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Pyridine nucleotides and calcium signalling in arterial smooth muscle: from cell physiology to pharmacology

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Abstract

It is generally accepted that the mobilisation of intracellular Ca^{2+} stores plays a pivotal role in the regulation of arterial smooth muscle function, paradoxically during both contraction and relaxation. However, the spatiotemporal pattern of different Ca^{2+} signals that elicit such responses may also contribute to the regulation of, for example, differential gene expression. These findings, among others, demonstrate the importance of discrete spatiotemporal Ca^{2+} signalling patterns and the mechanisms that underpin them. Of fundamental importance in this respect is the realisation that different Ca^{2+} storing organelles may be selected by the discrete or coordinated actions of multiple Ca^{2+} mobilising messengers. When considering such messengers, it is generally accepted that sarcoplasmic reticulum (SR) stores may be mobilised by the ubiquitous messenger inositol 1,4,5 trisphosphate. However, relatively little attention has been paid to the role of Ca^{2+} mobilising pyridine nucleotides in arterial smooth muscle, namely, cyclic adenosine diphosphate-ribose (cADPR) and nicotinic acid adenine dinucleotide phosphate (NAADP). This review will therefore focus on these novel mechanisms of calcium signalling and their likely therapeutic potential.
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Keywords: NAADP; cADPR; ADP-ribosyl cyclase; Ryanodine receptor; Sarcoplasmic reticulum; Lysosomes; Artery; Smooth muscle

Abbreviations: 8-bromo-cADPR, 8-bromo-cyclic adenosine diphosphate-ribose; ACh, acetylcholine; β-NAD^+, β-nicotinamide adenine dinucleotide (oxidized form); β-NADH, β-nicotinamide adenine dinucleotide (reduced form); β-$NADP^+$, β-nicotinamide adenine dinucleotide phosphate; BK_{Ca} channel, Ca^{2+}-activated potassium channel; cADPR, cyclic adenosine diphosphate-ribose; cAMP, cyclic adenosine monophosphate; cGDPR, cyclic guanosine diphosphate-ribose; cGMP, cyclic guanosine monophosphate; CICR, Ca^{2+}-induced Ca^{2+} release; ER, endoplasmic reticulum; ET-1, endothelin-1; FKBP, FK-506 binding proteins; HPV, hypoxic pulmonary vasoconstriction; IP_3, inositol 1,4,5 trisphosphate; IP_3R, inositol 1,4,5 trisphosphate receptor; mAChR, muscarinic acetylcholine receptors; NAADP, nicotinic acid adenine dinucleotide phosphate; $PGF_{2\alpha}$, prostaglandin $F_{2\alpha}$; PKA, protein kinase A; ROS, reactive oxygen species; RyR, ryanodine receptors; SERCA, sarcoplasmic/endoplasmic reticulum Ca^{2+} ATPase; SR, sarcoplasmic reticulum; STOC, spontaneous transient outward potassium current.

Contents

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1. Introduction

Consideration of the wide variety of processes regulated by changes in intracellular Ca^{2+} concentration, from fertilisation and gene expression to muscle contraction and cell death, can leave us in no doubt of the need for a versatile Ca^{2+} signalling system (Berridge et al., 2000). Consequently, not all stimuli that initiate a given cell response, say muscle contraction, do so by eliciting Ca^{2+} signals with a common spatiotemporal pattern (Berridge et al., 2000). Conversely, not all stimuli that increase intracellular Ca^{2+} concentration initiate a common cell response. Thus, Prentki et al. (1988) proposed that "Ca^{2+} fingerprints" may be generated in an agonist-specific manner. However, the precise mechanisms that underpin stimulus-specific Ca^{2+} signalling remain obscure.

We know that agonist specificity is determined, in part, by the release of Ca^{2+} from intracellular stores in a manner dependent on second messengers and their associated Ca^{2+} release channels. During pharmaco–mechanical coupling in smooth muscle, it has long been accepted that many G-protein-coupled receptors induce the production of inositol 1,4,5 trisphosphate (IP_3), which leads to the activation of one or more of the known IP_3R subtypes on the sarcoplasmic reticulum (SR) and release of Ca^{2+} from this store (Somlyo & Somlyo, 1994; Berridge et al., 2000). However, there is a growing body of evidence to support a role for Ca^{2+} mobilising pyridine nucleotides in the regulation of intracellular Ca^{2+} signalling in a number of cell types, including smooth muscle (Galione, 2002; Li et al., 2003). Consistent with this proposal, the enzymes for the synthesis and metabolism of nicotinic acid adenine dinucleotide phosphate (NAADP; Wilson et al., 1998; Patel et al., 2001; Yusufi et al., 2002) and cyclic adenosine diphosphate-ribose (cADPR; de Toledo et al., 1997; Wilson et al., 2001; Galione, 2002; Zhang et al., 2004) have been shown to be associated with smooth

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