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# Elevated SR $Ca^{2+}$ Channel Activity in Diabetic Skeletal Muscles

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Dysfunctions of skeletal muscles have been frequently reported in chronic diabetic mellitus (DM). In order to investigate the molecular mechanisms of abnormal function, the junctional sarcoplasmic reticulum (HSR) vesicles of skeletal muscles were prepared from the control and the streptozotocin-induced diabetic rats. The activity of SR $Ca^{2+}$-ATPase was increased by 14% and SR $^{45}Ca^{2+}$ uptake was three times higher in DM muscle. The great increase in $^{45}Ca^{2+}$ uptake may be due to not only the increase in $Ca^{2+}$-ATPase activity but the influx of $^{45}Ca^{2+}$ through the SR $Ca^{2+}$ release channel. The amount of SR $^{45}Ca^{2+}$ release was twice higher and the open probability (Po) of the channel was increased by 6~8 times in DM muscle. When the SR $Ca^{2+}$ release channel (ryanodine receptor) was reconstituted into planar lipid bilayer, the mean open time of the channel was increased in DM muscle while no difference in both slope conductance and frequency of channel opening. The $Ca^{2+}$-dependence of the channel was qualitatively similar in both muscles. The effects of various modulators were also investigated and ATP increased Po in only control channel. The scorpion venom, Lqh, decreased Po of the control channel while no effect of Lqh on the channel activity was observed in DM muscle. In conclusion, the activities of SR $Ca^{2+}$-ATPase and SR $Ca^{2+}$ release channel were increased in DM skeletal muscle.