Case # 283 High TG and Low HDL-C: AIM HIGH or Aim Low?

Hi Lipidaholics: A somber note to start. It is with sadness that I learned of the passing of "Dr. Framingham" Bill Kannel (Journal of Clinical Lipidology (2011) 5, 501–502). What a master epidemiologist, what a master author and what a very humble and nice human being who always had the time to discuss CVD. He will be missed. No one is likely to duplicate the mass volume of information he and his colleagues discovered.

Seems my schedule never lets up. I spent a full week at AHA learning, meeting old friends, new friends and planning for a very special next year. In the final Lipidaholics Anonymous newsletter which will be out in 2-3 weeks, expect a major, major announcement regarding yours truly. It is time for this senior citizen to plot the rest of my professional life and wouldn't it be great if I could get back to the uninhibited type of lipid/CV education all of you associate with me but are now restricted to PhRMA regulations. Many of you know my son Brad (only child) is the communications director for the majority leader in the US House of Representatives and it is time for my wife and I to be closer to him (he lives in northern Virginia). Enough said: stay tuned.

Here is a brand new article for all: Put together by the one and only Peter Toth and myself: Drug Safety Evaluation of Rosuvastatin. Expert Opinion On Drug Safety 2011 Nov;10(6):969-86. This is about as thorough as it gets on rosuvastatin! Reprints: peter.toth@srfc.com One of my color graphics is there.

Still time: Want to attend a world class all day Lipid Summit CME event (7 credits) on HDL complexities in Philadelphia on Saturday December 3 **for no cost**? Check out the faculty and agenda. You will see the fantastic HDL biogenesis animated slides I have developed – Download the brochure attached to this e-mail or visit http://www.vindicomeded.com/meetings/lipidsummit/Default.asp

For those interested in the stanol vs sterol war, please check this out:

http://www.nutraingredients-usa.com/Regulation/You-say-stanol-I-say-sterol-Raisio-clashes-with-Unilever-over-cholesterol-lowering-terminology

Now for the case: A provider writes: "I have a case that puzzles me. He is a 65 year old male who is statin intolerant and has been on Welchol 625 mg 6 tabs daily and also on Niaspan but only 500 mg daily. His only CV risk factors are his age and hyperlipidemia. Before therapy he had LDL-C values in the 150 mg/dL range and HDL-C in the 30s with normal TG levels." His most recent panel revealed a:

TC = 211, HDL-C = 35, LDL-C = 124 and TG = 259 mg/dL
Ratios: The TC/HDL-C = 6.02 and the TG/HDL-C = 7.4
Non-HDL-C = 176 mg/dL

"I have been reading that TG/HDL ratio is a good surrogate marker for dense particle LDL and that lowering the TG will affect the density. Therefore I am left with the question is this ratio high enough to add fenofibrate to lower his TGs? Would it be better to increase the Niaspan?" Now that the AIM HIGH trial finally has been published there are many questions on the current use of niacin in lipid management.

DAYSPRING DISCUSSION

As always the first chore is to estimate the degree of CV risk. The patient has two major risk factors for CHD, namely age and low HDL-C and thus he qualifies for Framingham Risk Scoring (FRS). He has at least two components of the metabolic syndrome (high TG and low HDL-C) and we were not given his glucose, BP or waist size. My guess he has at least one of those and is a true metabolic syndrome. His FRS is 14 points which gives him a calculated ten year risk of a CV event of 16%. Using the 2004 NCEP addendum paper, he therefore is classified as having moderately high CV risk and treatment (lifestyle and drugs) is to be considered if the LDL-C is > 130 mg/dL with an option to consider the therapy for an LDL-C between 100 and 130 mg/dL. The goal of therapy is to achieve an LDL-C of < 130 mg/dL with an option for < 100 mg/dL. Since his on treatment TG are > 200 mg/dL the corresponding non-HDL-C goals are at play and would be 160 with option for 130 mg/dL. For those who like to be aggressive you can postulate that if indeed the metabolic syndrome is present, it would elevate him to the high risk CV category and the optional LDL-C and non-HDL-C goals would be < 70 mg/dL and 100 mg/dL respectively.

I never fail to remind clinicians that due to lack of trial evidence there is absolutely no specific TG or HDL-C goal of therapy provided in NCEP ATP-III. They succinctly state that if on therapy (lifestyle and presumably a statin)the LDL-C goal has been achieved but the TG are > 200 mg/dL, then non-HDL-C becomes the secondary goal of therapy. For low HDL-C (<40/50 male/female) in the face of elevated TG, non-HDL-C also becomes the goal and if the low HDL-C is isolated (not associated with high TG) they suggest that in high risk patients a fibrate or niacin be added to the statin (keep in mind that in 2001 neither ezetimibe nor prescription omega-3 FA were available). Again because of a total lack of Level I evidence supporting a specific HDL-C goal, NCEP offers none. Do not get too confused here: NCEP is not against the lowering of TG or raising of HDL-C and indeed sort of encourage it using especially lifestyle and if needed FDA approved therapies, to achieve LDL-C ad non-HDL-C goals. Lowering TG reduces VLDL-C and that plus a rise in HDL-C and LDL-C lowering will improve non-HDL-C.

Need I remind my readers what exactly non-HDL-C is? Simply the cholesterol trafficked within the apoB-containing lipoproteins: i.e., LDL-C, Lp(a)-C, VLDL-C, remnant-C, and IDL-C. None of that cholesterol is in HDL particles and hence it is termed non-HDL-C. It is easily calculated by subtracting HDL-C from TC or in essence by adding LDL-C and VLDL-C (calculated as TG/5). If as NCEP suggested in 2001, CV risk begins at a TG is > 150 mg/dL, a normal VLDL-C is 150/5 or 30 mg/dL. Hence the non-HDL-C goal is determined by adding 30 to the desired LDL-C goal. As stated by Fredrickson, Levy and

Lees in their classic 1967 NEJM paper, lipid concentrations are simply surrogates or proxies of lipoprotein concentrations and thus non-HDL-C is simply an approximation of apolipoprotein B (apoB) concentrations. Since apoB particles when present in excess number initiate atherosclerosis, non-HDL-C is a free guesstimate of apoB. Because of its very long half-life, > 90% of apoB particles are low density lipoproteins (LDL) and thus LDL-C is also a predictor of apoB. In a lipoprotein world measuring (it cannot be calculated) apoB via protein immunoassay is a technique of quantifying LDL particle concentration. ApoB does not tell you very much about remnants or VLDL-P and it is amazing how many lipidologists erroneously think it does. ApoB is an LDL particle count. Lastly although LDL-C, non-HDL-C apoB and LDL-P (via NMR spectroscopy) all have high correlation, in way too many people, there is considerable discordance between actual particle numbers and lipid concentrations. When lipid concentrations and lipoprotein concentrations are discordant (do not agree), CV risk almost always follows measured apoB and LDL-P (rare exceptions exist).

So this man despite treatment with a bile acid sequestrant and a near placebo dose of Niaspan has not achieved either LDL-C or non-HDL-C goals. So in addition to further improvements in lifestyle (has to watch those carbs better) additional medication is indicated to achieve whatever goals the provider desires. Personally I consider this man as high risk and although NCEP would then give me the goal-option of an LDL-C and non-HDL-C of 70 and 100 mg/dl respectively, I would be looking at apoB and LDL-P (via NMR) and make all treatment decisions based on these parameters. Using the very recent advice of the expert panel from NLA, this is exactly the type of patient they would state is very reasonable to do advanced testing using measured apoB and/or LDL-P by NMR (the underscored words are theirs). Apart from NLA, also now providing apoB goals are Canadian guidelines, the ADA/ACC 2008 consensus statement, the AACC 2010 statement, 2011 European Atherosclerosis Society (EAS) TG-rich lipoprotein management guidelines, and the very new EAS/ECS lipid guidelines.

The odds are quite high that this is a patient who likely has an extremely high apoB and LDL-P and reduced total HDL-P. Some might be nervous using a sequestrant like Welchol in a patient with a TG elevation, but please keep in mind that although Welchol raises TG a bit, it lowers apoB and LDL-P (as a monotherapy or when added to a statin), so the TG rise is likely irrelevant. ApoB and LDL-P are your goals - not TG per se. Of course it is tough to lower apoB without attacking TG-rich lipoproteins. One of the reasons HDL-C is often low in IR (MetSynd) patients, is that the HDLs are a TG-rich lipoprotein, which means they are cholesterol-depleted HDLs, explaining the low HDL-C. We need to remember it is not only VLDLs that are TG-rich lipoproteins. In IR states, both LDLs and HDLs often are TG-rich and thus CE-poor (cholesteryl ester). What do CE-depleted HDLs and LDLs have in common – elevated LDL particle counts, explaining much of the associated CV risk.

The provider zeroed in on the TG/HDL-C ratio. That ratio is best used in drug naïve patients, not in those taking lipid modulating drug therapies. An abnormal ratio should lead to several assumptions that should ultimately be verified with advanced testing. In general (but there are exceptions) a ratio > 3.0 is associated with insulin resistance and

increased CV mortality and risk for MI. At levels above 3.8 there is 80% likelihood that the LDL particles are small (dense). Although that seems to strike fear in providers, what really should raise the alarm is that almost all drug-naïve patients with small LDLs (usually cholesterol-depleted) have elevations of LDL-P (apoB) and that is what drives the risk. The recent NLA statement stated there is no evidence that measuring LDL size can help a clinician make CV risk or treatment decisions. I'd disagree a bit: in drug-naïve patients small LDLs (as well as large VLDLs and a reduction in large HDLs) are a potential sign of insulin resistance, which is important to know. Thus LipoScience uses such parameters to report the LP-IR score (lipoprotein insulin resistance score (0-100 where values above or below 50 are associated with IR or insulin sensitivity respectively). Keep in mind that the lipoprotein abnormalities are seen in IR patients' decades before glycemic parameters become abnormal. As the NLA experts state, there is no trial data that therapeutic shifting of LDL size or HDL size for that matter, provides any clinical benefits and thus such parameters should not followed per se as goals of therapy. To answer another of the provider's questions, although proper treatment will likely improve the TG/HDL-C ratio, the ratio should not be a goal of therapy

Now back to the case: What are the potential therapeutic approaches? Remember NCEP would want the non-HDL-C (the apoB or LDL-P guestimate) to be brought to goal. If the LDL-P is high risk (> 1600 nmol/L) which is a likely (but please never assume you can predict LDL-P with a high degree of certainty using lipid concentrations – because you cannot) then I need to go back and see what the statin intolerance in this patient is all about – how many different and which statins did he use and at which doses? Since statins are the best a LDL-P lowering drugs, I'd try intermittent (off-label use) of a statin. I especially like pitavastatin (Livalo) in this circumstance because it has rather clean pharmacokinetics (perhaps due to its unique isopropyl group that no other statin has). In statin intolerant patients I start 1 mg every other day and go from there (while in this case continuing the Welchol but holding the 500 mg Niaspan). Recheck the particle count within a month. I'd push the Livalo until it was maxed (4 mg daily) or it also caused myalgia..

If the statin is tolerated but non-HDL-C or better yet LDL-P is still not at goal, then additional LDL-P lowering therapies are needed: after statins and Welchol, two are left namely ezetimibe (Zetia) or extended-release niacin (Niaspan). Both through very different mechanisms of action lead to additional LDL-P lowering. Note this man has a very different CV circumstance than that tested in the AIM HIGH trial. In that study, after all patients were titrated to 1500 or 2000 mg of Niaspan per day, statin or statin ezetimibe treatment was added to get LDL-C to < 70, (they achieved 61 mg/dL), non-HDL-C to < 100 mg/dL and apoB to < 80 mg/dL. However, despite getting to goal, the HDL-C was still low. Then, ½ of the patients had the Niaspan stopped and in its place they got a tiny dose of immediate release niacin to keep the trial blinded by inducing some flushing (not a dose that has any lipid/lipoprotein effect). So AIM HIGH had two treatment groups: One on statin or statin/ezetimibe and the other Niaspan 1500-2000 mg plus statin or statin/ezetimibe. Of course more ezetimibe was needed to get to LDL-C goal than in the non-Niaspan group. So they had two groups of folks who had NCEP goals of achieved (LDL-C and non-HDL-C) as well as ADA/ACC or AACC apoB goals.

No NMR data is available. Thus even though there was a 20% rise in HDL-C (more in the niacin than non-niacin group) outcomes were the same.

What we now know and do not know after AIM HIGH:

1) Even though these patients had CAD, over 92% were aggressively treated for a year or more and thus were likely to have very stable plaques. The trial was likely underpowered to generate enough events to differentiate the therapies. Although some believe it would have mattered continuing the trial (as the AHA trial discussant Phil Barter stated) would not have mattered unless you planned on doing it for twenty years. Otherwise it would have been a waste of money. Keeping the patients at apoB goal with statin alone or statin/ezetimibe combo or statin/Niaspan combo or statin/ezetimibe/Niaspan triple combo all worked equally well. They did not keep anyone on statin monotherapy unless LDL-C, non-HDL-C, apoB were at goal. So in my mind this trial showed us we have several therapies to achieve apoB goal.
2) Once someone is at LDL-P (apoB) goal, raising HDL-C without raising HDL-P may be futile. Others (Phil Barter) have stated that although Niaspan raised HDL-C by 25% to 42 mg/dL, there was a 9.8% rise to 38 mg/dL in the other group (due to statin) and thus the difference between the two groups was not that impressive. Who cares? The purpose of the trial was to show that niacin added to statin would help event reduction for any numbers of potential reasons including raising HDL-C. Well **we know it did not in persons with very stable plaque with aggressively treated apoB** and that is all we can conclude. Still open to conjecture is that niacin added to statin or statin/ezetimibe, would have worked had those folks not been at lipid or apoB goal. Almost certainly that will be tested in the much larger on-going HPS-THRIVE study in Europe and China. Stay tuned!
3) If niacin has pleiotropic effects, and many studies would support that it does, including increasing macrophage RCT, perhaps they do not matter if apoB is aggressively treated b below goal.
4) Maybe if niacin raised HDL-P (like a fibrate) additional benefit would have been seen (niacin raises HDL-C by increasing HDL size). Keep in mind that in VA-HIT gemfibrozil vs placebo worked by raising HDL-P even though the HDL-C rise was much less (6% or 1.8 ,g/dL) than what niacin induced in AIM HIGH. This is related to the fact that fibrates decrease (not enlarge) HDL size.
5) There is no reason at this time to stop niacin in any high risk patient where it has helped achieved apoB (LDL-P) goals. There is also no reason to add niacin if other therapies have achieved apoB or LDL-P goal.
6) At this time there is no Level I evidence to support the belief that therapeutically raising HDL-C with any available drug matters. Stay tuned for HPS THRIVE with statin/niacin and for the ongoing CETP inhibitor or modulator drug trials.

So, since niacin is an apoB lowering med, and does not raise HDL-P we do not need to AIM high, but rather start AIMING LOW (with respect to LDL-P, apo B or their less

accurate surrogates like LDL-C and non-HDL-C. If in this case if a statin can be used with the Welchol, use it. If goal is not achieved or if indeed no statin can be used the best combination to lower apoB is ezetimibe (Zetia) plus fenofibrate or fenofibric acid (Trilipix) (both at full dose). If you still do not get to apoB goal adding Niaspan is appropriate, but you must get up to > 1500 - 2000 mg. 500 is a placebo dose.

Fortunately in most patients we can use statins. The two best apoB lowering drugs to add to a statin are either ezetimibe (10 mg) or Niaspan (1500-2000 mg). After those two, Welchol is next in LDL-P lowering potency. Caution with using slow niacin with the hepatotoxicity at these high doses. Adding fenofibric acid and presumably fenofibrate to a low or moderate or high dose statin does not give any additional LDL-P lowering (you will get a 5% apoB lowering, represent VLDL-P reduction). Thus niacin and ezetimibe, not fibrates are what we need to use for LDL-P lowering beyond a statin. However if you think you need to raise HDL-P, use fenofibrate or fenofibric acid and do not forget the microvascular benefits that occur in diabetic patients. (off label use).

TAKE HOME POINTS:

Right now atherosclerosis is an apoB mediated, maladaptive inflammatory disease and the evidence in at risk patients with low HDL-C is to AIM LOW with apoB as your first target priority. What you should be doing to any HDL issues, is to wait for ongoing trials. Dan Rader, master HDL guru, at his AHA lecture stated we need to look at the associations so prevalent in persons with low HDL-C (high LDL-P, insulin resistance, diabetes, etc.), and not consider HDL-C as a goal of therapy per se.

REFERENCES OF THE WEEK:

1) Cardiovascular Consequences of Ovarian Disruption: A Focus on Functional Hypothalamic Amenorrhea in Physically Active Women Emma O'Donnell et al. J Clin Endo & metab. doi:10.1210/jc.2011-1223 With the premenopausal years typically considered to be cardioprotective in association with normal ovarian function, ovarian disruption in women with EAA is of importance. Further investigation of the short-term, and potentially long-term, cardiovascular consequences of hypoestrogenemia in women with EAA is recommended

2) The Incretin Axis in Cardiovascular Disease. Editorial by Jorge Plutzky, Circulation. 2011;124:2285-2289. Anything written by Dr P is a must. pathways originating in the gut can alter outcomes not in the veins but in the arterial system, where the pathological consequences of T2D occur.\

3) Inflammatory stress exacerbates hepatic cholesterol accumulation via increasing cholesterol uptake and de novo synthesis Lei Zhao, et al. Journal of Gastroenterology and Hepatology 26 (2011) 875–883 Inflammatory stress disrupted hepatic SREBP2-mediated

low-density lipoprotein receptor and HMGCoA-r feedback regulation resulting in exacerbated cholesterol accumulation in livers of mice and HepG2 cells.

4) Intestinal sterol transporters and cholesterol absorption inhibition. Harry R. Davis Current Opinion in Lipidology 2011, 22:467–478 Additional lipid-lowering agents are needed to fulfill an unmet medical need for those patients who do not achieve optimal LDL-C goals on statin monotherapy. The inhibition of cholesterol absorption is an important therapeutic strategy to reduce cholesterol levels.

DAYSPRING TRAVELS (December)

Madison and Brookfield, WI Tues and Wed this week
HDL Summit in Philadelphia (12/3)
Return to Philadelphia, PA (12/6)
Port Orange and Ocala, FL
Reno, NV
Valhalla, NY

BULLETIN BOARD:

1) **Health Diagnostic Labs in Richmond, VA:** Is the lab that I use in my practice on all patients. In addition to the NMR parameters I need, I get Lp(a) and Lp(a)-C (need both together to make rational apo(a) decisions), several inflammatory markers including Lp-PLA2 and myeloperoxidase (my HDL functionality test), important insulin resistance markers, the Omega-3 Index and much more. Markers of cholesterol absorption and synthesis now available. Their patient portal, a fantastic site for patients to learn about their results and obtain a lot of education is now up and running. It is my great pleasure to assist the entire HDL team in their collaborative mission to develop several types of lipid/lipoprotein and vascular biology educational programs. Visit HDL at http://www.myhdl.com/ to see what they offer to both your patients (in the way of health care coaching, an interactive web site) and how you can utilize their services.

   Apolipoprotein E isoform testing often confuses many. I have written a short piece on how to utilize them information. Please check out http://www.hdlabinc.com/sciencebulletin/v1i2/clinical-spotlight

You Tube discussions on CV testing (just search my name on You Tube).

2) FAMILIL HYPERCHOLESTEROLEMIA: There is a new AP for iPhones and iPADs with respect to Familial Hypercholesterolemia Please check it out at http://iphoneapplicationlist.com/app/id450486145/

Also check out this great video on lipoproteins in FH and recommend it to your FH patients http://www.youtube.com/watch?v=UIjkAPn2CRE

Finally another great site for patient information please visit:
http://www.pcna.net/clinical/patients/fh/index.php

3) Please visit www.lipidcenter.com (education professionals) to read many of my writings, view many of my slides and blogs and get all sorts of great lipid/lipoprotein info. Additions are continually made. Make sure you click on the professionals tab to see my work. There is also lots of good information for your patients on the other parts of the web site and new items are always being entered. This is one of the best (serious, high level) informational lipid/lipoprotein web sites. See pdfs of my slides.

4) **I continue to urge readers to** visit this web site http://vimeo.com/29987751 and listen to Dr. Bill Cromwell talk off the cuff on insulin resistance and understanding lipid and lipoprotein issues in such patients (if prompted for a password use Cromwell2011). **Do not fail to view this incredible short piece**: you will learn more in 15-20 minutes than by reading a lipid textbook.

3) **DAYSPRING Publications**

Free PDF Downloads of my work:

1) Moving beyond LDL-C: incorporating lipoprotein particle numbers and geometric parameters to improve clinical outcomes
http://www.dovepress.com/articles.php?article_id=5601

2) Understanding hypertriglyceridemia in women: clinical impact and management with prescription omega-3-acid ethyl esters
http://www.dovepress.com/articles.php?article_id=6607

At www.lipidcenter.com site check out the Pocket Guide on Lipid and Lipoproteins put together by Bill Cromwell, myself and Michael Richman. This is a very useful guide for those of you trying to incorporate LDL particle testing into your practice.

At www.lipidcenter.com (under education/professionals) check out: Lipid and Lipoprotein Biochemistry" and "Lipid Treatment Algorithm" and my brand new piece on HDL and Cholesterol trafficking.

http://www.lipidcenter.com/er_hcp_lipidstudy.php
http://www.lipidcenter.com/er_hcp_lipiddrugs.php
http://www.lipidcenter.com/pdf/HDL_Trafficking_of_Cholesterol.pdf

For those interested in what fenofibric acid with or without statin does to NMR parameters in dyslipoproteinemic patients: please see Peter H. Jones, Thomas Dayspring,

Carolyn Setze, Aditya Lele, Maureen Kelly, Kamlesh Thakker. Effects of Fenofibric Acid in Combination with Statin Therapy on LDL and HDL Particle Number in Patients with Mixed Dyslipidemia J Clin Lipidololgy 2011;5:202 or e-mail me for the actual poster.

For those of you who are NLA members, please check out my article in the Lipid Spin entitled Demystifying Lp(a) The Lipid Spin 2011;9:11-12, 33 Free for NLA members at http://www.lipid.org/publications/spin.php Click on Spring 2011

Article reviews: Check out my latest BioCritique postings on BioCritique (a CME) web site www.biocritique.com (free but simple registration required).

Pharmacologic Suppression of Hepatic ATP-Binding Cassette Transporter 1 Activity in Mice Reduces High-Density Lipoprotein Cholesterol Levels but Promotes Reverse Cholesterol Transport.
http://www.biocritique.com/secure/viewref.cfm?messageid=6f4eeb5f-4431-4ac9-8e75-6c12aaa2d6d7

Reliability of low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein B measurement. JH Contois, GR Warnick, AD Sniderman J Clin Lipidol 2011 7;5(4):264-72
http://www.biocritique.com/secure/viewref.cfm?messageid=c5bff5af-117d-4065-8fcc-2598e8a4d392

Xanthelasmata, arcus corneae, and ischaemic vascular disease and death in general population: prospective cohort study. M Christoffersen, R Frikke-Schmidt, P Schnohr, GB Jensen, BG Nordestgaard, A Tybjærg-Hansen BMJ 2011 1;343:d5497
http://www.biocritique.com/secure/viewref.cfm?messageid=9178cb5e-b341-4495-ad66-9e7a4cb15011

Correlation between circulating adiponectin levels and coronary plaque regression during aggressive lipid-lowering therapy in patients with acute coronary syndrome: subgroup analysis of JAPAN-ACS
http://www.biocritique.com/secure/viewref.cfm?messageid=bc3c6a70-b321-40b5-b315-1fade90b9b99

Effect of statins on cholesterol crystallization and atherosclerotic plaque stabilization.
http://www.biocritique.com/secure/viewref.cfm?messageid=efd9afd6-6782-4a26-8c79-209f0543fa8d

Medical management of diabetic retinopathy: fenofibrate and ACCORD Eye studies.
http://www.biocritique.com/secure/list.cfm?groupid=a37c869a-d617-4e63-a100-5c8bfcbe2763&sort=1&search=&filter=1

Effects of coadministered ezetimibe plus fenofibrate in mixed dyslipidemic patients with metabolic syndrome.

http://www.biocritique.com/secure/viewref.cfm?messageid=9e26f716-97d6-439a-8d8e-d21ac376b136

Cholesterol efflux potential and anti-inflammatory properties of high-density lipoprotein after treatment with niacin or anacetrapib. http://www.biocritique.com/secure/viewref.cfm?messageid=ae1d362e-7b8b-49b3-b3bd-2dcf4fe8fbd4

Efficacy of fenofibric Acid plus statins on multiple lipid parameters and its safety in women with mixed dyslipidemia. http://www.biocritique.com/secure/viewref.cfm?messageid=f61f3568-cc6c-4a07-b876-1933c5a43290

Clinical implications of discordance between low-density lipoprotein cholesterol and particle number. http://www.biocritique.com/secure/viewref.cfm?messageid=f0146d6c-ceec-4fe0-a39f-658bd538691e

National Lipid Association A great, very affordable organization for anyone interested in increasing their lipid knowledge. Please visit www.lipid.org for details. They are seeking new members at all times. Please consider joining your lipid colleagues (this is open to all healthcare professionals: you do not have to be a physician to join). The NLA has an informal journal called the "Lipid Spin" as well as the more sophisticated Journal of Clinical Lipidology. Chapter meetings are scheduled throughout the year. Try attending a meeting in your region. See the NLA web site.

The NLA also offers Lipid University which provides comprehensive, in-depth instruction on lipid science for medical sales professionals to better understand the pathophysiology of dyslipidemia and atherosclerosis and also the risk assessment models that influence clinical decision making. After attending the course, representatives test their clinical competency in a Level II proctored exam.

Lipid Board Certification: Interested?

Physicians: Visit www.lipidboard.org

Allied Healthcare practitioners visit: www.lipidspecialist.org

Lastly FREEDOM IS NOT FREE

The following great line will be repeated in perpetuity in my newsletter**: A veteran is someone who, at one point in his life, wrote a blank check made payable to his country for an amount of "up to and including my life."**

Also "Some people spend an entire lifetime wondering if they made a difference in the world. But the U.S. ARMED FORCES don't have that problem." ..... Ronald Reagan

If you have never heard of the Angel Flight, get a tissue and watch this video
http://www.youtube.com/watch?v=70Ikj1hZDnw

Please offer prayers or silent thoughts for our brave troops in Afghanistan and around the globe. Please check out the wounded warriors project: http://www.woundedwarriorproject.org or Salute to our Heroes http://www.saluteheroes.org/ They need our support.

Visit the SEAL - Naval Special Warfare Foundation web site (take a thorough tour of that site) and make a donation:

http://www.usnavysealfoundation.org/CONTRIBUTE.html

As always, Happy "Lipiding" to all

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