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Title: Late stage of experimental pulmonary fibrosis is modulated by collagen V

Mr. Alexandre 27162 Todorovic Fabro alexandretodofabro@yahoo.com.br MD [1], Mr. Mozar 27163 Suzigan de Almeida mozar.suzigan@gmail.com [2], Mr. Igor O. 27164 Minatel igorminatel@hotmail.com [3], Ms. Erika C. 27165 Prado erikaprando@yahoo.com.br [4], Prof. Dr Claudia 27166 Aparecida Rainho rainho@ibb.unesp.br [5], Prof. Dr P.R. Teodoro 27168 Walcy matrix@lim17.fm.usp.br [6], Prof. Dr Edwin R. 27175 Parra Cuentas erparra20003@yahoo.com.br MD [7], Prof. Dr Vera 27193 Capelozzi vcapelozzi@lim05.fm.usp.br MD [8] and Prof. Dr Helmut 900014 Popper helmut.popper@medunigraz.at [9]. [1] Department of Pathology, Faculdade de Medicina de Botucatu - UNESP, Botucatu, SP, Brazil, 18608-970 ; [2] Department of Pathology, Universidade de São Paulo, SP, Brazil, 01307-003 ; [3] Department of Pathology, Faculdade de Medicina de Botucatu - UNESP, Botucatu, SP, Brazil, 18608-970 ; [4] Department of Genetic, Instituto de Biociências de Botucatu - UNESP, Botucatu, SP, Brazil, 18608-970 ; [5] Department of Genetic, Instituto de Biociências de Botucatu - UNESP, Botucatu, SP, Brazil, 18608-970 ; [6] Department of Pathology, Universidade de São Paulo, SP, Brazil, 01246-903 ; [7] Department of Pathology, Universidade de São Paulo, SP, Brazil, 01246-903 ; [8] Department of Pathology, Universidade de São Paulo, SP, Brazil, 01246-903 and [9] Institute of Pathology - Graz University .

Body: Background: The IPF is a disease with high morbi-mortality. Several experimental models of pulmonary fibrosis (PF) has been proposed, however, a later stage of these models tend to go for the resolution of the fibrosis, but in different degrees of intensity depending on the strain. Thus these mechanisms in certain strains may participate in the progression of PF. Aims: To study the immune-fibrotic pattern in different models of PF in the late stage (21d). Methods: We used the models of Bleomycin-Balb/c (BLM), Paraquat-Balb/c, Bleomycin-C57BL/6 and BLM-IL17RA-KO-C57BL/6. We analyzed the amounts of total collagen (TC) and collagen V (Col5) through the morphometric evaluation by the picrosirius and IF. These data were validated by RT-PCR of Col5. Results: The peribronchiolar TC by PPM did not differ between the treated groups, but the peripheral interstitial TC was higher in the C57BL/6, independent of the absence of IL-17RA. The protein expression of Col5 was higher in IL-17RA-KO (75,5±9% X 52,7±13%; p=0,01) and lower in BLM-Balb/c (69,8±3,4% X 53,3±14,3%; p<0,05). Likewise, the gene expression of Col5 was also higher in the IL17RA-KO (p<0.0485) and lower in the BLM-Balb/c (p<0.0037) (Figure 1). Conclusion: The perpetuation of PF in fibrosis-susceptible mice is related to expression of Col5 in a IL-17-independent manner and this suggests that Col5 is an important component responsible for the development of PF.