City University of Hong Kong
Department of Biomedical Sciences
presents a seminar

"MDIG AND HUMAN CANCER, 20 YEARS OF MINING AND DIGGING."

by

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Date : 22 April 2016
Time: 10.30am to 12.00am
Venue: Meeting room 2-130, 1/F, Block 2, To Yuen Building, CityU

Abstract
Mineral dust-induced gene (mdig, also named Mina53) was first identified from alveolar macrophages of the coal miners with chronic lung inflammation or fibrosis, but how this gene is involved in lung diseases is poorly understood. Here we show that heterozygotic knockout of mdig (mdig+/-) ameliorates silica-induced lung fibrosis by altering the balance between Th17 cells and Treg cells. Relative to the wild type (WT) mice, infiltration of the macrophages and Th17 cells was reduced in lungs from silica-exposed mdig+/- mice. In contrast, an increased infiltration of the T regulatory (Treg) cells to the lung intestitium was observed in the mdig+/- mice treated with silica. Both the number of Th17 cells in the lung lymph nodes and the level of IL-17 in the bronchoalveolar lavage fluids were decreased in the mdig+/-mice in response to silica. Thus, these results suggest that mdig may contribute to silica-induced lung fibrosis by altering the balance between Th17 and Treg cells. Genetic deficiency of mdig impairs Th17 cell infiltration and function, but favors infiltration of the Treg cells, the immune suppressive T cells that are able to limit the inflammatory responses by repressing the Th17 cells and macrophages.seminar.

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All are welcome