1-O-D2-2 Oral Sessions**Induction of Autophagy Attenuates Imatinib-induced Cardiotoxicity**

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Imatinib, a tyrosine kinase inhibitor, was reported to induce cardiotoxicity. Autophagy is an intracellular bulk protein and organelle degradation process, but its roles in cardiac diseases are unclear. We examined whether imatinib induces myocyte autophagy and the role of autophagy in imatinib-induced cardiotoxicity using *in vitro* and *in vivo* experiments. In *in vitro* experiments, neonatal rat cardiac myocytes were treated with imatinib (10 μ M). Imatinib increased microtubule-associated protein light chain (LC) 3-II and acridine orange-stained autolysosome expression, suggesting that it induced autophagy. Consequently, imatinib induced mitochondrial reactive oxygen species (ROS) production and loss of mitochondrial membrane potential, assessed by the fluorescent indicator, MitoSOX and JC-1, respectively, leading to myocyte apoptosis. 3-methyl-adenine (3MA), an autophagic inhibitor, exacerbated imatinib-induced apoptosis. In *in vivo* experiments, C57BL/6 mice were treated with imatinib (200 mg/kg/day) for 5 weeks in the presence or absence of 3MA. Echocardiographic measurement showed that imatinib induced left ventricular (LV) dilatation and systolic dysfunction. Apoptosis and LC3-II expression in cardiac tissue were increased by imatinib. Co-treatment with 3MA and imatinib further impaired imatinib-induced cardiac apoptosis and LV dysfunction. Imatinib increases myocyte autophagy as a consequence of apoptosis and autophagy has a pro-survival role in imatinib-induced cardiac impairment.