control ratios (RCR) were measured using respiratory medias containing [K+] of 15, 37, 81, and 146 mM. In all measurements, the media contained (in mM): 1 EGTA, 20 HEPES, 5 MgCl₂, 5 KPO₄ and 1 g/l bovine serum albumin. pH was adjusted to 7.4 and the osmolality to 330 mOsm/kg H₂O using a 1:3 ratio of sucrose and mannitol. The RCR of kidney cortex mitochondria decreased when the [K⁺] was elevated compared to the media containing 15 mM K⁺ (5.2 ± 0.2 vs. 2.5 ± 0.2, 3.7 ± 0.2, 3.9 ± 0.2, and 3.0 ± 0.1, respectively). However, RCR of heart mitochondria was lowest at 37 mM (3.9 ± 0.3) and was highest at 146 mM K⁺ (10.1 ± 0.45). A two-way ANOVA showed that kidney cortex mitochondria have a different sensitivity towards K⁺ compared to heart mitochondria (interaction p < 0.05, treatment p < 0.05, and group p < 0.05). Gibenclamide (100 µM), an inhibitor of the ATP-sensitive K⁺ channel, increased RCR in kidney cortex mitochondria at 15 mM K⁺ (+32%), but significantly more at 146 mM K⁺ (+47%). Blockade of the voltage-gated K⁺ channel by 4-aminoypyridine (4-AP, 1 mM) together with glibenclamide improved RCR by +73% at 146 mM K⁺. Neither of the applied K⁺-channel blockers had any effect on the RCR of heart mitochondria. Mitochondria swelling at increasing [K⁺] were observed in kidney cortex mitochondria, measured as loss of absorbance at 540 nm. Kidney cortex mitochondria in K⁺-based media are non-functional in response to an increase of [K⁺]. Heart mitochondria do not display K⁺-sensitivity to the same degree, but rather increase respiratory control ratios (RCR) by +47%. Blockade of the voltage-gated K⁺ channel by 4-aminoypyridine (4-AP, 1 mM) together with glibenclamide improved RCR by +73% at 146 mM K⁺. Neither of the applied K⁺-channel blockers had any effect on the RCR of heart mitochondria. Mitochondria swelling at increasing [K⁺] were observed in kidney cortex mitochondria, measured as loss of absorbance at 540 nm. 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