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In vitro INFLUENCE OF β-ADRENERGIC RECEPTORS STIMULATION ON CONTRACTILE ACTIVITY OF SMOOTH MUSCLES

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β-Adrenergic receptors (β-ARs) have been sub-classified as β1- and β2-ARs since 1967. This sub-classification has led to the development of β1- and β2-AR antagonists and/or agonists which have been useful for the treatment of cardiovascular diseases and asthma. In early 1980s, a third β-AR, initially referred to as "atypical" and later called β3-AR has been found in a number of species, including man, and in 1989, the human, rat, and mouse β3-ARs were first cloned and characterized. β-ARs are known to mediate several important physiological functions. It has also been reported that β3-AR receptor plays a significant role in regulating lipolysis and thermogenesis in rodent and human adipose issues. Moreover, studies of β3-AR mRNA demonstrated that, β3-ARs exist in human heart, gall bladder, gastrointestinal tract, prostate, and urinary bladder detrusor tissue in addition to adipocytes.

Myometrial contractile activity is controlled by myogenic, neurogenic and hormonal mechanism. Uterine hyperactivity primarily associates with primary dysmenorrhoea and aperars to be the result of an action or interaction of several factors including: ovarian steroids, cervical obstruction, pituitary hormones and prostaglandins. But the exact mechanisms are largely unclear. Tocolytic agents such as β_2-adrenergic receptor agonists or calcium channel blockers are employed to inhibit myometrial contractions and increase uterine blood flow. In spite of their effectiveness, side effects of these drugs in long-term therapy limit its clinical uses. One of the major limitations for β_2-AR agonists use in clinical practice is their cardiovascular side effects. Another limiting factor for the use of selective β_2-AR agonists as tocolytic drugs is that their effect, if any, appears to be of relatively short duration. It has recently been reported that β_3-AR agonists exert a relaxant effect on human uterine contractions *in vitro* that is of equal potency to the relaxant effect of β_2-AR agonists.