According to the WHO 2012 pancreatic cancer is the 10th highest incidence and the 4th place in the 5-year survival rate in the world [1]. In Belarus died more than 700 people in 2010, and according to the 2012 – 809 [2]. Diagnosis of pancreatic cancer is difficult operation. The disease has no specific symptoms in the early stages. Patients seek treatment at III or IV stage when the tumor is large or metastatic.

For estimating the proliferative activity of tissue, the expression of the nuclear antigen Ki-67 is evaluated. This antigen is contained in the nucleus and its amount increases when the cell divides. Also, Ki-67 is a predictor of tumor disease and tumor response to chemotherapeutic treatment. This is determined in the following way: the lower the Ki-67 index, the worse the tumor reacts to chemotherapy treatment. And vice versa - the higher the Ki-67 index, the better the tumor will respond to chemotherapy [3, 4].

Material and methods. Clinical data and tumor tissue of 50 patients with pancreatic cancer at the age of 63,20437 ± 1,67 years served as a material for the study. They were on treatment at the Republican Scientific and Practical Center of Oncology and Medical Radiology, N.N. Alexandrov".

Determination of the expression's level of Ki-67 in patients with pancreatic cancer was performed by immunohistochemistry using DAKO reagents (Denmark) and visualization system (EnVision +).

As a result of the analysis of the expression's level of the proliferative antigen Ki-67, it was found that 36% (18 patients) had no expression of this protein or were detected in single cells (less than 1%), with prevalent ductal adenocarcinoma diagnosed in 33.33% of patients with IV stage of the tumor process. A positive reaction for Ki-67 was observed in 32 cases with prevalence of ductal adenocarcinoma and stage IV of the disease. Of these, 22% (11 patients) had high proliferative activity (>50% positively stained cells), a moderate level of expression was found in 12 (24%) patients, weak expression was found in 9 (18%).

Thus, as a result of the molecular-biological studies it was found that 64% of patients with pancreatic cancer showed expression of the proliferative antigen Ki-67, which may indicate tumor aggressiveness and unfavorable prognosis of the disease course.

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GENERATION AND ANALYSIS OF DECELLULARIZED LIVER SCAFFOLDS FOR REGENERATIVE MEDICINE

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Decellularization of the hepatic tissue for obtaining scaffolds and their subsequent recellularization by allogeneic cell cultures is a promising direction of tissue and organ bioengineering for a potential creation of a liver with full biocompatibility for transplantation. A careful multi-parameter evaluation of the functional and immunogenic properties of the various allogeneic cell cultures is important in recellularization.

Keywords: decellularization, recellularization, Kupffer cells, immunological properties.

Tissue engineering is a field of regenerative medicine aimed at recreating tissues that relies on 3 main pillars: cell cultures, scaffolds that provide structural support to the cells and bioactive molecules that direct their organization into tissues [2, 4, 5].

One of the main technical aspects that until recently limited the creation of tissue and organ transplants is the complexity of the framework selection, which would ensure adequate oxygen and nutrients transfer. This problem has been potentially solved with the development of methods for the decellularization of organs while preserving the structural and functional characteristics of their native microvascular network [1, 3, 4].

Decellularization-derived scaffolds have several advantages over other techniques. Firstly, they retain the native extracellular matrix 3D structure, which fosters cell repopulation and proper function. Secondly, they can incorporate growth factors and release important bioactive molecules upon degradation. Thirdly, they are available and easy to be obtained from humans and animals [1, 3, 5].

The aim of this study was to generate decellularized liver scaffolds from different species, analyze their morphological (architecture, level of residual cellularization and others), biochemical (molecular composition), mechanical (elasticity, durability, etc.) and immunological properties.

Methodological approaches for creating decellularized animal hepatic scaffolds have been generated, residual DNA has been determined and the bioreactor prototype for subsequent recellularization has been worked up. Primary hepatocytes, endotheliocytes and Kupffer cells cultures as well as mesenchymal stromal cells have been isolated and cultures have been established followed by cell culture cryobank creation.

Overall, such biomedical products can serve as effective models for testing pharmacological agents, also they have the potential to be used in clinical treatment as patient-specific transplants with full biocompatibility.

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IDENTIFICATION OF CLONAL REARRANGERS OF GENES OF ANTIGEN-RECOGNIZING RECEPTORS IN CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKEMIS

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The use of modern, high-tech molecular genetics methods of diagnosis in oncohematological practice has a wide practical potential. One of the most powerful methods to evaluate the effectiveness of therapy, compare the protocols of treatment, control the preservation of remission and predict the risk of relapse, is the definition of minimal residual disease (MRD).

Keywords: acute lymphoblastic leukemia, gene rearrangement, minimal residual disease, PCR screening, heteroduplex analysis.

Discrimination between polyclonality and monoclonality remains one of the goals in the differential diagnosis between normal lymphoid population and lymphoid neoplasia.

Currently several methods are used for detection of malignant lymphoid cell monoclonality. They are: 1) flow-cytometric immunophenotyping; 2) cytogenetical definition of chromosome aberrations; 3) polymerase chain reaction (PCR) analysis of breakpoint fusion regions of leukemia-specific chromosome translocations. The attractive approach to evaluate lymphoid cell monoclonality is PCR-based analyses of specific junction region rearrangement of immunoglobulin (Ig) and T cell receptor (TCR) genes [1, 3]. A junctional diversity of TCR and IG gene loci determines the clonal variety of normal T and B lymphocytes. Lymphoid tumors originate from