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Mechanisms for Protein Detected by Immunohistochemistry

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Description

Similar mechanisms are likely present in canine IPF as TGFβ protein was detected by immunohistochemistry in areas of fibrosis, and a receptor for, and a transcription factor known for promoting its intracellular effects, pSMAD2/3, were found in the epithelium. Interestingly, latent binding protein gene expression was decreased as was β8 integrin; these changes have been proposed to ultimately affect activation. Another extracellular matrix implicated in pulmonary fibrosis, thrombospondin-1, also appeared up regulated. Of note, circulating TGFβ1 concentrations in the periphery were higher in animals “predisposed” to pulmonary fibrosis compared to “non-predisposed” breeds. Alveolar interstitial fibrillin-2 immuno reactivity was up regulated in WHWTS as well. This is similar to what has been found the idiopathic interstitial pneumonias in humans.

Chemokines have also been implicated in the pathogenesis of IPF. Similarly, higher levels of and have been detected in broncho alveolar lavage fluid obtained from affected WHWTs compared to healthy dogs.21 Circulating levels of, but not, were reported in the same animals. In contrast, no differences in relative gene expression for were observed when comparing the lung biopsies of control vs. affected animals. In those affected, and immune reactivity was detected in bronchial airway epithelial cells. In other work, Activin B, a cytokine member of the TGFβ superfamily, was found to be up regulated in the broncho alveolar lavage fluid of WHWTs with canine IPF, but not Activin A. These studies suggest that similar mechanisms of action are acting in both human and animal forms of spontaneous pulmonary fibrosis, but they fail to explain how such pathways lead to distinct histologic patterns observed between species.

Overall, the discussions of the 2014 meeting emphasized a concept unveiled during the earlier meeting. Namely, those domestic animals develop spontaneously occurring interstitial lung diseases that can result in pulmonary fibrosis and share features with the human condition. While prior discussions centered on the possibility that some of these animals may develop disease identical to the human condition, the more recent histopathologic studies available suggest that domestic animals develop diseases with clinical manifestations similar to those of human IPF, but are likely distinct from that condition. Nevertheless, the group felt that, while identifying a model identical to the human condition would be preferable, a more realistic goal would be to simply identify better models of spontaneously occurring disease than those currently used today; domestic animals such as the WHWT might provide such a model.

Two important challenges hindering progress in this area remain present today. The first relates to difficulties inherent in communicating about these disorders considering that domestic animals with pulmonary fibrosis are simply referred to has having IPF (canine IPF), which is confusing as these animals do not appear to adequately mimic human IPF. The group recommends that the term “idiopathic interstitial pneumonia or IIP” be used as this reflects classifications used in human disease. For example, instead of canine IPF, the term canine IIP should be used, at least until these IIPs are better defined.

The above challenge is directly linked to gaps in disease definition due to an inadequate understanding of the clinical, histologic and radiographic manifestations of pulmonary fibrosis in distinct animal species. Greater understanding of IPF and related idiopathic interstitial pneumonias in humans came after defining their distinct clinical, radiographic, and histologic presentations. In fact, today, an accurate diagnosis of these human conditions remains dependent on the interpretation of the aggregate clinical, radiographic and histologic data. This knowledge laid the foundation for the emergence of standardized, placebo-controlled, and randomized clinical trials that culminated in the identification of anti-fibrotic drugs. Unfortunately, this information is not available for domestic animals. To date, studies correlating the clinical, radiographic and histologic presentations of domestic animals with fibrosing lung disease are very limited, and a clear classification of these disorders and diagnostic algorithms remain to be developed. Success in collecting such data would be greatly accelerated by the establishment of domestic animal clinical registries well linked to tissue and other biological sample repositories. Such repositories may be located at specialized veterinary centers with interest and expertise in this field. Undoubtedly, the resources needed to support such endeavor are significant and may originate in industry, private foundations and government agencies.

In short, domestic animals develop spontaneously occurring fibrosing lung disease that resembles the human condition. Although not identical, these models might be superior to those used today when testing mechanisms of action and the effectiveness of novel interventions. However, this will first require a better classification of fibrosing lung disorders in domestic animals based on clinical, radiographic and histologic presentations.

Obtaining the information needed to develop such classification would benefit from a registry of clinical data and biological samples. The molecular tools needed to test genetic variants and mechanisms of action and to unveil potential targets for intervention are available, but this will require access to well-defined biological specimens from nonhuman disease. The above effort will likely require support from industry, private foundations and government agencies.