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P1499
Board Number: B1301
Evolution of collagen IV and basement membrane enabling multicellularity.
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The emergence of basement membrane (BM) coincided with the appearance of animal multicellularity, suggesting that this specialized extracellular matrix (ECM) was a prerequisite. Ctenophora has recently emerged as the earliest-branching extant animal phylum, providing a unique opportunity to explore the evolutionary origin and association of BM in the transition to multicellularity. Here we characterized the ECM, with a focus on collagen IV, of Ctenophora and other basal phyla. We identified BM and collagen IV in Ctenophora, and show that the structural and genomic features of collagen IV are homologous to those of basal phyla and Bilateria. Yet, the ctenophoran features are more diverse and distinct, expressing up to twenty genes compared to six in vertebrates. We conclude that collagen IV and its variant, spongian short chain collagens, are primordial components enabling the transition to multicellularity, and that collagen IV, as a component of basement membrane, enabled the assembly of a fundamental architectural unit, which enabled epithelial tissue evolution.

P1500
Board Number: B1302
Imbalance of matrix metalloproteinase in the obesity-associated asthma.
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Asthma is a complex chronic inflammatory disorder. The increase in the prevalence of both asthma and obesity has prompted researchers to suggest that obesity may have an important influence in the development of asthma, or even worsen the pre-existing asthma. Recent advances have revealed that remodeling of the airways in asthma is due, at least in part, to deposition of excess of extracellular matrix (ECM) in the airway walls, worsening the airway obstruction. The metalloproteinases of extracellular matrix (MMPs) are key proteolytic enzymes that may play an important role in remodeling of the airways and/or severity of asthma. Obesity and asthma individually elevate the levels of TNF-α that this cytokine in turn lead to induction of metalloproteinase. Based on these findings, we found relevant to study the imbalance of MMPs and consequently the ECM elements in lung tissues (or bronchoalveolar lavage fluid) of obese-asthmatics in comparison with lean-asthmatics. Male C57BL6 mice were treated with a high-fat diet for 12 weeks to induce obesity, whereas lean mice received standard chow. Lean and obese mice were then sensitized and challenged with ovalbumin (OVA). We divided the experimental groups into the following groups: non-sensitized lean (NSL), non-sensitized obese (NSO), sensitized lean (SL) and sensitized obese (SO). SO mice exhibited an increase of collagen types I, III and IV compared with SL groups. Gelatinase activity in situ showed a high brightness in SO group, mainly in the peribronchiolar areas. The activity of MMP-8 and isoforms of MMP-9 were elevated in SO group. NSO and SL showed high activities of MMP-2 and MMP-12. SO exhibited less activity of
these enzymes in comparison with SL. In separate groups, SL and SO mice were treated with anti-TNF-α (2mg/kg). In mice treated with anti-TNF-α, it was noted decreases of MMP-2 and MMP-9 activities in SL and SO in lung tissue, and of MMP-9 in the bronchoalveolar lavage of SO. This study shown an imbalance of MMPs (-2, -8, -9, -12) in the lungs of obese and asthmatic mice. MMP-9 is present in chronic inflammation, and in elevated levels was noted in lungs of obese with/without asthma. The high deposition of collagen can indicate that the remodeling process is installed, supporting the asthma severity in obese mice. Anti-TNFα modulated the action of MMPs and can be an alternative to inhibit these enzymes in allergic inflammation. In view of the complexity of the MMPs, it is necessary to study its inhibitors, aiming a decrement of asthma in obese.

P1501
Board Number: B1303
Regulation of larval zebrafish wound healing through the formation of collagen projections.
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The larval zebrafish caudal-fin is structurally maintained by collagen-containing structures known as actinotrichia. Following amputation, these structures re-organize during the regenerative process, although little is known about the regulation of this remodeling. We performed a combination of second harmonic generation (SHG) imaging, immunofluorescence and long duration microscopy of live samples to examine the relationship between this collagen fiber re-organization and the mesenchymal and epithelial cell populations during caudal fin re-growth. With these techniques we identified collagen projections at the wound edge that appear to support, and possibly promote, the regenerative process in the fin. These projections initiate as an uneven healing plane along the amputated region of the fin by as early as 1 day post-amputation, extending in length during the second day post wound, resulting in a somewhat more even wound edge by 3 days post-wound. These collagen containing projections associate with a populations of mesenchymal cells and epithelial cells as healing progresses. Additionally, early ROS and downstream signaling pathways play a role in regulating the interactions between collagen fiber projection, mesenchymal cells and epithelial cells during wound repair.

P1502
Board Number: B1304
Biophysical properties of the corneal stroma influence apoptosis of myofibroblasts.
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Keratoablative corneal procedures such as Laser-assisted in situ keratomileusis (LASIK) involve wounding of the corneal stroma. Wounding and subsequent healing processes alter the biophysical attributes of the corneal stroma which, in turn, modulates a wide array of fundamental stromal cell behaviors including transdifferentiation of the quiescent keratocyte to activated fibroblasts and myofibroblasts (KFM). Myofibroblasts play a central role in the normal corneal wound healing process. Myofibroblasts are removed from the wound space through apoptosis. Excessive numbers or prolonged persistence of myofibroblasts have been associated with the development of corneal haze that can impair vision. While