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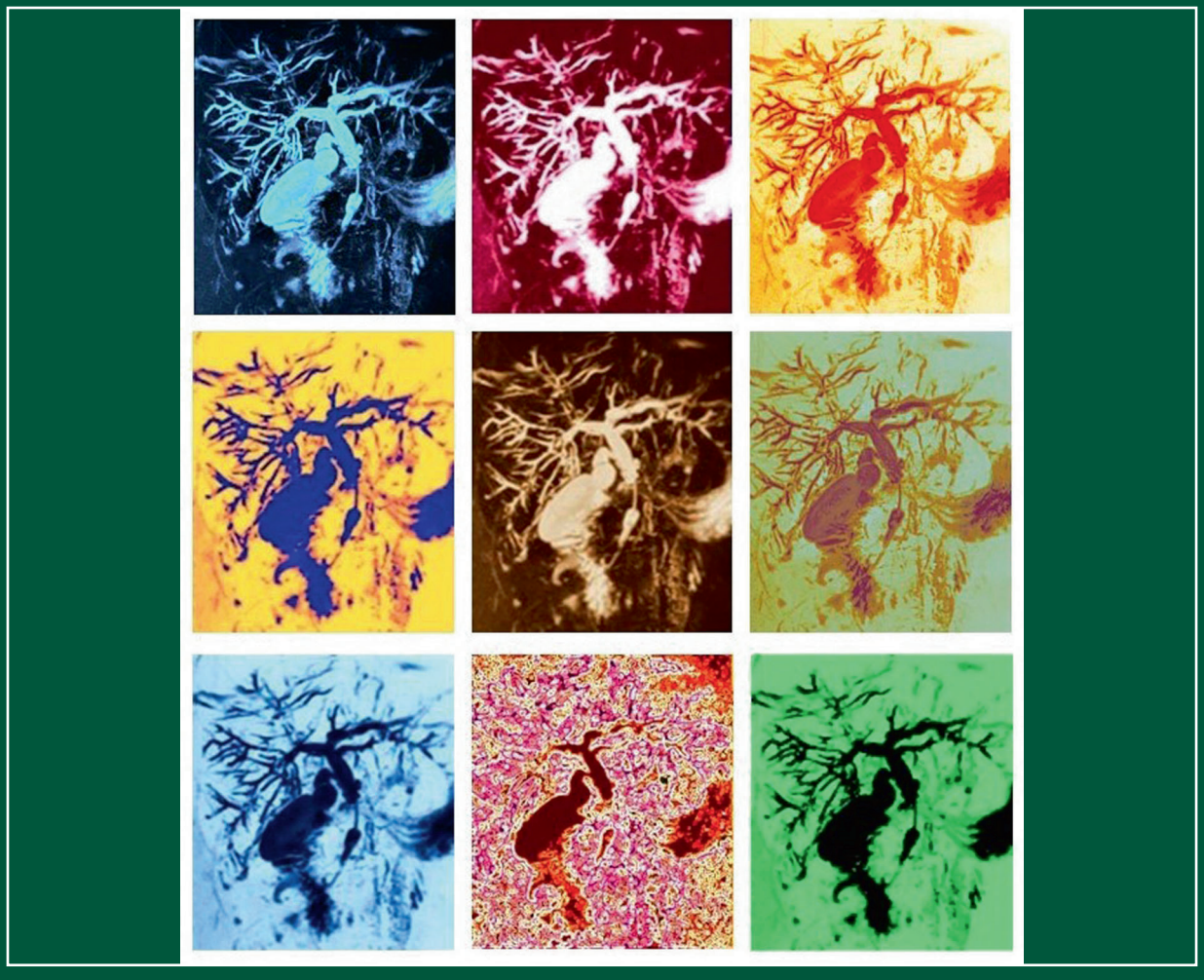
European Journal of Clinical Investigation

56TH ANNUAL SCIENTIFIC MEETING –
8–10 June 2022, Bari, Italy



Cholangiocarcinoma - 9 faces of the killer
It shows cholangiocarcinoma, an aggressive bile duct tumour with dismal prognosis,
It was captured during magnetic resonance cholangiopancreatography (MRCP)
Piotr Milkiewicz, Warsaw Poland

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ESCI EUROPEAN SOCIETY FOR CLINICAL INVESTIGATION

Official journal of the European Society for Clinical Investigation

European Journal of Clinical Investigation

THE JOURNAL OF THE EUROPEAN SOCIETY FOR CLINICAL INVESTIGATION

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Aims & Scope

The European Journal of Clinical Investigation (EJCI), in publication since 1970, is a peer-reviewed general-interest biomedical journal with a broad readership. It is the official journal of the European Society for Clinical Investigation (ESCI) and it is published monthly by Wiley. It considers any original contribution from the most sophisticated basic molecular sciences to applied clinical and translational research and evidence-based medicine across a broad range of subspecialties. The EJCI publishes reports of high-quality research that pertain to the genetic, molecular, cellular, or physiological basis of human biology and disease, as well as research that addresses prevalence, diagnosis, course, treatment, and prevention of disease. We are primarily interested in studies directly pertinent to humans, but submission of robust in vitro and animal work is also encouraged. Interdisciplinary work and research using innovative methods and combinations of laboratory, clinical, and epidemiological methodologies and techniques is of great interest to the journal. Several categories of manuscripts (for detailed description see below) are considered: editorials, original articles (also including randomized clinical trials, systematic reviews and meta-analyses), reviews (narrative reviews), opinion articles (including debates, perspectives and commentaries); and letters to the Editor.

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The Society holds one Annual Scientific Meeting each year, usually in April/May. Meetings are held in different cities in Europe. Other Society's activities include the sponsorship of Workshops and Postgraduate Courses and the encouragement of the exchange of Scientists between Laboratories.

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during readaptation after hypokinesia, adult rats reacted with a tendency to restore LVL, CP and a complete restoration of heart rate values. The recovery period in rat pups led to a decrease in the parameters of LVL (29%) and CP (23%) below the control values and a decrease in heart rate parameters by 27% of heart rate ($p < 0.05$).

Conclusions: Thus, unlike adult animals, a recovery period of two weeks is insufficient for young developing rats. This paper has been supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030).

56ASM-0104 | Nitric oxide effect on rat myocardial contractility during mobility restriction

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Background: Nitric oxide (NO) is a signaling molecule involved in the regulation of myocardial contractility. The action of many drugs used in cardiology is based on the release of NO, but the vascular and cardiac effects are not fully understood. Research on the role of NO in the body during motor activity limitation is of interest. There is evidence that prolonged limitation of mobility causes significant changes in the contractile function of the heart.

Materials and Methods: Experiments were carried out on random-bred albino rats. Restrictions of motor activity were achieved by placing rats in a small box: the first two days, the time of inactivity was 1 hour, and then increased by 2 hours every 2 days. By day 25, the time spent by animals in the cage-cases reached 23 hours. We determined the response of ventricular myocardial contractile function to the action of SNP (SNP at a dose of 10-6M) and against the background of L-NAME at a dose of 10 mg/kg. The contractile activity of myocardium was examined in vitro in a PowerLab setup equipped with a MLT 050/D Force Transducer (ADInstruments). We calculated the response of contraction force in response to pharmacological agents as a percentage of the initial force (100%). Experiments were performed in accordance with the regulatory guidelines for the treatment of laboratory animals.

Results: Under the action of SNP there was an increase in ventricular myocardial striatal contraction force by 23% ($p < 0.05$). Against the background of the action of L-NAME ventricular myocardial stripe contractile force with the addition of SNP increased by 55% compared with the baseline ($p < 0.05$).

Conclusions: The positive effect of SNP is increased 2.5-fold in rats growing under mobility restriction against the background of non-selective NO synthase blockade. This paper has been supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030).

56ASM-0105 | In vivo ultrasonographic evaluation of skeletal muscle and cardiac function and structure in animal models of neuromuscular disorders: a new approach to improve preclinical translational research

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Background: Neuromuscular disorders induce structural and functional muscle changes relevant for diagnosis and disease progression. The absence, in many cases, of specific therapies makes it necessary to improve predictability of pre-clinical studies also regarding methodology. Ultrasonography is a useful method for assessing quantitative changes in human muscle such as muscle size and presence of fat or fibrous tissue infiltrations through echodensity measures. Today, it is possible to apply ultrasound in preclinical settings obtaining more predictive data to translate in patients. We recently set up an ultrasonographic technique for ultrasound acquisition suitable for rodent skeletal muscle and validated this new approach to assess disease progression and pharmacological efficacy.

Materials and Methods: Ultrasonography experiments were carried out using the Vevo2100 set up equipped with a probe working at 40 HMz (cardiac acquisitions) and a probe working at 21 MHz (diaphragm and hindlimb acquisitions).

Results: By ultrasound, we showed that the treatment with growth hormone secretagogues prevent the FDL muscle loss occurring in a rat model of cisplatin induced cachexia. Subsequently, we showed that the long-term treatment with taurine of mdx mice, a model of Duchenne Muscular Dystrophy, exerted a protective action improving the left ventricular function as demonstrated by the restoration of ejection fraction, shortening fraction, and stroke volume values.

In mdx mice, the morphological and functional properties of diaphragm muscle were investigated showing a significant decrease in diaphragm contractile amplitude and a significant increase in mean pixel echodensity as an index of fibrosis.