Inhaled terbutaline attenuates hyperpnoea-induced bronchoconstriction and mast cell activation in athletes.

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Background: Anderson et al.¹,² proposed that β₂-adrenoceptor agonists taken by inhalation were superior to tablets for inhibiting exercise-induced bronchoconstriction (EIB) because sufficiently high concentration of drug was delivered directly to the airway to prevent mediator release from mast cells.

Aim: The aim of this study was to investigate the effect of a clinically recommended dose of β₂-adrenoceptor agonist on mast cell activation in response to dry air hyperpnoea in athletes with EIB.

Methods: A randomized, double blind, placebo controlled, cross over design in 18 subjects. Terbutaline (0.5 mg) or placebo was inhaled 15 min before 8 min of eucapnic voluntary hyperpnoea (EVH) with dry air at ~80% predicted maximum ventilation. The response to EVH was measured as the maximum % fall in FEV₁ from baseline after EVH. Mast cell activation was measured by urinary 11β-prostaglandin(PG)F₂α concentrations at baseline and 30, 60 min post-EVH.

Results: The ventilation achieved was 100±18 (SD) & 101±18 L.min⁻¹ (p>0.07) & the % fall in FEV₁ after EVH was of 17±9 and 8±6% after the placebo & terbutaline, respectively (p<0.001). Terbutaline afforded 54±35% bronchoprotection & 14 of the 18 subjects had <10% fall in FEV₁ after EVH. There was a significant increase in 11β-PGF₂α ng.mmol creatinine⁻¹ from baseline to after EVH following placebo (37±17 to 51±23 p<0.001), but not following terbutaline (36±14 to 40±18, p=0.103). The rise in 11β-PGF₂α post-EVH (14±14 vs 4±11) was significantly attenuated with terbutaline (p<0.01)

Conclusion: These data provide novel in vivo evidence of a mast cell stabilising effect of a single, therapeutic dose of terbutaline following bronchial provocation with EVH in athletes with EIB. The findings support the earlier proposal¹² that the concentration of β₂-adrenoceptor agonist achieved in the airway from a clinically recommended dose is sufficient to prevent the release of mast cell mediators and associated bronchoconstriction.


Key Words: mast cell, prostaglandin, terbutaline, eucapnic hyperpnoea