

# Regulation of smooth muscle contraction by monomeric non-RhoA GTPases

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Smooth muscle contraction in the cardiovascular system, airways, prostate and lower urinary tract is involved in the pathophysiology of many diseases, including cardiovascular and obstructive lung disease plus lower urinary tract symptoms, which are associated with high prevalence of morbidity and mortality. This prominent clinical role of smooth muscle tone has led to the molecular mechanisms involved being subjected to extensive research. In general smooth muscle contraction is promoted by three major signalling pathways, including the monomeric GTPase RhoA pathway. However, emerging evidence suggests that monomeric GTPases other than RhoA may be involved in signal transduction in smooth muscle contraction, including Rac GTPases, cell division control protein 42 homologue, adenosine ribosylation factor 6, Ras, Rap1b and Rab GTPases. Here, we review these emerging functions of non-RhoA GTPases in smooth muscle contraction, which has now become increasingly more evident and constitutes an emerging and innovative research area of high clinical relevance.

## 1 | INTRODUCTION

Smooth muscle contraction in the cardiovascular system, airways, lower urinary tract and prostate is primarily involved in the pathophysiology of many diseases, which have a high mortality and are also causing high individual and socio-economic burden, such as in cardiovascular and obstructive lung diseases plus lower urinary tract symptoms. Accordingly, inhibition of smooth muscle contraction is an important strategy in medical treatment of these diseases, which together affecting billions of patients worldwide. In line with the prominent role of smooth muscle tone in health, disease and therapy

the molecular mechanisms of contraction and relaxation were and are the subject of continuous research.

Smooth muscle contraction is induced by receptors, by mechanical and other stimuli, which subsequently activate intracellular, contraction-mediated signalling pathways. These pro-contraction signalling pathways all end in a few final mechanisms, which are absolute prerequisites for smooth muscle contraction, including myosin light chain (MLC) phosphorylation, actin polymerization, organization and attachment of filaments to membranes and anchoring of cells to the extracellular matrix (Kim, Appel, Vetterkind, Gangopadhyay, & Morgan, 2008; Puete, Lubomirov, & Pfister, 2009). Intracellular signalling pathways resulting in contraction include calcium-dependent mechanisms that parallel increases of calcium sensitivity, which was realized early during the exploration of smooth muscle contraction. The role of the small monomeric GTPase RhoA for signal transduction promoting vascular smooth muscle contraction by immediate increases of calcium sensitivity has been recognized since 1992

**Abbreviations:** ARF, adenosine ribosylation factor; BPH, benign prostatic hyperplasia; Cdc42, cell division control protein 42 homologue; Egac, exchange protein directly activated by cAMP; GDI, guanine nucleotide dissociation inhibitor; GDP, guanosine diphosphate; GEF, guanine nucleotide exchange factor; IP<sub>3</sub>, inositol-1,4,5-trisphosphate; MLC, myosin light chain; MLCc, myosin light chain-kinase; MYPT1, myosin targeting subunit.

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# Regulation And Contraction Of Smooth Muscle Progress In Clinical And Biological Research V245

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