Erratum

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The inhibition of α_1 -adrenoceptor-mediated contractions of rabbit pulmonary artery by Ca^{2+} -withdrawal, pertussis toxin and N-ethylmaleimide is dependent on agonist intrinsic efficacy

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Due to an unfortunate printing error the first two sentences of the Summary were confused. The correct version is now printed below.

Summary. Contractions were induced in rings of rabbit pulmonary artery with the preferential α_1 -adrenoceptor agonists, phenylephrine, methoxamine and St 587 [2-(2-chloro-trifluoromethyl-phenylimino)imidazolidine and the preferential α_2 -adrenoceptor agonists, clonidine and B-HT 920 [6-allyl-2-amino-5,6,7,8-tetrahydro-4H-thiazolo-(4,5-d)azepine]. Phenylephrine and methoxamine acted as full agonists whereas St 587, clonidine and B-HT 920 were partial agonists (intrinsic activities 0.62, 0.38 and 0.42, respectively). Experiments with α_1 - and α_2 -adrenoceptor antagonists indicated that the receptors involved are of the α_1 type only. Removal of extracellular Ca^{2+} inhibited maximal contractions to phenylephrine and methoxamine by 30% and 49%, respectively. The remaining contraction components of the full agonists were abolished by the "intracellular Ca^{2+} antagonist" TMB-8 [8-(N,N-diethylamino)octyl-3,4,5-trimethoxybenzoate]. Contractions to St 587, clonidine and B-HT 920 were virtually abolished in Ca^{2+} -free medium. Pretreatment of the donor rabbits with pertussis toxin (2.5 $\mu\text{g}/\text{kg}$ i.v., 5–6 days before sacrifice) attenuated the efficacies of the full agonists, phenylephrine and

methoxamine by only 24% and 17%, respectively, whereas maximal contractions to the partial agonists, St 587, clonidine and B-HT 920, were inhibited by 46%, 61% and 75%, respectively. Also the sulfhydryl reagent, N-ethylmaleimide (10 μM), reduced contractile efficacies of phenylephrine and methoxamine to a lesser degree than those of St 587, clonidine and B-HT 920. When agonists were used at equieffective concentrations (i.e. EC_{30-40} for phenylephrine and methoxamine, EC_{70-80} for St 587 and EC_{99} for clonidine and B-HT 920) the degree of inhibition by removal of extracellular Ca^{2+} , pertussis toxin and N-ethylmaleimide was similar for all agonists. These data suggest that a unitary α_1 -receptor may stimulate contractions via two different mechanisms. At a low degree of receptor stimulation, contractions are mediated by a pertussis toxin- and N-ethylmaleimide-sensitive influx of external Ca^{2+} . At a higher degree of receptor stimulation, an additional mechanism is activated which is insensitive to the two G protein inhibitors and mediated by Ca^{2+} mobilization from intracellular sites.