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Possible mechanisms of the inhibitory effect of the allogeneic dispersed biomaterial upon fibrosis of different etiology

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Statement of the problem: At present allogeneic decellularized biomaterials are successfully used to replace defects of different tissues. When properly selected, the collagen synthesis, somewhat stretched in time, being correlated with the gradual resorption of the biomaterials by macrophages results in the formation of the structurally complete collagen fibers with the adequate architectonics and prevention of scarring. The macrophages were established to play the main role not only in the lysis of the biomaterial fibers but also in building newly formed collagen fibers (matrix-formed macrophages). We have developed technology to obtain Dispersed Allogeneic Biomaterials (DAB), which allows inserting them in the form of suspension, which significantly expands the scope of their application to stimulate regeneration of different tissues.

Purpose: The purpose of this study is to reveal possible effect mechanisms of the macrophage population upon the development of the degenerative-dystrophic changes in tissues and the possibility to correct them using the allogeneic dispersed biomaterial.

Methodology & theoretical orientation: There have been carried out the experiments (on rats and rabbits) with pathology modelling leading to fibrosis and scarring (liver cirrhosis, corticosteroid glaucoma, myocardial infarction) and followed by the DAB insertion. After 3,7,14,30 and 90 days the material was investigated using the histological, electron microscopic, histochemical (Hale) and immunochistochemical methods (TGF-ß1, TNF-α, IL-1α, CD 68, Vimentin, Thy1, C-kit, GATA-4) and also by means of flow cytometry (CD 45, CD 90).

Conclusion: The results of the experiments showed that in case of fibrosis, the population reduction of macrophages and high TGF-ß1 expression were observed. Upon the DAB insertion, there was observed a substantial macrophage concentration increase of the haemopoietic and mesenchymal origin with pro-inflammatory M1 phenotype which led to the predominance of TNF-α expression in comparison of TGF-ß1 one. Against this background, cardio myoblasts (GATA-4+) which were then differentiated as cardio myocytes were revealed in the experiment with the infarction model. Thus the macrophage activity prevents fibrosis and contributes to its reduction. Under this condition the stem cells exhibit activity.