Introductions
There is growing evidence that changes in the lung microbiome are involved in chronic obstructive pulmonary disease (COPD) exacerbations. Cigarette smoke is the best known risk factor for COPD. Health concerns of microbes in tobacco are starting to be acknowledged. Other groups showed that tobacco flakes inhaled from cigarettes could carry bacteria into the lungs. Moreover, bacterial products such as LPS remain in cigarette smoke and could contribute to inflammation. Our group is one of the first suggesting that Toll-like receptors (TLRs) are involved in the pathogenesis of COPD. TLRs are activated by microbial products. Our in vitro studies suggest that cigarette smoke induces inflammation partly via microbial products. Our data emphasize that certain microorganisms and their products could become airborne during combustion. This information is relevant for (passive) smokers but also for women and children who use biomass for cooking indoors in developing countries. Better insight into cause could contribute to disease prevention and could open up new treatment strategies.

Results
Here we provided further evidence for a role of TLR9 in the development of COPD. First, we demonstrated that chronic (5 weeks) activation of pulmonary TLR9 in mice leads to lung inflammation, emphysema, and heart hypertrophy (Figure 1). Moreover, it is demonstrated that not all bacteria are destroyed during tobacco burning. When smoke from burning cigarettes is passed over agar plates, colony-forming units clearly follow smoke deposition patterns unrelated to tobacco flake transmission. When cigarette smoke is bubbled through a medium or agar plates, colony forming units clearly follow smoke deposition patterns. Cigarettes is passed over agar plates, colony forming units clearly follow smoke deposition patterns during tobacco burning. When smoke from burning cigarettes is bubbled through a medium or agar plates, colony forming units clearly follow smoke deposition patterns.

Perspective
Currently, a role for bioaerosol exposure in the development of COPD is still underappreciated. Our data emphasize that certain microorganisms and their products could become airborne during combustion. This information is relevant for (passive) smokers but also for women and children who use biomass for cooking indoors in developing countries. Better insight into cause could contribute to disease prevention and could open up new treatment strategies.