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ABSTRACTS

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Abstracts of the

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P6.10 Oral presentation

Structure and function: the fibrillar crimps of the sclera

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Crimps are a typical feature of tendons and of some ligaments where they are responsible for the non-linear behavior of the tissue at low strain values. Previous studies (Raspanti et al., 2005) have shown that in all tendons these visible crimps correspond to a distinctive buckling and/ or to a torsion of the collagen fibrils, aptly named fibrillar crimp (Franchi et al., 2007). These are exclusive of the collagen fibrils of tendons and ligaments, whose subfibrils run straight and parallel, while the collagen fibrils of most tissues, whose subfibrils follow an helical course, are almost infinitely flexible and can withstand extreme deformation without buckling. In the present study we investigated two tissues whose ultrastructure is closely related to that of tendons and ligaments, i.e. the sclera of mammals and the calcified tendons of birds. The tissues were observed by light microscopy, high-resolution scanning electron microscopy and atomic force microscopy. The sclera appears made of flattened fascicles of large and inhomogeneous collagen fibrils, running in all directions and following a curved or wavy course. The structure and appearance of each fascicle are identical to those of tendon, as are the numerous crimps which can be easily observed. The leg tendons of the tuckey, whose ultrastructure is initially similar to that of all other tendons, appear to be frozen by the mineralization process in their fully extended state when they become partly calcified in the adult. Their fibrils run straight and parallel and no longer reveal evident crimps, except for the faint trace that these leave even once the fibrils have been straightened. The presence of crimps in the uncalcified portion of turkey tendons is entirely corresponding with what was observed in all other tendons; their straightening with mineralization is also consistent with the mechanical behaviour if the tissue, since the crimps lose any function once the collagen fibrils are cemented and immobilized by the mineral phase. Less obvious was the presence of crimps in the scleral fascicles, because this tissue is maintained under continuous tension by the intraocular pressure (IOP) and is never able to relax and recoil as unloaded tendons do. A typical intraocular pressure of 20–30 mmHg would induce in the rat sclera a tension of $50-80~\mathrm{kN/m^2}$, which could still correspond to the toe region of the stress/strain curve. Therefore the crimps may be fully functional even in the sclera, confirming this tissue as a sort of hemispherical tendon.

References

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P6.11 Oral presentation

A neonatal pulse of corticosteroids modifies the expression of tenascin-C, elastin and smooth muscle actin and induces a complex alteration of rat lung development

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Introduction: Pre- and postnatal corticosteroids are used in perinatal medicine to improve pulmonary function in preterm infants. To mimic this clinical situation and elucidate its consequences, newborn rats were treated with dexamethasone (DX).

Methods: Newborn rats received DX at days P1-P4 (0.1-0.01 mg/kg). Alveolarization was quantified stereologically by estimating the formation of new septa between days P4-P60. The parenchymal expression of tenascin-C (TNC), smooth muscle actin (SMA), and elastin was observed by immunofluorescence and TNC-gene expression was measured by qRT-PCR.

Results: Under DX-treatment alveolarization was first delayed (P6–P10) but finished earlier (P36 versus P60) at the same level as the controls. TNC expression concentrated at the septal tips and was limited to the saccular and the early alveolar stage (P1–P14). DX treatment suppressed temporarily TNC gene expression around P4 and delayed but also prolonged TNC protein expression by some days. Elastin and SMA expression, concentrated at the septal tips, were also delayed by DX.

Discussion: Even if the final state of lung development was indistinguishable between treated and untreated animals, DX induced a complex alteration of the timing by impairing and accelerating the formation of alveolar septa. TNC is expressed, probably due to its mechano-sensing properties at the entrance ring of the alveolus. Its expression starts before and specifically at the sites of new septal formation and is co-localized with elastin and SMA expression. DX delays and prolongs TNC expression. We hypothesize that TNC, in combination with elastin and smooth muscles is involved in the control of alveolarization and may be important for lung regeneration.