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butylbenzoyl)-1-[(2-methylphenyl)-methylamino]-2-hydroxy-propane sulfonate. The peptide arginine vasopressin (AVP) increases CI- transport across the colonic epithelium by activating a V1a receptor. The objective of this study was to characterize the receptor mediating AVP-stimulated CI- transport and to determine whether the V1a receptor antagonist 4-(p-t-

butylbenzoyl)-1-[(2-methylphenyl)-methylamino]-2-hydroxy-propane sulfonate (TT-20) is able to block the stimulation by AVP of in vitro short circuit current and CI- transport by the T84 cell monolayer. In addition, TT-20 was tested as an inhibitor of AVP-stimulated 3H-inositol phosphate (IP3) formation in the COS-7 cell line expressing the AVP V1a receptor. Finally, AVP-stimulated 3H-IP3 formation was examined in membrane preparations obtained from the colon. AVP increased both short circuit current and CI- transport by T84 cells. The V1a receptor antagonist TT-20 (1 microM) decreased baseline short circuit current by 40% and reduced stimulation by AVP by 80%. CI-secretion was reduced by 40% by TT-20, whereas both short circuit current and CI- transport were inhibited by 80% by TT-20. In the COS-7 cell line, AVP stimulated IP3 formation and the activation of the V1a receptor was blocked by TT-20 (50 microM). IP3 formation was inhibited in a dose-dependent fashion by TT-20. In colonic membrane preparations, AVP stimulated IP3 formation, which was blocked by TT-20 (10 microM). The results show that AVP stimulates CI- secretion and phosphoinositide turnover in colonic epithelium, supporting the concept of a V1a receptor

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