Subject: Koffein sogar kontraproduktiv
Posted by Raiders-Fan on Tue, 01 May 2007 09:22:35 GMT
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Interleukin-1beta-induced inhibition of hair growth in vitro is mediated by cyclic AMP.

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J Invest Dermatol 1997 Jan;108(1):40-2.
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PMID: 8980284 [PubMed - indexed for MEDLINE]

Interleukin (IL)-1 has been shown to be a potent inhibitor of hair growth in vitro. We hypothesized that this cytokine might be a decisive factor causing hair loss during the lymphocytic attack in alopecia areata. Neither the intracellular pathways involved in hair growth inhibition mediated by IL-1beta nor the signal transduction processes within hair follicles in general are known. We therefore investigated the intracellular signals involved in human hair growth in vitro. Hair follicles were isolated from scalp biopsies by microdissection, and hair growth was measured daily by image analysis. We assessed intracellular signal transducing elements using specific inhibitors or activators either alone or in combination with IL-1beta. The calcium ionophore A 23187 induced a rapid and complete arrest of hair growth, and phorbol-12-myristate-13-acetate (PMA), genistein, or IL-1beta decreased hair growth by approximately 60%-80%. IL-1beta-elicited hair growth arrest was not antagonized by calphostin C, a specific inhibitor of protein kinase C. In contrast, coincubation of IL-1beta with pertussis toxin or H 1004 neutralized the effect of IL-1beta, and dibutyryl-cAMP and cholera toxin, an activator of adenylate cyclase, inhibited hair growth. These data suggest that cAMP acts as a second messenger for IL-1beta-induced inhibition of hair growth. Moreover, our data indicate that in vitro hair growth is dependent on intracellular Ca2+ levels and activation of tyrosine kinase as well as protein kinase C. We were unable to detect a signal transducing element responsible for enhanced hair growth in vitro.

Effect of caffeine on meiotic maturation of porcine oocytes.

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Zygote 2004 Feb;12(1):31-8.
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PMID: 15214577 [PubMed - indexed for MEDLINE]

This study was conducted to evaluate the effect of caffeine on the meiotic maturation of porcine oocytes. Oocyte-cumulus complexes were collected from slaughterhouse-derived ovaries and cultured for 24, 32 or 48 h in medium 199 supplemented with 10% fetal calf serum, 10 microg/ml FSH, 50 microg/ml sodium pyruvate and 50 microg/ml gentamicin in the presence or absence of 2.5 mM caffeine. Caffeine inhibited the meiotic resumption of pig oocytes effectively after 24 h of culture, and 95.5% of oocytes were arrested at the germinal vesicle (GV) stage (control 17.8%, $p < 0.05$). Prolonged culture with caffeine up to 32 h or 48 h, however, resulted in a significant decrease in the inhibitory effect (GV: 13.8% and 8.2%). The number of oocytes at metaphase II after 48 h of culture in the presence of caffeine was significantly lower than that in the control medium (65.3% vs 94.7%, $p < 0.05$). The withdrawal of caffeine after 24 h of culture resulted in

the resumption of meiotic maturation, and the oocytes reached metaphase II after 48 h. However, the ability of caffeine-treated oocytes to develop to blastocysts after artificial activation was lower than that of the control (5.5% vs 9.1%, $p < 0.05$). Caffeine treatment significantly increased cAMP levels in the oocytes after 24 h of culture, while both Cdc2 kinase and MAP kinase activation were inhibited in the oocytes. These results suggest that caffeine, similarly to other purine derivatives, prolongs the meiotic arrest of porcine oocytes at the GV stage, perhaps by its action of increasing the cAMP level and by the suppression of Cdc2 kinase and MAP kinase activities in the oocytes.

Inhibitory effect of anaphylactic shock by caffeine in rats.

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Int J Immunopharmacol 2000 Jun;22(6):411-8.
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PMID: 10727752 [PubMed - indexed for MEDLINE]

Caffeine is known to reduce evoked histamine secretion, but the effects of caffeine on anaphylactic shock have not been clarified. We have investigated the effects of caffeine on anaphylactic shock in rats. Systemic anaphylactic shock by compound 48/80 injection was monitored for 1 h. An IgE-dependent local anaphylactic shock was generated by sensitizing the skin with anti-dinitrophenyl (DNP) IgE followed 48 h later with an injection of antigen. Caffeine inhibited compound 48/80-induced anaphylatic shock to 40% with a dose of 1 mg/kg. Caffeine (0.1 mg/kg) inhibited to 56.4+/-0.4% passive cutaneous anaphylactic shock activated by anti-DNP IgE. Caffeine (5-20 mM) significantly inhibited histamine release from rat peritoneal mast cells (RPMCs) activated by compound 48/80 or anti-DNP IgE. Especially, caffeine (20 mM) inhibited by 96.7+/-0.5% histamine release activated by compound 48/80. Moreover, caffeine (1-20 mM) had a significant inhibitory effect on anti-DNP IgE-induced tumor necrosis factor-alpha production from RPMCs. The level of cAMP in RPMCs, when caffeine (20 mM) was added, increased significantly after 5-60 min compared with that of a normal control. These results indicate that caffeine inhibits immediate-type allergic reactions by inhibition of mast cell degranulation in vivo and in vitro.