Literature update week 18 (2019)

[1] *Shen XN, Lu Y, Tan CTY et al.* **Identification of inflammatory and vascular markers associated with mild cognitive impairment**. Aging 2019; 11:2403-2419.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31039131
ABSTRACT
Biochemical processes have been associated with the pathogenesis of mild cognitive impairment (MCI) and dementia, including chronic inflammation, dysregulation of membrane lipids and disruption of neurotransmitter pathways. However, research investigating biomarkers of these processes in MCI remained sparse and inconsistent. To collect fresh evidence, we evaluated the performance of several potential markers in a cohort of 57 MCI patients and 57 cognitively healthy controls. MCI patients showed obviously increased levels of plasma TNF-alpha (p = 0.045) and C-peptide (p = 0.004) as well as decreased levels of VEGF-A (p = 0.042) and PAI-1 (p = 0.019), compared with controls. In addition, our study detected significant correlations of plasma sTNFR-1 (MCI + Control: B = -6.529, p = 0.020; MCI: B = -9.865, p = 0.011) and sIL-2Ralpha (MCI + Control: B = -7.010, p = 0.007; MCI: B = -11.834, p = 0.003) levels with MoCA scores in the whole cohort and the MCI group. These findings corroborate the inflammatory and vascular hypothesis for dementia. Future studies are warranted to determine their potential as early biomarkers for cognitive deficits and explore the related mechanisms.

[2] *Carpenter M, Berry H, Pelletier AL*. **Clinically Relevant Drug-Drug Interactions in Primary Care**. American family physician 2019; 99:558-564.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31038898
ABSTRACT
Drug interactions are common in the primary care setting and are usually predictable. Identifying the most important and clinically relevant drug interactions in primary care is essential to patient safety. Strategies for reducing the risk of drug-drug interactions include minimizing the number of drugs prescribed, re-evaluating therapy on a regular basis, considering nonpharmacologic options, monitoring for signs and symptoms of toxicity or effectiveness, adjusting dosages of medications when indicated, and adjusting administration times. Inhibition or induction of cytochrome P450 drug metabolizing isoenzymes is the most common mechanism by which clinically important drug interactions occur. The antimicrobials most likely to affect the international normalized ratio significantly in patients receiving warfarin are trimethoprim/sulfamethoxazole, metronidazole, and fluconazole. An empiric warfarin dosage reduction of 30% to 50% upon initiation of amiodarone therapy is recommended. In patients receiving amiodarone, limit dosages of simvastatin to 20 mg per day and lovastatin to 40 mg per day. Beta blockers should be tapered and discontinued several days before clonidine withdrawal to reduce the risk of rebound hypertension. Spironolactone dosages should be limited to 25 mg daily when coadministered with potassium supplements. Avoid prescribing opioid cough medicines for patients receiving benzodiazepines or other central nervous system depressants, including alcohol. Physicians should consider consultation with a clinical pharmacist when clinical circumstances require the use of drugs with interaction potential.

[3] *Lefort C, Van Hul M, Delzenne NM et al.* **Hepatic MyD88 regulates liver Inflammation by altering the synthesis of oxysterols**. American journal of physiology. Endocrinology and metabolism 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31039009
ABSTRACT
This study aims to investigate the function of hepatic MyD88, a central adaptor of innate immunity, on metabolism. Although its role in inflammation is well known, we have recently discovered that MyD88 can also mediate energy, lipid and glucose metabolism.More precisely, we have reported that mice harboring hepatocyte-specific deletion of MyD88 (Myd88(DeltaHep)) were predisposed to glucose intolerance, liver fat accumulation and inflammation. However, the molecular events explaining the onset of hepatic disorders and inflammation remain to be elucidated. To investigate the molecular mechanism, Myd88(DeltaHep)and WT mice were challenged by two complementary approaches affecting liver lipid metabolism and immunity. The first one consisted of a short-term exposure to HFD whereas the second was an acute LPS injection. We discovered thatupon 3 days of HFD, Myd88(DeltaHep)mice displayed an increase in liver weight and liver lipids as compared to WT mice. Moreover, we found that bile acid and oxysterol metabolism were deeply affected by the absence of hepatic MyD88. Our data suggest that the negative feedback loop suppressing bile acid synthesis was impaired (i.e., ERK activity was decreased) in Myd88(DeltaHep)mice. Finally, the predisposed inflammation sensitivity displayed by Myd88(DeltaHep)mice may be caused by the accumulation of 25-OHC, an oxysterol linked to inflammatory response and metabolic disorders. This study highlights the importance of MyD88 on both liver fat accumulation and cholesterol-derived bioactive lipid synthesis. Those are two key features associated with metabolic syndrome. Therefore, investigating the regulation of hepatic MyD88 could lead to discover new therapeutic targets.

[4] *Chen Y, Dong J, Zhang X et al.* **Evacetrapib reduces prebeta-1 HDL in patients with atherosclerotic cardiovascular disease or diabetes**. Atherosclerosis 2019; 285:147-152.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31054484
ABSTRACT
BACKGROUND AND AIMS: Cholesteryl ester transfer protein (CETP) inhibitor-mediated induction of HDL-cholesterol has no effect on the protection from cardiovascular disease (CVD). However, the mechanism is still unknown. Data on the effects of this class of drugs on subclasses of HDL are either limited or insufficient. In this study, we investigated the effect of evacetrapib, a CETP inhibitor, on subclasses of HDL in patients with atherosclerotic cardiovascular disease or diabetes. METHODS: Baseline and 3-month post-treatment samples from atorvastatin 40mg plus evacetrapib 130mg (n=70) and atorvastatin 40mg plus placebo (n=30) arms were used for this purpose. Four subclasses of HDL (large HDL, medium HDL, small HDL, and prebeta-1 HDL) were separated according to their size and quantified by densitometry using a recently developed native polyacrylamide gel electrophoresis (PAGE) system. RESULTS: Relative to placebo, while evacetrapib treatment dramatically increased large HDL and medium HDL subclasses, it significantly reduced small HDL (27%) as well as prebeta-1 HDL (36%) particles. Evacetrapib treatment reduced total LDL, but also resulted in polydisperse LDL with LDL particles larger and smaller than the LDL subclasses of the placebo group. CONCLUSION:

Evacetrapib reduced prebeta-1 HDL and small HDL in patients with ASCVD or diabetes on statin. Prebeta-1 HDL and medium HDL are negatively interrelated. The results could give a clue to understand the effect of CETP inhibitors on cardiovascular outcomes.

[5] *De Backer G, Jankowski P, Kotseva K et al.* **Management of dyslipidaemia in patients with coronary heart disease: Results from the ESC-EORP EUROASPIRE V survey in 27 countries**. Atherosclerosis 2019; 285:135-146.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31054483
ABSTRACT
BACKGROUND AND AIMS: One of the objectives of the ESC-EORP EUROASPIRE V survey is to determine how well European guidelines on the management of dyslipidaemias are implemented in coronary patients. METHODS: Standardized methods were used by trained technicians to collect information on 7824 patients from 130 centers in 27 countries, from the medical records and at a visit at least 6 months after hospitalization for a coronary event. All lipid measurements were performed in one central laboratory. Patients were divided into three groups: on high-intensity LDL-C-lowering-drug therapy (LLT), on low or moderate-intensity LLT and on no LLT. RESULTS: At the time of the visit, almost half of the patients were on a high-intensity LLT. Between hospital discharge and the visit, LLT had been reduced in intensity or interrupted in 20.8% of the patients and had been started or increased in intensity in 11.7%. In those who had interrupted LLT or had reduced the intensity, intolerance to LLT and the advice of their physician were reported as the reason why in 15.8 and 36.8% of the cases, respectively. LDL-C control was better in those on a high-intensity LLT compared to those on low or moderate intensity LLT. LDL-C control was better in men than women and in patients with self-reported diabetes. CONCLUSIONS: The results of the EUROASPIRE V survey show that most coronary patients have a less than optimal management of LDL-C. More professional strategies are needed, aiming at lifestyle changes and LLT adapted to the need of the individual patient.

[6] *Perego C, Da Dalt L, Pirillo A et al.* **Cholesterol metabolism, pancreatic beta-cell function and diabetes**. Biochimica et biophysica acta. Molecular basis of disease 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31029825
ABSTRACT
Cholesterol plays an essential role in determining cell membrane physico-chemical characteristics and functions. A proper membrane structure is critical in pancreatic beta-cells for glucose-mediated insulin secretion, and alterations in cellular cholesterol content may negatively affect this process, leading to beta-cell dysfunction. The low density lipoprotein receptor (LDL-R) appears to play a relevant role in ss-cell dysfunction due to cholesterol accumulation. This observation raised the question of whether hypocholesterolemic drugs which increase LDL-R expression might bear diabetogenic properties, thus increasing the risk of new-onset diabetes or worsen glycaemic parameters in diabetic patients. Being at higher cardiovascular risk, diabetic patients are usually treated with hypolipidemic drugs to correct the atherogenic dyslipidemia characteristic of this pathological condition. Statin therapy has been associated with an increased incidence of new-onset diabetes (NOD), being the diabetogenic effect depending on the type and dose of statin. However, it is worth noting that the benefits on cardiovascular mortality largely exceed the increased risk associated with the development

of diabetes. Although genetic variants associated with lower levels of LDL-C are also associated with an increased NOD risk, clinical trials with lipid-lowering drugs other than statins, namely ezetimibe or monoclonal antibodies against PCSK9, did not observe an increase of developing diabetes. In summary, molecular evidence clearly points to a key role for cholesterol homeostasis in pancreatic beta-cell function which, in humans, is negatively affected by statins. Available data exclude that this could be the case for other hypocholesterolemic approaches, but long-term studies are warranted to explore this critical aspect.

[7] *Previsdomini M, Graziano E, Decosterd L et al.* **Severe rosuvastatin accumulation with rhabdomyolysis due to drug interactions and low cardiac output syndrome**. British journal of clinical pharmacology 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31050036
ABSTRACT

[8] *Khunti K, Jung H, Dans AL et al.* **Statin Use in Primary Prevention: A Simple Trial-Based Approach Compared With Guideline-Recommended Risk Algorithms for Selection of Eligible Patients**. The Canadian journal of cardiology 2019; 35:644-652.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31030865
ABSTRACT
BACKGROUND: Cardiovascular disease risk assessment tools help identify individuals likely to benefit from preventative therapies. In this study we compared outcomes using the American College of Cardiology/American Heart Association (ACC/AHA) risk algorithm and the Framingham Risk Score (FRS) tool in the Heart Outcomes Prevention Evaluation (HOPE)-3 study. METHODS: We compared outcomes using the ACC/AHA algorithm and the FRS with those seen in HOPE-3, which randomized participants to 10 mg rosuvastatin or placebo. The first coprimary outcome was the composite of death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke; second coprimary outcome additionally included heart failure, cardiac arrest, and revascularization. RESULTS: Relative risks using risk scores were similar to those observed in the HOPE-3. Hazards ratios for the first coprimary outcome according to risk categories of </= 10%, 10%-20%, and >/= 20% using the ACC/AHA algorithm were 0.82 (95% confidence interval [CI], 0.53-1.28), 0.72 (95% CI, 0.53-0.96), and 0.72 (95% CI, 0.55-0.93), and absolute risk reduction (ARR) of 0.18%, 1.33%, and 1.85%, respectively, over a median of 5.6 years. Corresponding results using the FRS were 0.69 (95% CI, 0.36-1.35), 0.73 (95% CI, 0.52-1.01), and 0.75 (95% CI, 0.60- 0.94); and ARR of 1.32%, 0.61%, and 1.43%. Hazard ratios for the second coprimary outcome were 0.77 (95% CI, 0.51-1.14), 0.73 (95% CI, 0.56-0.95), and 0.74 (95% CI, 0.58-0.94); and ARR of 0.36%, 1.49%, and 1.85%, using the ACC/AHA algorithm and 0.76 (95% CI, 0.41-1.41), 0.70 (95% CI, 0.52-0.95), and 0.76 (95% CI, 0.62-0.94); and ARR of 1.08%, 0.83%, and 1.56% using the FRS. CONCLUSIONS: The pragmatic HOPE-3 trial approach identifies in an ethnically diverse primary prevention population individuals at intermediate risk who benefit from statin therapy using simple clinical characteristics without the need for complex, currently used risk assessment tools.

[9] *Chang SN, Wu CK, Lai LP et al.* **The effect and molecular mechanism of statins on the expression of human anti-coagulation genes**. Cell Mol Life Sci 2019.

PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31053884

ABSTRACT

Statins are potent lipid-lowering drugs. Large prospective clinical trials have shown the anti-thrombotic effect of statins, e.g., preventing deep vein thrombosis. However, the mechanism underlying the beneficial effect of statins in reducing thrombus formation remains to be established. We, thus, conduct this study to investigate the potential molecular mechanisms. The cultured human hepatoma cells (HepG2) were used as the in vitro model. The human protein C gene promoter was cloned into the luciferase reporter to study the transcriptional regulation of human protein C gene. Wistar rats fed with simvastatin (5 mg/kg day) were used as the in vivo model. We found that simvastatin increased the expression of protein C in hepatocytes (361 +/- 64% and 313 +/- 59% after 2 h and 6 h of stimulation, respectively, both $p < 0.01$). In the animal study, the serum protein C levels were increased in the simvastatin-treated group (7 +/- 2.2 unit/ml vs 23.4 +/- 19.3 unit/ml and 23.4 +/- 18.2 unit/ml and 1 and 2 weeks of treatment, respectively, both $p < 0.05$). Regarding the possible molecular mechanism, we found that the level of hepatocyte nuclear factor 1alpha (HNF1alpha) was also increased in both the in vivo and in vitro models. We found that the protein C promoter activity was increased by simvastatin, and this effect was inhibited by HNF1alpha knockdown and constitutively active Rac1. Therefore, stains may modulate protein C expression through small GTPase Rac 1 and HNF1alpha.

[10] *Lopez-Fandino R*. **Role of dietary lipids in food allergy**. Critical reviews in food science and nutrition 2019:1-18.

PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31032635

ABSTRACT

The prevalence of food allergy is raising in industrialized countries, but the mechanisms behind this increased incidence are not fully understood. Environmental factors are believed to play a role in allergic diseases, including lifestyle influences, such as diet. There is a close relationship between allergens and lipids, with many allergenic proteins having the ability to bind lipids. Dietary lipids exert pro-inflammatory or anti-inflammatory functions on cells of the innate immunity and influence antigen presentation to cells of the adaptive immunity. In addition to modifying the immunostimulating properties of proteins, lipids also alter their digestibility and intestinal absorption, changing allergen bioavailability. This study provides an overview of the role of dietary lipids in food allergy, taking into account epidemiological information, as well as results of mechanistic investigations using in vivo, ex vivo and in vitro models. The emerging link among high-fat diets, obesity, and allergy is also discussed.

[11] *Carmena R, Betteridge DJ*. **Diabetogenic Action of Statins: Mechanisms**. Current atherosclerosis reports 2019; 21:23.

PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31037345

ABSTRACT

PURPOSE OF REVIEW: Observational studies and meta-analyses of randomized clinical trials data have revealed a 10-12% increased risk of new-onset diabetes (NOD) associated with statin therapy; the risk is increased with intensive treatment regimens and in people with features of the metabolic syndrome or prediabetes. The purpose of this review is to provide an updated

summary of what is known about the potential mechanisms for the diabetogenic effect of statins. RECENT FINDINGS: Hydroxyl methyl glutaryl coenzyme A reductase (HMGCoAR) is the target of statin therapy and the activity of this key enzyme in cholesterol synthesis is reduced by statins in a partial and reversible way. Mendelian randomization studies suggest that the effect of statins on glucose homeostasis reflect reduced activity of HMGCoAR. In vitro and in vivo data indicate that statins reduce synthesis of mevalonate pathway products and increase cholesterol loading, leading to impaired beta-cell function and decreased insulin sensitivity and insulin release. While this effect has been thought to be a drug class effect, recent insights suggest that pravastatin and pitavastatin could exhibit neutral effects on glycaemic parameters in patients with and without diabetes mellitus. The mechanisms by which statins might lead to the development of NOD are unclear. The inhibition of HMGCoAR activity by statins appears to be a key mechanism. It is difficult to offer a comprehensive view regarding the diabetogenic effect of statins because our understanding of the most widely recognized potential mechanisms, i.e. underlying statin-induced reduction of insulin sensitivity and/or insulin secretion, is still far from complete. The existence of this dual mechanism is supported by the results of a study in a large group of non-diabetic men, showing that a 46% higher risk of NOD in statin users compared to non-users was accompanied by a significant 12% reduction in insulin secretion and a 24.3% increase in insulin resistance. Although statin therapy is associated with a modest increase in the risk of NOD (about one per thousand patient-years), patients should be reassured that the benefits of statins in preventing cardiovascular disease (CVD) events far outweigh the potential risk from elevation in plasma glucose.

[12] *Thompson G, Parhofer KG*. **Current Role of Lipoprotein Apheresis**. Current atherosclerosis reports 2019; 21:26.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31041550
ABSTRACT
PURPOSE OF REVIEW: Lipoprotein apheresis is a very efficient but time-consuming and expensive method of lowering levels of low-density lipoprotein cholesterol, lipoprotein(a)) and other apoB containing lipoproteins, including triglyceride-rich lipoproteins. First introduced almost 45 years ago, it has long been a therapy of "last resort" for dyslipidaemias that cannot otherwise be managed. In recent years new, very potent lipid-lowering drugs have been developed and the purpose of this review is to define the role of lipoprotein apheresis in the current setting. RECENT FINDINGS: Lipoprotein apheresis still plays an important role in managing patients with homozygous FH and some patients with other forms of hypercholesterolaemia and cardiovascular disease. In particular, patients not achieving treatment goals despite modern lipid-lowering drugs, either because these are not tolerated or the response is insufficient. Recently, lipoprotein(a) has emerged as an important cardiovascular risk factor and lipoprotein apheresis has been used to decrease lipoprotein(a) concentrations in patients with marked elevations and cardiovascular disease. However, there is considerable heterogeneity concerning the recommendations by scientific bodies as to which patient groups should be treated with lipoprotein apheresis. Lipoprotein apheresis remains an important tool for the management of patients with severe drug-resistant dyslipidaemias, especially those with homozygous FH.

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[13] *Sinning D, Landmesser U*. **Is There a Need to Revise Goals in the Management of Dyslipidemias?** Current cardiology reports 2019; 21:51.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31041621
ABSTRACT
PURPOSE OF REVIEW: Current guidelines on the management of patients with dyslipidemias recommend specific risk-dependent low-density lipoprotein cholesterol (LDL-C) treatment goals. Recently, several randomized clinical trials have investigated further lowering of LDL-C in addition to statin therapy using novel therapeutic approaches and examined their effects on cardiovascular (CV) risk. This review summarizes newly available data on efficacy and safety of lowering LDL-C beyond statin therapy and below current treatment targets. RECENT FINDINGS: In patients at very high risk for CV events, a significant residual risk remains when failing to achieve significant LDL-C reduction on maximally tolerated statin therapy alone. Further lowering of LDL-C, even beyond current treatment targets, has been shown to be safe and was associated with a further reduced CV risk reduction. The relative risk reduction per change in LDL-C levels has been observed to be consistent even in patient populations achieving extremely low levels of LDL-C. In patients at very high CV risk, further lowering of LDL-C beyond statin therapy and present treatment targets has been observed to further reduce CV risk, which may be foremost relevant for patients at a particular high absolute CV risk, e.g., for patients with progressive and/or very extensive coronary disease.

[14] *Yang M, Liu Y, Wen C et al.* **Association between spousal diabetes status and diabetic retinopathy in Chinese patients with type 2 diabetes**. Diabetes & vascular disease research 2019:1479164119844695.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31046451
ABSTRACT
PURPOSE: To evaluate the association between spousal diabetes status and the prevalence of diabetic retinopathy in Chinese patients with type 2 diabetes. METHODS: A cross-sectional community-based study was performed in 1510 patients with type 2 diabetes in Shanghai, China. Non-mydriatic digital fundus photography was used to detect diabetic retinopathy. Spousal diabetes status was assessed using a standardised interview questionnaire. RESULTS: The prevalence of diabetic retinopathy was significantly lower in patients who had diabetic spouses, compared with those who did not (20.2% vs 29.1%, p 0.01). The fully adjusted odds ratio for diabetic retinopathy in those had diabetic spouses was decreased by 36% (odds ratio = 0.64, 95% confidence interval = 0.42-1.00, p = 0.048). The negative correlation between spousal diabetes status and diabetic retinopathy was presented in patients with the duration of diabetes 10 years, those with HbA1c 7% and those not using lipid-lowering drugs (odds ratio = 0.31, 95% confidence interval = 0.13-0.74, p = 0.0082; odds ratio = 0.50, 95% confidence interval = 0.27-0.94, p = 0.031; odds ratio = 0.58, 95% confidence interval = 0.37-0.92, p = 0.021, respectively). CONCLUSION: We demonstrated that spousal diabetes was associated with a lower diabetic retinopathy prevalence in Chinese patients with type 2 diabetes.

[15] *Kjellmo CA, Pop G, Lappegard KT, Hovland A*. **Intensive lipid lowering therapy reduces large, but not small, dense low-density lipoprotein particles measured by gel electrophoresis,**

in elderly patients with atrial fibrillation. European journal of preventive cardiology 2019:2047487319845966.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31032636
ABSTRACT

[16] *Doggrell SA*. **Clinical trials of eicosapentaenoic acid (EPA) prescription products for the treatment of hypertriglyceridemia**. Expert opinion on pharmacotherapy 2019:1-5.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31038369
ABSTRACT
INTRODUCTION: Hypertriglyceridemia is common and increases cardiovascular risk. Fish oil decreases triglyceride levels, but also increases low-density lipoprotein (LDL) cholesterol, which may negate any cardiovascular benefits. EPA, a component of fish oil, reduces triglyceride levels without increasing LDL cholesterol. Areas covered: Two forms of purified EPA ethyl ester are available on prescription. This review considers the clinical trials of these purified esters to treat hypertriglyceridemia and shows that the EPA ethyl esters reduce triglyceride levels and reduce cardiovascular events. Expert opinion: To date, the effects of the purified EPA ethyl esters on cardiovascular events have only been tested in subjects taking statins. With statin treatment, if hypertriglyceridemia persists, it may be worthwhile considering adding an EPA ethyl ester. However, as the fibrates reduce the triglyceride levels by similar amounts to the EPA ethyl esters, while increasing the levels of HDL cholesterol, they are an alternative to EPA ethyl esters in combination with statins. As the proprotein convertase subtilisin-kexin type 9 (PCSK9) inhibitors reduce triglycerides to a similar extent to the EPA ethyl ester, while reducing LDL cholesterol levels to a greater extent than the statins, they should be considered as an alternative to the statin/EPA ethyl ester combination.

[17] *Wu Q, Wang Q, Fu J, Ren R*. **Polysaccharides derived from natural sources regulate triglyceride and cholesterol metabolism: a review of the mechanisms**. Food & function 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31049523
ABSTRACT
With great changes in people's lifestyles, the incidence of hyperlipidaemia has dramatically increased in recent years. Numerous studies have demonstrated that natural polysaccharides have lipid lowering effects. In this review, the causes and mechanisms of hyperlipidaemia are discussed in order to better understand how polysaccharides alleviate hyperlipidaemia. Natural polysaccharides reduce triglyceride levels through ATGL-(PPAR-alpha)/(PGC-1alpha), (SREBP-1c)-ACC/FAS and ACC-CPT1 signal pathways, and exert cholesterol lowering effects via (SREBP-2)-HMGCR and bile acid biosynthesis pathways. Activation of adenosine monophosphate-activated protein kinase (AMPK) is the key factor that mediated the simultaneous regulation of both glucose and lipid metabolism by polysaccharides. The new discovery of polysaccharides increasing the production of endogenous H2S, an important physiological gaseous signaling molecule, is also discussed. Collectively, the current available data suggest that natural polysaccharides could be potentially developed as new and safe lipid-lowering drugs; yet further mechanistic and clinical studies are required during this long-term process.

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[18] *Oppi S, Luscher TF, Stein S*. **Mouse Models for Atherosclerosis Research-Which Is My Line?** Frontiers in cardiovascular medicine 2019; 6:46.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31032262
ABSTRACT
Atherosclerosis is one of the primary causes of cardiovascular disease and mortality. This chronic immunometabolic disease evolves during decades in humans and encompasses different organs and immune cell types, as well as local and systemic processes that promote the progression of the disease. The most frequently used animal model to study these atherogenic processes and inter-organ crosstalk in a short time frame are genetically modified mouse models. Some models have been used throughout the last decades, and some others been developed recently. These models have important differences in cholesterol and lipoprotein metabolism, reverse cholesterol transport pathway, obesity and diabetes as well as inflammatory processes. Therefore, the disease develops and progresses differently in the various mouse models. Since atherosclerosis is a multifaceted disease and many processes contribute to its progression, the choice of the right mouse model is important to study specific aspects of the disease. We will describe the different mouse models and provide a roadmap to facilitate current and future atherosclerosis researchers to choose the right model depending on their scientific question.

[19] *Cheng PC, Hsu SR, Li JC et al.* **Plasma Low-Density Lipoprotein Cholesterol Correlates With Heart Function in Individuals With Type 2 Diabetes Mellitus: A Cross-Sectional Study**. Frontiers in endocrinology 2019; 10:234.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31031709
ABSTRACT
Background: Heart failure is a frequent complication of type 2 diabetes mellitus (T2DM). Plasma cholesterol, particularly the proatherogenic low-density lipoprotein (LDL) cholesterol, impairs heart function by promoting atheroma formation and ventricular dysfunction. Considering the established effect of cholesterol on the cardiovascular system, we hypothesized that plasma LDL cholesterol may influence left ventricular function in individuals with T2DM. Methods: This cross-sectional study was conducted at a tertiary care hospital in Taiwan. Enrollment criteria were patients exceeding 21 years of age with T2DM who received antidiabetic and cholesterol-lowering medications. Candidates were excluded if they had heart failure, acute cardiovascular events, or familial hypercholesterolemia. Participants received blood sampling for plasma lipids after a 12-h fast, followed by transthoracic echocardiography in the cardiology clinic. Results: The study enrolled 118 participants who were divided into two groups according to their plasma LDL cholesterol levels. Demographic characteristics including age (69.7 vs. 66.9 years, P = 0.159), body mass index (26.2 vs. 25.9 kg/m(2), P = 0.66), diabetes duration (5.4 vs. 5.1 years, P = 0.48), hemoglobin A1c (7.2 vs. 7.5%, P = 0.225), and systolic blood pressure (129 vs. 130 mm Hg, P = 0.735) were similar between these groups. Moreover, all participants received similar antihypertensive medications. Participants with lower plasma LDL cholesterol levels had better heart function, as measured by the left ventricular ejection fraction (LVEF), than patients with higher LDL cholesterol levels (58.0 vs. 50.5%, P = 0.022). Multivariate regression analysis also showed an inverse correlation between plasma LDL cholesterol and left ventricular function (beta coefficient: -0.110, P = 0.024). Conclusion: This study observed an inverse correlation

between plasma LDL cholesterol and heart function in individuals with T2DM. Patients with higher levels of plasma LDL cholesterol had worse left ventricular function. Therefore, plasma LDL cholesterol may be a modifiable risk factor of heart failure in diabetes, but prospective studies are necessary to confirm this finding.

[20] *Alecu I, Bennett SAL*. **Dysregulated Lipid Metabolism and Its Role in alpha-Synucleinopathy in Parkinson's Disease**. Frontiers in neuroscience 2019; 13:328.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31031582
ABSTRACT
Parkinson's disease (PD) is the second most common neurodegenerative disease, the main pathological hallmark of which is the accumulation of alpha-synuclein (alpha-syn) and the formation of filamentous aggregates called Lewy bodies in the brainstem, limbic system, and cortical areas. Lipidomics is a newly emerging field which can provide fresh insights and new answers that will enhance our capacity for early diagnosis, tracking disease progression, predicting critical endpoints, and identifying risk in pre-symptomatic persons. In recent years, lipids have been implicated in many aspects of PD pathology. Biophysical and lipidomic studies have demonstrated that alpha-syn binds preferentially not only to specific lipid families but also to specific molecular species and that these lipid-protein complexes enhance its interaction with synaptic membranes, influence its oligomerization and aggregation, and interfere with the catalytic activity of cytoplasmic lipid enzymes and lysosomal lipases, thereby affecting lipid metabolism. The genetic link between aberrant lipid metabolism and PD is even more direct, with mutations in GBA and SMPD1 enhancing PD risk in humans and loss of GALC function increasing alpha-syn aggregation and accumulation in experimental murine models. Moreover, a number of lipidomic studies have reported PD-specific lipid alterations in both patient brains and plasma, including alterations in the lipid composition of lipid rafts in the frontal cortex. A further aspect of lipid dysregulation promoting PD pathogenesis is oxidative stress and inflammation, with proinflammatory lipid mediators such as platelet activating factors (PAFs) playing key roles in arbitrating the progressive neurodegeneration seen in PD linked to alpha-syn intracellular trafficking. Lastly, there are a number of genetic risk factors of PD which are involved in normal lipid metabolism and function. Genes such as PLA2G6 and SCARB2, which are involved in glycerophospholipid and sphingolipid metabolism either directly or indirectly are associated with risk of PD. This review seeks to describe these facets of metabolic lipid dysregulation as they relate to PD pathology and potential pathomechanisms involved in disease progression, while highlighting incongruous findings and gaps in knowledge that necessitate further research.

[21] *Andreou I, Stone PH, Ikonomidis I et al.* **Recurrent atherosclerosis complications as a mechanism for stent failure**. Hellenic journal of cardiology : HJC = Hellenike kardiologike epitheorese 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31034959
ABSTRACT
Stents are an indispensable tool in the percutaneous treatment of symptomatic coronary artery disease. Yet, stent failure due to restenosis or thrombosis may compromise their clinical benefit, carrying substantial morbidity and mortality. Despite improvements in device design

and adjunctive medical treatment, stent failure still occurs during long-term follow-up, suggesting that this may be an issue that persists for many years, perhaps indefinitely. Numerous studies during the last decade have highlighted the previously underappreciated pivotal role of atherosclerosis in stent failure. We review evolving evidence on the role of atherosclerosis in stent restenosis and thrombosis, differentiating between de novo in-stent atherosclerosis development (i.e., neoatherosclerosis) and progression of pre-existing underlying atherosclerosis (i.e., paleoatherosclerosis), a distinction with potentially important clinical implications. We conclude with a concept that provides a unifying pathophysiology for these significant problems in the field of interventional cardiology based on the progression and destabilization of atherosclerosis.

[22] *Lye CT, Mukherjee S, Burns SF*. **Combining Plant Sterols With Walking Lowers Postprandial Triacylglycerol to a Greater Extent Than Walking Alone in Chinese Men With Elevated Body Mass Index**. International journal of sport nutrition and exercise metabolism 2019:1-24.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31034251

ABSTRACT

This study examined if plant sterols and walking reduce postprandial triacylglycerol (TAG) concentrations in Chinese men with elevated body mass index (BMI, >/= 23.5 kg/m(2)). Fifteen Chinese men [mean (SD): age 25 (3) years; BMI 26.2 (1.5) kg/m(2)] completed four 10-day trials in random order with a 7-10 day washout between trials: (i) daily consumption of a control margarine whilst sedentary (C-S); (ii) daily consumption of margarine containing 2 g/day of plant sterols whilst sedentary (PS-S); (iii) daily consumption of a control margarine with 30 minutes daily walking (C-W) ; and (iv) daily consumption of margarine containing 2 g/day of plant sterols with 30 minutes daily walking (PS-W). On day 11 of each trial postprandial TAG were measured after a high-fat milkshake. The 5-hour total area under the TAG curve was 22%, 25% and 12% lower on PS-W (mean (SD): 8.9 (4.3) mmol5h/L) than C-S (11.4 (4.5) mmol5h/L; p = .005; d = .56), PS-S (11.9 (4.9) mmol5h/L; p = .004; d = .67) and C-W (10.1 (4.4) mmol5h/L; p = .044; d = .27) trials, respectively. Similarly, 5-hour incremental area for PS-W (4.5 (2.7) mmol5h/L) was 31%, 32% and 18% lower than C-S (6.6 (3.3) mmol5h/L; p = .005; d = .62), PS-S (6.6 (3.4) mmol5h/L; p = .004; d = .64) and C-W (5.5 (2.8) mmol5h/L; p = .032; d = .29). Ten days of daily plant sterol intake combined with walking presents an intervention strategy to lower postprandial TAG in Chinese men with elevated BMI.

[23] *Aghajanzadeh M, Ghannad F, Zamani M et al.* **Anti-inflammatory effect of rosuvastatin using diblock amphiphilic copolymer: Synthesis, characterization, in vitro and in vivo study**. Journal of biomaterials applications 2019:885328219847055.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31053039

ABSTRACT

In this study, we synthesized methoxy poly(ethylene glycol)-poly(epsilon-caprolactone) diblock copolymers as water soluble nanocarriers to investigate the in vivo anti-inflammatory characteristic of rosuvastatin-loaded nanocarriers. For determining the structure of prepared nanocarriers, we used proton nuclear magnetic resonance, gel permeation chromatography, Fourier-transform infrared spectroscopy, atomic force microscopy, and dynamic scanning calorimetry method. Nano-precipitation method was used for loading of rosuvastatin into

copolymeric nanocarriers. The goal of this study is investigation of the anti-inflammatory effects of rosuvastatin-loaded nanocarriers in comparison with indomethacin. The paw edema thickness was measured during 4 h after gavage of nanocarriers in acute inflammation-induced rats, and the ability of nanocarriers to inhibit the edema was calculated. Rosuvastatin was loaded in nanocarriers with a loading capacity of 9.38 +/- 0.96% and an encapsulation efficiency of 62.50 +/- 0.84%; moreover, rosuvastatin and rosuvastatin nanocarriers displayed considerable anti-inflammatory activity in this study. This study indicated that rosuvastatin and rosuvastatin nanocarriers have anti-inflammatory characteristic and we can conclude that in addition to lipid lowering affect, statins have potential for anti-inflammatory activity.

[24] *Ishihara-Yusa S, Fujimura T, Lyu C et al.* **Successful treatment of multiple eruptive adult xanthogranuloma concomitant with xanthelasma palpebrarum with probucol**. The Journal of dermatology 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31050040
ABSTRACT

[25] *Tamargo C, Sando K, Prados Y, Cowart K*. **Change in Proportion of Days Covered for Statins Following Implementation of a Pharmacy Student Adherence Outreach Program**. Journal of managed care & specialty pharmacy 2019; 25:588-592.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31039060
ABSTRACT
BACKGROUND: Nearly half of statin users discontinue therapy within the first year of treatment. Nonadherence to statin therapy may lead to an increased risk of atherosclerotic cardiovascular disease and, thus, higher costs due to hospitalizations. Value-based care models, such as accountable care organizations (ACO), are measured on adherence rates to statins through proportion of days covered (PDC). However, there is little research describing pharmacy student-based interventions within value-based care models. OBJECTIVES: To (a) identify mean change in PDC for statins following implementation of a pharmacy student adherence outreach program and (b) identify the proportion of patients converted to PDC >/= 0.80 following the implementation of the outreach program. METHODS: This single-center retrospective quasi-experimental study included patients actively enrolled in a Humana Medicare Advantage Prescription Drug (MA-PD) plan who completed at least 1 adherence outreach telephone call performed by a pharmacy student between January 1, 2017, and December 31, 2017. RESULTS: 99 patients met inclusion criteria. Atorvastatin was the most commonly prescribed statin (43%), followed by simvastatin (38%). Sixty-four percent of patients had a baseline PDC of < 0.80. Mean (SD) PDC was 0.66 (+/-0.24) before the pharmacy student adherence outreach intervention, and 0.79 (+/- 0.23)-a 0.13 increase-after the pharmacy student adherence outreach intervention (P < 0.001). Among patients who had PDC < 0.80 at baseline, 35% of patients (n = 35) were converted to PDC >/= 0.80 (P < 0.001), and 5% of patients with a baseline PDC >/= 0.80 had a decrease in PDC to < 0.80 following the intervention. CONCLUSIONS: Among patients enrolled in a Humana MA-PD plan within an ACO, mean PDC for statins increased following exposure to a pharmacy student adherence outreach program. One third of patients converted their PDCs to >/= 0.80 following the intervention. Value-based care programs may consider incorporating pharmacy student services to improve

adherence to statins. DISCLOSURES: No outside funding supported this research. The authors have no financial conflicts of interest to disclose. At the time of conducting this research, all authors were employed at Nova Southeastern University. Preliminary results were presented as a poster at the AMCP Managed Care & Specialty Pharmacy Annual Meeting; April 23-26, 2018; Boston, MA.

[26] *Kingstone LL, Currie GM, Torres C*. **The Pathogenesis, Analysis, and Imaging Methods of Atherosclerotic Disease of the Carotid Artery: Review of the Literature**. Journal of medical imaging and radiation sciences 2012; 43:84-94.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31052031
ABSTRACT
Cerebrovascular (CVA) accidents are the second leading cause of death worldwide and their numbers are increasing. Strokes can arise from several causes, with extracranial carotid artery atherosclerosis (CAS) being one of the leading causes. CAS causes these strokes either by diminishing blood flow distal to the diseased stenotic segment of the artery or, as more recently discovered, by a thromboembolic event of material from the plaque site itself. The specific etiology of CAS is unknown, but causative factors in the formation of atherosclerotic plaque of the carotid arteries have been linked to specific morphological areas within the plaque that may be vulnerable to rupture, leading to thromboemboli into the cerebrovascular circulation. The current means for imaging and reporting CAS is through the measurement of the severity of luminal diameter stenosis caused by atherosclerotic disease. Recent developments in medical imaging techniques have expanded the role of early imaging and detection of CAS. Although current practice uses luminal narrowing as the surrogate marker to assess CAS, it has been recently discovered that plaque morphology and composition may help predict the clinical behavior of CAS and better determine the necessary medical intervention or risk of stroke. Although a single optimized imaging modality for standard CAS imaging has not been established or agreed on, various modalities can provide key elements to a successful exam. This review article will evaluate the most commonly used methods for CAS imaging along with the new and upcoming uses, advantages, and limitations for advanced CAS imaging.

[27] *Mamann N, Lemale J, Karsenty A et al.* **Intermediate-Term Efficacy and Tolerance of Statins in Children**. J Pediatr 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31053349
ABSTRACT
OBJECTIVES: To evaluate the intermediate-term efficacy and tolerance of statins in children and adolescents with familial hypercholesterolemia. STUDY DESIGN: A total of 131 children or adolescents treated with statins for familial hypercholesterolemia were prospectively included. The efficacy of treatment was established by the percentage of children who achieved low density lipoprotein-cholesterol (LDL-C) levels <160 mg/dL during treatment. Treatment tolerance was evaluated by the occurrence of clinical or laboratory side effects, regularity of increases in height and weight, and pubertal development. RESULTS: The median duration of treatment with statins was 4 years. A median decrease of 32% in LDL-C levels was observed (P < .0001). The therapeutic target (LDL-C <160 mg/dL) was achieved in 67% of cases. Increases in height and weight and sexual maturation were not affected by the treatment. Minor side

effects were reported for 24 (18.4%) patients including 3 cases of a clinically asymptomatic increase in creatine phosphokinase (CPK) levels, 2 cases of an increase in CPK levels with muscular symptoms, 14 cases of myalgia without an increase in CPK levels, 3 cases of abdominal pain, 1 case of dysuria, and 1 case of diffuse pain. None of these side effects led to the discontinuation of statin therapy, although a change of statin was required in 7 cases. This new statin was tolerated in all cases. No patients had abnormal liver function during treatment. CONCLUSIONS: The results of this large cohort confirm the intermediate-term safety and efficacy of statin therapy in children with familial hypercholesterolemia.

[28] *Baker MA, Nandivada P, Mitchell PD et al.* **Omega-3 fatty acids are protective in hepatic ischemia reperfusion injury in the absence of GPR120 signaling**. Journal of pediatric surgery 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31036368
ABSTRACT
BACKGROUND: A single dose of IV fish oil (FO) before hepatic ischemia reperfusion injury (HIRI) increases hepatocyte proliferation and reduces necrosis in wild type (WT) mice. It has been suggested that the GPR120 receptor on Kupffer cells mediates FO's ability to reduce HIRI. The purpose of this study was to determine whether GPR120 is required for FO to reduce HIRI. METHODS: Sixty-four (n=8/group) adult male WT (C57BL/6) and GPR120 knockout (KO) mice received IV FO (1g/kg) or saline 1h prior to HIRI or sham operation. Mice were euthanized 24h postoperatively for analysis of hepatic histology, NFkappaB activity, and serum alanine transaminase (ALT) levels. RESULTS: FO pretreated livers had less necrosis after HIRI than saline pretreated livers in both WT (mean+/-SEM 25.9+/-7.3% less, P=0.007) and KO (36.6+/-7.3% less, P<0.0001) mice. There was no significant difference in percent necrosis between WT-FO and KO-FO groups. Sham groups demonstrated minimal necrosis (0-1.9%). Mean [95% CI] ALT after HIRI was significantly higher (P=0.04) in WT-Saline mice (1604U/L [751-3427]) compared to WT-FO (321U/L [150-686]) but was not significantly higher in KO-Saline mice compared to KO-FO. There were no differences in ALT between WT-FO and KO-FO mice who underwent HIRI or between groups who underwent sham surgery. There were no differences in NFkappaB or IKKbeta activation among groups as measured by Western blot analysis. CONCLUSIONS: IV FO pretreatment was able to reduce HIRI in GPR120 KO mice, suggesting the hepatoprotective effects of FO are not mediated by GPR120 alone.

[29] *Bonaparte A, Tansey C, Wiebe M et al.* **The effect of atorvastatin on haemostatic parameters in apparently healthy dogs**. The Journal of small animal practice 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31044427
ABSTRACT
OBJECTIVE: To determine the effect of atorvastatin on haemostatic parameters as measured by prothrombin time, activated partial thromboplastin time and thromboelastography in apparently healthy dogs administered 2 mg/kg orally once daily for 1 week. MATERIALS AND METHODS: Prospective study of 20 apparently healthy client-owned dogs at a small animal specialty hospital. Dogs had a baseline complete blood count, serum chemistry profile, fibrinogen, platelet count, prothrombin time, activated partial thromboplastin time and thromboelastography performed. Each dog was then administered approximately 2 mg/kg of

atorvastatin orally once daily for 1 week, and the laboratory tests were repeated. Adverse effects attributed to atorvastatin were recorded. RESULTS: All 20 enrolled dogs completed the study. Dogs received a median dose of 2.06 mg/kg (range 1.94 to 2.44 mg/kg) atorvastatin once daily, which was associated with a significant increase in pulse rate, mean corpuscular haemoglobin concentration, albumin and a significant decrease in mean corpuscular volume, cholesterol and lipase values compared with baseline. On thromboelastography, there was a significant increase in maximum amplitude, G, coagulation index, amplitude at 30 minutes, amplitude at 60 minutes and significant decrease in percentage of clot lysed at 30 minutes and percentage of clot lysed at 60 minutes values compared with baseline. Six dogs had a noticeable increase in appetite. CLINICAL SIGNIFICANCE: The results of this study suggest that atorvastatin may produce a procoagulant effect in dogs, although the clinical significance is unclear. Polyphagia was the most commonly reported adverse effect.

[30] *Qamar A, Giugliano RP, Sabatine MS*. **Interindividual and Intraindividual Responses to PCSK9 Inhibition-Reply**. JAMA cardiology 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31042267
ABSTRACT

[31] *Thompson PD*. **Interindividual and Intraindividual Responses to PCSK9 Inhibition**. JAMA cardiology 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31042263
ABSTRACT

[32] *Chuan J, Qian Z, Zhang Y et al.* **The association of the PCSK9 rs562556 polymorphism with serum lipids level: a meta-analysis**. Lipids in health and disease 2019; 18:105.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31036026
ABSTRACT
BACKGROUND: Studies had investigated the associations between proprotein convertase subtilisin/kexin type 9 SNP rs562556 and serum lipids levels and response to statin treatment, however, the results remained inconclusive. We conducted this meta-analysis to elucidate the relationship of rs562556 and serum lipids levels. METHODS: All eligible studies met the inclusion criteria were retrieved from multiple databases. Relative data were extracted from each study. Review Manager (version 5.3.5) and STATA 12.0 software was used to perform this meta-analysis. Pooled standardized mean difference (SMD) with 95% CI was employed to evaluate the association of rs562556 with serum lipids levels. RESULTS: A total of 7 eligible articles involving 4742 subjects were included in the final meta-analysis. The results revealed that the G carriers had lower levels of total cholesterol (SMD: 0.14, 95% Cl: 0.06-0.23, P = 0.001) and LDL-C(SMD: 0.13, 95% Cl: -0.55-0.22,P = 0.002) than the non-carriers. The statistical results also illustrated that the G carriers had lower relative risk (SMD: 1.38, 95% Cl: 1.02-1.85, P = 0.003) than the non-carriers. CONCLUSIONS: The results of the current meta-analysis for the first time indicated the relevance of rs562556 and lower serum cholesterol levels.

[33] *Wang JF, Zhang HM, Li YY et al.* **A combination of omega-3 and plant sterols regulate glucose and lipid metabolism in individuals with impaired glucose regulation: a randomized and controlled clinical trial**. Lipids in health and disease 2019; 18:106.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31043161
ABSTRACT
BACKGROUND: Lipid metabolism imbalance has been recognized as one of the major drivers of impaired glucose metabolism in the context of type 2 diabetes mellitus (T2DM), the rates of which are steadily increasing worldwide. Impaired glucose regulation (IGR) plays a vital role in the prevention and treatment of T2DM. The goal of this study was to further clarify whether the combination of plant sterols (PS) and omega-3 fatty acids yields any synergistic effect that enhances the prevention and treatment of IGR. METHODS: A total of 200 participants were randomized to receive PS and omega-3 fatty acids (n = 50), PS alone (n = 50), omega-3 fatty acids alone (n = 50), or placebo soy bean powder plus placebo capsules (n = 50) for 12 weeks. Patient characteristics including body composition, blood pressure, glucose metabolism (Fasting plasma glucose (FPG), fasting insulin (FINS), glycosylated hemoglobin (HbA1c), Homeostasis Model Assessment of Insulin Resistance (HOMA-IR)), lipid metabolism (TG, TC, HDL-C, LDL-C) and inflammatory factors (Hs-CRP, IL-6) were all monitored in these IGR individuals. RESULTS: Compared to the placebo group, the group receiving the combined intervention exhibited significantly decreased TG, HDL-C, FBG, HOMA-IR and HbA1c. Omega-3 fatty acids alone were associated with significant reductions in waistline, TG, FBG, HOMA-IR and Hs-CRP. PS alone was only associated with decreased TG and Hs-CRP. No interventions produced significant changes in body weight, BMI, blood pressure, FINS, body fat percentage, visceral fat rating, TC, LDL-C or IL-6. CONCLUSIONS: In summary, this study has demonstrated for the first time that PS, omega-3 fatty acids or the combination thereof significantly improved inflammation, insulin resistance, as well as glucose and lipid metabolism in IGR individuals. These findings may provide a scientific basis for the development of nutritional products incorporating PS and omega-3 fatty acids, and also for the development of nutritional supplement strategies aimed at preventing the development of disease in the IGR population.

[34] *Roopmani P, Krishnan UM*. **Corrigendum to "Harnessing the pleiotropic effects of atorvastatin-fenofibrate combination for cardiovascular stents" [Mater. Sci. Eng. C 92 (2018) 875-891]**. Materials science & engineering. C, Materials for biological applications 2019; 101:707-708.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31029364
ABSTRACT

[35] *Siniawski D, Masson W, Rossi E et al.* **[Eligibility for the indication of PCSK9 inhibitors according to the recommendations of different scientific societies]**. Medicina 2019; 79:104-110.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31048275
ABSTRACT
LDL-cholesterol (LDL-C) lowering is a primary objective in cardiovascular prevention. Recent studies demonstrated clinical benefit when proprotein convertase subtilisin/kexin-9 inhibitors (PCSK9i) were added to the treatment in patients who had not achieved the LDL-C goal despite

being treated with high intensity statins and ezetimibe, however the use of these drugs is limited by their cost. The American College of Cardiology, the Argentine Society of Cardiology and the European Society of Cardiology recommend an LDL-C goal less than 70 mg/dl in secondary prevention, determining thresholds of LDL-C to start treatment with PCSK9i of 70, 100 or 140 mg/dl respectively. In order to evaluate the lipid-lowering regimen prescribed in patients hospitalized for acute coronary syndrome or coronary revascularization and analyze the proportion of eligible to be treated with PCSK9i in a real and simulated scenario, we conducted a study that included 351 patients with coronary disease collected from an electronic database of a university hospital. The 48.4% received high intensity statins, 11.4% ezetimibe and 54.7% did not achieve the LDL-C goal of less than 70 mg/dL. Using a simulation model in which all would be treated with high intensity statins and ezetimibe, the eligibility to prescribe PCSK9i was 31.1%, 12.8% and 9.1% according to the C- LDL thresholds determined by the three scientific societies. Our study demonstrated a gap between the consensus recommendations for LDL-C lowering and the current practice that should be minimized to optimize the cost/effectiveness ratio in secondary prevention.

[36] *Deng F, Hao X, Tang Z et al.* **Carotid plaque magnetic resonance imaging and recurrent stroke risk: A protocol for systematic review and meta-analysis**. Medicine (Baltimore) 2019; 98:e15410.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31045796
ABSTRACT
BACKGROUND & AIMS: Carotid atherosclerotic plaque is an important cause of carotid artery stenosis. The features of carotid atherosclerotic plaque detected by relevant magnetic resonance imaging (MRI), such as lipid core, plaque hemorrhage, and fibrous cap rupture, have been confirmed to be associated with the occurrence of the first cerebral ischemic event. Meanwhile, the features of carotid atherosclerotic plaque can be used as biomarkers to predict the occurrence of cerebral ischemic event. However, the mechanism of recurrent stroke is still unclear. A systematic review and meta-analysis will be performed to summarize the association between features of carotid artery plaque detected by MRI and recurrent stroke, so as to find biomarkers that can predict recurrent stroke. METHODS: Electronic search will be performed in PUBMED, EMBASE, Cochrane Controlled Register of Trials (CENTRAL) from inception to October 30, 2018. We will include cohort studies with an average follow-up time of >1 month in which lipid-rich/necrotic cores (LRNC), intraplaque hemorrhage (IPH), and thinned or ruptured fibrous caps (TRFC) are associated with recurrent ipsilateral stroke or ischemic events. We will perform heterogeneity assessment before carrying out meta-analysis. According to the heterogeneity, we select random effect model or fixed effect model for meta-analysis of the included cohort studies. RESULTS: Review Manager 5.3 software will be used to calculate the combined hazard ratio value and 95% confidence interval (CI). This meta-analysis will provide high-quality data analysis of LRNC, IPH, and TRFC and ipsilateral recurrent stroke or ischemic events, including biomarkers as major predictors. CONCLUSION: The systematic review will provide evidence to assess the association between features of carotid plaque and ipsilateral recurrent stroke or ischemic events. PROSPERO REGISTRATION NUMBER: PROSPERO CRD42019124043.

[37] *Yang X, Zhang Q, Jiang G et al.* **The effects of statins on benign prostatic hyperplasia and the lower urinary tract symptoms: A Meta-analysis**. Medicine (Baltimore) 2019; 98:e15502.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31045838

ABSTRACT

BACKGROUND: The aim of this meta-analysis was to understand the relationship between statin with benign prostatic hyperplasia (BPH) and lower urinary tract symptoms (LUTS). METHODS: A systematic literature search was conducted using PubMed, Embase, Cochrane Library, Chinese Medical and Biological Literature Database, China HowNet, Vip, and Wanfang. We calculated pooled odds ratios (OR) and 95% CI and standardized mean difference (SMD). Using Stata 12.0 and Review 5.3 for meta-analysis. RESULTS: This meta-analysis included 11 articles and 49,128 participants. Results show statins could not reduce the incidence of BPH [OR = 0.77 (0.57, 1.03, P = .08]. For patients over 60 years old, statins could reduce the incidence of BPH [OR = 0.35 (0.22, 0.55), P < .0001]. Statins can slow down the progression of LUTS in BPH [SMD = -0.32 (-0.54, -0.10), P = .004], but there is no significant correlation between them in patients taking drugs for less than 1 year. CONCLUSION: Statins have no significant effect on the incidence of BPH, but statins can reduce the risk of BPH for patients over 60 years old. For patients with hyperlipidemia, the duration of medication is more than 1 year, which can slow down the progression of LUTS. However, more high-quality and large sample size studies are needed to further improve and verify.

[38] *Grefhorst A, Verkade HJ, Groen AK*. **The TICE Pathway: Mechanisms and Lipid-Lowering Therapies**. Methodist DeBakey cardiovascular journal 2019; 15:70-76.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31049152

ABSTRACT

Besides the well-known hepatobiliary pathway of cholesterol excretion into the feces, transintestinal cholesterol excretion (TICE) is a second major pathway through which cholesterol is disposed from the body. In the process of TICE, cholesterol is taken up from lipoprotein particles at the basolateral side of the enterocyte and translocates towards the apical side of the enterocyte. At the apical side, the ATP-binding cassette transporters G5 and G8 form a heterodimer that transports cholesterol into the intestinal lumen. A substantial amount of the secreted cholesterol is likely reabsorbed by the cholesterol influx transporter Niemann-Pick C1-Like 1 (NPC1L1) since recent data indicate that inhibition of NPC1L1 increases the efficacy of TICE for disposal of cholesterol via the feces. The pathways and proteins involved in intracellular cholesterol trafficking in the enterocyte have not yet been identified. Therefore, in addition to discussing known mediators of TICE, this review will also examine potential candidates involved in cholesterol translocation in the enterocyte. Both the cholesterol reuptake and efflux pathways can be influenced by pharmaceutical means; thus, the TICE pathway is a very attractive target to increase cholesterol excretion from the body and prevent or mitigate atherosclerotic cardiovascular disease.

[39] *Jia X, Lorenz P, Ballantyne CM*. **Poststatin Lipid Therapeutics: A Review**. Methodist DeBakey cardiovascular journal 2019; 15:32-38.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31049147

ABSTRACT

Literature update week 18 (2019)

Low-density lipoprotein cholesterol (LDL-C) is a well-established risk factor for atherosclerotic cardiovascular disease (ASCVD). Statins remain the first-line therapy for patients with elevated LDL-C and increased risk. However, many at-risk patients do not achieve adequate LDL-C lowering with statin monotherapy or do not tolerate statins because of side effects. Recent cardiovascular outcome trials involving ezetimibe and proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors have demonstrated efficacy of nonstatin therapies in further reducing LDL-C levels and ASCVD risk. This review highlights the available nonstatin therapeutic options and explores important novel therapeutic approaches currently under development.

[40] *Orringer CE*. **How Much Do Lipid Guidelines Help the Clinician? Reading Between the (Guide)lines**. Methodist DeBakey cardiovascular journal 2019; 15:16-22.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31049145
ABSTRACT
Although lipid guidelines provide updated, practical, and clinically relevant information that may be used in patient care, the continuing publication of new evidence and the inevitable treatment gaps present in all guidelines reinforce the importance of clinical judgment in shared decision making. This article explores the development of the 2013 American College of Cardiology/American Heart Association Blood Cholesterol Guidelines and the evidence base for managing patients with severe hypercholesterolemia, provides more recent high-quality evidence, and identifies existing treatment gaps that should be considered when caring for such patients. Although it was submitted prior to publication of the 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol, this review also includes key takeaway messages from the updated guideline.

[41] *Pownall HJ, Gotto AM, Jr.* **Cholesterol: Can't Live With It, Can't Live Without It**. Methodist DeBakey cardiovascular journal 2019; 15:9-15.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31049144
ABSTRACT
Given its role in many biochemical processes essential to life, cholesterol remains a topic of intense research. Of all the plasma lipids, cholesterol is distinctive because it is a precursor to steroidogenic molecules, some of which regulate metabolism, and its blood concentration in the form of low- and high-density lipoprotein cholesterol (HDL-C) are positive and negative risk factors for atherosclerotic cardiovascular disease (ASCVD). New research, however, has challenged the widely held belief that high HDL-C levels are atheroprotective and is showing that both low and high plasma HDL-C levels confer an increased risk of ASCVD. Furthermore, it is disputing the widely cited mechanism involved in reverse cholesterol transport. This review explores the evolution of cholesterol research starting with the Gofman and Framingham studies, the development of traditional and emerging lipid-lowering therapies, and the role of reverse cholesterol transport in HDL cardioprotection.

[42] *Toth PP, Banach M*. **Statins: Then and Now**. Methodist DeBakey cardiovascular journal 2019; 15:23-31.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31049146

ABSTRACT
The discovery of statins (3-hydroxy-3-methylglutaryl CoA reductase inhibitors) is a consequence of the highly targeted, arduous search for naturally occurring compounds that inhibit cholesterol biosynthesis. An enormous amount of basic scientific, genetic, and clinical research substantiated the role of lipoprotein-derived cholesterol in atherogenesis. Quantifying the impact of lipid lowering on cardiovascular event rates became an issue of utmost urgency. Although a variety of nonstatin drugs had been tested in clinical trials, they found limited utility in the clinical setting due to lack of mortality reduction or tolerability issues. As multiple prospective randomized statin trials began publishing their results, it became clear that reducing atherogenic lipoprotein burden with these drugs was highly efficacious, safe, and generally well tolerated. Statins have been shown to reduce risk for nonfatal MI, ischemic stroke, need for revascularization, and cardiovascular and all-cause mortality. They have also been shown to stabilize and even regress established atherosclerotic plaque. For the first 2 decades of their use, statin dosing was largely determined by risk-stratified low-density lipoprotein cholesterol (LDL-C) goals. More recently, there has been a transition away from LDL-C goal attainment with a focus more on cardiovascular risk and percent LDL-C reduction. Unfortunately, long-term adherence rates with statin therapy remain low and, even when used, they tend to be underdosed.

[43] *Warrington NM, Beaumont RN, Horikoshi M et al.* **Maternal and fetal genetic effects on birth weight and their relevance to cardio-metabolic risk factors**. Nature genetics 2019; 51:804-814.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31043758
ABSTRACT
Birth weight variation is influenced by fetal and maternal genetic and non-genetic factors, and has been reproducibly associated with future cardio-metabolic health outcomes. In expanded genome-wide association analyses of own birth weight (n = 321,223) and offspring birth weight (n = 230,069 mothers), we identified 190 independent association signals (129 of which are novel). We used structural equation modeling to decompose the contributions of direct fetal and indirect maternal genetic effects, then applied Mendelian randomization to illuminate causal pathways. For example, both indirect maternal and direct fetal genetic effects drive the observational relationship between lower birth weight and higher later blood pressure: maternal blood pressure-raising alleles reduce offspring birth weight, but only direct fetal effects of these alleles, once inherited, increase later offspring blood pressure. Using maternal birth weight-lowering genotypes to proxy for an adverse intrauterine environment provided no evidence that it causally raises offspring blood pressure, indicating that the inverse birth weight-blood pressure association is attributable to genetic effects, and not to intrauterine programming.

[44] *Onuora S*. **Daily atorvastatin safe for patients with RA**. Nature reviews. Rheumatology 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31048780
ABSTRACT

[45] *Qi W, Yan L, Liu Y et al.* **Simvastatin aggravates impaired autophagic flux in NSC34-hSOD1G93A cells through inhibition of geranylgeranyl pyrophosphate synthesis**. Neuroscience 2019.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31051215
ABSTRACT
Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease characterized by selective loss of motor neurons. Statins are widely used as cholesterol-lowering drugs and significantly reduce the risk of cardiovascular and cerebrovascular diseases. Increasing evidence indicates the protective effects of statins against certain neurodegenerative diseases. However, in ALS, many studies have found that statins might accelerate disease progression and shorten survival, although the exact mechanism is unclear. In the present study, we investigated the effect of simvastatin on NSC34cells stably transfected with the G93A mutation in human SOD1 (NSC34-hSOD1G93A cells), a recognized in vitro model of ALS. Our results showed that simvastatin caused a decrease in cell viability and the accumulation of autophagic vacuoles with elevated levels of LC3 II/I and P62 in NSC34-hSOD1G93A cells. Conversely, these outcomes were completely reversed by co-incubation with mevalonate, farnesyl pyrophosphate (FPP) or geranylgeranyl pyrophosphate (GGPP) but not cholesterol. In addition, inhibition of geranylgeranyl transferase I by GGTI-286 led to similar alterations in cell viability and autophagic marker levels. These results indicated that the cytotoxic effect of simvastatin on NSC34-hSOD1G93A cells might be due to the aggravation of autophagic flux impairment through the inhibition of GGPP synthesis.

[46] *Domenech M, Casas R, Ruiz-Leon AM et al.* **Effects of a Novel Nutraceutical Combination (Aquilea Colesterol((R))) on the Lipid Profile and Inflammatory Biomarkers: A Randomized Control Trial**. Nutrients 2019; 11.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31035469
ABSTRACT
BACKGROUND: Cholesterol-lowering nutraceuticals are useful in the management of moderate hypercholesterolemia. METHODS: In a parallel-group, randomized, placebo-controlled double-blind trial we evaluated the effects on plasma total cholesterol, low-density lipoprotein cholesterol (LDL-c), and inflammatory biomarkers of a nutraceutical combination (Aquilea Colesterol((R))) containing phytosterols (1.5 g), red yeast rice providing monacolin K (10 mg), hydroxytyrosol (5 mg), and plasma cholesterol values >5.17 mmol/L (>200 mg/dL) and LDL-c >2.97 mmol/L (>115 mg/dL). At baseline and at one and three months we recorded dietary habits; anthropometric parameters; blood pressure; lipid profile; fasting glucose; liver, renal, and muscle function tests, C-reactive protein (hs-CRP); and interleukin-6. RESULTS: 13 men and 27 women (mean age 61.8 years) completed the trial; 20 participants received the nutraceutical and 20 received placebo. No adverse effects were noted. Compared to placebo, at one and three months the nutraceutical reduced total cholesterol by 11.4% and 14.1%, LDL-c by 19.8% and 19.7%, and apolipoprotein B by 12.4% and 13.5%, respectively ($p < 0.001$; all). hs-CRP decreased significantly ($p = 0.021$) in the nutraceutical group. CONCLUSION: The nutraceutical Aquilea Colesterol((R)) is useful for reducing total cholesterol, LDL-c, and inflammation in individuals with moderate hypercholesterolemia.

[47] *Lee SG, Lee SJ, Thuy NVP et al.* **Synergistic protective effects of a statin and an angiotensin receptor blocker for initiation and progression of atherosclerosis**. PloS one 2019; 14:e0215604.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31050669
ABSTRACT
AIM: Although the atheroprotective effects of statins and angiotensin II receptor blockers (ARBs) are well-established, little is known about their additive effects, especially during the early period of atherosclerosis. The aim of this study was to investigate whether combination of a statin and an ARB exerts synergistic anti-atherosclerotic effects, and to elucidate the mechanisms of combined effects. METHODS: Atherosclerotic plaques were developed in arteries of 23 rabbits using a high-cholesterol diet (HCD) and intra-arterial balloon inflation. Rabbits received one of five different treatment strategies for 4 weeks: positive control [n = 5, HCD]; negative control [n = 3, regular chow diet]; statin [n = 5, HCD and rosuvastatin 10 mg]; ARB [n = 5, HCD and olmesartan 20 mg]; and combination [n = 5, HCD and statin+ARB]. RESULTS: Histological analysis demonstrated that development of atherosclerotic plaques was inhibited more in combination group than in statin group (P = 0.001). Although macrophage infiltration identified by RAM11 staining was not significantly different between combination and individual treatment groups (31.76+/-4.84% [combination] vs. 38.11+/-6.53% [statin; P = 0.35] or 35.14+/-2.87% [ARB; P = 0.62]), the relative proportion of pro-inflammatory M1-macrophages was significantly lower in combination group than in ARB group (3.20+/-0.47% vs. 5.20+/-0.78%, P = 0.02). Furthermore, M2-macrophage polarization was higher in combination group than in statin group (17.70+/-3.04% vs. 7.86+/-0.68%, P = 0.001). CONCLUSION: Combination treatment with a statin and an ARB produced synergistic protective effects for atherosclerosis initiation and progression, which may be attributed to modulation of macrophage characteristics in the early period of atherosclerosis.

[48] *Munkhaugen J, Tore Vethe N, Wang Fagerland M et al.* **Statin-associated muscle symptoms in coronary patients - design of a randomized study**. Scandinavian cardiovascular journal : SCJ 2019:1-23.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31030568
ABSTRACT
Objectives Estimate the effect of atorvastatin on muscular symptom intensity in coronary patients with subjective statin-associated muscle symptoms (SAMS) and to determine the association with blood levels of atorvastatin and its metabolites, to obtain an objective marker for true SAMS. Design A randomized, double-blinded, cross-over study will include 80 coronary patients with subjectively reported SAMS during ongoing atorvastatin therapy or previous muscle symptoms that led to discontinuation of atorvastatin. Patients will be randomized to 7-weeks treatment with atorvastatin 40 mg/day in the first period and matched placebo in the second 7-weeks period, or placebo in the first period and atorvastatin in the second period. Each period is preceded by 1-week wash-out. A control group (n = 40) without muscle symptoms will have 7 weeks open treatment with atorvastatin 40 mg/day. Blood samples will be collected at baseline and at the end of each treatment period, and muscular symptoms will be rated by the patients weekly using a Visual Analogue Scale (VAS). The primary outcome is the difference in aggregated mean VAS scores between the last three weeks of atorvastatin

treatment and of placebo treatment. The main purpose is to develop an objective marker for true SAMS, by comparing SAMS associated with blinded atorvastatin treatment with blood concentrations of atorvastatin and its metabolites. Diagnostic and discrimination performance will be determined. Conclusions The study provides new knowledge on SAMS in coronary patients and may contribute to more personalized statin treatment and monitoring, fewer side-effects and consequently improved adherence and lipid management in future practice.

[49] *Wada S, Koga M, Minematsu K et al.* **Baseline Carotid Intima-Media Thickness and Stroke Recurrence During Secondary Prevention With Pravastatin**. Stroke 2019:Strokeaha119024968.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31035902
ABSTRACT
Background and Purpose- As a prespecified post hoc analysis of the J-STARS (Japan Statin Treatment Against Recurrent Stroke) Echo Study, the 5-year stroke recurrence rate according to the baseline mean carotid intima-media thickness (IMT) with and without pravastatin treatment was investigated. Methods- Patients were randomly assigned to receive pravastatin 10 mg/day (pravastatin group) or control group (nonstatin treatment; 1:1) for 5 years. Baseline mean IMT of the common carotid artery was measured by ultrasonography. Cox proportional hazards models were used to investigate whether the stroke (any ischemic stroke, atherothrombotic brain infarction, or lacunar infarction) recurrence rate was different according to tertiles of baseline mean IMT. Results- A total of 793 patients, including 388 in the pravastatin group and 405 in the control group, were investigated. In the control group, Cox proportional hazards models showed that participants in the highest tertile IMT group (>/=0.931 mm) had a higher rate of atherothrombotic brain infarction than those in the lowest tertile IMT group (<0.812 mm; [hazard ratio, 9.08; 95% CI, 1.15-71.43]). Patients in the pravastatin group had a lower risk of atherothrombotic brain infarction than those in the control group only in the highest tertile IMT group by the log-rank test (P value=0.045). Conclusions- Long-term pravastatin administration may prevent the occurrence of atherothrombotic brain infarction in noncardioembolic infarction patients with the highest tertile IMT. Clinical Trial Registration- URL: https://www.clinicaltrials.gov . Unique identifier: NCT00361530.

[50] *Feng X, Sureda A, Jafari S et al.* **Berberine in Cardiovascular and Metabolic Diseases: From Mechanisms to Therapeutics**. Theranostics 2019; 9:1923-1951.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31037148
ABSTRACT
Cardiovascular and metabolic diseases (CVMD) are the leading causes of death worldwide, underscoring the urgent necessity to develop new pharmacotherapies. Berberine (BBR) is an eminent component of traditional Chinese and Ayurvedic medicine for more than 2000 years. Recently, BBR has attracted much interest for its pharmacological actions in treating and/or managing CVMD. Recent discoveries of basic, translational and clinical studies have identified many novel molecular targets of BBR (such as AMPK, SIRT1, LDLR, PCSK9, and PTP1B) and provided novel evidences supporting the promising therapeutic potential of BBR to combat CVMD. Thus, this review provides a timely overview of the pharmacological properties and

therapeutic application of BBR in CVMD, and underlines recent pharmacological advances which validate BBR as a promising lead drug against CVMD.

[51] *Wang L, Zhang B, Wang H et al.* **[Establishment and application of China Elderly Dietary Guideline Index 2018 in the elderly of 15 provinces (autonomous regions and municipalities)in China]**. Wei sheng yan jiu = Journal of hygiene research 2019; 48:41-48.
PM: http://www.ncbi.nlm.nih.gov/pubmed/?term=31032766

ABSTRACT

OBJECTIVE: The China Elderly Dietary Guidelines Index 2018(CDGI(2018)-E) was established to evaluate the dietary quality and analyze its influencing factors of the elderly aged 60 and above in 15 provinces(autonomous regions and municipalities) in China. METHODS: Based on Dietary Guidelines Index 2007(CDGI-2007), the equal weight continuity scoring method was used, Chinese residents' dietary guidelines(2016) and a balanced diet pagoda as the basis, to establish Chinese Dietary Guidelines Index 2018, and used the data of China Health and Nutrition Survey in 2015 to evaluate the elderly aged 60 and above of dietary quality status and it's influencing factors of 15 provinces(autonomous regions and municipality) in our country. RESULTS: The CDGI(2018)-E score which ranges from 0 to 110 points, includes 13 evaluation index, consists of three major categories; Class "adequate intake": cereals and tubers(the percentage energy from carbohydrate and other grain and dry beans), fruit, vegetables(total vegetables and the rate of dark vegetables) soybean and nuts and dairy products; Class "moderate intake": meat and poultry, eggs and aquatic products; Class "limited intake": oil, salt and wine. In 2015, the mean of CDGI(2018)-E score for the elderly over 60 years old of 15 provinces(autonomous regions and municipality) in China was 53. 79 points(median 53. 42 points), high income level and high education level was higher, the eastern region was higher than the western and central region, and the urban higher than the rural. The scores of limited intake category were higher, while the scores of moderate and adequate intake category were mainly distributed within 0-15 and 0-25. The first five indicators of low dietary score of the elderly aged 60 and above in 15 provinces(autonomous regions and municipality)of China were dairy products, fruits, other grains and dry beans, meat and poultry in turn. The highest score class of protein, carbohydrate, dietary fiber, retinol, riboflavin and niacin intake was higher than the other groups. CONCLUSION: A series of nutrients related to cardiovascular disease risk, such as protein, fat, vitamins, sodium and potassium are different in the elderly population with different scores. The diet quality of the elderly is relatively low, and the consumption of dairy products, aquatic products and fruit needed to be improved. The nutrition education and the intervention work should be mainly targeted at the elderly in western and central regions, rural, low income and education levels.