1st Summer School
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Instructions for abstract preparation
- Abstracts should be written in English.
- Font: Arial 10 pt,
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Title: One initial capital letter followed by lower case letters.
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Structure your abstract in: **Aims**, **Methods and results** and **Conclusions**.

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An abstract example can be found here:

NADPH oxidase 4 protects against development of endothelial dysfunction and atherosclerosis in LDL receptor deficient mice

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Aims: Endothelial dysfunction is an early step in the development of atherosclerosis. Increased formation of superoxide anions by NADPH oxidase Nox1, 2, and 5 reduces nitric oxide availability and can promote endothelial dysfunction. In contrast, recent evidence supports a vasoprotective role of H_2O_2 produced by main endothelial isoform Nox4. Therefore, we analysed the impact of genetic deletion of Nox4 on endothelial dysfunction and atherosclerosis in the low-density lipoprotein receptor (Ldlr) knockout model.
Methods and results: Ex vivo analysis of endothelial function by Mulvany myograph showed impaired endothelial function in thoracic aorta of $Nox4^{-/-}/Ldlr^{-/-}$ mice. Further progression of endothelial dysfunction due to high-fat diet increased atherosclerotic plaque burden and galectin-3 staining in $Nox4^{-/-}/Ldlr^{-/-}$ mice compared with $Ldlr^{-/-}$ mice. Under physiological conditions, loss of Nox4 does not influence aortic vascular function. In this setting, loss of Nox4-derived H_2O_2 production could be partially compensated for by nNOS upregulation. Using an innovative optical coherence tomography approach, we were able to analyse endothelial function by flow-mediated vasodilation in the murine saphenous artery in vivo. This new approach revealed an altered flow-mediated dilation in $Nox4^{-/-}$ mice, indicating a role for Nox4 under physiological conditions in peripheral arteries in vivo.
Conclusions: Nox4 plays an important role in maintaining endothelial function under physiological and pathological conditions. Loss of Nox4-derived H_2O_2 could be partially compensated for by nNOS upregulation, but severe endothelial dysfunction is not reversible. This leads to increased atherosclerosis under atherosclerotic prone conditions.