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Call for original papers on "Influence of sex and gender on biology of atherosclerotic cardiovascular disease"

There are differences in the risk for atherosclerotic cardiovascular diseases (ASCVD) between men and women. These differences stem from various biological aspects related to sex and gender and should be considered when assessing the risk for ASCVD in clinical practice, planning clinical trials, and performing *in vivo* and even *in vitro* experiments in research settings. *Atherosclerosis*, the journal of the European Atherosclerosis Society (EAS), is now **calling for the submission of Original Research Papers for a Special Issue related to the role of sex and gender biology in ASCVD**. These manuscripts will undergo a regular review process and in case of acceptance will go online within the usual time of processing. Submissions are encouraged from all fields related to the topic including clinical, translational, and basic research.

The submitted Original Research Articles will be handled by Elena Osto, Jeanine Roeters van Lennep, and Lale Tokgözoğlu as Guest Editors and Katariina Öörni as Co-Editor of *Atherosclerosis*. They will decide on the peer reviewers of the submitted articles. If a manuscript is accepted for publication, these Original Research Articles will appear printed together in a combined issue of the journal containing roughly a dozen in-depth review articles on the sex and gender biology of ASCVD. The collection aims to provide the most comprehensive, insightful, and current overview of the clinical and translational aspects and basic research related sex and gender differences in ASCVD. The topics and authors for these review articles have already been decided for this project. The publication is planned for spring/summer 2023 and is expected to receive a high visibility. **Accepted papers will be published with promotional open access for a one-year period, free of charge.**

For preparation of the Original Research manuscripts please see the "Guide for authors"

Deadline for submission of the first draft of Original Research Papers is December 31st, 2022. This call is only open for Original Research Articles and no review articles are allowed. Please select "Special issue: Gender biology in ASCVD" as article type at submission.

To submit your paper go to: Editorial Manager®

Atherosclerosis newsletter

Simona Negrini and Arnold von Eckardstein

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Statin therapy for the primary prevention of cardiovascular disease: Pros

Statin therapy for the primary prevention of cardiovascular disease: Cons

In this “Pros” debate, Razavi et al. discuss the benefits of statin pharmacotherapy in primary prevention of cardiovascular disease while examining over three decades worth of basic science, translational, and clinical research in the setting of clinical practice guidelines. In the “Cons” debate, Duray and Redberg highlight how strategies to reduce the risk of cardiovascular disease should avoid a focus on cholesterol levels and subsequent pharmacological therapy and should instead redouble efforts to improve the lifestyle factors that are far more consequential to the development of cardiovascular disease and overall good health.

Impact of NAFLD and its pharmacotherapy on lipid profile and CVD

Increasing evidence suggests that, in addition to traditional metabolic risk factors such as obesity, hypercholesterolemia, hypertension, diabetes mellitus, and insulin resistance (IR), nonalcoholic fatty liver disease (NAFLD) is an emerging driver of atherosclerotic cardiovascular disease (ASCVD) via multiple mechanisms, mainly by disrupting lipid metabolism. The lack of pharmaceutical treatment has spurred substantial investment in the research and development of NAFLD drugs. However, many reagents with promising therapeutic potential for NAFLD also have considerable impacts on the circulating lipid profile. In this review, Wang et al. summarize the mechanisms linking lipid dysregulation in NAFLD to the progression of ASCVD, they highlight the potential risks of/benefits to ASCVD conferred by NAFLD pharmaceutical treatments and discuss potential strategies and next-generation drugs for treating NAFLD without unwanted side effects.

Long-term tracking and population characteristics of lipoprotein (a) in the Cardiovascular Risk in Young Finns Study

Lipoprotein(a) (Lp(a)) is synthesized by the liver and composed of one molecule of low density lipoprotein (LDL)-like particle containing apolipoprotein-B and one of apolipoprotein(a) linked by a disulfide bridge. The physiological role of Lp(a) is unknown, but elevated Lp(a) levels are associated with increased risk of atherosclerotic cardiovascular disease outcomes, such as coronary heart disease, myocardial infarction, ischemic stroke, and aortic valve calcification and its levels are under strict

genetic control. Distribution of Lp(a) is often highly skewed and large proportion of individuals have very low or nearly undetectable levels of Lp(a). Finns have lower Lp(a) levels than central Europeans, but it is unknown whether there are differences within Finland, especially between the eastern and western parts of the country with known genetic duality and persistent differences in cardiovascular disease rates. Raitakari et al. examined the long-term stability of Lp(a) levels over 25 years in the Cardiovascular Risk in Young Finns Study (YFS), and the characteristics of individuals with different Lp(a) levels, including their geographical origin within Finland.

In YFS, the first large baseline examination was conducted in 1980. Several follow-ups during the past 40 years were conducted to investigate the determinants of cardiometabolic health. Lp(a) levels were measured in 1986, 2001, 2007 and 2011. Tracking of Lp(a) was estimated by calculating Spearman's rank order correlations between the study years, and by cross-tabulating how many individuals diagnosed with either elevated or non-elevated Lp(a) levels in 1986, 2001 and 2007 remained in the same category in the latest follow-up in 2011.

Most individuals (87–94%) who had a high Lp(a) level (>30 mg/dl) in any of the previous study years had a high level also in 2011. On average, median Lp(a) levels were consistently ∼20% higher in individuals originating from eastern Finland compared to those from western Finland, but there were no differences in the distribution of known genetic determinants between eastern and western Finns that would have explained the observed difference. High Lp(a) level (≥50 mg/dL) was associated with increased risk of cardiovascular disease, but not with the development of pre-clinical phenotypes, including carotid plaques, intima-media thickness, elasticity or brachial endothelial function.

These data confirm that Lp(a) levels remain very stable over a life time. The 20% higher Lp(a) levels observed in individuals originating from eastern Finland compared to those originating from western Finland are in line with the genetic duality between eastern and western parts of Finland.

Sex differences of lipoprotein(a) levels and associated risk of morbidity and mortality by age: The Copenhagen General Population Study

Lipoprotein(a) (Lp(a)) is a well-known causal risk factor for cardiovascular morbidity and mortality. It is currently recommended by the European Society of Cardiology, jointly with the European Atherosclerosis Society and the Canadian Cardiovascular Society, to screen all women and men for high levels of (Lp(a) once in a lifetime. More than 90% of the variance in Lp(a) levels is explained by genetics, and the distribution varies with different ethnicities. Little is known about the effect of age and sex on Lp(a) levels, and it is largely unknown if the same elevation in Lp(a) confers the same increase in risk in women and men. Bay Simony et al. investigated whether Lp(a) levels and associated risks of morbidity and mortality by age are similar in women and men.

37,545 women and 32,497 men from the Copenhagen General Population Study were included in the analysis. The main endpoint was myocardial infarction but similar analyses for ischemic heart disease, ischemic stroke, aortic valve stenosis, heart failure, cardiovascular mortality, and all-cause mortality were conducted.

Plasma Lp(a) increased with age, and an additional increase was found in women around age 50. In women, levels were 27% higher after menopause and 12% lower during hormone replacement therapy. Adjustment for estimated Glomerular Filtration Rate (GFR) in both sexes and plasma estradiol in women resulted in attenuated sex differences in Lp(a) levels. In sex and age stratified multivariable adjusted models, Lp(a) >40 mg/dL *versus* <10 mg/dL was associated with increased risk of myocardial infarction, ischemic heart disease, aortic valve stenosis, and heart failure (men only), but not statistically significant with risk of ischemic stroke, cardiovascular mortality, or all-cause mortality.

Lp(a) levels increased modestly around age 50 selectively in women; however, risk of morbidity and mortality for high Lp(a) was similar in women and men above age 50. This implies that elevated Lp(a) above age 50 is a relatively more common cardiovascular risk factor in women, pointing toward repeat measurements in women above age 50.