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the efficacy of muscle renewal. In fact, during ageing a progressive decline of muscle mass, strength and quality take place, a condition termed sarcopenia[4]. Although no specific therapy is presently available to counteract the onset and progression of sarcopenia, it has been demonstrated that physical exercise may efficiently mitigate the age-related muscle atrophy. In particular, we demonstrated that an adapted aerobic physical exercise (treadmill running) leads to the reactivation of nuclear activity, increasing transcriptional and post-transcriptional processes in both myofibers and SCs of old skeletal muscles[5,6].

The process of SC activation is finely regulated by the expression of specific factors, among which the paired box protein 7 (Pax7) and the myogenic differentiation factor D (MyoD). Although largely investigated in SC nuclei, the distribution and function of Pax7 and MyoD in myonuclei are poorly known. We have therefore focused our attention on the possible effects that age-related atrophy as well as exercise-related nuclear reactivation may induce on the fine distribution and relative amounts of Pax7 and MyoD in myonuclei of old mice. Our results shed light on the possible functional role played by Pax7 and MyoD in the myonuclear response to physical exercise and, more generally, in skeletal myofiber regeneration.

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HISTOCHEMICAL STUDY OF MYOCARDIAL ISCHAEMIC TISSUE IN ADVANCED PUTREFACTION: PRELIMINARY RESULTS

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Histologic diagnosis in forensic context it's often hampered by autolysis and putrefaction. Specimens from autopsies performed on exhumed cadavers or on bodies in advanced state of decomposition render recognition of pathological alterations quite difficult. Moreover tissue decomposition can sometimes simulates non-existent histopathological processes[1]. In forensic pathology practice it is common to encounter heavily putrefied bodies. In these cases to prove the possible presence of an acute myocardial infarction as the cause of death is very important. Moreover, the appearance of myocardial ischemia can be masked or even imitated by autolysis and putrefaction[2].

The use of hematoxylin-eosin (HE) stain is not sufficient to investigate decomposed myocardial tissue[3]. The use of histochemical techniques could help in the histopathological analysis of this type of material to increase the diagnostic specificity[4]. The purpose of this research is the evaluation of the efficacy of histochemical stains to identify ischemic areas in the putrefied myocardial tissue.

Heart tissue specimens was taken from eight cases of macroscopically evident acute myocardial infarction (AMI) during diagnostic autopsies. Specimens was obtained from an area containing ischemic and non-ischemic myocardium. One tissue fragment was immediately fixed in a 10% buffered formalin and used as control. Specimens from AMI were placed in an open case and stored at controlled room temperature, ranging between 16 °C and 20 °C. At time interval of 15 and 30 days of putrefaction the samples of AMI tissues were fixed 42 hours in 10% formalin, processed and embedded in paraffin. Sections of 4µ were cut from paraffin blocks and stained with standard HE and Mallory trichrome stain.

Preliminary results showed in all cases: after 15 days of putrefaction HE stains was no longer able to detect AMI areas. Instead, after 30 days of decomposition, Mallory trichrome showed strongly positive staining of non-ischemic cardiac fibers (red colored), while ischemic myocardium was very less intense.

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