

**TEMPERATURE-DEPENDENT EXPRESSION
OF SARCOLEMMA POTASSIUM CURRENTS
IN ATRIAL AND VENTRICULAR MYOCYTES
OF THE RAINBOW TROUT HEART**

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Introduction

The shape and duration of the cardiac action potential (AP) differ greatly among different vertebrate species and different types of cardiac myocytes. Numerous K^+ conductances of the sarcolemma, including transient outward currents, inwardly rectifying currents and delayed rectifier currents, contribute to the great variability of the cardiac AP waveforms (Snyders, 1999). The diversity of K^+ channel expression in cardiac myocytes allows fine tuning of the AP shape and provides efficient means of regulating cardiac excitability and maintaining electrical stability of the heart.

We recently showed that adaptation to cold decreases the mechanical refractoriness of the rainbow trout atrial and ventricular muscle (Aho & Vornanen, 1999). The shorter refractory period and faster rate of force restitution after cold-acclimation are, at least partly, due to the improved Ca^{2+} handling by sarcoplasmic reticulum (SR) as they were cancelled by ryanodine, a blocker of SR Ca^{2+} -release channels. Because mechanical and electrical restitution are closely connected and K^+ currents contribute to electrical refractoriness of the

cardiac muscle, we hypothesised that K^+ channels might be involved in temperature adaptation of trout cardiac function. Therefore, we examined whether K^+ conductances of the rainbow trout cardiac myocytes are affected by thermal acclimation.

Methods

Rainbow trout were acclimated at +4°C or +18°C for a minimum of three weeks. Standard patch-clamp methods in whole-cell configuration were used to record ionic currents and APs in enzymatically isolated cells at either $10 \pm 1^\circ\text{C}$ or $20 \pm 1^\circ\text{C}$ (Vornanen, 1998).

Results

After blocking other membrane currents, K^+ conductances of the sarcolemma were measured by clamping the membrane from a holding potential of -80 mV to various voltages between -120 and +20 mV (Fig. 1). Thermal acclimation had a strong effect on the sarcolemmal K^+ conductance of the rainbow trout cardiac myocytes and the effect was qualitatively similar in atrial and ventricular cells. The inward current at the negative side of the K^+ equilibrium potential was strongly reduced by cold-acclimation which was especially clear in ventricular cells where this current component is well developed. In ventricular cells, the slope conductance between -120 and -80 mV (at 20°C) was 0.737 ± 0.094 nS pF^{-1} for WA trout but only 0.269 ± 0.035 nS pF^{-1} for CA trout ($P < 0.001$), indicating about 64% reduction after acclimation to cold. In contrast to the inward current, the density of the outward current at the positive voltages was increased after cold-acclimation. The densities of the outward current at +20 mV (20°C) were 2.85 ± 0.35 and 0.96 ± 0.30 pA pF^{-1} ($p < 0.001$) in ventricular cells of CA and WA fish, respectively. The current densities in atrial cells were 8.93 ± 0.61 and 1.88 ± 0.32 pA pF^{-1} ($P < 0.001$) for CA and WA fish, respectively. Thus, the steady-state current-voltage relations indicate that acclimation to cold suppresses inward K^+ current(s) but enhances late outward K^+ current(s) in the cardiac myocytes of the rainbow trout heart.

The inward current at the negative side of the K^+ equilibrium potential was inhibited by $100 \mu\text{M Ba}^{2+}$ indicating it as the background inward rectifier current. The outward current at positive voltages was completely blocked by $2 \mu\text{M E-4031}$ and by $100 \mu\text{M}$ sotalol, specific blockers of the rapid component of the delayed rectifier.

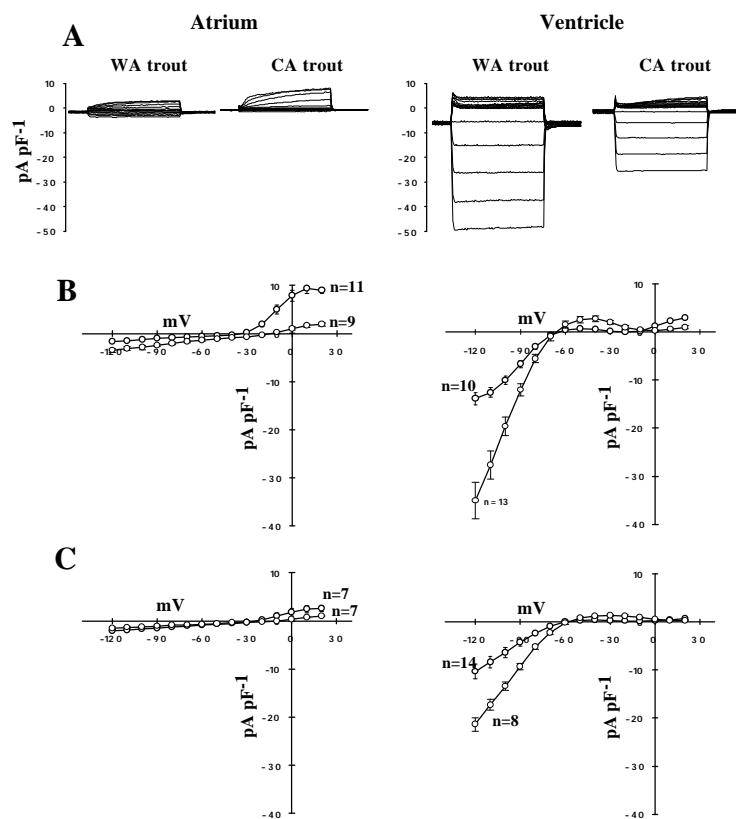


Figure 1. Steady-state current-voltage relations of rainbow trout atrial (left) and ventricular (right) myocytes. Original current recordings are shown in A. Mean current-voltage relations at 20°C and 10°C are shown in B and C, respectively.

Conclusions

The present experiments indicate that two major components of the sarcolemmal K^+ conductance of the rainbow trout cardiac myocytes are the E-4031 sensitive delayed rectifier current, I_{Kr} and the Ba^{2+} -sensitive background inward rectifier, I_{K1} . Furthermore, it is shown that thermal acclimation modifies these K^+ conductances in opposite manner: I_{Kr} is increased and I_{K1} is decreased by cold acclimation. The cold-induced increase in I_{Kr} can be regarded as a compensatory adaptation which limits AP duration and decreases refractoriness of the heart (Aho and Vornanen, 2000) and thereby allows compensatory increase in heart rate and cardiac output in the cold. The explanation for the reduction of I_{K1} is less evident, but may involve energy savings by reducing the demand for ATP-dependent ion pumping across the sarcolemma.

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References

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