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Dear ASCEPT Secretariat,

Subject: Report for IUTOX/IUPHAR travel grant

As a honored recipient of IUTOX/IUPHAR travel grant from ASCEPT, I have attended the conference during 2nd – 7th of July, presented my poster on the 4th of July, manned the booth for ASCEPT on 5th of July, and returned to Australia on 13th of July.

The conference was outstanding for academic and social events, I enjoyed meeting new friends, exchange opinions, broadening my knowledge through it.

There were a variety of topics discussed in the conference. I was most interested in one session that Na⁺/Ca²⁺ exchanger (NCX) was discussed in depth, as I work in the same area for my PhD study. Prof Annunziato from Department of Neuroscience, University of Naples Federico II, Italy gave a very interesting talk clarifying the different expression of NCX products in the development of ischemic damage and the related modulation by NGF. Another expert in this

area: Prof. Iwamoto from the Department of Pharmacology, Fukuoka University, Japan talked about the most important and complicated part in this area: inhibitors. In addition, Prof. Bers demonstrated the function of NCX in normal electrophysiological condition and during heart failure. Finally, Prof. Herchuelz from University Libre de Bruxelles, Belgium demonstrated his research in the molecular biological aspect of NCX using NCX1 overexpressed and knockout models. All these talks filled in the gaps of our knowledge in the area of NCX and would be very instructive for the future study.

At last, I would like to express my gratitude to ASCEPT for giving me this great opportunity to attend this conference. It would be beneficial for my research career.

With regards,

Yours truly,

JUN ZHAO

P.S. My abstract for IUPHAR conference is attached.

MODULATION OF NITRIC OXIDE DONORS ON THE $\text{Na}^+/\text{Ca}^{2+}$ EXCHANGER IN RAT ISOLATED AORTA

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The $\text{Na}^+/\text{Ca}^{2+}$ exchanger (NCX) is a bi-directional transmembrane ion transporter that is involved in regulating the intracellular $[\text{Ca}^{2+}]$ in most tissues. Lowering the concentration of extracellular sodium ($[\text{Na}^+]_o$) results in contraction of rat aortic rings by inducing Ca^{2+} inflow through NCX. Previous studies have suggested that in the presence of low $[\text{Na}^+]_o$, nitric oxide is released from endothelial cells and inhibit NCX. The aim of the present study was to examine the effects of sodium nitroprusside (SNP) on low $[\text{Na}^+]_o$ -induced contraction of endothelium-denuded aortic rings isolated from male Sprague-Dawley rats. 30nM SNP produced a greater relaxation response in rings precontracted with low $[\text{Na}^+]_o$ (1.18mM) than thromboxane A_2 -mimetic U46619 (n=5, $P<0.05$) or 80mM KCl (n=5, $P<0.001$). These results indicate that constriction by Ca^{2+} entry through NCX is highly sensitive to inhibition by nitric oxide, which may explain why the endothelium dampens NCX mediated constriction.