

Role of adenosine A2 receptors in regulation of cerebral blood flow during induced hypotension

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The mechanisms responsible for vascular autoregulation in the brain during changes in mean arterial blood pressure are ambiguous. Potentially, adenosine, a purine nucleoside and potent vasodilator, may be involved as earlier studies have documented an increase in brain adenosine concentrations with cerebral ischemia and hypotension. Consequently, we tested the hypothesis that adenosine is involved in vasodilatation during hypotension within the autoregulatory range (>50 mm Hg) by exposing adenosine 2a receptor (A2aR) knockout and wild type (WT) mice to short (2 to 5 mins) periods of hypotension. We found that autoregulation was significantly ($P < 0.05$) impaired by 29% in A2a knockout mice as compared with WT animals. Furthermore, the A2R antagonist (A2a>A2b:10–85>1), ZM-241385, in a dose (1, 5, 10 mg/kg, intraperitoneally)-related manner, attenuated autoregulation in WT mice. In knockout mice treated with ZM-241385 (5 and 10 mg/kg), autoregulation was further impaired indicating that A2b receptors also participated in cerebral vasodilatation. Treatment with dipyridamole (1.0 mg/kg) that increases extracellular concentrations of adenosine improved autoregulation in the A2aR knockout mice. We would conclude that adenosine through both A2a and A2b receptors is involved in physiologic vascular regulation during hypotension even within the autoregulatory range.

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Introduction

The brain has the capacity to modulate cerebral blood flow (CBF) during changes in blood pressure. This phenomenon is known as autoregulation (Lassen, 1959). The mechanism(s) responsible for the regulation of cerebral arteriolar tone during autoregulation is unclear, but a number of theories have been proposed (Paulson *et al.*, 1990), including the myogenic (Heistad and Kontos, 1983), neural (Hamel, 2006), and metabolic hypotheses (Heistad and Kontos, 1983). All three hypotheses entail biochemical (or transmitter) signal(s) whose action leads to a change in arterial diameter. A number of biochemical factors have been proposed to have such a role. For example, *in vivo* and *in vitro* studies

have suggested involvement of arachidonic acid metabolites (Harder *et al.*, 2000; Roman, 2002), nitric oxide (Jones *et al.*, 2003), calcitonin gene-related peptide (Hong *et al.*, 1994; Wei *et al.*, 1992), to mention a few, in vasoreactivity during alteration in mean arterial blood pressure (MABP) or intraluminal pressure.

Another potential signaling compound is the purine nucleoside adenosine, which is a potent vasodilator (Berne *et al.*, 1974; Ngai *et al.*, 2001). Brain adenosine concentrations are increased during hypoxia (Winn *et al.*, 1981; Zetterstrom *et al.*, 1982), ischemia (Winn *et al.*, 1979) and neuronal activation (Winn *et al.*, 1980a). However, there is controversy as to whether adenosine is elevated in brain during hypotension. To resolve this controversy, we performed studies to test the hypothesis that adenosine is involved in autoregulation during decreases in MABP and to determine the relative contribution of the adenosine 2a receptor (A2aR) and the 2b receptor (A2bR). We restricted our analysis to changes in MABP within the autoregulatory range and used short periods of decreases in MABP in wild type

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