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ABSTRACT BOOK

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"Precision medicine for healthy ageing"

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Guest Editor:

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Contents

Abstract number

PL | Plenary speakers

PL-1-PL-5

Workshop invited speakers

W1 | Phagocyte biology
W2 | Mitochondria, ageing and disease
W2/W4 | Mitochondria, ageing and disease & Hepato-Gastroenterology/Lipids
W3 | Cardiology-CIBERCV
W4/W7 | Hepato-Gastroenterology/Lipids & Obesity-CIBEROBN
W5/W7 | Omics & Obesity-CIBEROBN
W6 | Precision nutrition
W7 | Obesity-CIBEROBN

W1-L1-W1-L19
W2-L1-W2-L18
W2/W4-L1-W2/W4-L4
W3-L1-W3-L18
W4/W7-L1-W4/W7-L7
W5/W7-L1-W5/W7-L14
W6-L1-W6-L9
W7-L1-W7-L4

Oral presentations

W1 | Phagocyte biology
W2 | Mitochondria, ageing and disease
W3 | Cardiology-CIBERCV
W4/W7 | Hepato-Gastroenterology/Lipids & Obesity-CIBEROBN
W7 | Obesity-CIBEROBN

W1-O1-W1-O15
W2-O1-W2-O10
W3-O1-W3-O14
W4/W7-O1-W4/W7-O10
W7-O1-W7-O4

Posters by theme

W1 | Phagocyte biology
W1 | Phagocyte biology
W2 | Mitochondria, ageing and disease
W2 | Mitochondria, ageing and disease
W2 | Mitochondria, ageing and disease
W3 | Cardiology-CIBERCV
W3 | Cardiology-CIBERCV
W3 | Cardiology-CIBERCV
W4 | Hepato-Gastroenterology/Lipids
W5 | Omics
W6 | Precision nutrition
W7 | Obesity-CIBEROBN
W7 | Obesity-CIBEROBN
W9 | Miscellaneous Medical Topics

Poster session 1
Poster session 2
Poster session 1
Poster session 2
Poster session 3
Poster session 1
Poster session 2
Poster session 3
Poster session 3
Poster session 2
Poster session 3
Poster session 1
Poster session 3
Poster session 3

P1-T-P20-T
P21-T-P35-T
P36-T-P55-T
P56-T-P72-T
P1-F-P34-F
P73-T-P102-T
P103-T-P128-T
P35-F-P68-F
P69-F-P90-F
P129-T-P161-T
P91-F-P99-F
P162-T-P191-T
P100-F-P125-F
P126-F-P142-F

Posters by session

W1 | Phagocyte biology
W2 | Mitochondria, ageing and disease
W3 | Cardiology-CIBERCV
W7 | Obesity-CIBEROBN
W1 | Phagocyte biology
W2 | Mitochondria, ageing and disease
W3 | Cardiology-CIBERCV
W5 | Omics
W2 | Mitochondria, ageing and disease
W3 | Cardiology-CIBERCV
W4 | Hepato-Gastroenterology/Lipids
W6 | Precision nutrition
W7 | Obesity-CIBEROBN
W9 | Miscellaneous Medical Topics

Poster session 1
Poster session 1
Poster session 1
Poster session 1
Poster session 2
Poster session 2
Poster session 2
Poster session 2
Poster session 3
Poster session 3
Poster session 3
Poster session 3
Poster session 3
Poster session 3

P1-T-P20-T
P36-T-P55-T
P73-T-P102-T
P162-T-P191-T
P21-T-P35-T
P56-T-P72-T
P103-T-P128-T
P129-T-P161-T
P1-F-P34-F
P35-F-P68-F
P69-F-P90-F
P91-F-P99-F
P100-F-P125-F
P126-F-P142-F

Poster session 1 Thursday, 31 May, 12:30-14:30

Poster session 2 Thursday, 31 May, 17:30-19:00

Poster session 3 Friday, 1 June, 12:30-14:30

P121-T | Immunohistological detection of FBN1 expression in mouse aorta

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Marfan syndrome (MFS) is a systemic hereditary autosomal dominant disease characterized by connective tissue disorders. Acute aortic syndrome (AAS) including aortic dissection is the cause of death in over 90% of untreated patients. Fibrillin 1 (FBN1) has been identified as the major causing gene of MFS, as nearly 3000 FBN1 mutations has been associated with Marfan disease, but rarely with dissection events. FBN1 is an important structural protein which regulates microfibril stability and assembly. FBN1 mutations disrupt microfibril formation, and eventually weaken the connective tissue. Therefore, dysregulation in FBN1 content could play an important role in aneurysm formation. Histopathology of aneurysm is characterized by an enlargement and weakened aortic medial layer, with fibrosis and disorganization and fragmentation of the elastic fibers.

We have used two different models of Marfan mice to analyze in aorta gene expression changes in Fbn1.

1. A mouse heterozygous for an allele of Fbn1 (Fbn1C1039G/+) containing a mutation, frequently found in MFS patients.
2. A mouse model based on lentiviral-mediated Fbn1 silencing.

Our results indicate that lentivirus encoding Fbn1 specific shRNA efficiently downregulated aortic Fbn1, leading aortic dilation and medial degeneration. Given the difficulties to detect Fbn1 expression in mouse aortic extracts by Western Blot, we have set up an immunohistological protocol to detect Fbn1 based on digestion of the aortic tissue with elastase. This approach has allowed us to monitor the levels of Fbn1 protein expression in wild type, in Fbn1-deficient mice, and compare them Fbn1 expression in Marfan patients.

P122-T | The influence of methoxamine on the isolated heart chronotropy and inotropy

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It had earlier been shown that the combined blockade of β 1-, β 2-, and α 1-ARs is more effective than the selective

blockade of β -ARs, which is widely used in the treatment of cardiac pathologies. α 1-AR of the heart participates in numerous physiologic processes, such as inotropy, genes transcription, protein synthesis, glucose metabolism and inhibition of apoptosis. These findings attest to the need of further studies to develop novel approaches in the treatment of cardiac pathologies.

The experiments were carried out on albino rats aged 20 weeks. The rats were anesthetized intraperitoneally with 25% urethane (800 mg/kg body weight). The heart was perfused in a Langendorff System (ADInstruments) with carbogen-oxygenated Krebs-Henseleit solution *ex vivo*. The retrograde perfusion was driven by constant hydrostatic pressure of 60–65 mmHg. To stimulate α 1-ARs, methoxamine (MX, an agonist affecting all subtypes of α 1-ARs, Sigma) was used at the concentrations of 10^{-10} – 10^{-8} M. The signals were recorded in a PowerLab 8/35 system (ADInstruments) with the help of LabChart Pro 8.0 software. The data was processed statistically using Microsoft Excel software and Student's *t* test.

The stimulation of α 1-ARs with MX led to bradycardia in the isolated heart. It was also observed that all studied concentrations of methoxamine induced a negative inotropic reaction of the isolated left ventricle of rats. The intensity of the negative inotropic effect depended on concentration of the agonist. Decreased heart rate and myocardium contractility with the activation of α 1-AR may be secondary to decreased ICa via the activation of protein kinase C. It is quite possible that α 1-AR participates in more delicate regulations of cardiac function and it is most likely that the effects of this stimulation depends on activities of other receptors and different intracellular systems.

Work supported by Program of Competitive Growth of KFU and Russian Foundation for Basic Research.

P123-T | The influence of If inhibition on the myocardium electrical activity

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“Funny currents” (If) play a decisive role in the creation of automatic activities in the cells of mammalian pacemakers. Recently, new data were obtained indicating a possible involvement of If in the performance of working cardiomyocytes. This work was designed to study changes in the shape of the action potential (AP) of rat atrial cardiomyocytes induced by a specific inhibition of If with 10^{-5} M