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Title: Effect of cigarette smoke on peripheral blood Th17 (PBTh17) cells from COPD patients. Role of acetylcholine

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Body: PBTh17 cells producing IL-17A and IL-22, as well as Acetylcholine (ACh) are involved in the systemic inflammation of several autoimmune and inflammatory diseases, including COPD. We investigated the effect of cigarette smoke extract (CSE) and the role of ACh on the expression of RORgt, IL-17A and IL-22 in PBTh17 cells during systemic inflammation in COPD. ACh, IL-17A, IL-22 and RORgt levels as well as the colocalization of ACh with IL-17A, IL-22 and RORgt were evaluated (flowcytometry) in PBT-lymphocytes (PBT) from COPD patients (n=16), healthy smokers (HS) (n=12) and healthy control subjects (HC) (n=13). Furthermore, PBT cells from COPD (n=6) patients and PBT cells from HC (n=6) stimulated in vitro with CSE (10 %) were cultured for 72 hrs in the presence or absence of Tiotropium (Spiriva®) (20 nM) and Olodaterol (1 nM) alone or in combination. The colocalization of ACh with IL-17A, IL-22 and RORgt was significantly increased in PBT cells from COPD patients when compared to HC and HS subjects as well as in PBT cells from HC stimulated with CSE when compared with unstimulated cells. Tiotropium and Olodaterol alone reduced the increased levels of colocalization between ACh with IL-17A, IL-22 and RORgt in cultured PBT-cells from COPD patients and in PBT-cells from HC subjects stimulated with CSE. We suggests that cigarette smoke might be able to increase the levels of ACh in PBT cells promoting the switch into PBTh17 cells which produce IL-17A and IL-22 during the systemic inflammation in COPD. The use of anticholinergics as Tiotropium and long-acting β2-agonists as Olodaterol might prevent these events. Funded by: Boehringer Ingelheim Pharma GmbH & Co. KG, Biberach, Germany.