Discipline MCP5853

Concentration area: 5131

Creation: 10/02/2022

Activation: 10/02/2022

Credits: 2

Workload:

| Theory | Practice | Study | Duration | Total |
|----------|----------|----------|----------|----------|
| (weekly) | (weekly) | (weekly) | | |
| 5 | 10 | 15 | 1 weeks | 30 hours |

Professors:

Raul Cavalcante Maranhao

Raul Dias dos Santos Filho

Objectives:

OBJECTIVES: This course has as main objective that students become familiar with research methods in the extensive area of ​​dyslipidemia/atherosclerosis prevention. At the end of the course, students are expected to develop a critical sense to adequately analyze the value and limitations of the various types of studies that are used in the area (metabolism and pathophysiology studies, cross-sectional association studies, longitudinal risk assessment studies , retrospective studies using propensity scores, Mendelian randomization studies, genomic wide association studies (GWAS), lipid intervention studies, and meta-analyses aimed at preventing cardiovascular disease). Finally, the student must be able to properly analyze the literature in the area and develop a basic or clinical study that assesses the dyslipidemia/atherosclerosis relationship

Rationale:

RATIONALE: Cardiovascular diseases are the main cause of morbidity and mortality in the world including Brazil. In this way, its prevention plays an important role in Public Health. Dyslipidemias and their association with other cardiovascular risk factors play an important role in the genesis of atherosclerosis. The training of researchers in this area is extremely important for the understanding of pathophysiology, evaluation of cardiovascular risk and development of appropriate preventive interventions. The understanding of the methods used to investigate lipid metabolism, its association with other risk factors for atherosclerosis, predictive risk models of clinical outcomes and intervention are fundamental for the development of researchers in the area. The course is focused on health professionals who work with cardiovascular, endocrinology, nutritional and pharmaceutical research.

Content:

PROGRAM: 1. Lipid Metabolism: a. Metabolism and kinetic studies: role and limitations 2. Clinical Risk Stratification a. Cross sectional vs. Longitudinal studies (INTERHEART vs. FRAMINGHAM). Kaplan Maier and time to event analyses (COX models). b. How to incorporate new biomarkers in clinical practice: relative risk, discrimination (C statistic), calibration and reclassification (IDI and NRI). 3. Genetic studies: Mendelian randomization and genome wide association studies (GWAS), dyslipidemias and atherosclerosis biomarkers. Genetic scores on

cardiovascular risk prediction. 4. Interventional randomized controlled studies in dyslipidemias: how to separate robust from inadequate studies. 5. Critical analysis on metaanalysis use in clinical lipidology

Type of Assessment:

EVALUATION FORM: 1-Seminars and discussions of key studies during the classes.

Notes/Remarks:

OBSERVATIONS: Minimal number of students: 5 Maximal number of students : 10

Bibliography:

1. McQueen MJ, Hawken S, Wang X, Ounpuu S, Sniderman A, Probstfield J, Steyn K, Sanderson JE, Hasani M, Volkova E, Kazmi K, Yusuf S; INTERHEART study investigators. Lipids, lipoproteins, and apolipoproteins as risk markers of myocardial infarction in 52 countries (the INTERHEART study): a case-control study. Lancet. 2008 ;372:224-33. 2. Ingelsson E, Schaefer EJ, Contois JH, McNamara JR, Sullivan L, Keyes MJ, Pencina MJ, Schoonmaker C, Wilson PW, D'Agostino RB, Vasan RS. Clinical utility of different lipid measures for prediction of coronary heart disease in men and women. JAMA. 2007 ;298:776-85. 3. Wilson PW. Challenges to improve coronary heart disease risk assessment. JAMA. 2009;302:2369-70. 4. Lloyd-Jones DM, Liu K, Tian L, Greenland P. Narrative review: Assessment of C- reactive protein in risk prediction for cardiovascular disease. Ann Intern Med 2006 145:35-42. 5. Patel AP, Wang M, Pirruccello JP et al. Lp(a) (Lipoprotein[a]) Concentrations and Incident Atherosclerotic Cardiovascular Disease. New Insights From a Large National Biobank. Arterioscler Thromb Vasc Biol 2021; 41:465-474 6. Zacho J, Tybjaerg-Hansen A, Jensen JS, Grande P, Sillesen H, Nordestgaard BG. Genetically elevated C-reactive protein and ischemic vascular disease. N Engl J Med. 2008;359:1897-1908 7. Voight BF, Peloso GM, Orho-Melander M et al. Plasma HDL cholesterol and risk of myocardial infarction: a mendelian randomisation study. Lancet. 2012 ;380:572-80 8. Clarke R, Peden JF, Hopewell JC et la. Genetic Variants Associated with Lp(a) Lipoprotein Level and Coronary Disease. N Engl J Med 2009;361:2518-28. 9. Nissen SE, Wolski K. Effect of rosiglitazone on the risk of myocardial infarction and death from cardiovascular causes. N Engl J Med 2007;356:2457-71. 10. Rist PM, Buring JE, Ridker PM et al. Lipid levels and the risk of hemorrhagic stroke among women. Neurology 2019;92:e2286-e2294 11. Yeboa J, McClelland RL, TS Polonzky et al. Comparison of Novel Risk Markers for Improvement in Cardiovascular Risk Assessment in Intermediate-Risk Individuals. JAMA. 2012;308(8):788-795 12. Dormuth CR, Hemmelgarn BR, Paterson JM et al. Use of high potency statins and rates of admission for acute kidney injury: multicenter, retrospective observational analysis of administrative databases. BMJ 2013;346:f880 doi: 10.1136/bmj.f880 13. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta- analysis of data from 170 000 participants in 26 randomised trials. Lancet 2010;376:1670-81. 14. Carneiro MC, Miname MH, Gagliardi AC et al. The removal from plasma of chylomicrons and remnants is reduced in heterozygous familial hypercholesterolemia subjects with identified LDL receptor mutations: study with artificial emulsions. Atherosclerosis 2012; 221(1):268-74.

Class type:

Presencial