0.29±0.12, $p<0.05$), The protein expression levels of RYR₂ was significantly higher in the aged dogs than in the adult dogs (0.18±0.21 vs 0.08±0.36, $p<0.05$), There were not significantly different protein expression levels of SECRA₂ and Calpain I in two groups.

Conclusion These data suggest that the change of mRNA and protein expression of CaV1.2 and RYR₂ of LVDCC may serve as the molecular basis of I_{Ca-L} remodeling respectively in aged dogs. This plays an important role in the predisposition to developing atrial fibrillation (AF) due to ageing.

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MOLECULAR MECHANISMS OF IONIC REMODELING IN L-TYPE VOLTAGE DEPENDENT CALCIUM CHANNEL OF AGE-RELATED CHANGE IN ATRIAL OF CANINE

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Purpose The present study aims to investigate the molecular basis of L-type voltage dependent calcium channel (LVDCC) in adult and aged canine.

Methods The action potential duration (APD₉₀), amplitude of action potential plateau, I_{Ca-L} peak current density were of LVDCC, recorded by patch clamp technique. The mRNA gene and protein expression levels of α1c subunit (CaV1.2), sarcoplasmic reticulum Ca²⁺-ATPase (SECRA₂), Calpain-I, ryanodine receptor (RyR₂) were detected by semi-quantitative RT-PCR.

Results I_{Ca-L} peak current density was (-14.04±0.82 pA/pF), in adult group compared with (-8.11±0.54 pA/pF, $p<0.05$) in the aged group and action potential duration to 90% repolarisation (APD₉₀) of aged group was significantly decreased. The mRNA gene expression levels of CaV1.2 was significantly lower in the aged dogs (0.9±0.35) than in the adult dogs (2.38±0.4, $p<0.05$), The mRNA gene expression levels of RYR₂ was significantly higher in the aged dogs (4.39±4.68) than in the adult dogs (1.49±1.69, $p<0.05$). There were not significantly different gene expression levels of SECRA₂ and calpain I in two groups; The protein expression levels of CaV1.2 were significantly lower in the aged dogs than in the adult dogs (0.13±0.10 vs