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The Role of Synthetic Biology Offers a Bottom Up Engineering

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Description

Only a profound understanding of the structure and function of cells - either as single units or in the context of tissues and whole organisms - will allow a comprehension of what happens in pathological conditions and provides the means to fight disease. The Cell Biology and Infection (BCI for Biologie Cellulaire et Infection) department was created in 2002 at the Institut Pasteur in Paris to develop a research program under the umbrella of cell biology, infection biology and microbiology. Its visionary ambition was to shape a common framework for cellular microbiology, and to interface the latter with hard sciences like physics and mathematics and cutting-edge technology. This concept, ahead of time, has given high visibility to the field of cellular microbiology and quantitative cell biology, and it has allowed the successful execution of highly interdisciplinary research programs linking a molecular understanding of cellular events with disease. Now, the BCI department embraces additional pathologies, namely cancer and neurodegenerative diseases. Here, we will portray how the integrative research approach of BCI has led to major scientific breakthroughs during the last ten years, and where we see scientific opportunities for the near future.

Synthetic biology offers a bottom–up engineering approach that intends to understand complex systems via design-build-test cycles. Embryonic development comprises complex processes that originate at the level of gene regulatory networks in a cell and emerge into collective cellular behaviors with multicellular forms and functions. Here, we review synthetic biology approaches to development that involve building de novo developmental trajectories or engineering control in stem cell-derived multicellular systems. The field of synthetic developmental biology is rapidly growing with the help of recent advances in artificial gene circuits, self-organizing organoids, and controllable tissue microenvironments. The outcome will be a blueprint to decode principles of morphogenesis and to create programmable organoids with novel designs or improved functions.

In addition to the above, the group also discussed that noted similarities between dogs and humans began to dissipate when evaluating the pulmonary histopathologic findings present in these conditions. In 18 WHWT with canine IPF, a pattern resembling NSIP was predominant rather than a pattern of UIP.11 NSIP, for nonspecific interstitial pneumonia, is another histologic pattern observed in another type of idiopathic interstitial pneumonia. In contrast to UIP, the NSIP pattern is more homogenous throughout the lung, shows more cellularity and less fibrosis, and fibroblastic foci are not typical; this entity is considered responsive to immunosuppression in many circumstances in humans.12 The majority of the dogs tested showed multifocal areas of accentuated subpleural and peribronchiolar fibrosis with what was reported as occasional honeycombing and “profound” alveolar epithelial changes, and fibroblastic foci were not seen. In some cases, intra-alveolar organizing fibrosis adjacent to interstitial mature collagen deposition was observed, especially in more severely affected areas. Interestingly, severe pulmonary lesions were more frequent in the caudal than in the cranial lung lobes. The increased availability of high-resolution computed tomography data from affected dogs, coupled with the limited pathologic data already available, support the contention that the canine form of IPF is in fact NSIP. Case cohort studies are now ongoing at the University of Edinburgh Veterinary School to assess the clinical response of affected dogs to immuno-suppressive therapy with prednisolone and mycophenolate to determine if this indeed behaves as NSIP. Furthermore, while the ground glass attenuation and mosaic pattern on HRTC can be associated with hypersensitivity pneumonitis, this condition can be excluded as it is not recognized in the dog. One confounding factor, however, is the often-late presentation of affected dogs with extensive fibrosis making measurable response to trial therapy problematic.

In evaluating 9 cats carrying a diagnosis of pulmonary fibrosis based on radiographic findings, investigators found focally increased soft tissue attenuation, masses and ventral consolidations that exhibited no improvement with dorsal vs. ventral recumbence.13 On histology, pulmonary fibrosis in these cats was evident with type II pneumocyte hyperplasia and smooth muscle hypertrophy. Epithelial metaplasia was present in one case. However, they also observed changes consistent with a broncho-interstitial

pattern, alveolar pattern, pulmonary masses, pulmonary bullae, pleural effusions and cardiomegaly. Overall, the findings suggested highly variable radiographic characteristics, which might mimic pulmonary fibrosis, but also other conditions such as asthma, pneumonia, pulmonary edema and neoplasia.

In another study, 23 cats with a histology of UIP were investigated.14 Most were middle-aged to older cats (median 8.7 years) with no obvious sex or breed predisposition. Symptoms included respiratory distress and cough. Duration of signs was less than 6 months in 17 cats. Exam revealed tachypnea, inspiratory or mixed inspiratory and expiratory effort, and adventitial lung sounds. Radiographic changes included dense patchy or diffuse interstitial, bronchiolar, and alveolar infiltrates. BALF revealed mild neutrophilic inflammation in 6 cases, with no consistent pathogen identified. Response to steroids is poor and most cats died within days to months.

Overall, the group remained impressed with the similarities observed in symptoms, lung examination, abnormalities in oxygenation and imaging studies, and outcomes when comparing humans and domestic animals with pulmonary fibrosis. However, the differences observed in histopathology strongly argue against these being identical conditions. This prompted discussions regarding mechanisms of action and several presentations were devoted to this topic.

To date, there is consensus that IPF and other forms of fibrosing lung disease are likely triggered by certain exposures in the setting of host genetics that render the lung epithelium susceptible to injury. In turn, epithelial cell injury leads to its dysfunction and the subsequent elicitation of intracellular pathways responsible for the overexpression of soluble profibrotic growth factors. Of these, transforming growth factor-β (TGFβ) is considered the most influential, but many other activated signals exert pro-fibrotic activity.

Similar mechanisms are likely present in canine IPF as TGFβ protein was detected by immunohistochemistry in areas of fibrosis, and a receptor for TGFβ, TFGβRI and a transcription factor known for promoting its intracellular effects, pSMAD2/3, were found in the epithelium.19 Interestingly, latent binding TGFβ protein gene expression was decreased as was β8 integrin; these changes have been proposed to ultimately affect TGFβ activation. Another extracellular matrix implicated in pulmonary fibrosis, thrombospondin-1, also appeared upregulated. Of note, circulating TGFβ1 concentrations in the periphery were higher in animals “predisposed” to pulmonary fibrosis compared to “nonpredisposed” breeds.20 Alveolar interstitial fibrillin-2 immunoreactivity was upregulated in WHWTS as well. This is similar to what has been found the idiopathic interstitial pneumonias in humans.