generic enoxaparin group. Hence, be careful to use generic enoxaparin in patients who had percutaneous coronary intervention.

**Basic Science, Animal Models and Preclinical Studies**

(TCTA A-146 to TCTA A-149)

**TCTA A-146**

**MicroRNA-21 Expression and its Association with Leukocytes Infiltration on the Early Phase of Coronary Microembolization**

Zhangwei Chen, Jiayi Ma, Jiayin Qian, Shuifei Chang, Aijun Sun, Yunzeng Zou, Junbo Ge

**Zhongshan hospital, Shanghai, China**

**Background:** MicroRNAs are small non-coding RNAs that regulate gene expression at the post-transcriptional level by either degradation or translational repression of a target mRNA. It has been demonstrated that miR-21 plays a critical role on inflammatory response after acute myocardial infarction. However, it is unknown about myocardial expression of miR-21 after coronary microembolization (CME) in mini-pigs.

**Methods:** Ten mini-pigs were enrolled in this study, including sham-operation group (n=5) and CME group (n=5). Troponin T and interleukin-6 (IL-6) were detected on baseline, 6 hour and 7 days after CME. Myocardial expressions of miR-21 were detected by Real-time PCR method. Myocardium specimens were embedded in paraffin for hematoxylin and eosin (HE) staining, while number of leukocytes infiltration was analyzed by Leica DFC 320 digital soft.

**Results:** Compared with sham-operation group, serum level of troponin T and IL-6 were increased significantly after 6 hours in CME group (Troponin T: 0.64±0.239 vs. 0.02±0.01 mg/ml, P<0.05; IL-6: 140±30.8 vs. 35.4±21.4 ng/ml, P<0.05). Leukocyte infiltration on microembolization associated myocardium was also increased markedly after CME. We found that myocardial expression of miR-21 was increased significantly at 7-day after CME (CME vs. sham: 3.72±1.51, P<0.033), which also had a positive relation (r=0.656, P=0.046) with average number of leukocytes infiltration on microembolization area.

**Conclusion:** Myocardial miR-21 expression was increased after coronary microembolization, which could be involved in the process of leukocytes infiltration.

**TCTA A-147**

**TongXinLuo Protects Human Cardiac Microvascular Endothelial Cells from Hypoxia/Reoxygenation Injury by Inducing Autophagy via the MEK/ERK Pathway**

Hehe Cui, Xiangdong Li, Na Li, Kang Qi, Qing Li, Chen Jin, Qian Zhang, Lian Liu, Jiang, Yuejin Yang

**Fuwai Hospital, Beijing, China**

**Background:** In contrast to cardiomycocytes, autophagy in cardiac microvascular endothelial cells (CMECs) during ischemia/reperfusion (I/R) injury has not been fully investigated. Tongxinluo (TXL), which is a traditional Chinese medicine formulation consists of extracts or powders from Radix ginseng, Bathus martensi, Hirudo, Eucommia ulmoides, Scleropodium subspinosa, Periostracum cicadae, Ratosterae rhizoma, Semen ziziphi spinosae, Lignum dalbergiae odoriferae, Lignum santals albi, and Borneol. was previously demonstrated to be vascular protective. This study was designed to elucidate the role of autophagy and its regulatory mechanisms by TXL in CMECs subjected to IR injury.

**Methods:** CMECs were exposed to varying concentrations of TXL solution for 30 min and subjected to hypoxia/reoxygenation (HR) each for 2 h to determine the optimal working concentration. The autophagy inhibitor 3-methyladenine (3-MA), the autophagy promoter rapamycin, and the MEK inhibitor PD98059 were used to further investigate the role and the modulatory mechanism of autophagy in CMECs.

**Results:** The results indicated that HR significantly induced autophagy, as identified by an increased number of monodansylcadaverine (MDC)-positive CMECs, increased autophagosome formation, and a higher type II/type I of light chain 3 (LC3-II/LC3-I) ratio (p < 0.05), but not Beclin-1 expression. The inhibition of autophagy using 3-MA was found to be prosapotic whereas the induction of autophagy by rapamycin was antiapoptotic, which was reflected by index such as flowcytometric apoptotic rates, expression of Bcl, Bax, and Cytochrome c (p < 0.05). TXL enhanced autophagy and decreased apoptosis in a dose-dependent manner. TXL reached its largest anti-apoptotic effect in CMECs at 800 μg/ml (Mean±SEM, 9.92±0.49%) in the 800 μg/ml TXL group vs. 21.04±1.11% in the H/R group, p < 0.05), and MDC-positive cell rate of the 800 μg/ml TXL group was significantly higher than that of the H/R group (p < 0.05). 3-MA attenuated the TXL-promoted autophagy and antiapoptotic effects (p < 0.05), whereas the induction of autophagy by rapamycin was antiapoptotic, which was reflected by index such as flowcytometric apoptotic rates, expression of Bcl, Bax, and Cytochrome c (p < 0.05). TXL enhanced autophagy and decreased apoptosis in comparison to TXL alone. TXL upregulated the phosphorylation of MEK and ERK, but ERK phosphorylation was abrogated by PD98059, which also decreased autophagy and increased apoptosis in comparison to TXL alone.

**Conclusion:** These results suggest that autophagy is a protective mechanism in CMECs subjected to ischemia/reperfusion injury and that TXL can promote autophagy via activation of the MEK/ERK pathway.