

Aspirin Effect on Tobacco Smoke Withdrawal-Induced Anxiety In Female Rats

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ABSTRACT

Chronic exposure to cigarette smoke produces neuroinflammation and long-term changes in neurotransmitter systems, especially glutamatergic systems. We examined the effects of cigarette smoke on astroglial glutamate transporters as well as NF- κ B expression in mesocorticolimbic brain regions including the prefrontal cortex (PFC) and the nucleus accumbens (NAc). The behavioral consequences of cigarette smoke exposure on withdrawal-induced anxiety-like behavior were assessed using open field (OF) and light/dark (LD) tests. Sprague-Dawley rats were randomly assigned to 5 experimental groups: a control group exposed only to standard room air, a cigarette smoke exposed group treated with saline vehicle, two cigarette smoke exposed groups treated with aspirin (15mg/kg and 30mg/kg, respectively). Lastly, a group treated only with aspirin (30 mg/kg). Cigarette smoke exposure was performed for 2hr/day, 5days/week, for 31 days. Behavioral tests were conducted weekly, 24hrs after cigarette smoke exposure, during acute withdrawal. At the end of week 4, rats were given either saline or aspirin 45 min before cigarette exposure for 11 days. Cigarette smoke increased withdrawal-induced anxiety, and 30 mg/kg aspirin attenuated this effect. Cigarette smoke exposure increased the relative mRNA and the protein level for nuclear factor κ B (NF κ B) in the PFC and the NAc, and aspirin treatment reversed this effect. In addition, cigarette smoke decreased the relative mRNA and the protein levels of glutamate transporter 1 (GLT-1) and the cystine-glutamate transporter (xCT) in the PFC and the NAc, while aspirin treatment normalized their expression. Thus, cigarette smoke caused neuroinflammation, alterations in relative mRNA glutamate transporter expression, and increased anxiety-like behavior, and these effects were attenuated by aspirin treatment.