

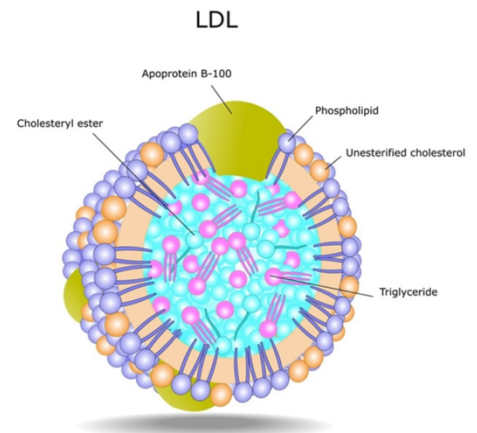
Hyperlipidemia refers to elevated plasma cholesterol or triglyceride levels or both. Cholesterol is a waxy substance that travels through the bloodstream on proteins called lipoproteins. At normal levels, cholesterol has many important functions. It is one of the key components of cell membranes. Cholesterol is also an important precursor molecule for the synthesis of vitamin D and the steroid hormones, including the adrenal gland hormones cortisol and aldosterone, as well as the sex hormones progesterone, estrogen, and testosterone.

Hyperlipidemia, more commonly known as high cholesterol, is a condition in which there are high levels of lipids in the blood. This is characterized by increased levels of low-density lipoprotein (LDL) particles and decreased levels of high-density lipoprotein (HDL) particles. The main cause of secondary hyperlipidemia includes unhealthy lifestyle habits in which a major risk factor is mainly poor diet i.e. with a fat intake greater than 40 percent of total calories, saturated fat intake greater than 10 percent of total calories; and cholesterol intake greater than 300 milligrams per day.<sup>1</sup> Other lifestyle factors such as obesity, smoking, heavy alcohol use, and lack of exercise can also lead to hyperlipidemia.

High triglyceride levels can be an independent condition resulting from fatty liver disease with liver inflammation. High serum triglyceride levels can also result from high cholesterol when high cholesterol has catalyzed the onset of liver inflammation.

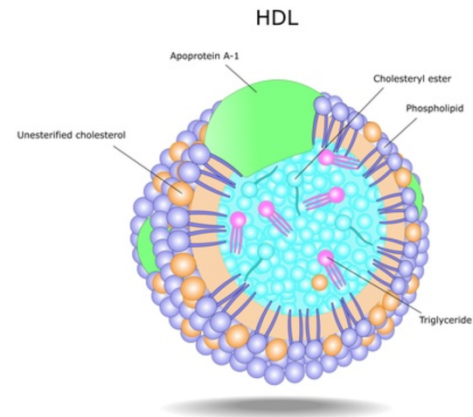
### Low-Density Lipoproteins

There are two types of lipoproteins that carry cholesterol to and from the cells. LDL and very low-density lipoprotein (VLDL) are both known as “bad cholesterol” because LDL carries cholesterol from the liver to the cells and VLDL contains the highest amount of triglycerides. LDL carries the majority of the cholesterol that is in the circulation and is one of the most prevalent risk factors contributing to the evolution of atherosclerosis and consequent vascular disease. LDL receptors in the liver play a major role in determining plasma LDL levels (a low number of receptors is associated with high plasma LDL levels while a high number of hepatic LDL receptors is associated with low plasma LDL levels).<sup>2</sup>



### High-Density Lipoproteins

HDL is known as “good cholesterol” because it carries cholesterol out of the bloodstream and back to the liver. HDL particles play an important role in reverse cholesterol transport from peripheral tissues to the liver, which is one potential mechanism by which HDL may be anti-atherogenic.<sup>2</sup> The pathway by which excess cholesterol from peripheral cells, such as macrophages, in the vessel wall is transported to the liver for excretion is through HDL. In addition, HDL particles have anti-oxidant, anti-inflammatory, anti-thrombotic, and anti-apoptotic properties, which may also contribute to their ability to inhibit atherosclerosis.<sup>2</sup>



HDL particles are enriched in cholesterol and phospholipids. Phospholipids and most likely cholesterol are added to ApoA-I, the main protein constituent of HDL, by the ATP-binding cassette (ABCA1) transporter. ApoA-I is secreted from the liver and intestine in a relatively lipid-poor state.<sup>2</sup>

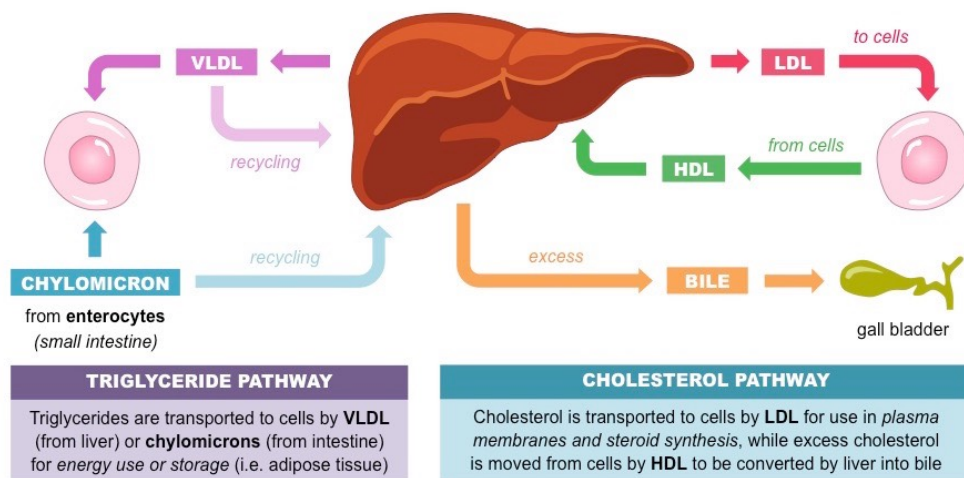
### The Liver and Cholesterol

The liver is central to the regulation of cholesterol levels in the body. The liver synthesizes not only the cholesterol, but also the HDL, LDL and VLDL. Cholesterol is both synthesized by the liver cells and taken in with food intake. The rate limiting enzyme in cholesterol synthesis is the HMG-CoA reductase. The HMG-CoA reductase in the

hepatocytes is under negative feedback regulations. When the sterol level is high, the liver cells will reduce the HMG-CoA reductase level and less cholesterol will be synthesized.<sup>6</sup> LDL receptors in the liver can bind with the LDL and control the level of LDL in the blood as well. The amount of LDL receptors in the liver is also under the negative feedback control. When the LDL is high in the blood, the LDL receptors will be decreased and less LDL will be released into the blood.<sup>6</sup>

Unhealthy eating habits, heavy alcohol use, or certain medications such as statins can cause liver injury and malfunctioning. This can lead to the loss of the liver's feedback control mechanism. Regardless of the high level of LDL and cholesterol in the blood, the liver keeps synthesizing the cholesterol and maintaining high amounts of LDL receptors causing high cholesterol in the blood.

A poorly functioning or deficient liver also can't synthesize the proper amount of HDL particles. This leads to a decrease in the number of ApoA-I, the main protein constituent of HDL, and the resulting less cholesterol which has been absorbed from food intake being brought back to the liver to be excreted. This causes an imbalance between LDL and HDL levels which can contribute to hyperlipidemia as well as atherosclerosis.



### Fatty Liver Disease and Cholesterol

Hypercholesterolemia is also a main cause of nonalcoholic fatty liver disease (NAFLD) as well as atherosclerosis. High cholesterol also can turn fatty liver disease (steatosis) into a more serious condition, nonalcoholic steatohepatitis (NASH) which may progress into hepatic fibrosis, cirrhosis, or hepatocellular carcinoma. Increased cholesterol synthesis or cholesterol overload can also cause free cholesterol accumulation in the liver. The presence of the increased levels of cholesterol in liver can significantly increase the risk factor of the “second hit” and amplify the impact of the “second hit” triggering the onset of liver inflammation and speeding up the irreversible progression of NASH. The accumulation of free cholesterol in the liver can also cause liver injury because of the activation of Kupffer cells (KCs) and Satellite cells (HSCs) which promotes inflammation and fibrogenesis. In addition, free cholesterol accumulation in liver mitochondria induces mitochondrial dysfunction, which results in increasing production of reactive oxygen species (ROS), and triggers the unfolded protein response in the endoplasmic reticulum (ER) causing ER stress and apoptosis.<sup>11,12</sup>

Patients who have hyperlipidemia with high cholesterol may or may not have high triglycerides levels in their blood. If patients have fatty accumulation in the liver (the “first hit” of fatty liver disease), having high cholesterol as well can trigger the “second hit” and the onset of liver inflammation and triglycerides in the blood begin to rise. High triglycerides may contribute to hardening of the arteries or thickening of the artery walls (arteriosclerosis) which increases the risk of stroke, heart attack and heart disease. Extremely high triglycerides can also cause acute pancreatitis, inflammation of the pancreas pancreatitis.

## Diet and Cholesterol

Small, dense LDL particles are particularly atherogenic while large buoyant LDL cholesterol is not known to be atherogenic.<sup>7</sup> Levels of small, dense LDL particles are closely correlated with dietary carbohydrate intake. Fructose also increases levels of small, dense LDL particles more than glucose does. Dietary saturated fat increases levels of both HDL cholesterol and large buoyant LDL cholesterol.<sup>7</sup>

An abundance of small dense LDL particles are seen in association with hypertriglyceridemia, low HDL levels, obesity, type 2 diabetes and infectious and inflammatory states.<sup>2</sup> Small dense LDL particles have a decreased affinity for the LDL receptor resulting in a prolonged retention time in the circulation.<sup>2</sup> Additionally, they more easily enter the arterial wall and bind more avidly to intra-arterial proteoglycans, which traps them in the arterial wall.

## Complications

Hyperlipidemia, in particular elevated LDL (hypercholesterolemia), is one of the most prevalent risk factors contributing to the evolution of atherosclerosis and consequent vascular disease.<sup>1</sup> Atherosclerosis frequently remains asymptomatic until plaque stenosis reaches 70 to 80% of the vessel's diameter. Atherosclerosis originates after underlying endothelial damage occurs, which appears to stem from the loss of nitric oxide within the endothelium.<sup>1</sup> This process leads to increased inflammation directly around the site of dysfunction, permitting the accumulation of lipids within the innermost layer of the endothelial wall. The lipids are then engulfed by macrophages, leading to the establishment of "foam cells."<sup>1</sup> This cholesterol build-up within the "foam cells" causes subsequent mitochondrial dysfunction, apoptosis, and, ultimately, necrosis of the underlying tissues.<sup>1</sup> Smooth muscle cells encapsulate the pack of "foam cells" or debris, which produces a fibrotic plaque that inhibits the underlying lipids (debris) from being destroyed.

Complications from undertreated or untreated hyperlipidemia include all types of vascular disease, which may prove fatal down the road.<sup>1</sup> These include, but are not limited to, coronary artery disease, peripheral artery disease, cerebrovascular accidents, aneurysms, type II diabetes, high blood pressure, and even death.<sup>1</sup>

Western medicine typically prescribes Statin medications which are a class of drugs that are lipid-lowering. Unfortunately, Statin medication complications include myopathy, renal injury, arthralgia, extremity pains, nausea, myalgia, elevated liver enzymes/hepatotoxicity, diarrhea, and rhabdomyolysis.<sup>1</sup>

## Statins and High Cholesterol

Although statins are the most common medication prescribed for hyperlipidemia, not all patients can take it or will benefit from it. The most serious risk of these drugs is rhabdomyolysis with acute renal failure and even death. About 5-10% of individuals are unable to tolerate statins. For some, allergies can be the causative issue and, if taken, can lead to acute kidney failure. This type of patient may rely on diet and exercise as their only option to control their cholesterol levels. For others, rhabdomyolysis and chronic fatigue can be developed in combination with severe muscle aches as a result of a side effect of statin medications. This is because statins lower the amount of coenzyme Q10 (CoQ10), which is important for muscle function. Simvastatin, for example, has been shown to decrease levels of CoQ10 by 40%.<sup>9</sup> It's important for individuals who have hyperlipidemia to exercise and maintain a healthy lifestyle, but statin medications can make this more difficult as they increase exercise-induced skeletal muscle injury. In a study done, the muscle pain prevented even moderate exertion during everyday activities in 38% of the patients with myalgia on statins.<sup>11</sup>

Patients who are on statins with no current side effects can develop long-term damage with high-dose and continued use. These include both liver and kidney damage. A large retrospective cohort study comparing long-term statin users with a matched group of nonusers found an association between statin treatment and an increased incidence of acute and chronic renal disease.<sup>10</sup>

## **Wellness Recommendation**

### Hyperlipidemia

Along with a healthy lifestyle and diet adjustment, the wellness recommendation for hyperlipidemia includes Brown and LC balancer. Brown is a liver Yin formula that helps to improve the structure and function of the liver. Through

improving liver function, the liver will resume its feedback control mechanism in LDL and cholesterol synthesis which helps reduce the LDL and cholesterol in the blood to the normal range. By improving the liver function, the synthesis of ApoA-I, the main protein constituent of HDL, can be restored which helps to increase the HDL levels in the blood. Brown and LC Balancer also helps reduce liver inflammation and triglyceride levels in the blood and clears fat accumulation in the liver. By repairing liver damage, the treatment also helps reduce the liver enzyme levels to the normal range.

Herbal ingredients in Brown have been shown to significantly reduce total cholesterol levels and serum lipid levels, contain antioxidant properties to help combat plaque formation, and increase HDL levels.<sup>3,4</sup> LC Balancer, a kidney Yin formula, helps to enhance microcirculation by improving microcapillary structure which improves overall blood flow and nutrient absorption. Herbal ingredients in LC Balancer have also been shown to address hyperlipidemia through reducing plasma total cholesterol and triglycerides, while elevating plasma HDL-cholesterol.<sup>5</sup> Patients with high cholesterol and triglyceride, high LDL and VLDL, and low HDL can experience improvement in 3-4 weeks with the use of Brown and LC Balancer. Patients can have a significant improvement in 6 weeks. Blood tests that monitor both liver enzyme and lipid levels are recommended to monitor the progress. 6 weeks to 3 months of the protocol may be required for patients to achieve desired levels of cholesterol, triglyceride, and lipoproteins. For patients who have borderline high triglycerides levels, the use of Brown and LC Balancer should help reduce it to the normal range. If patients have liver inflammation with very high levels of triglycerides, Levera is also required to help reduce liver inflammation and bring down the triglycerides levels to the normal range.

For patients who are over 50 or have poor kidney function, Xcel is also recommended to help support toxin and waste secretion. Symptoms of waste build up in the blood from the enhanced liver activity include anxiety, hot sensations, insomnia, fatigue or flu like symptoms. Xcel can help improve adrenal and kidney function and enhance the kidney's filtration and balancing of minerals for effective waste secretion.

#### Atherosclerosis

If the patient has both atherosclerosis and hyperlipidemia, the wellness recommendation also includes CV formula. CV Formula helps remove blood stasis and nurture heart Qi and Blood. It helps improve heart blood circulation, reduce blood vessel restriction, remove the atherosclerotic plaque, and repair artery damage. Radix Salviae, an herb used in CV Formula, has been shown to have many cardiovascular benefits including decreasing the development of atherosclerosis. Radix Salviae has the ability to inhibit oxidative stress which disrupts adhesion molecules and prevents LDL from oxidation, which in turn reduce atherosclerotic areas in the abdominal and thoracic aorta.<sup>8</sup> Radix Salviae also has been shown to decrease levels of pro-inflammatory cytokines which decrease the susceptibility of plaque formation.<sup>8</sup> During the initial 2-4 weeks of protocol, Myogen, B-2 and Qi Booster are also recommended to help clear the dissolved wastes and reduce the irritation to the heart muscle. Patients should have symptom improvement with 1-3 days of treatment. 4- 6 weeks of treatment is required for significant improvement.

#### **Selected Case Studies**

##### Case 1: Normalization of Platelet Count, Liver Enzymes, Triglycerides, and Body Weight

*Roopa Chari, MD, CA*

A 48-year-old female presents with hypothyroidism, trouble sleeping, and difficulties with weight loss. Since her twenties, she has been struggling with her weight and has tried numerous diets. When she would eat carbohydrates, she noticed it affected her weight dramatically and she could not keep the weight off, which has affected her emotional health and self-confidence. She initially weighed 141 lbs. and was hoping to lose 10-15 lbs. Her trouble sleeping has also been a major issue with her quality of life. She can only get 5 hours of sleep a night and wakes up, unable to fall back asleep. The patient has been taking certain Glandular products from Xymogen for her thyroid and adrenal function for the past two years.

The patient had her initial medical consultation with Dr. Chari in August 2017. Dr. Chari had ordered a food sensitivity test to create a dietary plan based on the results of said testing. The results of her food sensitivity test showed she was to avoid alcohol, dairy, eggs, gluten, and even certain vegetables. Dr. Chari also ordered comprehensive lab tests to assess her liver enzymes, thyroid function and complete blood count. Those results showed that her liver

enzymes were very elevated: AST was 65 IU/L, ALT was 99 IU/L and GGT was 98 IU/L (normal liver enzyme levels range from 0-60 IU/L). Her platelet count was also a concern since it was decreased at 124 x10E3/uL (normal range 150-379 x10E3/uL). In early September 2017, she started Wei Laboratories supplements consisting of LC Balancer (1/3 dose) to help with her microcirculation and Spring Juice (half dose) to aid in her digestion. At the end of September, Brown Formula was added to help with her liver function, trying to specifically help with her stress response to her weight loss and sleeping issues. Formula C was also added to aid in her connective tissue structure throughout her body.

In October 2017, the patient reported her clothes were fitting better and she noticed she was not always hungry. Her sleep had also increased by an hour each night; from 5 to 6 hours. On October 2, 2017, she continued Wei products consisting of Brown, LC Balancer, Formula C, KS, and Xcel all at full dose (which were ordered to help with her kidney function and kidney heat symptoms affecting her sleep). Dr. Chari started weaning her off the Glandular products from Xymogen at this time. At the end of October, she was taking Brown, LC Balancer, Xcel, KS, Probiosis and Luna all at full dose (Probiosis and Luna were added for digestive health). By the end of October, she ceased taking all Glandular products from Xymogen. She reported that her sleep was dramatically improving. She was sleeping 6 hours per night and also found it easy to fall back asleep upon waking up in the middle of the night.

In November 2017, she was finishing up her 3-month diet plan and her products consisted of Brown, LC Balancer, Xcel and KS at full dose. The patient reported that she now was 129 pounds and had lost 12 pounds with the dietary program from Dr. Chari and Wei formulas.

In December 2017, Dr. Chari repeated lab work to check on the status of her liver enzymes and platelet count. Her AST had decreased from 65 IU/L to 17 IU/L, ALT decreased from 99 IU/L to 20 IU/L and GGT decreased from 98 IU/L to 20 IU/L. However, her platelet count remained low at 128 x10E3/uL. Focus was then placed on improving her platelet count and continuing to remove any liver heat affecting her thyroid levels. At the end of December 2017, her protocol consisted of Brown, Levera, Cirrhonin, and Lido all at 2/3 dose, taking these products in the morning. Stemgen and Glucogen were also added at a 2/3 dose in the evening to nurture her spleen and improve her platelet count.

In February 2018, the patient went through another series of lab testing, ordered by Dr. Chari, to see how her blood levels have changed after being on the Wei products. After 5 months of working with Dr. Chari, dieting and using Wei products, her platelet count increased to 174 x10E3/uL which was now within normal range. This was a big improvement from August 2017, when her platelet count was only at 124 x10E3/uL. Her liver enzymes continued to stay within normal range: AST 14 IU/L, ALT 21 IU/L and GGT 30 IU/L. Her Triglycerides also had improved with treatment (see table 1 below).

Dr. Chari had a follow-up medical consult with her in March 2018. The patient reported her sleep has continually been better and her energy levels were great. Overall the patient reported that the biggest improvement for her was that she kept off and maintained her weight loss with the program from Dr. Chari and the Wei products. Even after stopping her diet plan in January 2018, where she allowed herself to eat a lot of carbs and drink wine occasionally, she was able to keep that 12 pounds off and her sleep quality was sustained. The patient was extremely happy with the results and now feels like a new person!

Table 1: Patient's blood work results from Aug 2017 to Feb 2018

	8/9/2017	12/ 9/2017	2/26/2018
Platelets (150- 379 x10E3/uL)	124 x10E3/uL	128 x10E3/uL	174 x10E3/uL
AST (0-40 IU/L)	65 IU/L	17 IU/L	14 IU/L
ALT (0-32 IU/L)	99 IU/L	20 IU/L	21 IU/L
GGT (0-60 IU/L)	98 IU/L	20 IU/L	30 IU/L
Triglycerides (0-149 mg/dL)	171 mg/dL	85 mg/dL	<b>109 mg/dL</b>

## Case 2: Successful Healing of Complex Set of Symptoms including High Cholesterol

*Jacklin Arastouzadeh, B.A UCLA, LA.c, Dipl. Ac, N.B.A.O., Qualified Medical Evaluator, Former UCLA Research Acupuncturist, CA*

A long-term patient (patient for ten years now), age 55 and male, came for first time treatment in 2001. He had been diagnosed with Hepatitis B, thoracic arthritis, high blood pressure, high cholesterol, diverticulitis, allergies, prostate problems, hemorrhoids, and shoulder problems. The patient considered himself dying at that point.

A comprehensive protocol composed of 30 sessions for a total length of eight months (one session per week) was prescribed. It was composed of acupuncture and herbal treatment including Wei Laboratories' Brown and LC Balancer and additional self-made herbs.

After six months, the protocol had eliminated all symptoms except for the thoracic arthritis. A preventive maintenance schedule (once a week) has been applied ever since. It just needed the right herbal mix and acupuncture to cure a complex set of symptoms. The patient has been loyal for ten years now.

### References:

1. Hill MF, Bordon B. Hyperlipidemia. [Updated 2020 Sep 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK559182/>
2. Feingold KR, Grunfeld C. Introduction to Lipids and Lipoproteins. [Updated 2018 Feb 2]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK305896/>
3. de Souza Zanchet, M. Z., Nardi, G. M., de Oliveira Souza Bratti, L., Filippin-Monteiro, F. B., & Locatelli, C. (2017). Lycium barbarum Reduces Abdominal Fat and Improves Lipid Profile and Antioxidant Status in Patients with Metabolic Syndrome. *Oxidative medicine and cellular longevity*, 2017, 9763210. <https://doi.org/10.1155/2017/9763210>
4. Ma, Z. F., Zhang, H., Teh, S. S., Wang, C. W., Zhang, Y., Hayford, F., Wang, L., Ma, T., Dong, Z., Zhang, Y., & Zhu, Y. (2019). Goji Berries as a Potential Natural Antioxidant Medicine: An Insight into Their Molecular Mechanisms of Action. *Oxidative medicine and cellular longevity*, 2019, 2437397. <https://doi.org/10.1155/2019/2437397>
5. Yamamoto M, Uemura T, Nakama S, Uemiya M, Kumagai A. Serum HDL-cholesterol-increasing and fatty liver-improving actions of Panax ginseng in high cholesterol diet-fed rats with clinical effect on hyperlipidemia in man. *Am J Chin Med.* 1983;11(1-4):96-101. doi: 10.1142/S0192415X83000161. PMID: 6660221.
6. Trapani, L., Segatto, M., & Pallottini, V. (2012). Regulation and deregulation of cholesterol homeostasis: The liver as a metabolic "power station". *World journal of hepatology*, 4(6), 184–190. <https://doi.org/10.4254/wjh.v4.i6.184>
7. DiNicolantonio, J. J., Lucan, S. C., & O'Keefe, J. H. (2016). The Evidence for Saturated Fat and for Sugar Related to Coronary Heart Disease. *Progress in cardiovascular diseases*, 58(5), 464–472. <https://doi.org/10.1016/j.pcad.2015.11.006>
8. Wang L, Ma R, Liu C, et al. *Salvia miltiorrhiza*: A Potential Red Light to the Development of Cardiovascular Diseases. *Curr Pharm Des.* 2017;23(7):1077–1097. doi:10.2174/1381612822666161010105242
9. Deichmann, R., Lavie, C., & Andrews, S. (2010). Coenzyme q10 and statin-induced mitochondrial dysfunction. *The Ochsner journal*, 10(1), 16–21.
10. Acharya T, Huang J, Tringali S, Frei CR, Mortensen EM, Mansi IA.. Statin use and the risk of kidney disease with long-term follow-up (8.4-year study) *Am J Cardiol.* 2016;117:647–55.
11. Parker, B. A., & Thompson, P. D. (2012). Effect of statins on skeletal muscle: exercise, myopathy, and muscle outcomes. *Exercise and sport sciences reviews*, 40(4), 188–194. <https://doi.org/10.1097/JES.0b013e31826c169e>
12. Kim, E. J., Kim, B. H., Seo, H. S., Lee, Y. J., Kim, H. H., Son, H. H., & Choi, M. H. (2014). Cholesterol-induced non-alcoholic fatty liver disease and atherosclerosis aggravated by systemic inflammation. *PLoS one*, 9(6), e97841. <https://doi.org/10.1371/journal.pone.0097841>

13. Wouters K, van Gorp PJ, Bieghs V, Gijbels MJ, Duimel H, Lütjohann D, Kerksiek A, van Kruchten R, Maeda N, Staels B, van Bilsen M, Shiri-Sverdlov R, Hofker MH. Dietary cholesterol, rather than liver steatosis, leads to hepatic inflammation in hyperlipidemic mouse models of nonalcoholic steatohepatitis. *Hepatology*. 2008 Aug;48(2):474-86. doi: 10.1002/hep.22363. PMID: 18666236.