876

MeSH Heading (Major)

Carrier Proteins|*AN; Hypertriglyceridemia|*BL; Lipoproteins, HDL Cholesterol|*BL; Phosphatidylcholine-Sterol O-Acyltransferase|*AN

MeSH Heading

Aged; Human; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

1079-5642

Country of Publication

UNITED STATES

Section ARecord 19 from database: **MEDLINE**[Go To The Top](#)**Title**

Cholestyramine therapy for dyslipidemia in non-insulin-dependent diabetes mellitus. A short-term, double-blind, crossover trial.

Author

Garg A; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052.

Source

Ann Intern Med, 1994 Sep, 121:6, 416-22

OBJECTIVE: To assess clinical efficacy and tolerability of cholestyramine therapy in patients with dyslipidemia and non-insulin-dependent diabetes mellitus (NIDDM). **DESIGN:** A randomized, double-blind, crossover study of cholestyramine (8 g twice daily) compared with placebo for a period of 6 weeks each. **SETTING:** Metabolic Unit and the Lipid and Diabetes Clinics at the Department of Veterans Affairs Medical Center, Dallas, Texas. **PATIENTS:** 21 patients with NIDDM that was well controlled using either glyburide or insulin therapy and with low-density lipoprotein (LDL) cholesterol levels more than 3.36 mmol/L (130 mg/dL) and fasting plasma triglyceride levels less than 3.4 mmol/L (300 mg/dL). **MEASUREMENTS:** During the last week of each period, for 5 consecutive days fasting plasma lipids and lipoproteins were measured, and plasma glucose levels were determined at 3, 7, and 11 a.m. and at 4 and 8 p.m. Daily urinary glucose excretion was measured for 3 days and glycosylated hemoglobin concentrations were determined on days 28 and 38 of the study periods. **RESULTS:** In this short-term study, when compared with placebo, cholestyramine reduced total cholesterol by 18% (95% CI, 14% to 22%) and LDL cholesterol by 28% (CI, 21% to 35%). Although cholestyramine therapy increased plasma triglyceride levels by 13.5% (CI, 1% to 26%), very-low density lipoprotein cholesterol and high-density lipoprotein cholesterol levels remained unchanged. Cholestyramine therapy improved glycemic control; mean plasma glucose values were lower by 13% (CI, 5% to 21%), a median reduction in urinary glucose excretion of 0.22 g/d was observed ($P < 0.001$), and a tendency to lower glycosylated hemoglobin concentration was noted. The doses of glyburide and insulin did not change during the study, and body weight remained stable. Constipation was the main side effect, and two patients dropped out of the study because of cholestyramine intolerance. **CONCLUSIONS:** In carefully selected male patients with NIDDM and high LDL cholesterol and normal triglyceride levels, cholestyramine therapy effectively reduces LDL levels and also may improve glycemic control. The long-term efficacy of cholestyramine therapy in patients with NIDDM needs further evaluation.

Language of Publication

English

Unique Identifier

94330646

MeSH Heading (Major)

Cholestyramine|*TU; Diabetes Mellitus, Non-Insulin-Dependent|*CO; Hypercholesterolemia|BL/*DT/ET

MeSH Heading

Adult; Aged; Double-Blind Method; Female; Human; Lipids|BL; Lipoproteins, LDL Cholesterol|BL; Male; Middle Age; Risk Factors; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0003-4819

Country of Publication

UNITED STATES

Section ARecord 20 from database: **MEDLINE**[Go To The Top](#)**Title**

Addressing the spectrum of hypercholesterolemia [see comments]

Author

Grundy SM; Mazzaferri EL

Address Karl Loren <http://www.oralchelation.com/technical/grundy1.htm>

University of Texas Southwestern Medical Center at Dallas, USA.

Source

Hosp Pract (Off Ed), 1996 Jun, 31:6, 43-8, 53-5, 59; discussion 60

Abstract

The past decade has seen a major shift in management: Trials of HMG-CoA reductase inhibitors have suggested that cholesterol reduction offers greater protection against coronary artery disease than does antihypertensive therapy. Five patient vignettes provide guidelines for initiating therapy. The agents should be prescribed with restraint, often not until other measures have been exhausted.

Language of Publication

English

Unique Identifier

96281858

MeSH Heading (Major)

Coronary Disease|ET/*PC; Hypercholesterolemia|CO/*TH; Lovastatin|*AA/TU

MeSH Heading

Adult; Aged; Algorithms; Anticholesteremic Agents|TU; Case Report; Diet, Fat-Restricted; Exercise; Female; Human; Male; Middle Age; Risk Factors; Smoking Cessation

Publication Type

JOURNAL ARTICLE

ISSN

8750-2836

Country of Publication

UNITED STATES

HealthGate Documents

Section B

Record 1 from database: **MEDLINE**

[Go To The Top](#)

Title

Influence of exchanging carbohydrate for saturated fatty acids on plasma lipids and lipoproteins in men.

Author

Wolf RN; Grundy SM

Address

Source

J Nutr, 1983 Aug, 113:8, 1521-8

Abstract

This study was designed to determine effects of reducing intake of total fat and increasing carbohydrate (glucose) on plasma lipoproteins. Eleven men were investigated. They were given two diets for 1 month each. One diet contained 40% of calories as fat with 20% saturated fatty acids, 10% monounsaturates and 10% polyunsaturates. The other diet contained 30% fat with equal amounts of each type of fatty acid. The 10% of fat removed from the latter was replaced by glucose. Six patients had significant reductions of cholesterol in total plasma and low density lipoprotein (LDL) on the 30% fat; for the group as a whole; however, the decrease was

not statistically significant. Total triglycerides increased modestly (15%) and high density lipoprotein (HDL)-cholesterol fell significantly (14%) on replacement of 40% fat with 30% fat. Seven patients also were given a 30% fat diet containing fatty acids in the same proportions as in the 40% fat diet. A similar response was noted as when fatty acids were given in equal ratios. This study indicates that response to reduction in fat content is inconsistent. The majority of patients were responders; others, however, were not.

Language of Publication

English

Unique Identifier

83267829

MeSH Heading (Major)

Dietary Fats|*AD; Lipids|*BL

MeSH Heading

Adult; Aged; Cholesterol|BL; Dose-Response Relationship, Drug; Fatty Acids, Unsaturated|AD; Food, Formulated; Glucose|AD; Human; Lipoproteins, HDL|BL; Lipoproteins, LDL|BL; Male; Middle Age; Milk Proteins|AD; Structure-Activity Relationship; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0022-3166

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Dietary Fats); 0 (Fatty Acids, Unsaturated); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, HDL); 0 (Lipoproteins, LDL Cholesterol); 0 (Milk Proteins); 0 (Triglycerides); 50-99-7 (Glucose); 57-88-5 (Cholesterol)

Section B

Record 2 from database: **MEDLINE**

[Go To The Top](#)

Title

Incorporation of radioactive phospholipid into subclasses of high-density lipoproteins.

Author

Tall AR; Blum CB; Grundy SM

Address**Source**

Am J Physiol, 1983 May, 244:5, E513-6

Abstract

The incorporation of orally administered phospholipid into plasma high-density lipoproteins (HDL) was studied in three subjects. Plasma was analyzed by equilibrium density gradient ultracentrifugation, 5, 6, and 8 h after ingestion of 1.1 g [3H-choline, 14C-dilinoleoyl]phosphatidylcholine. At all time points in all subjects, there was a peak of phosphatidylcholine specific activity in fractions of density approximately 1.10-1.13 g/ml, corresponding to the subclass previously designated HDL2a. There was also a more variable, smaller peak of specific activity of phospholipids in HDL2b (1.063-1.100 g/ml) and in fractions of density approximately 1.19 g/ml. In the 1.10-1.13 fraction, 97 and 71%, respectively, of the 3H and 14C radioactivity were in phospholipids. The 3H/14C ratio was similar in phospholipids of HDL subfractions, the d less than 1.07 fraction, and in the administered phospholipid. The results show preferential transfer or exchange or absorbed phosphatidylcholine

into specific subclasses of HDL. <http://www.oralchelation.com/technical/grundy1.htm>

Language of Publication

English

Unique Identifier

83201561

MeSH Heading (Major)

Lipoproteins, HDL|BI/*BL; Phospholipids|*BL

MeSH Heading

Cholesterol Esters|BL; Human; Kinetics; Middle Age; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0002-9513

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Cholesterol Esters); 0 (Lipoproteins, HDL); 0 (Phospholipids); 0 (Triglycerides)

Section B

Record 3 from database: **MEDLINE**

[Go To The Top](#)

Title

Increased low density lipoprotein production associated with obesity.

Author

Kesaniemi YA; Grundy SM

Address

Source

Arteriosclerosis, 1983 Mar-Apr, 3:2, 170-7

Abstract

Turnover rates of the apolipoprotein of low density lipoproteins (apoLDL) and cholesterol balance were determined in six obese men and six control men. The two groups were of similar age and matched for apoLDL concentrations. Levels of plasma total cholesterol in obese patients (209 +/- 14 SEM mg/dl) were similar to controls (225 +/- 17 mg/dl). LDL-cholesterol was numerically but not statistically lower in obese subjects (111 +/- 18 mg/dl) compared to controls (145 +/- 13 mg/dl). Synthetic rates of apoLDL in contrast were higher in obese patients (1450 mg/day) than in controls (934 mg/day) (p less than 0.002). Three factors could explain the similar concentrations of LDL-cholesterol in obese and control subjects, despite overproduction of apoLDL in the obese. First, LDL was diluted into a larger plasma pool in obese patients; second, fractional catabolic rates of apoLDL were somewhat greater in obese men than in controls; and third, obese patients had higher ratios of protein-to-cholesterol in LDL. The production of apoLDL for all patients was not correlated with total body synthesis of cholesterol. The major finding of this study was that obese patients have increased turnover of apoLDL, not necessarily reflected by high concentrations of LDL-cholesterol. This high turnover rate itself may raise the risk for coronary heart disease in obese patients.

Language of Publication

English

MeSH Heading (Major)

Lipoproteins, LDL|*BI/BL; Obesity|BL/*ME

MeSH Heading

Apolipoproteins|BI; Cholesterol|BL; Human; Kinetics; Lipoproteins, HDL|BL; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (apolipoprotein LDL); 0 (Apolipoproteins); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, HDL); 0 (Lipoproteins, LDL Cholesterol); 57-88-5 (Cholesterol)

Section B

Record 4 from database: **MEDLINE**

[Go To The Top](#)

Title

Normocholesterolemic tendon xanthomatosis with overproduction of apolipoprotein B.

Author

Vega GL; Illingworth DR; Grundy SM; Lindgren FT; Connor WE

Address

Source

Metabolism, 1983 Feb, 32:2, 118-25

Abstract

This report describes a 46-yr-old man with normocholesterolemic tendon xanthomatosis. He had severe bilateral xanthomas of Achilles tendons and small lesions on patellar tendons; biopsy of the latter revealed a fibroxanthoma of high cholesterol content. He did not have clinical evidence of atherosclerotic disease. The patient's total cholesterol (TC) and triglycerides (TG) were 245 and 258 mg/dl, respectively. LDL-TC was 168 mg/dl and HDL-TC was 32 mg/dl. VLDL consisted mainly of small particles (SfO 20-100) which were unusually rich in apolipoproteins B and E (and low in apo Cs). Plasma LDL-apo B was not increased (85-120 mg/dl), but VLDL-apo B was distinctly elevated (58 mg/dl). The synthesis rate of apoLDL (29.9 mg/kg/d) was increased markedly compared to a matched control (13.9 mg/kg/d) and to a patient with familial hypercholesterolemia (15.9 mg/kg/d). The concentration of apoLDL in our patient was not increased; this was because of an associated high FCR (0.484 day⁻¹). His HDL was relatively low in TC but high in TG, which caused an increase in HDL_{2b}. The patient's xanthomata may have been the result of an overproduction of apo B possibly combined with a defect in HDL metabolism.

Language of Publication

English

Unique Identifier

83140985

MeSH Heading (Major)

Apolipoproteins|*BI/BL; Cholesterol|*BL; Xanthomatosis|*BL/PA

MeSH Heading

Case Report; Human; Lipids|BL; Lipoproteins|BL; Male; Middle Age; Sterols|ME; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Tendons|PA

Publication Type

JOURNAL ARTICLE

ISSN

0026-0495

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoproteins B); 0 (Apolipoproteins); 0 (Lipoproteins); 0 (Sterols); 57-88-5 (Cholesterol)

Section B

Record 5 from database: **MEDLINE**[Go To The Top](#)**Title**

Overproduction of low density lipoproteins associated with coronary heart disease.

Author

Kesaniemi YA; Grundy SM

Address**Source**

Arteriosclerosis, 1983 Jan-Feb, 3:1, 40-6

Abstract

The turnover rates of low density lipoprotein-apolipoprotein (apoLDL) were determined in eight men with coronary heart disease (CHD) and seven men matched for age, weight, and plasma lipid levels who were used for controls. The CHD patients were normocholesterolemic (plasma cholesterol = 204 +/- 8 mg/dl sem) as were the control subjects (227 +/- 15 mg/dl). The concentrations of plasma LDL cholesterol and apoLDL were similar for the two groups. In contrast, the synthetic rates of apoLDL were higher in the CHD patients (20.0 +/- 1.8 mg/kg/day) than in the controls (12.9 +/- 1.1 mg/kg/day) (p less than 0.01). The ratios of protein-to-cholesterol in LDL averaged 19% higher in the CHD patients. These patients with CHD maintained normal LDL levels despite an over-production of apoLDL because of an increased capacity for LDL removal; their fractional catabolic rates of apoLDL averaged 43% higher than those of the controls. These findings indicate that some patients with CHD have abnormalities in the turnover of apoLDL, even with normal concentrations of LDL; these abnormalities may contribute to accelerated atherosclerosis.

Language of Publication

English

Unique Identifier

83126219

MeSH Heading (Major)

Coronary Disease|*ME; Lipoproteins, LDL|*BI/ME

MeSH Heading

Aged; Apolipoproteins|BI; Cholesterol|ME; Dietary Fats|ME; Human; Male; Middle Age; Support, U.S. Gov't,

Publication Type

JOURNAL ARTICLE

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (apolipoprotein LDL); 0 (Apolipoproteins); 0 (Dietary Fats); 0 (Lipoproteins, LDL Cholesterol); 57-88-5 (Cholesterol)

Section B

Record 6 from database: **MEDLINE**

[Go To The Top](#)

Title

Effect of bile acid conjugation pattern on bile acid metabolism in normal humans.

Author

Hardison WG; Grundy SM

Address**Source**

Gastroenterology, 1983 Mar, 84:3, 617-20

Abstract

Six male subjects were fed taurine 0.5 g six times daily for 2 wk to determine the effect of a shift in bile acid conjugation pattern upon bile acid metabolism. Duodenal bile acids were analyzed, and bile acid pool size, daily fecal excretion, and biliary excretion rate were quantified. In addition, daily biliary excretion rate of cholesterol and phospholipid were quantified, and biliary saturation with cholesterol was estimated. The dose of taurine caused reversal of the bile acid glycine-to-aurine conjugation ratio. Total bile acid pool size decreased, as did the pool size of chenodeoxycholic acid. Pool sizes of cholic and deoxycholic acids did not change. Daily fecal bile acid excretion decreased slightly. Biliary secretion rates of cholesterol, phospholipid, and bile acids did not change, nor did biliary cholesterol saturation. Pool size can decrease because of increased bile acid catabolism or decreased synthesis. The fact that bile acid excretion failed to increase, and actually decreased slightly, suggests that the effect is upon bile acid synthesis. In normal humans, the effect is small and probably physiologically unimportant. In special cases, however, such as during ursodeoxycholic acid therapy, the effect of shifting conjugation pattern may become important.

Language of Publication

English

Unique Identifier

83106321

MeSH Heading (Major)

Bile Acids and Salts|*ME

MeSH Heading

Adult; Aged; Chenodeoxycholic Acid|ME; Cholesterol|ME; Cholic Acids|ME; Chromatography, High Pressure Liquid; Deoxycholic Acid|ME; Duodenum|AN; Feces|AN; Glycine|ME; Human; Male; Middle Age; Phospholipids|ME; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Taurine|AD/ME

Publication Type

ISSN

0016-5085

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Bile Acids and Salts); 0 (Cholic Acids); 0 (Phospholipids); 107-35-7 (Taurine); 474-25-9 (Chenodeoxycholic Acid); 56-40-6 (Glycine); 57-88-5 (Cholesterol); 83-44-3 (Deoxycholic Acid)

Section B

Record 7 from database: **MEDLINE**[Go To The Top](#)

Title

Effects of haem infusion on biliary secretion of porphyrins, haem and bilirubin in man.

Author

McCormack LR; Liem HH; Strum WB; Grundy SM; Muller-Eberhard U

Address

Source

Eur J Clin Invest, 1982 Jun, 12:3, 257-62

Abstract

Employing a continuous bile collection, we measured the bile secretion of porphyrins, haem (iron protoporphyrin IX regardless of oxidation state) and bilirubin in five healthy subjects. The baseline values for the flow of porphyrins in the bile were: 4.7 +/- 1.9 nmol/h uroporphyrin, 27.3 +/- 3.8 nmol/h coproporphyrin and 39.2 +/- 11.7 nmol/h protoporphyrin. Bile haem flow was 59.7 +/- 12.6 nmol/h, and that of bilirubin 23.8 +/- 8.2 nmol/h. Following haem injection (6.4 mmol/kg) the flow of protoporphyrin but not of the other porphyrins was reduced, and the bile haem flow increased (232 +/- 109.5 nmol/h), while the flow of bilirubin did not increase significantly. A few patients with representative porphyrias showed the expected increase in copro- and protoporphyrin in the bile. The patient with coproporphyrinemia exhibited a bile flow of coproporphyrin of 1470 +/- 133 nmol/h and of protoporphyrin of 334 +/- 29 nmol/h; haem infusion significantly reduced the bile flow of both porphyrins (to 649 +/- 101 for copro- and 215 +/- 36 nmol/h for protoporphyrin). The patient with protoporphyrinemia had an increased protoporphyrin flow, yet haem infusion caused no reduction in protoporphyrin flow (106 +/- 7 after v. 81.4 +/- 13 nmol/h before haem). In conclusion, we found that haem and porphyrins are normal constituents of bile, and that injected haem appears in bile. Bile bilirubin did not rise within 12 h after haem infusion a finding which warrants further investigation.

Language of Publication

English

Unique Identifier

82261790

MeSH Heading (Major)

Bile|*SE; Bilirubin|*SE; Heme|*AD/SE; Porphyrins|*SE

MeSH Heading

Adult; Aged; Coproporphyrins|SE; Female; Human; Injections; Male; Middle Age; Porphyria|GE/ME; Protoporphyrins|SE; Skin Diseases|ME; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN Karl Loren
0014-2972

<http://www.oralchelation.com/technical/grundy1.htm>

Country of Publication
ENGLAND

CAS Registry/EC Number

0 (Coproporphyrins); 0 (Porphyrins); 0 (Protoporphyrins); 14875-96-8 (Heme); 635-65-4 (Bilirubin)

Section B

Record 8 from database: MEDLINE

[Go To The Top](#)

Title

National Cooperative Gallstone Study: the effect of chenodeoxycholic acid on lipoproteins and apolipoproteins.

Author

Albers JJ; Grundy SM; Cleary PA; Small DM; Lachin JM; Schoenfield LJ

Address

Source

Gastroenterology, 1982 Apr, 82:4, 638-46

Abstract

Subjects in the National Cooperative Gallstone Study undergoing 12 mo of therapy with chenodeoxycholic acid for the dissolution of gallstones (low-dose, 375 mg/day, n = 252; high-dose, 750 mg/day, n = 253) had a mean increase in serum cholesterol of 20 mg/dl as compared with a 5 mg/dl increase in the placebo group (n = 258). The effect of chenodeoxycholic acid on lipoproteins was determined in a random subset of the high-dose (n = 136) and placebo (n = 143) groups. For men, the mean baseline adjusted estimated low-density lipoprotein cholesterol level at 12 mo was significantly higher in the high-dose group than in the placebo group (159 vs. 148 mg/dl, p less than 0.01), whereas among women this difference was not demonstrated. Change in low-density lipoprotein cholesterol level was inversely related to baseline cholesterol to an equivalent degree in each group among men and women. Women in the high-dose group had significantly lower very-low-density lipoprotein cholesterol levels than did the corresponding placebo group (27 vs. 32 mg/dl, p less than 0.003). Very-low-density lipoprotein cholesterol levels did not differ significantly between the high-dose and placebo group in men. Treatment did not significantly affect the levels of high-density lipoprotein cholesterol or apoproteins A-I, A-II, or B. Chenodeoxycholic acid therapy produces an increase in total cholesterol and low-density lipoprotein cholesterol but does not alter high-density lipoprotein cholesterol levels.

Language of Publication

English

Unique Identifier

82139931

MeSH Heading (Major)

Apolipoproteins|*BL; Chenodeoxycholic Acid|*TU; Cholelithiasis|BL/*DT; Lipoproteins|*BL

MeSH Heading

Cholesterol|BL; Female; Human; Lipoproteins, HDL|BL; Lipoproteins, LDL|BL; Lipoproteins, VLDL|BL; Male; Middle Age; Sex Factors; Support, U.S. Gov't, P.H.S.

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0016-5085

Country of Publication

UNITED STATES

<http://www.oralchelation.com/technical/grundy1.htm>**CAS Registry/EC Number**

0 (Apolipoprotein A-I); 0 (Apolipoprotein A-II); 0 (Apolipoproteins B); 0 (Apolipoproteins); 0 (Lipoproteins); 0 (Lipoproteins, HDL); 0 (Lipoproteins, VLDL); 474-25-9 (Chenodeoxycholic Acid); 57-88-5 (Cholesterol)

Section BRecord 9 from database: **MEDLINE**[Go To The Top](#)**Title**

Metabolism of cholesterol and plasma triglycerides in nonketotic diabetes mellitus.

Author

Abrams JJ; Ginsberg H; Grundy SM

Address**Source**

Diabetes, 1982 Oct, 31:10, 903-10

Abstract

The metabolism of cholesterol and plasma triglycerides (TG) was studied in 14 diabetic men: these patients did not have marked obesity nor did they develop ketoacidosis without insulin. Before insulin therapy, measurements were made of (1) plasma lipoproteins, (2) postheparin lipolytic enzymes, (3) turnover to TG in very-low-density lipoproteins (VLDL) and chylomicrons, (4) cholesterol balance, and (5) biliary lipids. After baseline measurements, the patients were treated with enough long-acting insulin to maintain their fasting plasma glucose in the range of 100--125 mg/dl. When plasma glucose and lipid levels reached a new steady state, all of the above measurements were repeated. Before insulin, most patients had fasting hypertriglyceridemia. This was due mainly to overproduction of VLDL-TG. Insulin therapy lowered both synthesis and concentrations of VLDL-TG to near normal. Also, patients with normotriglyceridemia, both before and during insulin therapy, had essentially normal clearance of chylomicrons. Those with high fasting TG had delayed clearance of chylomicrons, but clearance returned to normal in most with insulin therapy. Postheparin lipolytic enzymes were not decreased. Before insulin, synthesis rates of cholesterol and bile acids usually were greater than normal, and bile commonly was supersaturated with cholesterol. During insulin therapy, synthesis of both cholesterol and bile acids remained elevated, possibly because of imperfect control of hyperglycemia.

Furthermore, saturation of bile with cholesterol was accentuated by insulin therapy.

Language of Publication

English

Unique Identifier

83106069

MeSH Heading (Major)

Cholesterol|*ME; Diabetes Mellitus|BL/DT/*ME; Lipoproteins|*BL/ME

MeSH Heading

Aged; Bile|AN; Chylomicrons|ME; Human; Insulin|TU; Lipids|AN/ME; Lipoproteins, VLDL|BL/ME; Male; Middle Age; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0012-1797

Country of Publication<http://www.oralchelation.com/technical/grundy1.htm>

UNITED STATES

CAS Registry/EC Number

0 (Chylomicrons); 0 (Lipoproteins); 0 (Lipoproteins, VLDL); 0 (Triglycerides); 11061-68-0 (Insulin); 57-88-5 (Cholesterol)

Section BRecord 10 from database: **MEDLINE**[Go To The Top](#)**Title**

Hypertriglyceridemia: mechanisms, clinical significance, and treatment.

Author

Grundy SM

Address**Source**

Med Clin North Am, 1982 Mar, 66:2, 519-35

Abstract

The association between hypertriglyceridemia and coronary heart disease is explored followed by a discussion of the mechanisms of the disorder and guidelines on patient evaluation and treatment.

Language of Publication

English

Unique Identifier

82171950

MeSH Heading (Major)

Triglycerides|*BL

MeSH Heading

Apolipoproteins|ME; Atherosclerosis|ET; Chylomicrons|ME; Human; Lipoproteins, HDL|ME; Lipoproteins, LDL|ME; Lipoproteins, VLDL|ME

Publication Type

JOURNAL ARTICLE; REVIEW

ISSN

0025-7125

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoproteins B); 0 (Apolipoproteins); 0 (Chylomicrons); 0 (Lipoproteins, HDL); 0 (Lipoproteins, VLDL); 0 (Triglycerides)

Section BRecord 11 from database: **MEDLINE**[Go To The Top](#)**Title**

Author

Zierenberg O; Grundy SM

Address

Source

J Lipid Res, 1982 Nov, 23:8, 1136-42

Abstract

The metabolic fate of 1 of 3H/14C-labeled dinoleoglycerophosphocholine was studied in five patients after oral administration. The 3H label was in choline and 14C was in the two linoleic acid residues. More than 90% of both isotopes was absorbed from the intestine. Seventy to 90% of the 3H radioactivity in blood was linked to phosphatidylcholine (PC) whereas 14C was associated with both PC and nonpolar lipids. At peak activity, the 3H/14C ratio of plasma PC was twice that of oral PC; this suggests that most oral PC was hydrolyzed to lysolecithin before absorption. The mean maximum concentration in total blood volume was 20% of the administered dose for 3H and 28% for 14C. Examination of lipoproteins revealed that the specific activity of PC in high density lipoprotein (HDL) was 2 to 6 times higher than in apoB-containing lipoproteins, and to 2 to 20 times than that of red blood cells or total blood. Thus, absorbed PC seemingly was incorporated preferentially into the HDL fraction of plasma.

Language of Publication

English

Unique Identifier

83084428

MeSH Heading (Major)

Intestinal Absorption*; Phosphatidylcholines|*ME

MeSH Heading

Absorption; Feces|AN; Female; Human; Kinetics; Lipoproteins|BL; Male; Middle Age; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Time Factors

Publication Type

JOURNAL ARTICLE

ISSN

0022-2275

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (polyene phosphatidylcholine); 0 (Lipoproteins); 0 (Phosphatidylcholines)

Section B

Record 12 from database: **MEDLINE**

[Go To The Top](#)

Title

Influence of polyunsaturated fats on composition of plasma lipoproteins and apolipoproteins.

Author

Vega GL; Groszek E; Wolf R; Grundy SM

Address

Source

Abstract

The mechanisms of the hypocholesterolemic effect of polyunsaturated fats (PUSF) are not well understood. One possibility is that these fats uniquely reduce the cholesterol content of lipoproteins. The present study was carried out to determine specifically whether the ratio of cholesterol-to-protein (or apoB) in LDL (or other lipoproteins) is reduced by PUSF; also, lipoprotein composition was examined for other possible changes. Eight men and two women with different levels of plasma cholesterol were studied on the metabolic ward for 8 weeks. They were given a diet high in saturated fats (SF) for 4 weeks and another rich in PUSF for 4 weeks. On PUSF diets, mean plasma cholesterol decreased by 25% (SF = 296 +/- 27 (SEM) vs. PUSF = 223 +/- 21 mg/dl) as did total plasma apoB (155 +/- 8 vs. 116 +/- 13 mg/dl). LDL-Cholesterol decreased by 26%, and LDL-apoB fell by 29%. The mean ratio of cholesterol-to-apoB did not change significantly (SF = 1.52 +/- 0.04 vs. PUSF = 1.48 +/- 0.07). Likewise, HDL-cholesterol decreased by 15% (SF = 51 +/- 5 vs. PUSF = 43 +/- 4 mg/dl), and total plasma apoA-I was reduced by 19% (95 +/- 15 vs. 77 +/- 6 mg/dl); also, no change in the cholesterol-to-apoA-I in HDL was noted. Finally, there were no changes in cholesterol/apoB or triglyceride/apoB ratios in VLDL despite a 23% decrease in plasma triglycerides on PUSF. Thus, the hypocholesterolemic effect of PUSF was uniform for all lipoproteins and usually was accompanied by a corresponding decrease in concentrations of apoprotein constituents.

Language of Publication

English

Unique Identifier

83032277

MeSH Heading (Major)

Apolipoproteins[*BL; Dietary Fats*PD; Fats, Unsaturated*PD; Lipoproteins*BL

MeSH Heading

Adult; Aged; Female; Human; Lipoproteins, HDL|BL; Lipoproteins, LDL|BL; Lipoproteins, VLDL|BL; Male; Middle Age; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0022-2275

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoproteins); 0 (Dietary Fats); 0 (Fats, Unsaturated); 0 (Lipoproteins); 0 (Lipoproteins, HDL); 0 (Lipoproteins, VLDL)

Section B

Record 13 from database: **MEDLINE**

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Title

Pretreatment biliary lipid composition in white patients with radiolucent gallstones in the National Cooperative Gallstone Study.

Author

Hofmann AF; Grundy SM; Lachin JM; Lan SP; Baum RA; Hanson RF; Hersh T; Hightower NC Jr; Marks JW; Mekhjian H; Shaefer RA; Soloway; RD; Thistle JL; Thomas FB; Tyor MP

Address

Source

Gastroenterology, 1982 Oct, 83:4, 738-52

Abstract

Biliary lipid classes (bile acids, phospholipids, cholesterol) as well as individual biliary bile acids were measured in duodenal bile samples obtained before treatment from 284 white men and 264 white women participating in the National Cooperative Gallstone Study. The patients had radiolucent gallstones present in visualizing gallbladders. Calculated biliary cholesterol saturation was significantly higher in women (143 +/- 43, mean +/- SD, vs. 132 +/- 39 for men). Chenodeoxycholic acid was the major biliary bile acid in both sexes (40.0 +/- 9.9 in men; 38.8 +/- 9.3 in women, NS). Cholic acid was the second most common bile acid, constituting 32.9 +/- 8.8 in men and 31.8 +/- 8.9 in women (NS). When other demographic and clinical characteristics, including serum lipids, were related with biliary lipid composition, only percent ideal body weight correlated significantly. The partial correlation coefficient adjusted for percent ideal body weight indicated that the proportion of chenodeoxycholic acid correlated negatively with the mole fraction of cholesterol in bile in men, but not in women. Multiple regression analyses showed that bile saturation could not be predicted reliably from any clinical, chemical, or radiologic measurement in either sex. Published data for biliary lipid composition in individuals with biliary disease showed considerable overlap with the National Cooperative Gallstone Study data reported here, suggesting that cholesterol gallstone disease is not caused solely by increased biliary cholesterol saturation.

Language of Publication

English

Unique Identifier

82262647

MeSH Heading (Major)

Bile|*AN; Cholelithiasis|*ME/RA; Lipids|*AN

MeSH Heading

Adult; Aged; Bile Acids and Salts|AN; Body Weight; Caucasoid Race; Chenodeoxycholic Acid|AN; Cholesterol|AN; Deoxycholic Acid|AN; Female; Human; Male; Middle Age; Phospholipids|AN; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0016-5085

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Bile Acids and Salts); 0 (Phospholipids); 474-25-9 (Chenodeoxycholic Acid); 57-88-5 (Cholesterol); 83-44-3 (Deoxycholic Acid)

Section B

Record 14 from database: **MEDLINE**

[Go To The Top](#)

Title

Lack of effect of tocopherol on plasma lipids and lipoproteins in man.

Author

Kesaniemi YA; Grundy SM

Source

Am J Clin Nutr, 1982 Aug, 36:2, 224-8

Abstract

The influence of D,L-alpha-tocopherol (vitamin E) on the plasma total and very low-density lipoprotein, low density lipoprotein, and high-density lipoprotein cholesterol and triglyceride was studied in one normolipidemic and four hypertriglyceridemic subjects. Overall vitamin E caused no decrease in plasma total, very low-density and low-density lipoprotein cholesterol and triglyceride concentrations and no increase in high-density lipoprotein cholesterol level. D,L-alpha-Tocopherol does not seem to have any consistent effect on plasma lipids and lipoproteins in these patients.

Language of Publication

English

Unique Identifier

82253686

MeSH Heading (Major)

Lipids*BL; Lipoproteins|*BL; Vitamin E|*PD

MeSH Heading

Cholesterol|BL; Coronary Disease|CO; Female; Human; Hyperlipoproteinemia Type IV|BL/CO; Lipoproteins, HDL|BL; Lipoproteins, LDL|BL; Lipoproteins, VLDL|BL; Male; Middle Age; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0002-9165

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (very low density lipoprotein triglyceride); 0 (Lipoproteins); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, HDL); 0 (Lipoproteins, LDL Cholesterol); 0 (Lipoproteins, VLDL); 0 (Triglycerides); 1406-18-4 (Vitamin E); 57-88-5 (Cholesterol)

Section B

Record 15 from database: **MEDLINE**

[Go To The Top](#)

Title

Effects of AOMA on cholesterol metabolism in man.

Author

Crouse JR; Grundy SM; Johnson JH

Address

Source

Metabolism, 1982 Jul, 31:7, 733-9

Abstract

A new cholesterol-lowering agent, surfomer (AOMA), has been developed that blocks cholesterol absorption and lowers plasma cholesterol in animals. To evaluate AOMA in man, we studied its effects on plasma cholesterol, cholesterol absorption, fecal excretion of cholesterol and its bacterial degradation products,

coprostanol and coprostanone, and percent saturation of gallbladder bile with cholesterol in 20 individuals chosen for hyperlipidemia. These patients had low density lipoprotein cholesterol (LDL-C) of 215 +/- 29 mg/dl. Two dose levels of AOMA were compared (10.8 and 5.4 grams daily), each for 1 mo in a study that combined features of inpatient and outpatient investigation. AOMA was tolerated well by all volunteers. There was a statistically significant correlation between percent absorption and LDL-C in both the control and AOMA treated states. AOMA lowered mean plasma cholesterol and LDL-C by 9.1% and 12.9% at the high dose and by 6.4% and 8.3% at the low dose, respectively. Triglyceride (control = 223 +/- 58 mg/dl, treatment = 232 +/- 85 mg/dl), high density lipoprotein cholesterol (HDL-C: control = 50 +/- 11 mg/dl, treatment = 50 +/- 13 mg/dl), and other lipoprotein lipids were not affected. AOMA lowered cholesterol absorption by 25% on the high dose. For 18/20 patients there was a statistically significant (p less than 0.001) correlation (r = 0.74) between percent LDL-C reduction and percent absorption inhibition. For these patients, presumably, variable effectiveness of the agent in inhibiting absorption was the most important predictor of individual responsiveness although individual variation in other cholesterol regulatory mechanisms also played a role. Two other patients showed marked LDL-C reduction at unusually low levels of absorption inhibition. We also had the opportunity to compare the effects of AOMA with neomycin in 8 volunteers. Neomycin was 50% more effective in lowering LDL-C than AOMA; however, it was twice as effective in inhibition absorption as well. AOMA dramatically reduced fecal excretion of cholesterol bacterial conversion products; whereas cholesterol per se accounted for only 50% of total neutral steroid excretion in the control state, it accounted for 93% of steroid excretion when patients were administered 10.8 grams of AOMA daily. In four patients studied there was no adverse effect of AOMA on gallbladder saturation with cholesterol; in fact, the percent saturation tended to decrease with AOMA in these four patients.

Language of Publication

English

Unique Identifier

82219189

MeSH Heading (Major)

Cholesterol*ME; Hypercholesterolemia*DT; Hyperlipidemia*DT; Polymers*TU; Succinates*TU

MeSH Heading

Adult; Aged; Bile|AN; Comparative Study; Feces|AN; Female; Human; Intestinal Absorption; Lipoproteins, LDL|ME; Male; Middle Age; Neomycin|TU; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0026-0495

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Lipoproteins, LDL Cholesterol); 0 (Polymers); 0 (Succinates); 1404-04-2 (Neomycin); 57-88-5 (Cholesterol); 71251-04-2 (surfomer)

Section BRecord 16 from database: **MEDLINE**[Go To The Top](#)**Title**

Significance of low density lipoprotein production in the regulations of plasma cholesterol level in man.

Author Karl Loren

<http://www.oralchelation.com/technical/grundy1.htm>

Kesaniemi YA; Grundy SM

Address

Source

J Clin Invest, 1982 Jul, 70:1, 13-21

Abstract

To determine whether production or catabolism of low density lipoprotein (LDL) is the major factor controlling LDL concentrations in subjects with plasma cholesterol levels from low-normal to mildly elevated, measurements of apoprotein of LDL (apoLDL) turnover were performed in 16 patients with various plasma cholesterol concentrations. Cholesterol balance studies were done simultaneously in 13 of these patients. Plasma concentrations of apoLDL and LDL-cholesterol were positively correlated with synthetic rates of apoLDL ($r = 0.74$, P less than 0.001; $r = 0.50$, P less than 0.05, respectively). No correlation was noted between the fractional catabolic rate for apoLDL and apoLDL levels (or LDL-cholesterol). For further analysis, the patients were divided into three groups with stepwise increases in apoLDL concentrations. When apoLDL levels rose significantly, from 83 ± 5 SEM to 122 ± 2 to 149 ± 5 mg/dl, synthetic rates for apoLDL also increased significantly from 11.6 ± 12 to 17.0 ± 0.9 to 23.8 ± 1.8 mg/d/kg ideal weight. In contrast, the fractional catabolic rate of apoLDL was not different among the three groups (0.32 ± 0.03 vs. 0.29 ± 0.02 vs. $0.33 \pm 0.03/d$). No relation was noted between synthesis of total body cholesterol (or bile acids) and concentrations, production rates, or removal of apoLDL. Thus, concentrations of apoLDL and LDL-cholesterol in these subjects with plasma cholesterol levels from low-normal to mildly elevated were regulated mainly by synthetic rates of apoLDL and not by LDL catabolism.

Language of Publication

English

Unique Identifier

82214463

MeSH Heading (Major)

Cholesterol|BI/*BL; Lipoproteins, LDL|*BI/*BL

MeSH Heading

Adult; Aged; Bile Acids and Salts|BI; Female; Human; Hypercholesterolemia, Familial|ME; Kinetics; Lipids|BL; Lipoproteins|BL; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0021-9738

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Bile Acids and Salts); 0 (Lipoproteins); 0 (Lipoproteins, LDL Cholesterol); 0 (Triglycerides); 57-88-5 (Cholesterol)

Section B

Record 17 from database: **MEDLINE**

[Go To The Top](#)

Title

Author

Barrett-Connor E; Grundy SM; Holdbrook MJ

Address

Source

Am J Epidemiol, 1982 May, 115:5, 657-63

Abstract

Most previous studies of hyperlipidemia in diabetics are based on patients in specialty clinics or reflect an era when diabetics consumed a high-fat, low-carbohydrate diet. In this paper, data from a defined adult population aged 20-79 years in an upper middle class community in Southern California, 1972-1974, were used to ascertain the relationship of hyperlipidemia to diabetes in a current community-based population. All (n = 358) diabetics as defined by history and/or fasting hyperglycemia (fasting plasma glucose, greater than or equal to 140 mg/dl) were compared with all (n = 4387) nondiabetics defined as euglycemic (fasting plasma glucose, less than 110 mg/dl) with no personal or family history of diabetes. In both men and women 50 years of age and older, the mean cholesterol level and the prevalence of categorical hypercholesterolemia were not significantly different in diabetics vs. nondiabetics, whereas the mean triglyceride level and the prevalence of categorical hypertriglyceridemia were significantly higher in diabetics vs. nondiabetics. Case-control comparisons of 356 diabetics matched for age and obesity with 356 nondiabetics confirmed the significantly higher triglyceride levels in diabetes. Conversely, hypertriglyceridemia was associated with diabetes in 29 per cent of nonobese men and 25 per cent of obese men, and in 10 per cent of non-obese women and 21 per cent of obese women. The biologic mechanism of hypertriglyceridemia in diabetics is discussed.

Language of Publication

English

Unique Identifier

82203358

MeSH Heading (Major)

Diabetes Mellitus|BL/*CO; Hyperlipoproteinemia|*CO

MeSH Heading

Adult; Aged; California; Cholesterol|BL; Female; Human; Male; Middle Age; Obesity|CO; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0002-9262

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Triglycerides); 57-88-5 (Cholesterol)

Section B

Record 18 from database: **MEDLINE**

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Title

Effects of interruption of the enterohepatic circulation of bile acids on the transport of very low density-lipoprotein triglycerides.

Author Karl Loren

<http://www.oralchelation.com/technical/grundy1.htm>

Beil U; Crouse JR; Einarsson K; Grundy SM

Address

Source

Metabolism, 1982 May, 31:5, 438-44

Abstract

An increase in plasma very low density lipoprotein-triglycerides (VLDL-TG) is seen frequently during treatment with bile acid-binding resins. The purpose of this study was to determine whether this increment in VLDL-TG is due mainly to an increase in synthesis of VLDL, or to an enhanced catabolism. Three types of patients were studied: (1) 7 normotriglyceridemic subjects. (2) 4 obese patients, and (3) 9 hypertriglyceridemic patients. Before treatment they underwent a study of VLDL-TG kinetics that employed multicompartmental analysis of specific activity curves following injection of 3H-glycerol. The patients were then treated with a bile acid-binding resin, either cholestyramine or colestipol, for several weeks to several months. At the end of the treatment period, they were readmitted to the hospital for a second study of VLDL-TG kinetics. The patients showed a variable response to resin therapy. Many had an increase in concentrations of VLDL-TG, but others had no change or even a slight decrease. However, analysis of the data showed a high correlation between change in production rates of VLDL-TG and change in concentration. Also, when the data for the 20 patients were combined, there was a statistically significant increase in both synthetic rates and concentrations of VLDL-TG; in contrast, the fractional catabolic rate (FCR) was unchanged by therapy. Therefore, our data show that when treatment with bile acid sequestrants causes an increase in plasma VLDL-TG, the increase is due to an increment in production and not to a decrease in catabolism.

Language of Publication

English

Unique Identifier

82194821

MeSH Heading (Major)

Bile Acids and Salts*|BL; Enterohepatic Circulation*|; Lipoproteins, VLDL*|BL; Triglycerides*|BL

MeSH Heading

Adult; Aged; Human; Hypercholesterolemia|BL; Hyperlipoproteinemia Type IV|BL; Male; Middle Age; Obesity|BL; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0026-0495

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (very low density lipoprotein triglyceride); 0 (Bile Acids and Salts); 0 (Lipoproteins, VLDL); 0 (Triglycerides)

Section B

Record 19 from database: **MEDLINE**

[Go To The Top](#)

Title

Optimizing the effect of plant sterols on cholesterol absorption in man.

Author

Address

Source

Am J Clin Nutr, 1982 Apr, 35:4, 697-700

Abstract

During three experimental periods, nine adults were hospitalized on a metabolic ward and fed a meal containing 500 mg of cholesterol as a component of scrambled eggs. In addition, the meal contained: 1) no additive, 2) 1 g beta-sitosterol, or 3) 2 g beta-sitosteryl oleate. Stools for the succeeding 5 days were analyzed to determine the percentage of the cholesterol in the test meal that was absorbed. The addition of beta-sitosterol resulted in a 42% decrease in cholesterol absorption; the beta-sitosteryl oleate caused a 33% reduction. These results indicate that the judicious addition of beta-sitosterol or beta-sitosteryl oleate to meals containing cholesterol-rich foods will result in a significant decrease in cholesterol absorption, with a consequent decrease in plasma cholesterol.

Language of Publication

English

Unique Identifier

82179369

MeSH Heading (Major)

Cholesterol, Dietary|*ME; Sitosterols|*PD

MeSH Heading

Absorption; Adult; Eggs; Feces|AN; Human; Structure-Activity Relationship; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0002-9165

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Cholesterol, Dietary); 0 (Sitosterols); 3712-16-1 (beta-sitosterol oleate); 5779-62-4 (sitosterol)

Section B

Record 20 from database: **MEDLINE**

[Go To The Top](#)

Title

Triglyceride and cholesterol metabolism in primary hypertriglyceridemia.

Author

Beil U; Grundy SM; Crouse JR; Zech L

Address

Source

Arteriosclerosis, 1982 Jan-Feb, 2:1, 44-57

Abstract

To determine mechanisms of elevated plasma triglycerides (TG) in patients with primary hypertriglyceridemias, simultaneous studies were carried out on kinetics of very low density lipoprotein-triglycerides (VLDL-TG) and synthesis of cholesterol and bile acids. Sixteen hypertriglyceridemic patients with familial combined

hyperlipidemia (FCHL) and 12 patients with poorly classified, primary hypertriglyceridemia were studied, and their results were compared to a series of normal and obese subjects previously studied in our laboratory. The mean value for transport (synthesis) of VLDL-TG in patients with FCHL was about twice normal. Although the upper normal synthesis rates overlapped with transport rates of some patients with FCHL, it appeared that the major cause of hypertriglyceridemia in FCHL was an elevated production of VLDL-TG. However, the height of the plasma TG in FCHL patients also was influenced by individual clearance capacities for VLDL-TG, and fractional clearance rates in several seemed particularly low. Synthesis rates for cholesterol and/or bile acids were high in several patients with FCHL, suggesting simultaneous overproduction of VLDL-TG and sterols; however, increased synthesis of both was not observed in all the patients. Most patients with poorly classified hypertriglyceridemia had over-production of VLDL-TG, but an apparent reduction in clearance was common. In these patients, increased synthesis of cholesterol and bile acids was infrequent. Our results indicate that abnormally high production of VLDL-TG seemed to be the major factor in causing primary hypertriglyceridemia, but that clearance capacity can play an important role in determining the the severity of the TG elevation.

Language of Publication

English

Unique Identifier

82134533

MeSH Heading (Major)

Bile Acids and Salts*BI; Cholesterol*BI/BL; Hyperlipidemia, Familial Combined|BL/ET/*ME; Lipoproteins, VLDL|BL/*ME; Triglycerides*BL/*ME

MeSH Heading

Adult; California; Hospitals, Veterans; Human; Lipoprotein Lipase|BL; Middle Age; Obesity|BL/ME; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

Country of Publication

UNITED STATES

CAS Registry/EC Number

EC 3.1.1.34 (Lipoprotein Lipase); 0 (Bile Acids and Salts); 0 (Lipoproteins, VLDL); 0 (Triglycerides); 57-88-5 (Cholesterol)

HealthGate Documents

Section C

Record 1 from database: **MEDLINE**

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Title

Effect of ascorbate supplementation on low density lipoprotein oxidation in smokers.

Author

Fuller CJ; Grundy SM; Norkus EP; Jialal I

Address

Department of Clinical Nutrition, University of Texas-Southwestern Medical Center, Dallas, USA.

Source

Atherosclerosis, 1996 Jan, 119:2, 139-50

The oxidative modification of low density lipoprotein (LDL) may play a role in the pathogenesis of atherosclerosis. Furthermore, evidence of oxidized LDL (ox-LDL) has been found in vivo. Supplementation of some animal models with antioxidants has been shown to retard the formation of aortic atherosclerosis. Ascorbate (vitamin C) is a highly potent aqueous-phase antioxidant in plasma, which has been shown in vitro to retard LDL oxidation. Cigarette smokers have reduced concentrations of ascorbate in their plasma, and their LDL may be more prone to oxidation. Hence, the objective of the present study was to examine the effect of ascorbate depletion and supplementation on the propensity of LDL to oxidize in smokers in a 6-week study. Nineteen healthy smokers followed a low ascorbate diet (< or = 30 mg/day) for 2 weeks, then were randomly assigned to receive placebo or 1000 mg ascorbate per day for 4 weeks. Blood was taken at 0 and 4 weeks of supplementation for study of LDL oxidative susceptibility. LDL was oxidized with 5 mumol/l copper. The ascorbate-supplemented group had significant increases in plasma ascorbate. The placebo group showed no change in the time course of LDL oxidation between 0 and 4 weeks. However, the ascorbate-supplemented group has a significant reduction in LDL oxidative susceptibility as measured by thiobarbituric acid-reactive substances (TBARS) and the formation of conjugated dienes. The ascorbate-supplemented group demonstrated significantly increased lag phase and decreased oxidation rate at 4 weeks compared to 0 weeks. No changes were found in the placebo group. The ascorbate-supplemented group showed no biochemical signs consistent with increased body iron stores. Supplementation of otherwise healthy smokers for 4 weeks with 1000 mg ascorbate per day resulted in increased plasma ascorbate and reduced LDL oxidative susceptibility.

Language of Publication

English

Unique Identifier

96404213

MeSH Heading (Major)

Antioxidants|*PD; Ascorbic Acid|BL/*PD; Atherosclerosis|ME/*PC; Lipoproteins, LDL|*BL; Smoking|*BL

MeSH Heading

Adult; Beta Carotene|BL; Cholesterol|BL; Female; Free Radicals; Human; Lipid Peroxidation|DE; Lipoproteins, HDL|BL; Male; Middle Age; Oxidation-Reduction|DE; Support, U.S. Gov't, P.H.S.; Thiobarbituric Acid Reactive Substances|AN; Vitamin E|BL

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0021-9150

Country of Publication

IRELAND

Section CRecord 2 from database: **MEDLINE**[Go To The Top](#)**Title**

Relationships of generalized and regional adiposity to insulin sensitivity in men.

Author

Abate N; Garg A; Peshock RM; Stray Gundersen J; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052, USA.

Source

Abstract

The relative impacts of regional and generalized adiposity on insulin sensitivity have not been fully defined. Therefore, we investigated the relationship of insulin sensitivity (measured using hyperinsulinemic, euglycemic clamp technique with [3-3H]glucose turnover) to total body adiposity (determined by hydrodensitometry) and regional adiposity. The latter was assessed by determining subcutaneous abdominal, intraperitoneal, and retroperitoneal fat masses (using magnetic resonance imaging) and the sum of truncal and peripheral skinfold thicknesses. 39 healthy middle-aged men with a wide range of adiposity were studied. Overall, the intraperitoneal and retroperitoneal fat constituted only 11 and 7% of the total body fat. Glucose disposal rate (Rd) and residual hepatic glucose output (rHGO) values during the 40 mU/m².min insulin infusion correlated significantly with total body fat (r = -0.61 and 0.50, respectively), subcutaneous abdominal fat (r = -0.62 and 0.50, respectively), sum of truncal skinfold thickness (r = -0.72 and 0.57, respectively), and intraperitoneal fat (r = -0.51 and 0.44, respectively) but not to retroperitoneal fat. After adjusting for total body fat, the Rd and rHGO values showed the highest correlation with the sum of truncal skinfold thickness (partial r = -0.40 and 0.33, respectively). We conclude that subcutaneous truncal fat plays a major role in obesity-related insulin resistance in men, whereas intraperitoneal fat and retroperitoneal fat have a lesser role.

Language of Publication

English

Unique Identifier

95340882

MeSH Heading (Major)

Adipose Tissue|*ME; Insulin|*PD; Obesity|*ME

MeSH Heading

Adult; Fatty Acids, Nonesterified|ME; Glucose|ME; Human; Insulin Resistance; Liver|ME; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0021-9738

Country of Publication

UNITED STATES

Section C

Record 3 from database: **MEDLINE**

[Go To The Top](#)

Title

Interactions between microsomal triglyceride transfer protein and apolipoprotein B within the endoplasmic reticulum in a heterologous expression system.

Author

Patel SB; Grundy SM

Address

Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, Texas 75235-9052, USA.

Source

J Biol Chem, 1996 Aug, 271:31, 18686-94

Abstract

When apolipoprotein B (apoB) is expressed in heterologous cells, it is not secreted but retained and degraded within the endoplasmic reticulum (ER). We have previously characterized carboxyl-terminal truncated forms of apoB expressed in COS cells and have shown that these proteins were readily synthesized but retained within the ER and degraded, if the size of the truncated protein was larger than apoB 29. Below this size, the smaller the size of the apoB truncates, the greater the extent of secretion, although >50% of these smaller proteins were also degraded within the ER. In the present study, we demonstrate that this secretory defect can be overcome by coexpression with microsomal triglyceride transfer protein (MTP); moreover, this complementation is inversely related to the size of apoB. Secretion of apoBs larger than B29 required the coexpression of MTP and, in the presence of MTP, was oleate-responsive. MTP, in the presence or absence of oleate supplementation, had little or no effect on the secretion of the shorter truncates. We discovered, however, that MTP was physically associated with all forms of apoB intracellularly (B13-B41). The association of MTP with apoB 41 was stable to high salt washing, as well as to low pH, suggesting that these interactions may be hydrophobic in nature. In addition to the interaction with MTP, apoB was also found to be associated with calnexin, confirming previous studies, and with proteins bearing the KDEL retention signal. However, studies on overexpression of human calnexin and tunicamycin inhibition of glycosylation showed that interaction with calnexin was not necessary for the formation or secretion of apoB 41-containing lipoproteins; moreover, in the presence of MTP, the association of calnexin with apoB 41 was transient or absent. These data suggest that for apoB to attain a folded state sufficient to escape the quality control of the ER, it needs to obtain neutral lipid (supplied by MTP), as well as its ability to keep it packaged as a rudimentary lipoprotein, dependent on its size being larger than B29.

Language of Publication

English

Unique Identifier

96324947

MeSH Heading (Major)

Apolipoproteins B|CH/GE/*ME; Carrier Proteins|*ME; Endoplasmic Reticulum|DE/*ME; Microsomes|*ME

MeSH Heading

Amino Acid Sequence; Animal; Calcium-Binding Proteins|ME; Cell Line; Human; Lipids|CH; Molecular Sequence Data; Molecular Structure; Oleic Acids|PD; Oligopeptides|GE; Peptide Fragments|CH/GE/ME; Recombinant Proteins|CH/GE/ME; Signal Peptides|CH/GE/ME; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Tunicamycin|ME

Publication Type

JOURNAL ARTICLE

ISSN

0021-9258

Country of Publication

UNITED STATES

Section CRecord 4 from database: **MEDLINE**[Go To The Top](#)**Title**

Bimodal distribution of cholesteryl ester transfer protein activities in normotriglyceridemic men with low HDL cholesterol concentrations.

Author

Tato F; Vega GL; Grundy SM

Address

Source

Arterioscler Thromb Vasc Biol, 1995 Apr, 15:4, 446-51

Abstract

Increased plasma activities of cholesteryl ester transfer protein (CETP) theoretically could lower HDL cholesterol levels due to enhanced transfer of cholesteryl esters from HDL to apo B-containing lipoproteins. To determine whether high CETP activities are associated with isolated hypoalphalipoproteinemia, CETP activities were measured in 109 adult men with HDL cholesterol < 35 mg/dL, plasma triglycerides < 200 mg/dL, and LDL cholesterol < 160 mg/dL; the results were compared with those of 50 normolipidemic (HDL cholesterol > 40 mg/dL) male subjects. CETP activities were assayed in vitro and expressed as the percent of [3H]cholesteryl ester transferred from HDL3 to LDL during a 16-hour incubation. In addition, postheparin plasma activities of lipoprotein lipase (LPL) and hepatic triglyceride lipase (HTGL) were determined in 71 patients with a low HDL cholesterol level. Distributions of CETP activities were unimodal in control subjects (mean +/- SD, 23.1 +/- 5.0%), but they were bimodal in the low-HDL patients. Among the latter, 27 patients had elevated CETP activities (40.8 +/- 4.6%), whereas 82 patients had CETP activities that overlapped the normal range (26.14 +/- 7.6%). Low-HDL patients with normal CETP activities had 20% lower LPL activities (P = .01), 25% higher HTGL activities (P = .03), and 63% lower LPL/HTGL ratios (P < .001) than those of low-HDL patients with increased CETP activity. Furthermore, mean LPL and HTGL activities in the low-HDL patients with elevated CETP activities were in the normal range. Another important distinction between the two subgroups with low HDL was that the subgroup with high CETP activity had only a 30% prevalence of coronary heart disease compared with a 70% prevalence in the subgroup with normal CETP activity (P < .01). These findings suggest that elevated CETP activity may be a significant factor in causing low HDL cholesterol levels in a distinct subgroup of normolipidemic patients with low HDL cholesterol levels.

Language of Publication

English

Unique Identifier

95268979

MeSH Heading (Major)

Carrier Proteins[*BL]; Lipoproteins, HDL Cholesterol[*BL]; Triglycerides[*BL]

MeSH Heading

Adult; Human; Lipoprotein Lipase|AN; Liver|ME; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

1079-5642

Country of Publication

UNITED STATES

Section C

Record 5 from database: **MEDLINE**

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Title

Relation between cholesterol ester transfer protein activities and lipoprotein cholesterol in patients with hypercholesterolemia and combined hyperlipidemia.

Author

Address

Center for Human Nutrition, UT Southerwestern Medical Center, Dallas, TX 75235-9052, USA.

Source

Arterioscler Thromb Vasc Biol, 1995 Jan, 15:1, 112-20

Abstract

Cholesterol ester transfer protein (CETP) promotes the transfer of cholesterol esters among different lipoprotein classes-high-density lipoproteins (HDL), very-low-density lipoproteins, intermediate-density lipoproteins, and low-density lipoproteins (LDL). The current study was carried out to determine whether CETP activities are correlated with lipoprotein cholesterol levels in a large number of patients having elevated LDL cholesterol and normal triglycerides (hypercholesterolemia) and elevated LDL cholesterol and high triglycerides (combined hyperlipidemia). Compared with 50 normolipidemic male patients, 113 hypercholesterolemic patients had a 42% higher mean activity of CETP, and approximately 60% of these patients had CETP activities outside the normal range. A similar elevation of CETP was observed in 47 patients with combined hyperlipidemia. Furthermore, in those with combined hyperlipidemia, CETP activities were highly correlated with LDL cholesterol, non-HDL cholesterol, and non-HDL/HDL ratios. Similar high correlations were obtained by combining normotriglyceridemic patients with and without elevated LDL cholesterol. Since patients with elevated LDL cholesterol had a significantly lower mean level of HDL cholesterol, a high CETP activity also was related to a reduced HDL cholesterol level. Our results are consistent with this concept, although they do not constitute final proof that high CETP activities contribute to elevated cholesterol concentrations and reduced HDL cholesterol levels in patients with hypercholesterolemia and in those with combined hyperlipidemia.

Language of Publication

English

Unique Identifier

95268925

MeSH Heading (Major)

Carrier Proteins|*BL; Cholesterol|*BL; Hypercholesterolemia|*ME; Hyperlipidemia, Familial Combined|*ME; Lipoproteins|*BL

MeSH Heading

Aged; Human; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

1079-5642

Country of Publication

UNITED STATES

Section C

Record 6 from database: **MEDLINE**

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Title

Metabolism of low density lipoproteins in nephrotic dyslipidemia: comparison of hypercholesterolemia alone and combined hyperlipidemia.

Author

Vega GL; Toto RD; Grundy SM

Address Karl Loren

<http://www.oralchelation.com/technical/grundy1.htm>

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas, USA.

Source

Kidney Int, 1995 Feb, 47:2, 579-86

Abstract

High levels of low-density lipoprotein cholesterol (LDL) (hypercholesterolemia) are commonly present in the nephrotic syndrome. Another pattern of dyslipidemia in nephrotic patients is an elevation of both cholesterol and triglyceride levels (combined hyperlipidemia). It has been postulated that the underlying cause of nephrotic dyslipidemia is an hepatic overproduction of apolipoprotein B (apo B)-containing lipoproteins. To examine this hypothesis, the metabolism of LDL-apo B was compared between nephrotic patients with hypercholesterolemia and with combined hyperlipidemia. Thirteen patients (7 with hypercholesterolemia, and 6 with combined hyperlipidemia) underwent measurements of turnover rates of autologous LDL apo B. The results were compared to normolipidemic controls and to patients with primary combined hyperlipidemia previously studied in our laboratory. Nephrotic patients with hypercholesterolemia generally had: (a) lower fractional catabolic rates of LDL apo B than normolipidemic healthy individuals; (b) LDL particles enriched in cholesterol; but (c) no overproduction of LDL apo B. In contrast, patients with combined hyperlipidemia were found to have: (a) high fractional catabolic rates for LDL apo B compared to normolipidemic controls; (b) cholesterol-poor LDL particles; and (c) markedly elevated production rates for LDL. Also, for the group as a whole, there was a positive correlation between plasma triglyceride levels and fractional catabolic rates. These data indicate that the metabolism of LDL is strikingly different between the two forms of nephrotic dyslipidemia. Although there may be common mechanisms contributing to LDL levels in nephrotic patients, there also appears to be a divergence of mechanisms depending on whether hypertriglyceridemia is associated with hypercholesterolemia.

Language of Publication

English

Unique Identifier

95239977

MeSH Heading (Major)

Hyperlipidemia|*BL/*CO; Lipoproteins, LDL|*BL; Nephrotic Syndrome|*BL/*CO

MeSH Heading

Adolescence; Adult; Aged; Comparative Study; Female; Human; Hypercholesterolemia|BL/CO; Male; Middle Age; Reference Values; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0085-2538

Country of Publication

UNITED STATES

Section C

Record 7 from database: **MEDLINE**

[Go To The Top](#)

Title

Atherogenic dyslipidemia: lipoprotein abnormalities and implications for therapy.

Author

Grundy SM

Address

Source

Am J Cardiol, 1995 Feb, 75:6, 45B-52B

Abstract

Atherogenic dyslipidemia is a lipoprotein profile combining 4 specific abnormalities: borderline-high total cholesterol levels; high triglyceride concentrations; small, dense, low-density lipoprotein (LDL) particles; and low high-density lipoprotein (HDL) concentrations. It is a predisposing factor to premature coronary artery disease (CAD), although separating and calculating the contribution of each abnormality to the risk of CAD is difficult, especially since the abnormalities often appear in this combination. The ratio of total cholesterol to HDL cholesterol is currently the most powerful single predictor of risk in dyslipidemic patients. Therapy for atherogenic dyslipidemia includes dietary changes aimed at decreasing intake of cholesterol-raising fatty acids and achieving weight reduction; exercise, which confers many of the benefits of weight reduction; and, when those measures fail to correct the lipid and lipoprotein profile, drug therapy. Nicotinic acid reduces triglyceride and cholesterol levels while raising HDL concentrations, but up to half of patients cannot tolerate its adverse effects. Fibric acids effectively lower triglyceride levels and are generally well tolerated but have little beneficial effect on the cholesterol profile. Statins offer marked reductions in total, LDL, and very low-density lipoprotein cholesterol levels and cause modest increases in HDL concentration. Combination therapy can enhance the efficacy of the individual drugs.

Language of Publication

English

Unique Identifier

95168167

MeSH Heading (Major)

Atherosclerosis|BL/*ET/*TH; Hyperlipidemia|BL/*CO/*TH; Lipoproteins|*BL

MeSH Heading

Coronary Disease|BL/ET/TH; Human; Risk Factors

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0002-9149

Country of Publication

UNITED STATES

Section C

Record 8 from database: **MEDLINE**

[Go To The Top](#)

Title

Efficacy of low-dose cholesterol-lowering drug therapy in men with moderate hypercholesterolemia.

Author

Denke MA; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas.

Source

Arch Intern Med, 1995 Feb, 155:4, 393-9

Abstract

OBJECTIVE: To test the potency of low-dose cholesterol-lowering drug therapy in patients with moderate

hypercholesterolemia and to evaluate the effectiveness for cholesterol lowering of a safe regimen to be used in primary prevention of coronary heart disease. DESIGN: The efficacy of three drug regimens (cholestyramine resin, 8 g/d; cholestyramine resin, 8 g/d, plus lovastatin, 5 mg/d; and lovastatin, 20 mg/d) was tested in 26 men aged 31 to 70 years with moderate hypercholesterolemia after a Step-One cholesterol-lowering diet. Each drug period was 3 months in duration, interspersed by a 1-month period of the Step-One diet only. Blood for lipid and lipoprotein measurements was obtained on 5 different days during the last 2 weeks of each drug and diet-only period. RESULTS: Cholestyramine resin therapy at 8 g/d achieved a significant reduction in low-density lipoprotein cholesterol levels from 4.47 mmol/L (173 mg/dL) to 3.90 mmol/L (151 mg/dL) (P < .005). The addition of 5 mg of lovastatin to cholestyramine therapy achieved even lower levels, averaging 3.39 mmol/L (131 mg/dL) (P < .005). Lovastatin therapy at 20 mg/d produced lowering of low-density lipoprotein cholesterol levels similar to that of the low-dose combination. CONCLUSIONS: Low-dose combination drug therapy for the management of hypercholesterolemia appears to be an effective means of lowering cholesterol levels that remain persistently elevated after dietary therapy, at the same time, it should carry a low risk of toxic effects.

Language of Publication

English

Unique Identifier

95150746

MeSH Heading (Major)

Anticholesteremic Agents|*TU; Hypercholesterolemia|BL/*DT

MeSH Heading

Adult; Aged; Cholestyramine|TU; Drug Therapy, Combination; Human; Lipids|BL; Lipoproteins|BL; Lovastatin|TU; Male; Middle Age; Severity of Illness Index; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Treatment Outcome

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE

ISSN

0003-9926

Country of Publication

UNITED STATES

Section C

Record 9 from database: **MEDLINE**

[Go To The Top](#)

Title

Role of low-density lipoproteins in atherogenesis and development of coronary heart disease.

Author

Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235-9052.

Source

Clin Chem, 1995 Jan, 41:1, 139-46

Abstract

There is a strong association between increased blood concentrations of low-density lipoprotein (LDL) and severity of coronary atherosclerosis. Multiple mechanisms affect hypercholesterolemia, e.g., diet, aging, hormones, and genetics. LDL receptors apparently are also important--through down-regulation, defects in structure, or decreased numbers--as are changes in LDL binding characteristics caused by alterations in

apolipoprotein B content or structure. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1911111/> Current concepts of LDL metabolism are extensively reviewed, including the role of modified or oxidized LDL in atherogenesis.

Language of Publication

English

Unique Identifier

95112404

MeSH Heading (Major)

Atherosclerosis|*BL; Coronary Disease|*BL; Lipoproteins, LDL|*BL

MeSH Heading

Human; Hypercholesterolemia|BL; Lipoproteins, LDL Cholesterol|BL; Risk Factors

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0009-9147

Country of Publication

UNITED STATES

Section C

Record 10 from database: **MEDLINE**

[Go To The Top](#)

Title

Comparison of a high-carbohydrate diet with a high-monounsaturated-fat diet in patients with non-insulin-dependent diabetes mellitus.

Author

Garg A; Bonanome A; Grundy SM; Zhang ZJ; Unger RH

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

N Engl J Med, 1988 Sep 29, 319:13, 829-34

Abstract

We compared a high-carbohydrate diet with a high-fat diet (specifically, a diet high in monounsaturated fatty acids) for effects on glycemic control and plasma lipoproteins in 10 patients with non-insulin-dependent diabetes mellitus (NIDDM) receiving insulin therapy. The patients were randomly assigned to receive first one diet and then the other, each for 28 days, in a metabolic ward. In the high-carbohydrate diet, 25 percent of the energy was in the form of fat and 60 percent in the form of carbohydrates (47 percent of the total energy was in the form of complex carbohydrates); the high-monounsaturated-fat diet was 50 percent fat (33 percent of the total energy in the form of monounsaturated fatty acids) and 35 percent carbohydrates. The two diets had the same amounts of simple carbohydrates and fiber. As compared with the high-carbohydrate diet, the high-monounsaturated-fat diet resulted in lower mean plasma glucose levels and reduced insulin requirements, lower levels of plasma triglycerides and very-low-density lipoprotein cholesterol (lower by 25 and 35 percent, respectively; P less than 0.01), and higher levels of high-density lipoprotein (HDL) cholesterol (higher by 13 percent; P less than 0.005). Levels of total cholesterol and low-density lipoprotein (LDL) cholesterol did not differ significantly in patients on the two diets. These preliminary results suggest that partial replacement of complex carbohydrates with monounsaturated fatty acids in the diets of patients with NIDDM does not increase the level of LDL cholesterol and may improve glycemic control and the levels of plasma triglycerides and HDL cholesterol.

Language of Publication

MeSH Heading (Major)

Diabetes Mellitus, Non-Insulin-Dependent[*DH; Dietary Carbohydrates*AD; Dietary Fats*AD; Fatty Acids, Monounsaturated]*AD

MeSH Heading

Adult; Aged; Blood Glucose|AN; Cholesterol|BL; Comparative Study; Energy Intake; Human; Insulin|AD; Lipoproteins, LDL Cholesterol|BL; Lipoproteins, VLDL|BL; Middle Age; Random Allocation; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0028-4793

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Blood Glucose); 0 (Dietary Fats); 0 (Fatty Acids, Monounsaturated); 0 (Lipoproteins, LDL Cholesterol); 0 (Lipoproteins, VLDL Cholesterol); 0 (Lipoproteins, VLDL); 0 (Triglycerides); 11061-68-0 (Insulin); 57-88-5 (Cholesterol)

Section C

Record 11 from database: **MEDLINE**

[Go To The Top](#)

Title

Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels.

Author

Bonanome A; Grundy SM

Address

Department of Clinical Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

N Engl J Med, 1988 May 12, 318:19, 1244-8

Abstract

We studied the metabolic effects of stearic acid (18:0) on plasma lipoprotein levels in 11 subjects during three dietary periods of three weeks each. The three liquid-formula diets, which were used in random order, were high in palmitic acid (16:0), stearic acid, and oleic acid (18:1), respectively. Caloric intakes were the same during the three periods. As compared with the values observed when the subjects were on the high-palmitic-acid diet, plasma total cholesterol decreased by an average of 14 percent during consumption of the high-stearic-acid diet (P less than 0.005) and by 10 percent during consumption of the high-oleic-acid diet (P less than 0.02). Low-density lipoprotein cholesterol levels fell by 21 percent in subjects on the high-stearic-acid diet (P less than 0.005) and by 15 percent in subjects on the high-oleic-acid diet (P less than 0.005). No significant differences were observed in the plasma levels of triglycerides or high-density lipoprotein cholesterol among the three diets. Measurements of the intestinal absorption of palmitic, stearic, and oleic acids revealed essentially complete absorption of each during the three dietary periods. The oleic acid content of plasma triglycerides and cholesteryl esters increased significantly during the high-stearic-acid period, suggesting that stearic acid is rapidly converted to oleic acid. We conclude that stearic acid appears to be as effective as oleic acid in lowering

plasma cholesterol levels when either replaces palmitic acid in the diet. <http://www.ncbi.nlm.nih.gov/pubmed/88202052>

Language of Publication

English

Unique Identifier

88202052

MeSH Heading (Major)

Cholesterol|*BL; Lipoproteins|*BL; Stearic Acids|*AD

MeSH Heading

Diet; Fatty Acids|ME; Human; Intestinal Absorption; Lipoproteins, HDL Cholesterol|BL; Lipoproteins, LDL Cholesterol|BL; Male; Middle Age; Oleic Acids|AD; Palmitic Acids|AD; Triglycerides|BL

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0028-4793

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Fatty Acids); 0 (Lipoproteins); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, LDL Cholesterol); 0 (Oleic Acids); 0 (Palmitic Acids); 0 (Stearic Acids); 0 (Triglycerides); 112-80-1 (Oleic Acid); 57-10-3 (Palmitic Acid); 57-11-4 (stearic acid); 57-88-5 (Cholesterol)

Section C

Record 12 from database: **MEDLINE**

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Title

Dietary therapy for different forms of hyperlipoproteinemia.

Author

Grundy SM

Address

Source

Circulation, 1987 Sep, 76:3, 523-8

Abstract

Diet is the first line of therapy for hypercholesterolemia. The major dietary factors raising the plasma cholesterol are saturated fatty acids, cholesterol, and excess total calories. For almost all forms of hyperlipidemia, the first principle of dietary therapy is to reduce saturated fatty acids, decrease cholesterol, and curtail excess calories. In patients with severe hypercholesterolemia, marked restrictions of diet may be necessary. For these patients, drugs may be required to control cholesterol levels. However, the majority of patients with elevated plasma cholesterol can achieve a satisfactory reduction of cholesterol levels by diet, and drugs will not be necessary. Dietary therapy alone is adequate for most patients with familial forms of hypertriglyceridemia, but for a few patients drugs are required.

Language of Publication

English

Unique Identifier

87302142

MeSH Heading (Major)

Hyperlipoproteinemia|*DH

MeSH HeadingAnticholesteremic Agents|TU; Cholesterol|BL; Cholesterol, Dietary|AD; Chylomicrons|BL; Energy Intake; Fatty Acids|AD; Human; Hypercholesterolemia|DH; Hypercholesterolemia, Familial|DH; Hyperlipidemia|DH/GE; Hyperlipoproteinemia Type IV|DH; Hyperlipoproteinemia Type V|DH

Publication Type

JOURNAL ARTICLE

ISSN

0009-7322

Country of Publication

UNITED STATES

CAS Registry/EC Number0 (Anticholesteremic Agents); 0 (Cholesterol, Dietary); 0 (Chylomicrons); 0 (Fatty Acids); 57-88-5 (Cholesterol)

Section C

Record 13 from database: **MEDLINE**[Go To The Top](#)**Title**

Influence of sucrose polyester on plasma lipoproteins, and cholesterol metabolism in obese patients with and without diabetes mellitus.

Author

Grundy SM; Anastasia JV; Kesaniemi YA; Abrams J

Address**Source**

Am J Clin Nutr, 1986 Nov, 44:5, 620-9

Abstract

Sucrose polyester (SPE) is a nonabsorbable substitute for fat. This study examined its effects in 10 obese patients, 6 with diabetes mellitus. Three diabetics had hypertriglyceridemia. Most patients were studied in three periods: weight maintenance, caloric restriction + SPE, and caloric restriction without SPE. Nondiabetics generally tolerated SPE better than diabetics. In nondiabetic patients caloric restriction + SPE produced a decrease in total cholesterol and in LDL-cholesterol of 20% and 26%, respectively. In normotriglyceridemic diabetic patients caloric restriction + SPE had an effect on plasma lipoproteins similar to that of nondiabetics. In diabetics with hypertriglyceridemia caloric restriction (with or without SPE) caused a marked reduction in plasma triglycerides. In all patients caloric restriction reduced cholesterol balance and presumably cholesterol synthesis. The feeding of SPE caused increased outputs of fecal neutral steroids suggestive of decreased absorption of cholesterol; SPE also frequently caused a mild increase in fecal acidic steroids (bile acids).

Language of Publication

English

Unique Identifier87022836

MeSH Heading (Major)

Cholesterol*BL; Lipoproteins*BL; Obesity*BL; Obesity in Diabetes*BL; Sucrose*AA/AE/PD

MeSH Heading

Aged; Comparative Study; Diarrhea|CI; Energy Intake; Feces|AN; Female; Human; Male; Middle Age; Steroids|AN; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0002-9165

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Lipoproteins); 0 (Steroids); 121854-29-3 (sucrose polyester); 57-50-1 (Sucrose); 57-88-5 (Cholesterol)

Section C

Record 14 from database: **MEDLINE**

[Go To The Top](#)

Title

Comparison of monounsaturated fatty acids and carbohydrates for lowering plasma cholesterol.

Author

Grundy SM

Address

Source

N Engl J Med, 1986 Mar 20, 314:12, 745-8

Abstract

To examine the effects of dietary fatty acids and carbohydrate on plasma lipids and lipoproteins, 11 patients with a mean plasma total cholesterol level of 251 +/- 10 mg per deciliter were studied on a metabolic ward during three dietary periods, each lasting four weeks. A liquid diet rich in monounsaturated fatty acids ("High-Mono") and a diet low in fat ("Low-Fat") were compared with a diet high in saturated fatty acids ("High-Sat"). The High-Sat and High-Mono diets contained 40 percent of their total calories as fat and 43 percent as carbohydrate; the Low-Fat diet had 20 percent fat and 63 percent carbohydrate. Body weight was kept constant by adjusting total caloric intake. As compared with the High-Sat diet, both the High-Mono and Low-Fat diets lowered plasma total cholesterol (by 13 percent and 8 percent, respectively) and low-density lipoprotein cholesterol (by 21 percent and 15 percent, respectively). As compared with the High-Sat diet, the Low-Fat diet raised triglyceride levels and significantly reduced plasma high-density lipoprotein cholesterol. In contrast, the High-Mono diet had no effect on levels of triglycerides or high-density lipoprotein cholesterol. The ratio of low-density to high-density lipoprotein cholesterol was also significantly lower when the High-Mono diet rather than the Low-Fat diet was followed. Therefore, in short-term studies in which liquid diets are used and body weight is kept constant, a diet rich in monounsaturated fatty acids appears to be at least as effective in lowering plasma cholesterol as a diet low in fat and high in carbohydrate.

Language of Publication

English

Unique Identifier

86146770

MeSH Heading (Major)

Cholesterol*BL; Dietary Carbohydrates*PD; Dietary Fats*PD; Fatty Acids, Unsaturated*PD

Aged; Comparative Study; Female; Human; Lipoproteins, HDL Cholesterol|BL; Lipoproteins, LDL Cholesterol|BL; Male; Middle Age; Oleic Acids|PD; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0028-4793

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Dietary Fats); 0 (Fatty Acids, Unsaturated); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, LDL Cholesterol); 0 (Oleic Acids); 0 (Triglycerides); 112-80-1 (Oleic Acid); 57-88-5 (Cholesterol)

Section C

Record 15 from database: **MEDLINE**[Go To The Top](#)**Title**

Effect of combined supplementation with alpha-tocopherol, ascorbate, and beta carotene on low-density lipoprotein oxidation [see comments]

Author

Jialal I; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

Circulation, 1993 Dec, 88:6, 2780-6

Abstract

BACKGROUND. Data continue to accumulate supporting a proatherogenic role for oxidized low-density lipoprotein (Ox-LDL). Antioxidant micronutrients such as ascorbate, alpha-tocopherol, and beta carotene, levels of which can be favorably manipulated by dietary measures without side effects, could be a safe approach in inhibiting LDL oxidation. In fact, in vitro studies have shown that all three antioxidants can inhibit LDL oxidation. The present study was undertaken to ascertain both the safety and antioxidant effect of combined supplementation with alpha-tocopherol, ascorbate, and beta carotene on LDL oxidation. **METHODS AND RESULTS.** The effect of combined supplementation with alpha-tocopherol (800 IU/d) plus ascorbate (1.0 g/d) and beta carotene (30 mg/d) on copper-catalyzed LDL oxidation was tested in a randomized, placebo-controlled study in two groups of 12 male subjects over a 3-month period. Blood samples for the lipoprotein profile, antioxidant levels, and LDL isolation were obtained at baseline and at 3 months. Neither placebo nor combined antioxidant therapy resulted in any side effects or exerted an adverse effect on the plasma lipoprotein profile. Compared with placebo, combined antioxidant therapy resulted in a significant increase in plasma ascorbate and lipid standardized alpha-tocopherol and beta carotene levels (2.6-, 4.1-, and 16.3-fold, respectively). At baseline, there were no significant differences in the time course curves and kinetics of LDL oxidation as evidenced by the thiobarbituric acid reacting substances (TBARS) assay and the formation of conjugated dienes. However, at 3 months, combined supplementation resulted in a twofold prolongation of the lag phase and a 40% decrease in the oxidation rate. The combined antioxidant group was also compared with a group that received 800 IU of alpha-tocopherol only. Although the combined antioxidant group had significantly higher ascorbate and beta carotene levels than the group supplemented with alpha-tocopherol alone, there were no significant differences between the two groups with respect to LDL oxidation kinetics. **CONCLUSIONS.** Combined supplementation with ascorbate, beta carotene, and alpha-tocopherol is not superior to high-dose alpha-tocopherol alone in inhibiting LDL oxidation. Hence, alpha-tocopherol therapy should be favored in future coronary prevention trials

involving antioxidants.

<http://www.oralchelation.com/technical/grundy1.htm>

Language of Publication

English

Unique Identifier

94074088

MeSH Heading (Major)

Ascorbic Acid|*AD; Carotene|*AD; Lipoproteins, LDL|*BL; Vitamin E|*AD

MeSH Heading

Adult; Atherosclerosis|PC; Diet; Drug Synergism; Human; Kinetics; Lipid Peroxidation|DE; Male; Middle Age; Oxidation-Reduction; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Thiobarbituric Acid Reactive Substances|ME

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0009-7322

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Lipoproteins, LDL); 0 (Thiobarbituric Acid Reactive Substances); 1406-18-4 (Vitamin E); 36-88-4 (Carotene); 50-81-7 (Ascorbic Acid); 7235-40-7 (Beta Carotene)

Section C

Record 16 from database: **MEDLINE**

[Go To The Top](#)

Title

Mechanisms and treatment of dyslipidemia of renal diseases.

Author

Toto RD; Vega GL; Grundy SM

Address

University of Texas Southwestern Medical Center, Dallas.

Source

Curr Opin Nephrol Hypertens, 1993 Sep, 2:5, 784-90

Abstract

Dyslipidemia is commonly observed in nephrotic syndrome, in chronic renal failure, and after renal transplantation. The patterns of dyslipidemia, however, differ among these three conditions, and the origins and mechanisms responsible for abnormalities in lipoprotein metabolism in each are not well understood. Whether these dyslipidemias contribute to the development of atherosclerosis and coronary heart disease is uncertain, but it is probable that they do. Important questions are whether an attempt should be made to treat the various renal dyslipidemias, and if so, by what means. Also of current interest are dyslipidemias in the nephrotic syndrome, chronic renal failure (uremia), and the post-renal transplantation state.

Language of Publication

English

Unique Identifier

95006520

MeSH Heading (Major)

Hyperlipidemia|BL/*ET/TH; Kidney Diseases|BL/*CO

MeSH HeadingAnimal; Human; Hydroxymethylglutaryl CoA Reductases|AI; Kidney Failure, Chronic|BL/CO; Kidney Transplantation|AE; Nephrotic Syndrome|BL/CO

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

1062-4821

Country of Publication

UNITED STATES

CAS Registry/EC NumberEC 1.1.1.88 (Hydroxymethylglutaryl CoA Reductases)

Section C

Record 17 from database: **MEDLINE**[Go To The Top](#)**Title**

Variability in cholesterol content and physical properties of lipoproteins containing apolipoprotein B-100.

Author

Abate N; Vega GL; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center at Dallas 75235.

Source

Atherosclerosis, 1993 Dec, 104:1-2, 159-71

Abstract

The primary objective of this study was to determine the variability in cholesterol carrying capacity of low density lipoproteins (LDLs) and other apolipoprotein B (apo B)-containing lipoproteins in normolipidemic men. One hundred and fifty-nine normolipidemic men, ages 21 to 73 years, were enrolled. In addition to determining plasma lipids and lipoproteins, three primary measurements were made: ratios of cholesterol to apo B in LDL; the electrophoretic pattern of LDL, i.e. pattern A, AB, or B; and levels of cholesterol in all lipoproteins other than high density lipoproteins (nonHDL-cholesterol) along with total apo B. First, the data revealed that about 85% of the variability of LDL-cholesterol levels can be accounted for by LDL-apo B levels, whereas the remaining 15% can be explained by differences in LDL-cholesterol/apo B ratios. Second, LDL electrophoretic pattern A was the predominant pattern in young adult men, but in older men the pattern shifted increasingly to AB and B. And third, there was a high correlation between nonHDL-cholesterol levels and total apo B levels, which suggests that nonHDL-cholesterol can be used as a relatively accurate surrogate for total apo B levels in normolipidemic individuals.

Language of Publication

English

Unique Identifier94190340

MeSH Heading (Major)

Apolipoproteins B|*AN; Cholesterol|*AN; Lipoproteins|*CH

MeSH Heading

Karl Loren

<http://www.oralchelation.com/technical/grundy1.htm>

Adult; Aged; Electrophoresis, Polyacrylamide Gel; Human; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0021-9150

Country of Publication

IRELAND

CAS Registry/EC Number

0 (apolipoprotein B-100); 0 (Apolipoproteins B); 0 (Lipoproteins); 57-88-5 (Cholesterol)

Section C

Record 18 from database: **MEDLINE**[Go To The Top](#)**Title**

Xanthogranulomatosis in an adult: lipid analysis of xanthomas and plasma.

Author

Garvey WT; Grundy SM; Eckel R

Address**Source**

J Am Acad Dermatol, 1987 Jan, 16:1 Pt 2, 183-7

Abstract

Xanthomatosis in the absence of hyperlipidemia is unusual but has been associated with compositional abnormalities of lipoprotein particles. An adult who developed juvenile xanthogranulomatosis in association with oral contraceptive ingestion is reported. Plasma lipids and lipoprotein electrophoresis were normal, as in a few other patients reported with this disorder. However, analysis of cutaneous xanthoma and plasma by thin-layer and gas-liquid chromatography revealed that cholesterol was the principal lipid in xanthoma and that there were no unusual sterols in plasma or tissue. Possible mechanisms of xanthoma formation are discussed. Thus juvenile xanthogranulomatosis should be considered in adults with normolipemic xanthomatosis.

Language of Publication

English

Unique Identifier

87138531

MeSH Heading (Major)

Lipids*AN; Xanthogranuloma, Juvenile|CI/*ME/PA

MeSH Heading

Adult; Biopsy; Case Report; Cholestanols|AN; Cholesterol|AN; Contraceptives, Oral, Synthetic|AE; Female; Human; Sitosterols|AN; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0190-9622

Country of Publication

UNITED STATES

<http://www.oralchelation.com/technical/grundy1.htm>**CAS Registry/EC Number**

0 (Cholestanols); 0 (Contraceptives, Oral, Synthetic); 0 (Sitosterols); 57-88-5 (Cholesterol); 5779-62-4 (sitosterol)

Section CRecord 19 from database: **MEDLINE**[Go To The Top](#)**Title**

Long-term changes in cholesterol biosynthesis and the effect of plasmapheresis therapy in a hypercholesterolemia homozygote.

Author

Levy RA; Ostlund RE Jr; Goldberg AC; Grundy SM

Address**Source**

Metabolism, 1986 May, 35:5, 415-8

Abstract

Synthesis of cholesterol was measured in a familial hypercholesterolemia homozygote on four occasions from age 1.1 to 9.9 years by the sterol balance technique. Both the fecal neutral steroid and fecal bile acid components of sterol balance were elevated initially. Over the decade of study, neutral steroid excretion/kg declined 61% whereas bile acid excretion/kg was unchanged. Chronic plasmapheresis therapy every two weeks for 3.4 years reduced plasma low-density lipoprotein cholesterol 54% but had little effect on the rate of cholesterol biosynthesis.

Language of Publication

English

Unique Identifier

86202736

MeSH Heading (Major)

Cholesterol|*BI/BL; Hypercholesterolemia, Familial|BL/ME/*TH; Plasmapheresis|*

MeSH Heading

Bile Acids and Salts|BI; Case Report; Child; Feces|AN; Follow-Up Studies; Homozygote; Human; Long-Term Care; Male; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0026-0495

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Bile Acids and Salts); 57-88-5 (Cholesterol)

Section C

[Go To The Top](#)

Title
Clofibrate, caloric restriction, supersaturation of bile, and cholesterol crystals.

Author
Kesäniemi YA; Grundy SM

Address

Source
Scand J Gastroenterol, 1983 Oct, 18:7, 897-902

Abstract
Lipid composition, cholesterol saturation, and cholesterol crystal formation of gallbladder bile were studied in seven type-IV hyperlipoproteinemic subjects who did not have gallstones. Thereafter, biliary cholesterol solubilization was overloaded, first by clofibrate and then by caloric restriction treatment. Initially increased cholesterol saturation was still increased by both clofibrate and caloric restriction treatment, but none of the subjects developed cholesterol crystals in bile, indicating that they had a mechanism to maintain cholesterol in solution in the bile despite remarkable supersaturation. This suggests that the patients who are at risk of developing gallstones can be better selected by cholesterol crystal analysis of bile samples than by analysis of lipid composition of bile.

Language of Publication
English

Unique Identifier
84223750

MeSH Heading (Major)
Bile Acids and Salts|*AN; Cholesterol|*PH; Clofibrate|AN/*TU; Energy Intake|*; Hyperlipoproteinemia Type IV|*PP/TH

MeSH Heading
Aged; Crystallization; Female; Human; Lipids|AN; Male; Middle Age; Solubility; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type
JOURNAL ARTICLE

ISSN
0036-5521

Country of Publication
NORWAY

CAS Registry/EC Number
0 (Bile Acids and Salts); 57-88-5 (Cholesterol); 637-07-0 (Clofibrate)

HealthGate Document

Section D

Record 1 from database: MEDLINE
[Go To The Top](#)

Title

Influence of weight reduction on plasma lipoproteins in obese patients.

Author

Wolf RN; Grundy SM

Address

Source

Arteriosclerosis, 1983 Mar-Apr, 3:2, 160-9

Abstract

To determine the influence of weight reduction on plasma lipoproteins, studies were carried out in 15 nondiabetic patients of varying degrees of obesity and four obese insulin-dependent diabetics. All studies were carried out on a metabolic ward and patients underwent three dietary periods: Period I, 4 to 5 weeks of weight maintenance in the obese state; Period II, caloric restriction to 1000 kcal/day to a weight loss of within 10% of ideal body weight; and Period III, again weight maintenance for 4 to 5 weeks near ideal body weight. Similar results were obtained for both nondiabetics and diabetics. Many patients had mildly elevated plasma triglycerides in Period I; they fell to the normal range in Period II and remained low in Period III. Total cholesterol levels decreased early in Period II, but levels began to rise near the end of caloric restriction, and in Period III, they were similar to Period I. Low density lipoprotein cholesterol levels followed a pattern similar to that of total cholesterol. High density lipoprotein cholesterol was relatively low in Period I (38 +/- 2 mg/dl +/- SEM); throughout weight loss, levels tended to rise, and in Period III, the average high density lipoprotein cholesterol was significantly higher (46 +/- 2 mg/dl).

Language of Publication

English

Unique Identifier

83177697

MeSH Heading (Major)

Body Weight|*; Lipoproteins|*ME; Obesity|BL/CO/*ME

MeSH Heading

Adult; Cholesterol|BL; Diabetes Mellitus|CO; Diet, Reducing; Energy Intake; Female; Human; Hypertension|ET; Lipoproteins, HDL|BL; Lipoproteins, LDL|BL; Lipoproteins, VLDL|BL; Male; Middle Age; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (HDL-triglyceride); 0 (Lipoproteins); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, HDL); 0 (Lipoproteins, LDL Cholesterol); 0 (Lipoproteins, VLDL Cholesterol); 0 (Lipoproteins, VLDL); 0 (Triglycerides); 57-88-5 (Cholesterol)

Section D

Record 2 from database: **MEDLINE**

[Go To The Top](#)

Title

Very low density lipoprotein metabolism in non-ketotic diabetes mellitus: effect of dietary restriction.

Author

Ginsberg H; Grundy SM

Source

Diabetologia, 1982 Nov, 23:5, 421-5

Abstract

We have measured the turnover of very low density lipoprotein (VLDL) triglyceride as well as plasma glucose, insulin and non-esterified fatty acid levels in nine mildly obese non-ketotic, insulinopenic diabetic subjects before and during an energy restricted diet. During the baseline period, subjects were hypertriglyceridaemic, hyperglycaemic and insulinopenic. During dietary restriction (mean weight loss: 2.3 +/- 0.4 kg) plasma triglyceride fell from 8.4 +/- 3.0 to 3.4 +/- 0.89 mmol/l (mean +/- SEM: p less than 0.05), and plasma glucose fell from 13.9 +/- 1.7 to 9.8 +/- 1.4 mmol/l (p less than 0.01). Neither fasting plasma insulin nor the insulin response to an oral glucose load changed. Plasma non-esterified fatty acid concentrations remained constant as well. During the baseline period, the transport rate of VLDL-triglyceride in the diabetic subjects was more than twice that in an age-weighted matched control group (27.4 +/- 2.9 versus 12.1 +/- 0.8 mg/kg ideal body weight per h). The fractional catabolic rates were similar in the two groups (0.20 +/- 0.05 versus 0.21 +/- 0.02/h). During energy restriction of the diabetic subjects, the VLDL-triglyceride transport rate fell to 17.4 +/- 2.9 mg/kg ideal body weight per h (p less than 0.05 versus baseline) while the fractional catabolic rate remained constant at 0.21 +/- 0.06/h (NS versus baseline). These data indicate that the major abnormality in triglyceride metabolism in these non-ketotic, insulinopenic diabetic patients was over-production of VLDL-triglyceride.

Language of Publication

English

Unique Identifier

83080204

MeSH Heading (Major)

Diabetes Mellitus|*BL; Diet|*; Energy Intake|*; Lipoproteins, VLDL|*BL; Triglycerides|*BL

MeSH Heading

Aged; Blood Glucose|AN; Body Weight; Fatty Acids, Nonesterified|BL; Female; Human; Insulin|BL; Male; Middle Age; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0012-186X

Country of Publication

GERMANY, WEST

CAS Registry/EC Number

0 (very low density lipoprotein triglyceride); 0 (Blood Glucose); 0 (Fatty Acids, Nonesterified); 0 (Lipoproteins, VLDL); 0 (Triglycerides); 11061-68-0 (Insulin)

Record 3 from database: **MEDLINE**[Go To The Top](#)**Title**

Treatment of dyslipidemia in non-insulin-dependent diabetes mellitus with lovastatin.

Author

Garg A; Grundy SM

Address

Veterans Administration Medical Center, Dallas, Texas.

Source

Abstract

Coronary artery disease (CAD) is the leading cause of death among whites with non-insulin-dependent diabetes mellitus (NIDDM). Several risk factors--dyslipidemia induced by NIDDM, obesity, hypertension and hyperglycemia--likely contribute to accelerated atherosclerosis. The dyslipidemia in NIDDM is characterized by abnormalities in composition and metabolism of very low density lipoproteins, low-density lipoproteins (LDL) and high-density lipoproteins (HDL). However, because of the lack of long-term prospective epidemiologic studies, the relative importance of lipoprotein risk factors in the causation of CAD in diabetic patients is not clear. The World Health Organization Multinational Study of vascular disease in diabetics observed increased prevalence of CAD in diabetic populations with relatively high levels of plasma cholesterol and supports the concept that lowering cholesterol levels may significantly reduce coronary risk in NIDDM. To determine the effectiveness of lovastatin, an inhibitor of HMG CoA reductase, for lowering cholesterol levels, 16 patients with NIDDM and mild to moderate increases in plasma cholesterol were given lovastatin (20 mg twice daily) in a randomized, double-blind, placebo-controlled manner for 4 weeks. Compared with the placebo, lovastatin reduced concentrations of total cholesterol (233 +/- 10 vs 172 +/- 7 mg/dl [standard error of the mean], p less than 0.001), LDL cholesterol (140 +/- 9 vs 101 +/- 6 mg/dl, p less than 0.001), and LDL apolipoprotein-B (108 +/- 16 vs 80 +/- 16 mg/dl, p less than 0.001). Plasma triglycerides and very low density lipoprotein cholesterol levels also decreased by 31 and 42%, respectively. Although HDL cholesterol levels did not increase, the total cholesterol/HDL cholesterol ratio decreased significantly with lovastatin therapy. No adverse effects were noted and glycemic control was well-maintained.(ABSTRACT TRUNCATED AT 250 WORDS)

Language of Publication

English

Unique Identifier

89047169

MeSH Heading (Major)

Diabetes Mellitus, Non-Insulin-Dependent|*CO; Hydroxymethylglutaryl CoA Reductases|*AI;
Hypercholesterolemia|*DT/ET; Lovastatin|*TU

MeSH Heading

Clinical Trials; Comparative Study; Coronary Disease|PC; Double-Blind Method; Female; Human; Male;
Random Allocation; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0002-9149

Country of Publication

UNITED STATES

CAS Registry/EC Number

EC 1.1.1.88 (Hydroxymethylglutaryl CoA Reductases); 75330-75-5 (Lovastatin)

Record 4 from database: **MEDLINE**

[Go To The Top](#)

Title

Combination drug therapy for familial combined hyperlipidemia.

Author

East C; Bilheimer DW; Grundy SM

Address

Source

Ann Intern Med, 1988 Jul 1, 109:1, 25-32

Abstract

STUDY OBJECTIVE: To compare the efficacy of gemfibrozil and colestipol with gemfibrozil and lovastatin in patients with familial combined hyperlipidemia. **DESIGN:** A prospective, randomized trial. **SETTING:** An outpatient clinical research center in a tertiary care center. **PATIENTS:** Seventeen patients with familial combined hyperlipidemia documented by studies of first-degree relatives; nine patients with type 2b hyperlipoproteinemia, and eight patients with type 4 hyperlipoproteinemia. **INTERVENTIONS:** Baseline lipid, lipoprotein, and apolipoprotein levels were obtained during control periods on diet alone and on gemfibrozil therapy. Patients then received gemfibrozil and colestipol or gemfibrozil and lovastatin in a randomized order. **MEASUREMENTS AND MAIN RESULTS:** In patients with type 2b hyperlipoproteinemia, gemfibrozil alone significantly reduced total cholesterol by 11%, and low density lipoprotein (LDL)-apolipoprotein B by 18%, did not change LDL-cholesterol, and raised high density lipoprotein (HDL)-cholesterol levels by 26%. Addition of either colestipol or lovastatin reduced LDL-cholesterol levels by 17% and 25%, respectively, compared to gemfibrozil alone. However, colestipol mitigated the HDL-cholesterol raising effect of gemfibrozil and did not further reduce LDL-apolipoprotein B levels. In contrast, addition of lovastatin caused an additional reduction of LDL-apolipoprotein B 19% compared with gemfibrozil alone. In patients with type 4 hyperlipoproteinemia, gemfibrozil alone reduced triglycerides by 40%, raised HDL-cholesterol by 26%, and increased LDL-cholesterol levels by 29%. The addition of either colestipol or lovastatin reduced LDL-cholesterol levels by 34% and 33%, respectively (compared with gemfibrozil alone), but greater reductions of LDL-apolipoprotein B (30% with lovastatin compared with 15% with colestipol, compared with gemfibrozil alone), and increases in HDL-cholesterol levels (8% increase with lovastatin compared with 10% decrease with colestipol, compared to gemfibrozil alone) were seen with the lovastatin combination. **CONCLUSIONS:** Although gemfibrozil with either colestipol or lovastatin favorably altered lipoprotein levels in patients with hypertriglyceridemia and familial combined hyperlipidemia, the combination of gemfibrozil and lovastatin appeared superior overall.

Language of Publication

English

Unique Identifier

88239259

MeSH Heading (Major)

Antilipemic Agents|*TU; Colestipol|*TU; Hyperlipidemia, Familial Combined|BL/*DT; Lovastatin|*TU; Pentanoic Acids|*TU; Polyamines|*TU; Valerates|*TU

MeSH Heading

Adult; Cholelithiasis|DI; Comparative Study; Drug Therapy, Combination; Female; Human; Hypercholesterolemia, Familial|DT; Hyperlipoproteinemia Type IV|DT; Male; Middle Age; Random Allocation; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Ultrasonography

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0003-4819

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Antilipemic Agents); 0 (Pentanoic Acids); 0 (Polyamines); 0 (Valerates); 25812-30-0 (Gemfibrozil); 50925-79-6 (Colestipol); 75330-75-5 (Lovastatin)

[Go To The Top](#)**Title**

Effects of lovastatin on the levels, structure, and atherogenicity of VLDL in patients with moderate hypertriglyceridemia.

Author

Gianturco SH; Bradley WA; Nozaki S; Vega GL; Grundy SM

Address

University of Alabama, Birmingham 35294-0012.

Source

Arterioscler Thromb, 1993 Apr, 13:4, 472-81

Abstract

The purpose of this study was to determine whether lovastatin treatment reduced very low density lipoprotein (VLDL) abnormalities in hypertriglyceridemic subjects. Lovastatin reduced plasma triglyceride levels and the levels of total VLDL, intermediate density lipoprotein (IDL), and low density lipoprotein (LDL) cholesterol. The numbers of VLDL particles of Sf 100-400 and Sf 60-100 but not Sf 20-60 particles were reduced by lovastatin, as was the amount of cholesteryl ester per particle. All VLDL subspecies bound to the LDL receptor of cultured human fibroblasts with similar, high affinities on both placebo and lovastatin, but VLDL Sf 100-400 and VLDL Sf 60-100 caused less suppression of 3-hydroxy-3-methyl glutaryl coenzyme A reductase activity after lovastatin therapy, indicating reduced LDL receptor-mediated cholesterol delivery. The average decrease in reductase suppression by VLDL Sf 100-400 after lovastatin was 32%, similar to the 34% average decrease in cholesteryl ester content of VLDL Sf 100-400 after lovastatin. Although statistical significance was not achieved, there was a trend toward decreased VLDL Sf 100-400-induced rapid, receptor-mediated triglyceride accumulation in P388D1 macrophages after lovastatin. Taken together, these observations suggest that lovastatin may be of potential benefit in decreasing the atherosclerotic complications of hypertriglyceridemia.

Language of Publication

English

Unique Identifier

93222123

MeSH Heading (Major)

Atherosclerosis|*ET; Hypertriglyceridemia|CO/*DT; Lipoproteins, VLDL|*BL/ME; Lovastatin|*TU

MeSH Heading

Adult; Aged; Cholesterol|BL; Human; Lipids|BL; Lipoproteins|BL/ME; Male; Middle Age; Receptors, Cell Surface|ME; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

CLINICAL TRIAL; CONTROLLED CLINICAL TRIAL; JOURNAL ARTICLE

ISSN

1049-8834

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (lipoproteins, IDL); 0 (Lipids); 0 (Lipoproteins); 0 (Lipoproteins, VLDL); 0 (Receptors, Cell Surface); 0 (Receptors, Lipoprotein); 0 (Triglycerides); 57-88-5 (Cholesterol); 75330-75-5 (Lovastatin)

[Go To The Top](#)

Title

Activities of lipoprotein lipase and hepatic triglyceride lipase in postheparin plasma of patients with low concentrations of HDL cholesterol.

Author

Blades B; Vega GL; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

Arterioscler Thromb, 1993 Aug, 13:8, 1227-35

Abstract

Previous investigations have shown that abnormalities in the postheparin plasma levels of the lipolytic enzymes, lipoprotein lipase (LPL) and hepatic triglyceride lipase (HTGL), are correlated with variations in plasma high-density lipoprotein cholesterol (HDL-C) levels. The present study was performed to determine correlations between the postheparin plasma activities of these two enzymes and HDL levels in a sizable number of subjects with low HDL-C levels. Two types of low-HDL subjects were investigated: 159 male subjects with low HDL-C (< 40 mg/dL) and normal triglyceride (< 250 mg/dL) levels (the low-HDL group) and 80 male subjects with low HDL-C (< 40 mg/dL) and elevated triglyceride (> or = 250 mg/dL) levels (the low-HDL/high-TG group). Postheparin plasma activities of LPL and HTGL were determined in these two groups, and these levels were compared with those obtained from 51 normolipidemic (normal-HDL) male subjects. Postheparin LPL activities were significantly lower in the low-HDL and low-HDL/high-TG groups (mean +/- SD, 9.9 +/- 2.9 and 10.4 +/- 3.0 mmol/h per liter, respectively; P < .001 for both) compared with the normal-HDL group (12.5 +/- 3.7 mmol/h per liter). Conversely, postheparin HTGL activities were significantly higher in the low-HDL and low-HDL/high-TG groups (39.3 +/- 16.2 and 44.4 +/- 16.7 mmol/h per liter, respectively; P < .001 for both) compared with the normal-HDL group (29.7 +/- 11.3 mmol/h per liter). Consequently, mean LPL/HTGL ratios were markedly lower in the two low-HDL groups compared with the normal-HDL group.(ABSTRACT TRUNCATED AT 250 WORDS)

Language of Publication

English

Unique Identifier

93344371

MeSH Heading (Major)

Heparin|*PD; Lipase|*BL; Lipoprotein Lipase|*BL; Lipoproteins, HDL Cholesterol|*BL; Liver|*EN

MeSH Heading

Aged; Human; Hypertriglyceridemia|BL; Male; Middle Age; Osmolar Concentration; Reference Values; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

1049-8834

Country of Publication

UNITED STATES

CAS Registry/EC Number

EC 3.1.1.3 (Lipase); EC 3.1.1.34 (Lipoprotein Lipase); 0 (Lipoproteins, HDL Cholesterol); 0 (Triglycerides); 9005-49-6 (Heparin)

Title

Association between a specific apolipoprotein B mutation and familial defective apolipoprotein B-100.

Author

Soria LF; Ludwig EH; Clarke HR; Vega GL; Grundy SM; McCarthy BJ

Address

Gladstone Foundation Laboratories for Cardiovascular Disease, University of California, San Francisco 94140-0608.

Source

Proc Natl Acad Sci U S A, 1989 Jan, 86:2, 587-91

Abstract

Familial defective apolipoprotein (apo) B-100 is a genetic disease that leads to hypercholesterolemia and to an increased serum concentration of low density lipoproteins that bind defectively to the apoB,E(LDL) receptor. The disorder appears to result from a mutation in the gene for apoB-100. Extensive sequence analysis of the two alleles of one subject heterozygous for the disorder has revealed a previously unreported mutation in the codon for amino acid 3500 that results in the substitution of glutamine for arginine. This same mutant allele occurs in six other, unrelated subjects and in eight affected relatives in two of these families. A partial haplotype of this mutant apoB-100 allele was constructed by sequence analysis and restriction enzyme digestion at positions where variations in the apoB-100 are known to occur. This haplotype is the same in three probands and four affected members of one family and lacks a polymorphic Xba I site whose presence has been correlated with high cholesterol levels. Thus, it appears that the mutation in the codon for amino acid 3500 (CGG----CAG), a CG mutational "hot spot," defines a minor apoB-100 allele associated with defective low density lipoproteins and hypercholesterolemia.

Language of Publication

English

Unique Identifier

89098975; GENBANK/M14162

MeSH Heading (Major)

Apolipoproteins B|*GE; Hypercholesterolemia, Familial|*GE

MeSH Heading

Alleles; Amino Acid Sequence; Base Sequence; Cloning, Molecular; DNA|GE; Genetic Vectors; Genotype; Haplotypes; Human; Lipoproteins, LDL|ME; Molecular Sequence Data; Mutation; Pedigree; Polymorphism, Restriction Fragment Length; Receptors, LDL|ME; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0027-8424

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoproteins B); 0 (Receptors, LDL); 9007-49-2 (DNA)

Record 8 from database: **MEDLINE**

[Go To The Top](#)

Title

Hyperlipoproteinemia: metabolic basis and rationale for therapy.

Author Karl Loren

<http://www.oralchelation.com/technical/grundy1.htm>

Grundy SM

Address

Source

Am J Cardiol, 1984 Aug 27, 54:5, 20C-26C

Abstract

Most forms of hyperlipoproteinemia are the result of at least 1 to 4 basic defects of lipoprotein metabolism. Hypercholesterolemia is most commonly due to decreased activity of receptors for low-density lipoproteins (LDL). A deficiency of LDL receptors can be caused by either a genetic defect in the structure of the receptor or metabolic suppression of receptor synthesis by genetic factors or dietary saturated fatty acids and cholesterol. An elevation of triglycerides in chylomicrons or very low density lipoproteins (VLDL) can be secondary to a reduced activity of lipoprotein lipase, and an increase in the catabolic remnants of these lipoproteins can be due to an abnormal isoform of apolipoprotein E, the apolipoprotein that mediates hepatic uptake of lipoprotein remnants. Finally, hepatic overproduction of VLDL can produce hypertriglyceridemia, or if there is a concomitant defect in clearance of lipoproteins, an accentuated increase of VLDL, remnants or LDL will occur. Thus, lipoprotein overproduction can give rise to multiple lipoprotein phenotypes in a single family. Specific therapy of hyperlipoproteinemia should be directed toward correcting these metabolic defects.

Language of Publication

English

Unique Identifier

84303892

MeSH Heading (Major)

Hyperlipoproteinemia|ET/*ME/TH

MeSH Heading

Chylomicrons|ME; Human; Hypercholesterolemia|ME; Intestines|ME; Lipoproteins, HDL|ME; Lipoproteins, LDL|ME; Lipoproteins, VLDL|ME; Liver|ME; Receptors, Cell Surface|ME; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0002-9149

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Chylomicrons); 0 (Lipoproteins, HDL); 0 (Lipoproteins, VLDL); 0 (Receptors, Cell Surface); 0 (Receptors, LDL); 0 (Triglycerides)

Record 9 from database: **MEDLINE**

Title

Excess body weight. An underrecognized contributor to high blood cholesterol levels in white American men [see comments]

Author

Denke MA; Sempos CT; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas.

Source

Arch Intern Med, 1993 May 10, 153:9, 1093-103

BACKGROUND: The influence of body weight on serum lipids is often overlooked in clinical practice. **METHODS:** The association between body weight adjusted for height as calculated by body-mass index (BMI) and serum lipid and lipoprotein levels in white men was examined using the second National Health and Nutrition Examination Survey (NHANES II). Lipid results were categorized into six different levels of BMI: (1) 21.0 kg/m² or lower, (2) 21.1 to 23.0 kg/m², (3) 23.1 to 25.0 kg/m², (4) 25.1 to 27.0 kg/m², (5) 27.1 to 30.0 kg/m², and (6) greater than 30.0 kg/m², and three age groups: (1) young men (20 through 44 years), (2) middle-aged men (45 through 59 years), and (3) older men (60 through 74 years). **RESULTS:** Using linear trend analysis, changes in BMI from categories 2 to 5 in young men were associated with a total cholesterol level 0.59 mmol/L (23 mg/dL) higher (P < .01), a non-high-density lipoprotein (non-HDL) cholesterol level 0.70 mmol/L (27 mg/dL) higher (P < .01), and a low-density lipoprotein (LDL) cholesterol level 0.59 mmol/L (23 mg/dL) higher (P = .03). For middle-aged men and older men, the same change in BMI was associated with smaller but still significant differences in total cholesterol levels (higher by 0.31 mmol/L [12 mg/dL] [P < .01] and 0.28 mmol/L [11 mg/dL] [P < .01], respectively) and non-HDL cholesterol levels (higher by 0.37 mmol/L [14 mg/dL] [P < .01] and 0.25 mmol/L [10 mg/dL] [P < .01], respectively), whereas the LDL cholesterol levels were unchanged. Although advancing age may blunt the BMI-associated differences in total and LDL cholesterol levels, the BMI-associated differences in triglyceride levels (higher by 0.70 to 1.33 mmol/L [62 to 118 mg/dL] [P < .001]) and HDL cholesterol levels (lower by 0.18 to 0.39 mmol/L [7 to 15 mg/dL] [P < .001]) were of similar magnitude in all age groups. **CONCLUSION:** Excess body weight is associated with deleterious changes in the lipoprotein profile. Higher BMI was associated at all ages with higher plasma triglyceride level, lower HDL cholesterol level, and higher total and non-HDL cholesterol levels. In young men, the higher total cholesterol level was reflected mainly in the LDL cholesterol level; in middle-aged and older men, in the non-HDL fraction. Programs to reduce coronary heart disease by improving lipid levels should include more emphasis on achieving and maintaining ideal body weight.

Language of Publication

English

Unique Identifier

93243849

MeSH Heading (Major)

Body Weight[*]; Cholesterol[*]BL; Hypercholesterolemia|BL/*ET; Obesity|BL/*CO/PP

MeSH Heading

Adult; Aged; Apolipoproteins B|BL; Diet; Health Surveys; Human; Lipoproteins, HDL Cholesterol|BL; Lipoproteins, LDL Cholesterol|BL; Male; Middle Age; Nutrition Surveys; Support, U.S. Gov't, P.H.S.; Triglycerides|BL; United States; Whites

Publication Type

JOURNAL ARTICLE

ISSN

0003-9926

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoproteins B); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, LDL Cholesterol); 0 (Triglycerides); 57-88-5 (Cholesterol)

Record 10 from database: **MEDLINE**[Go To The Top](#)

Title Karl Loren <http://www.oralchelation.com/technical/grundy1.htm>
Oxidized LDL and atherogenesis: relation to risk factors for coronary heart disease.

Author
Grundy SM

Address
Center for Human Nutrition, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source
Clin Cardiol, 1993 Apr, 16:4 Suppl 1, I3-5

Abstract
According to a new theory, a critical step in atherogenesis is oxidation of low-density lipoprotein (LDL) within the arterial wall. Direct data supporting this theory are limited, but indirect evidence suggests that oxidized LDL plays a role in atherogenesis. An important question is whether the LDL-oxidation hypothesis conforms to what is known about other risk factors for coronary heart disease (CHD), such as hypertension, smoking, low high-density lipoprotein (HDL) levels, and diabetes mellitus. Perhaps a unified theory of atherogenesis could be formulated if these risk factors exert their atherogenic actions in part by promoting, facilitating, or permitting the oxidation of LDL.

Language of Publication
English

Unique Identifier
93230723

MeSH Heading (Major)

Coronary Arteriosclerosis|*BL; Lipid Peroxidation|*PH; Lipoproteins, LDL|*BL

MeSH Heading

Cholesterol|BL; Human; Hypertriglyceridemia|BL; Lipoproteins, HDL|BL; Receptors, LDL|PH; Risk Factors; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0160-9289

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Lipoproteins, HDL); 0 (Lipoproteins, LDL); 0 (Receptors, LDL); 57-88-5 (Cholesterol)

Record 11 from database: **MEDLINE**

[Go To The Top](#)

Title

Four new mutations in the apolipoprotein B gene causing hypobetalipoproteinemia, including two different frameshift mutations that yield truncated apolipoprotein B proteins of identical length.

Author
Young SG; Pullinger CR; Zysow BR; Hofmann-Radvani H; Linton MF; Farese RV Jr; Terdiman JF; Snyder SM; Grundy SM; Vega GL; et; al

Address
Gladstone Institute for Cardiovascular Disease, San Francisco, CA 94141-9100.

Source
J Lipid Res, 1993 Mar, 34:3, 501-7

Familial hypobetalipoproteinemia can be caused by mutations in the apolipoprotein (apo)B gene that interfere with the translation of a full-length apoB molecule. Frequently, a truncated apoB molecule can be detected in the plasma lipoproteins of affected subjects. In this report, we characterize four different apoB gene mutations causing hypobetalipoproteinemia that are associated with the synthesis of truncated apoB proteins. Two of the mutations are nonsense mutations caused by single nucleotide substitutions; these mutations are associated with the production of apoB-32.5 (1473 amino acids) and apoB-82 (3733 amino acids). The other two mutations are single nucleotide deletions (of apoB cDNA nucleotides 7295 and 7359, respectively). The altered reading frames created by these different frameshift mutations terminated with the same stop codon, and both therefore yielded a truncated protein of identical size: apoB-52.8 (2395 amino acids). The two apoB-52.8 proteins differ, however, in the number of novel carboxyl-terminal amino acids introduced by the frameshift. The buoyant density of lipoproteins containing the truncated apoBs was inversely related to the length of the truncated apoB. ApoB-32.5 was present only in high density lipoproteins (HDL) and the $d > 1.21$ g/ml fraction, whereas apoB-82 was present almost exclusively in very low density lipoproteins (VLDL). ApoB-52.8 was present primarily in VLDL, intermediate density lipoproteins (IDL), and low density lipoproteins (LDL); trace amounts were observed in the HDL.

Language of Publication

English

Unique Identifier

93224835

MeSH Heading (Major)

Apolipoproteins B|BL/*GE; Frameshift Mutation|*; Hypobetalipoproteinemia|BL/*GE; Mutation|*

MeSH Heading

Adult; Aged; Base Sequence; Cholesterol|BL; Female; Human; Lipoproteins, VLDL|BL; Male; Middle Age; Molecular Sequence Data; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0022-2275

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoproteins B); 0 (Lipoproteins, VLDL); 57-88-5 (Cholesterol)

Record 12 from database: **MEDLINE**[Go To The Top](#)**Title**

Two patterns of LDL metabolism in normotriglyceridemic patients with hypoalphalipoproteinemia.

Author

Vega GL; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

Arterioscler Thromb, 1993 Apr, 13:4, 579-89

Abstract

The objective of this study was to determine whether normotriglyceridemic patients with low levels of high density lipoprotein (HDL) cholesterol have concomitant defects in the metabolism of low density lipoproteins (LDLs). To address this question, measurements of turnover rates of apolipoprotein A-I (apo A-I) and LDL apolipoprotein B (apo B) were made in 36 middle-aged men with low HDL cholesterol (< 40 mg/dL), normal triglyceride (< 250 mg/dL), and normal total cholesterol (< or = 90th percentile) levels. Similar measurements were made in eight hypertriglyceridemic men having low HDL levels. For control, turnover rates of LDL apo B were measured in 24 healthy, normolipidemic men, and apo A-I kinetics were determined in 20 other healthy men with normal HDL cholesterol levels. In all patients with low HDL levels, fractional catabolic rates (FCRs) for apo A-I were increased compared with control subjects; in contrast, input rates for apo A-I in low-HDL patients were similar to control. Hypertriglyceridemic patients had significantly higher FCRs for LDL (0.463 +/- 0.040 pool/day, [mean +/- SEM]) than control subjects (0.328 +/- 0.008 pool/day, p < 0.001). In normolipidemic patients having low HDL, a bimodal pattern of LDL-apo B kinetics was observed. For 23 low-HDL patients, FCRs for LDL apo B averaged 0.450 +/- 0.017 pool/day and were significantly higher than control values. Additionally, in these patients, levels of very low density lipoprotein plus intermediate density lipoprotein (VLDL+IDL) cholesterol and VLDL+IDL apo B were higher than in control subjects (54 +/- 3 versus 32 +/- 3 mg/dL and 25 +/- 2 versus 18 +/- 1 mg/dL, respectively). The remaining 13 low-HDL patients had lower and essentially normal FCRs for LDL (0.300 +/- 0.009 pool/day); these patients also had relatively low levels of cholesterol and apo B in VLDL+IDL. Thus, two patterns of LDL kinetics were present in normotriglyceridemic patients with low HDL levels. One pattern was indistinguishable from that typically present in patients with hypertriglyceridemia, whereas the other was similar to normal control subjects. These two patterns of LDL-apo B kinetics may reflect different mechanisms for the causation of low HDL cholesterol concentrations.

Language of Publication

English

Unique Identifier

93222137

MeSH Heading (Major)

Hypolipoproteinemia*BL; Lipoproteins, HDL*BL; Lipoproteins, LDL*BL; Triglycerides*BL

MeSH Heading

Adult; Aged; Apolipoprotein A-I|AN; Apolipoproteins B|BL; Human; Hypertriglyceridemia|BL; Kinetics; Lipoproteins, HDL Cholesterol|BL; Male; Middle Age; Reference Values; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

1049-8834

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Apolipoprotein A-I); 0 (Apolipoproteins B); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, HDL); 0 (Lipoproteins, LDL); 0 (Triglycerides)

Record 13 from database: **MEDLINE**

[Go To The Top](#)

Title

Rationale and management of hyperlipidemia of the nephrotic syndrome.

Author

Grundy SM; Vega GL

Address Karl Loren

<http://www.oralchelation.com/technical/grundy1.htm>

Department of Clinical Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

Am J Med, 1989 Nov, 87:5N, 3N-11N

Abstract

Hyperlipidemia is usually present in patients with the nephrotic syndrome. The most common lipid abnormality is hypercholesterolemia, although as the disorder progresses, hypertriglyceridemia may develop. Elevated plasma lipids have two potential vascular consequences, namely, atherosclerosis and progression of renal failure. Neither of these complications has been proven with certainty, but there is growing evidence to indicate that both may be long-term consequences of the nephrotic syndrome. Therefore, effective therapy of hyperlipidemia, particularly elevated cholesterol levels, is needed as a protection against these complications. Since nephrotic hypercholesterolemia frequently is severe, dietary therapy, although a valuable adjunct, will not normalize cholesterol levels in most nephrotic patients. Thus, if effective serum cholesterol lowering is to be achieved, drug therapy will be required. Bile acid-binding resins have been shown to lower cholesterol levels in nephrotic patients, but the decline in cholesterol concentrations is usually insufficient to produce a marked reduction in coronary risk. Nicotinic acid theoretically should be useful for treatment of nephrotic hyperlipidemia, but it has not been adequately tested. The new drugs that inhibit cholesterol synthesis, e.g., lovastatin, appear to be highly promising for treating elevations of both serum cholesterol and triglycerides in the nephrotic syndrome. However, testing of these drugs in this condition has been limited, and the possibility of significant side effects in an appreciable portion of patients has not been ruled out. Of particular concern is the development of severe myopathy that can produce myoglobinuria and acute renal failure. This side effect is relatively rare in patients without the nephrotic syndrome, but its prevalence in the latter condition has not been determined. The fibric acids will lower triglyceride levels in nephrotic patients, but they are not effective in lowering cholesterol levels; consequently, they probably have little role in the treatment of nephrotic hypercholesterolemia. Finally, the drug probucol will lower cholesterol levels in nephrotic patients, although not to desirable levels; still, probucol could prove useful in combination with other cholesterol-lowering drugs.

Language of Publication

English

Unique Identifier

91090140

MeSH Heading (Major)

Hyperlipidemia[*DT/ET/ME; Nephrotic Syndrome]*CO/ME

MeSH Heading

Human

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0002-9343

Country of Publication

UNITED STATES

Record 14 from database: **MEDLINE**

[Go To The Top](#)

Title

Food safety and health effects of canola oil.

Author

Dupont J, White PJ; Johnston KM; Hegger IA; McDonald BE; Grundy SM; Bonanome A

Address

Department of Food and Nutrition, Iowa State University, Ames.

Source

J Am Coll Nutr, 1989 Oct, 8:5, 360-75

Abstract

Canola oil is a newly marketed vegetable oil for use in salads and for cooking that contains 55% of the monounsaturated fatty acid; oleic acid, 25% linoleic acid and 10% alpha-linolenate [polyunsaturated fatty acid (PUFA)], and only 4% of the saturated fatty acids (SFAs) that have been implicated as factors in hypercholesterolemia. It is expressed from a cultivar of rapeseed that was selectively bred from old varieties in Canada to be very low in erucic acid--a fatty acid suspected to have pathogenic potential in diets high in the original rapeseed oil in experimental animals. Canola oil is free of those problems. It is the most widely consumed food oil in Canada, and has been approved for Generally Recognized as Safe (GRAS) status by the Food and Drug Administration (FDA) of the United States Department of Health and Human Services. The fatty acid composition of canola oil is consistent with its use as a substitute for SFAs, in meeting the dietary goals recommended by many health associations: an average diet containing about 30% of calories as fat made up of less than 10% SFAs, 8-10% PUFAs in a ratio of linoleic to linolenic acids between 4:1 and 10:1, the remainder being monounsaturated fatty acids. No single oil meets these current recommendations for ratios of PUFA/monounsaturated/polyunsaturated fatty acid ratios as the sole source of cooking and salad oil.

Language of Publication

English

Unique Identifier

90110761

MeSH Heading (Major)

Dietary Fats|AE/*AN; Fatty Acids|*AN; Fatty Acids, Unsaturated|*AN; Plant Oils|AE/*AN

MeSH Heading

Consumer Product Safety; Human

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0731-5724

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Dietary Fats); 0 (Fatty Acids); 0 (Fatty Acids, Unsaturated); 0 (Plant Oils)

Record 15 from database: **MEDLINE**

[Go To The Top](#)

Title

Monounsaturated fatty acids and cholesterol metabolism: implications for dietary recommendations [see comments]

Author

Grundy SM

Address

Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas 75235.

Source

J Nutr, 1989 Apr, 119:4, 529-33

Dietary fat is known to affect serum concentrations of total and lipoprotein cholesterol. However, all components of dietary triglycerides--saturated, monounsaturated, and polyunsaturated fatty acids--do not have identical effects on serum cholesterol levels. Until recently, most attention has been given to saturated fatty acids, which raise cholesterol levels, and polyunsaturated fatty acids, which are thought by many to lower cholesterol levels. Monounsaturates in contrast have been given little attention. However, recent studies carried out in our laboratory and in others have shown that monounsaturates can have favorable effects when substituted for saturated fatty acids in the diet. In this exchange, the monounsaturates reduce low density lipoprotein (LDL) cholesterol levels, but do not lower high density lipoprotein (HDL) cholesterol levels. In contrast, an HDL-lowering action has been noted for polyunsaturates. Also, monounsaturates appear to alter lipoproteins more favorably than carbohydrates, which can raise triglycerides and lower HDL cholesterol levels. Therefore, monounsaturated fatty acids appear to have more potential for use in cholesterol-lowering diets than previously recognized.

Language of Publication

English

Unique Identifier

89199115

MeSH Heading (Major)

Cholesterol*BL; Dietary Fats|AD/*PD; Fatty Acids, Monounsaturated*PD

MeSH Heading

Comparative Study; Dietary Carbohydrates|PD; Fatty Acids, Unsaturated|PD; Human; Lipids|BL; Lipoproteins|BL

Publication Type

JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

ISSN

0022-3166

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Dietary Fats); 0 (Fatty Acids, Monounsaturated); 0 (Fatty Acids, Unsaturated); 0 (Lipoproteins); 57-88-5 (Cholesterol)

Record 16 from database: **MEDLINE**[Go To The Top](#)**Title**

Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins [see comments]

Author

Denke MA; Grundy SM

Address

Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas.

Source

Am J Clin Nutr, 1992 Nov, 56:5, 895-8

Abstract

The effects of lauric acid (C12:0) on plasma lipids and lipoproteins were compared with the effects of palmitic acid (C16:0) and oleic acid (C18:1) in a metabolic-diet study of 14 men by using liquid-formula diets fed for 3 wk each in random order. Lauric acid was supplied in a synthetic high-lauric oil, palmitic acid was provided by

<http://www.cholesterol.com/Health/Grain1.htm>
palm oil and oleic acid in oleic-rich sunflower seed oil. The high lauric oil resulted in higher concentrations of plasma total cholesterol (4.94 +/- 0.75 mmol/L [mean +/- SE]) and LDL cholesterol (3.70 +/- 0.57 mmol/L) when compared with high-oleic sunflower oil (4.44 +/- 0.54 and 3.31 +/- 0.44 mmol/L, respectively), but did not raise total and LDL cholesterol concentrations as much as did palm oil (5.17 +/- 0.65 and 3.93 +/- 0.51 mmol/L, respectively). No differences were noted in plasma triglycerides or HDL cholesterol. Lauric acid raises total and LDL cholesterol concentrations compared with oleic acid, but is not as potent for increasing cholesterol concentrations as is palmitic acid.

Language of Publication

English

Unique Identifier

93035070

MeSH Heading (Major)

Dietary Fats|AD/*PD; Lauric Acids|AD/*PD; Lipids|*BL; Lipoproteins|*BL; Palmitic Acids|AD/*PD

MeSH Heading

Adult; Aged; Cholesterol|BL; Comparative Study; Human; Lipoproteins, HDL Cholesterol|BL; Lipoproteins, LDL Cholesterol|BL; Male; Middle Age; Oleic Acids|AD/PD; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Triglycerides|BL

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0002-9165

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Dietary Fats); 0 (Lauric Acids); 0 (Lipids); 0 (Lipoproteins); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, LDL Cholesterol); 0 (Oleic Acids); 0 (Palmitic Acids); 0 (Triglycerides); 112-80-1 (Oleic Acid); 143-07-7 (lauric acid); 57-10-3 (Palmitic Acid); 57-88-5 (Cholesterol)

Record 17 from database: **MEDLINE**

[Go To The Top](#)

Title

Comparison of effects of high and low carbohydrate diets on plasma lipoproteins and insulin sensitivity in patients with mild NIDDM.

Author

Garg A; Grundy SM; Unger RH

Address

Veterans Affairs Medical Center, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

Diabetes, 1992 Oct, 41:10, 1278-85

Abstract

Previous studies indicate that diets rich in digestible carbohydrates improve glucose tolerance in nondiabetic individuals, but may worsen glycemic control in NIDDM patients with moderately severe hyperglycemia. The effects of such high-carbohydrate diets on glucose metabolism in patients with mild NIDDM have not been studied adequately. This study compares responses to an isocaloric high-carbohydrate diet (60% of total energy from carbohydrates) and a low-carbohydrate diet (35% of total energy from carbohydrates) in 8 men with mild NIDDM. Both diets were low in saturated fatty acids, whereas the low-carbohydrate diet was rich in monounsaturated fatty acids. The two diets were matched for dietary fiber content (25 g/day). All patients were

randomly assigned to receive first one and then the other diet, each for a period of 21 days, in a metabolic ward. Compared with the low-carbohydrate diet, the high-carbohydrate diet caused a 27.5% increase in plasma triglycerides and a similar increase in VLDL-cholesterol levels; it also reduced levels of HDL cholesterol by 11%. Plasma glucose and insulin responses to identical standard breakfast meals were studied on days 4 and 21 of each period, and these did not differ significantly between the two diets. At the end of each period, a euglycemic hyperinsulinemic glucose clamp study with simultaneous infusion of [3-3H]glucose revealed no significant changes in hepatic insulin sensitivity; and peripheral insulin-mediated glucose disposal remained unchanged (14.7 +/- 1.4 vs. 16.5 +/- 2.3 microM.kg-1.min-1 on the high-carbohydrate and low-carbohydrate diets, respectively).(ABSTRACT TRUNCATED AT 250 WORDS)

Language of Publication

English

Unique Identifier

93012496

MeSH Heading (Major)

Diabetes Mellitus, Non-Insulin-Dependent|*BL; Dietary Carbohydrates|*PD; Insulin|*PD; Lipoproteins|*BL

MeSH Heading

Analysis of Variance; Blood Glucose|ME; Cholesterol|BL; Comparative Study; Energy Intake; Glucose Clamp Technique; Glycosuria; Hemoglobin A, Glycosylated|AN; Human; Insulin Infusion Systems; Male; Middle Age; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.; Time Factors; Triglycerides|BL

Publication Type

JOURNAL ARTICLE

ISSN

0012-1797

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Blood Glucose); 0 (Dietary Carbohydrates); 0 (Hemoglobin A, Glycosylated); 0 (Lipoproteins); 0 (Triglycerides); 11061-68-0 (Insulin); 57-88-5 (Cholesterol)

Record 18 from database: **MEDLINE**

[Go To The Top](#)

Title

The place of HDL in cholesterol management. A perspective from the National Cholesterol Educational Program [published erratum appears in Arch Intern Med 1989 Apr;149(4):940] [comment] [see comments]

Author

Grundy SM; Goodman DW; Rifkind BM; Cleeman JI

Address

National Cholesterol Education Program, National Heart, Lung, and Blood Institute, Bethesda, Md.

Source

Arch Intern Med, 1989 Mar, 149:3, 505-10

Abstract

The guidelines developed by the Adult Treatment Panel of the National Cholesterol Education Program identified low density lipoprotein (LDL) as the major atherogenic lipoprotein, and high levels of LDL-cholesterol as the primary target for cholesterol-lowering therapy. Low levels of high density lipoprotein (HDL)-cholesterol were recognized as a major risk factor for coronary heart disease. This report reexamines in depth the

recommendations of the Adult Treatment Panel on HDL cholesterol. Two major questions are discussed: (1) Should HDL-cholesterol levels be measured in all adults, as recommended for total cholesterol? (2) Should patients found to have a low serum HDL [corrected]-cholesterol level (less than 35 mg/dL [less than 0.91 mmol/L]) enter medical therapy to raise the level? The guidelines of the Adult Treatment Panel are reaffirmed as appropriate from the current perspective. These guidelines recommend that HDL-cholesterol levels be determined in patients deemed to be at high risk for coronary heart disease and suggest that HDL measurement is optional for individuals with borderline-high total levels. The guidelines of the Adult Treatment Panel recommend that low HDL-cholesterol levels be raised mainly by hygienic means (ie, smoking cessation, weight loss, aerobic exercise). When drug therapy is required for high LDL-cholesterol levels in the presence of low HDL levels, cholesterol-lowering drugs that concomitantly raise HDL should be given first priority.

Language of Publication

English

Unique Identifier

89149255

MeSH Heading (Major)

Cholesterol, Dietary*AD; Coronary Disease*PC; Lipoproteins, HDL Cholesterol*BL; Lipoproteins, LDL Cholesterol*BL

MeSH Heading

Human; Mass Screening; Risk Factors; United States

Publication Type

COMMENT; JOURNAL ARTICLE; REVIEW; REVIEW, MULTICASE

ISSN

0003-9926

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Cholesterol, Dietary); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, LDL Cholesterol)

Record 19 from database: **MEDLINE**

[Go To The Top](#)

Title

Effect of dietary supplementation with alpha-tocopherol on the oxidative modification of low density lipoprotein.

Author

Jialal I; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235-9052.

Source

J Lipid Res, 1992 Jun, 33:6, 899-906

Abstract

Much data has accumulated supporting a proatherogenic role for oxidized low density lipoprotein (Ox-LDL). Micronutrient antioxidants such as alpha-tocopherol, the principal lipid-soluble antioxidant, assume potential significance because levels can be manipulated by dietary measures without resulting in side effects. Co-incubation of LDL in vitro with alpha-tocopherol inhibits its oxidative modification. Hence the effect of dietary supplementation with alpha-tocopherol on the time course of copper-catalyzed oxidation of LDL was tested in a randomized placebo-controlled single-blind study. Two groups of 12 male subjects were given either placebo or alpha-tocopherol (800 IU/day) for a period of 12 weeks. Alpha-tocopherol therapy did not result in any side

effects or exert an adverse effect on the plasma lipid and lipoprotein profile. While the lipid standardized alpha-tocopherol levels were similar at baseline, the supplemented group had 3.3-fold and 4.4-fold higher levels compared to placebo at 6 and 12 weeks, respectively. In the 15 subjects in whom both plasma and LDL alpha-tocopherol levels were quantitated, there was a significant correlation ($r = 0.79$, P less than 0.0001). At baseline there were no significant differences in the time course curves of thiobarbituric acid-reacting substances (TBARS) activity or conjugated diene formation between the alpha-tocopherol and placebo groups. However, at both 6 and 12 weeks the mean levels of TBARS activity and conjugated diene formation were lower in the alpha-tocopherol group; the most significant differences were manifest at the 3-h time point. Also at both 6 and 12 weeks the mean rate of oxidation was lower in the alpha-tocopherol group.²⁺

Language of Publication

English

Unique Identifier

92381409

MeSH Heading (Major)

Lipoproteins, LDL|BL/*DE; Vitamin E|*AD

MeSH Heading

Administration, Oral; Adult; Carotene|BL; Human; Kinetics; Male; Middle Age; Oxidation-Reduction|DE; Placebos; Single-Blind Method; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.; Thiobarbiturates|BL

Publication Type

CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

ISSN

0022-2275

Country of Publication

UNITED STATES

CAS Registry/EC Number

0 (Lipoproteins, LDL); 0 (Placebos); 0 (Thiobarbiturates); 1406-18-4 (Vitamin E); 36-88-4 (Carotene); 504-17-6 (thiobarbituric acid); 7235-40-7 (Beta Carotene)

Record 20 from database: **MEDLINE**

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Title

beta-Carotene inhibits the oxidative modification of low-density lipoprotein.

Author

Jialal I; Norkus EP; Cristol L; Grundy SM

Address

Center for Human Nutrition, University of Texas Southwestern Medical Center, Dallas 75235.

Source

Biochim Biophys Acta, 1991 Oct 15, 1086:1, 134-8

Abstract

Several lines of evidence indicate that oxidized LDL (Ox-LDL) may promote atherogenesis. Hence, the role of antioxidants in the prevention of LDL oxidation needs to be determined. beta-Carotene, in addition to being an efficient quencher of singlet oxygen, can also function as a radical-trapping antioxidant. Since previous studies have failed to show that beta-carotene inhibits LDL oxidation, we re-examined its effect on the oxidative modification of LDL. For these studies, LDL was oxidized in both a cell-free (2.5 microM Cu²⁺ in PBS) and a cellular system (human monocyte macrophages in Ham's F-10 medium). beta-Carotene inhibited the oxidative modification of LDL in both systems as evidenced by a decrease in the lipid peroxide content (thiobarbituric-

acid-reacting substances activity), the negative charge of LDL (electrophoretic mobility) and the formation of conjugated dienes. By inhibiting LDL oxidation, beta-carotene substantially decreased its degradation by macrophages. beta-Carotene (2 microM) was more potent than alpha-tocopherol (40 microM) in inhibiting LDL oxidation. Thus, beta-carotene, like ascorbate and alpha-tocopherol, inhibits LDL oxidation and might have an important role in the prevention of atherosclerosis.

Language of Publication

English

Unique Identifier

92062712

MeSH Heading (Major)

Antioxidants|*PD; Carotene|*PD; Lipoproteins, LDL|*DE/ME

MeSH Heading

Cell-Free System; Human; In Vitro; Macrophages|ME; Oxidation-Reduction|DE; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Publication Type

JOURNAL ARTICLE

ISSN

0006-3002

Country of Publication

NETHERLANDS

CAS Registry/EC Number

0 (Antioxidants); 0 (Lipoproteins, LDL); 36-88-4 (Carotene); 7235-40-7 (Beta Carotene)