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Título	Role of A_1 and A_{2A} adenosine receptor agonists in adipose tissue inflammation induced by obesity in mice
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Resumo	Adenosine receptors are expressed in adipose tissue and control physiological and pathological events such as lipolysis and inflammation. The aim of this study was to evaluate the activity of N^6-cyclopentyladenosine (CPA), a potent and selective A_1 adenosine receptor agonist; 2-*p*-(2-carboxyethyl)phenethylamino-5′-*N*-ethylcarboxyamidoadenosine hydrochloride (CGS-21680), an A_{2A} adenosine receptor agonist; and 5′-*N*-ethylcarboxamidoadenosine (NECA), a potent non-selective adenosine receptor agonist on adipose tissue inflammatory alterations induced by obesity in mice. Swiss mice were fed with a high-fat diet for 12 weeks and agonists were administered in the last two weeks. Body weight, adiposity and glucose homeostasis were evaluated. Inflammation in adipose tissue was assessed by evaluation of adipokine production and macrophage infiltration. Adenosine receptor signaling in adipose tissue was also evaluated. Mice that received CGS21680 presented an improvement in glucose homeostasis in association with systemically reduced inflammatory markers (TNF-α, PAI-1) and in the visceral adipose tissue (TNF-α, MCP-1, macrophage infiltration). Activation of p38 signaling was found in adipose tissue of this group of mice. NECA-treated mice presented some improvements in glucose homeostasis associated with an observed weight loss. Mice that received CPA presented only a reduction in the *ex vivo* basal lipolysis rate measured within visceral adipose tissue. In conclusion, administration of the A_{2A} receptor agonist to obese mice resulted in

	improvements in glucose homeostasis and adipose tissue inflammation, corroborating the idea that new therapeutics to treat obesity could emerge from these compounds.
Fomento	