# European Respiratory Society
# Annual Congress 2013

**Abstract Number:** 1110

**Publication Number:** 3493

**Abstract Group:** 4.1. Clinical respiratory physiology, exercise and functional imaging

**Keyword 1:** Asthma - management **Keyword 2:** Exercise **Keyword 3:** Lung injury

**Title:** Effect of terbutaline on hyperpnoea-induced bronchoconstriction and urinary Clara cell protein release in athletes

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**Body:** Repeated injury of the airway epithelium caused by hyperpnoea of poorly conditioned air has been proposed as a key factor in the pathogenesis of exercise-induced bronchoconstriction (EIB) in athletes. In animals, the short-acting β2-agonist terbutaline has been shown to reduce dry airflow-induced bronchoconstriction and shedding of the airway epithelial cells. Our aim was to test the efficacy of terbutaline in attenuating hyperpnoea-induced bronchoconstriction and airway epithelial injury in athletes. Twenty-three recreational athletes with EIB participated in a randomised, double-blind, placebo controlled, cross-over study. Athletes completed an 8 min eucapnic voluntary hyperpnoea (EVH) test with dry air on two separate days 15 min after inhaling 0.5 mg terbutaline or a placebo. Forced expiratory volume in 1 sec ($FEV_1$) and urinary concentration of the Clara cell protein CC16 (a marker of airway epithelial stress) were measured before and for up to 60 min after EVH. The maximum fall in $FEV_1$ of 18 ± 8% (SD) on placebo was reduced to 8 ± 5% following terbutaline ($P<0.001$). Terbutaline gave bronchoprotection (i.e., post-EVH $FEV_1$ fall <10%) to 18 (83%) athletes. EVH caused an increase in urinary excretion of CC16 in both conditions ($P<0.001$), and terbutaline significantly reduced this rise (delta pre- to post-challenge CC16 increase 416 ± 495 ρg·mmol creatinine$^{-1}$ after placebo vs 315 ± 523 ρg·mmol creatinine$^{-1}$ after terbutaline, $P=0.016$). The results suggest that inhalation of a single therapeutic dose of terbutaline offers significant protection against hyperpnoea-induced bronchoconstriction and may attenuate acute airway epithelial stress in athletes.