Effect of transdermal nicotine administration on exercise endurance

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Introduction
Nicotine is the second most widely consumed psychostimulant in the world (after caffeine) and it is generally accepted that nicotine promotes many of the characteristics provided by other stimulants such as caffeine, cocaine and amphetamines, including increased alertness, improved coordination, increased endurance and positive mood changes. Although mechanisms and sites of action are debated, it is likely that increased activity of adrenergic, dopaminergic and serotonergic pathways within the medial basal forebrain (medial septum, nucleus accumbens, preoptic area of the hypothalamus) are involved in this arousal [Boutrel & Koob, 2004].

Previous results from our group [Bridge et al, 2000] showed that caffeine supplementation improved exercise endurance by ~20% and significantly reduced perception of exertion. As no effects were observed on indicators of peripheral metabolic stress or on the release of prolactin, it was concluded that caffeine may be acting on a central pathway other than those in the hypothalamus which are sensitive to temperature.

Despite the wealth of literature describing the effects of caffeine on exercise performance and discussion of its mechanism, to our knowledge there have been no attempts to replicate these findings with nicotine. The aim in this study was, therefore, to determine the effects nicotine might have on exercise endurance, perception of exertion and a range of physiological variables.

Methods
Following local ethics approval, twelve healthy, non-smoking, males gave their written informed consent to visit the laboratory on four occasions. Visit 1 was an incremental exercise test to exhaustion to determine the workload equivalent to 65% of Peak Power for subsequent rides. Visit 2 was a familiarisation ride to minimise any learning effects. Separated by a week, visits 3 and 4 involved subjects cycling to exhaustion at 18°C wearing a 7mg transdermal nicotine patch (NIC) or colour-matched placebo (CON); water was available ad libitum. Heart rate, expiratory gases and perceived exertion (Borg scale) were measured and blood samples taken every 10 minutes. Trials were single-blind, randomized and counterbalanced.

Results
Subjects were exercising at approximately 75% of their maximal O₂ uptake with no differences in cadence between trials. Ten out of twelve subjects cycled for longer with nicotine administration (Fig. 1) and this resulted in a significant ~13% improvement in performance (p<0.05) with time-to-fatigue being 61 and 70 mins for CON and NIC, respectively. No differences were observed for perceived exertion, heart rate or Vₑ. There were no differences in concentrations of blood glucose, lactate or circulating fatty acids.

Discussion/Conclusion
Nicotine improved exercise endurance by 13 ± 5% and in the absence of any effect on the usual peripheral markers such as ventilation, heart rate and blood metabolites, we conclude that nicotine prolongs endurance by a central mechanism that may involve nicotinic receptor activation and/or altered activity of dopaminergic pathways.

References