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DEVELOPMENT OF A BIOREACTOR FOR BLOOD VESSELS TISSUE ENGINEERING

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Cardiovascular disease (CVD) is considered the leading cause of mortality and morbidity in America and worldwide, causing 31.5% of all global deaths[1]. To date, the golden standard procedure to treat CVD is to bypass the blocked vessels with an autologous vein harvested from the patient through coronary bypass graft surgery[2]. However, this treatment presents limitations due to native blood vessels availability and long-term failure. Hence, human tissue engineered blood vessels (TEBV) could represent a valid therapeutic alternative thanks to the possibility to produce patient specific vessels starting from autologous stem cells. Moreover, TEBV represents an excellent platform for drug screening of pharmacological candidates prior to pre-clinical animal studies[3]. Here we report the design and development of a bioreactor for TEBV generation allowing different parameters setting such as duty and rest cycles, pressure and frequency of stimulation. Perivascular progenitor cells, from both mouse and human, are embedded in a gelatin methacrylate (GELMA) scaffold, a denatured collagen-based matrix that thanks to its natural origin drive cell adhesion and differentiation[4]. This cell-GELMA solution is poured into an opposite designed mold and, after UV polymerization, a blood vessel like structure of about 3 mm of diameter and 20 mm of length is obtained. These artificial vessels are cultured for at least 10 days to ensure proper cellular differentiation, with or without perfusion stimulation, and then analyzed by histological staining and immunofluorescence assay.

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Hoxb1 FUNCTION IN THE DEVELOPING MOUSE AUDITORY SYSTEM RHOMBOMERE 4-DERIVED

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Hoxb1 gene is essential for the specification of rhombomere 4 (r4)-derived auditory sensory and motor neurons contributing to the formation of specific auditory subcircuits. As we previously showed, R4 largely contributes to the motor cochlear efferent neurons and Hoxb1 loss strongly prevents the proper development of the auditory system. As a matter of fact, Hoxb1 mutants display an increased auditory threshold that leads to severe hearing impairments. It is known that medial olivo-cochlear motoneurons (MOCs) which synapse with outer hair cells (OHCs) are involved in the cochlear amplification mechanism. Indeed, we found a strong morphological damage of the OHCs and the total absence of MOCs when Hoxb1 function was abolished in r4[1]. A hypothesis is that MOC neuron endings could play a trophic function on OHCs and that the physical interaction between MOCs and OHCs is essential for proper maturation and functioning of OHCs[2]. In order to assess if the degeneration of OHCs and consequently altered hearing thresholds might be caused by the absence of synaptic/trophic stimulation of OHCs from the MOC fibers, we analyzed Hoxb1 mutant for the dorsal (sensory) and the ventral (motor) domain respectively. The sensory cochlear populations were affected in Hoxb1flox Atoh1-Cre and Hoxb1flox Ptf1a-Cre mice, whereas the olivocochlear motoneurons were deleted by using Hoxb1flox Nkx2.2-Cre mutants. The transmission and scanning electron microscopy's study showed that the absence of Hoxb1 in sensory domain of r4 does not impair the proper development of OHCs, which maintain a regular morphology and fail to reproduce the severe phenotype observed in Hoxb1null mutants. On the other hand, our preliminary data on Hoxb1flox Nkx2.2-Cre mutants seem to highlight a key role for MOCs, which origin from this domain, on OHC survival and sound amplification.

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PERIPHERAL NEUROPATHY RELATED TO OBESITY: A MICROANATOMICAL STUDY OF SCIATIC NERVE IN OBESE RATS

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Obesity and to a greater extent type-2 diabetes are associated with impaired glucose utilization and peripheral neuropathy. Different studies demonstrated that diet-induced obese rats develop whole-body insulin resistance and sensory neuropathy associated with reduced sensory nerve conduction velocity, thermal hypoalgesia and decreased intraepidermal nerve fiber density in the skin. The prediabetic stage is a leading cause of peripheral neuropathy, accounting for approximately 35.5% of undiagnosed cases. Moreover, dyslipidemia may contribute to the development of peripheral neuropathy. The aim of this study was to evaluate the effects of a high-fat diet (HFD) on the sciatic nerve in the rodent model of diet-induced obesity (DIO). DIO rats exposed to high-fat diet *ad libitum*, provide a useful animal model sharing several common features with human obesity. DIO rats were studied after 5 weeks when the obese phenotype appeared and after 17 weeks of the HFD. They were compared to the control rats with no fat diet (CHOW). Histochemical, immunohistochemical and immunochemical analysis were performed to evaluate nerve fiber changes of the sciatic nerve. Systolic blood pressure, glycaemia and insulin levels were higher in DIO rats only after 17 weeks of the HFD. No changes in total cholesterol and triglycerides were found. An increase of thiobarbituric reactive substances and oxidated proteins was observed in the serum of DIO rats compared to CHOW. Axon area and myelin thickness did not change in large and small nerve fibers in DIO rats. A decrease of 200-kDa neurofilament immunoreactivity and a reduced expression of myelin basic like-protein were observed in obese rats compared to the control. An inflammatory condition, with an increase of interleukin-β and oxidative stress were detected in the sciatic nerve of the obese rat. Our findings support the hypothesis that obesity, characterized by hyperglycaemia and adipose tissue accumulation, may represent a risk factor for neuropathy.