Abstract No. **40**
Category: **Heart Failure and Cardiomyopathies**
Title: **Resveratrol prevents right ventricular hypertrophy remodeling and loss of function but not pulmonary arterial hypertension in a murine model of pulmonary hypertension**
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Abstract:

Right ventricle function (RV) is a key mechanism involved in pulmonary hypertension (PAH), which is associated with clinical prognosis. Resveratrol (RES) is a phenolic compound with cardio-protective effect in PAH, although the underlying mechanisms remained to be elucidated. In this regard, we decided to analyze the echocardiographic, macro- and histological changes in lungs and heart, hemodynamics and contractile function in cardiomyocytes isolated from animals with monocrotaline-induces PAH (MC) and treated with MC plus RES. The standardized MC model showed an increased amount of muscular arteries, percentage of luminal occlusion, in the lungs of treated rats with MC, which was similar to RES-treated rats, there was also an increased amount of fibrosis in the RV compared to controls, but less than MC-treated rats.

There were no significant differences in the weight of the heart and lungs between MC and RES-treated rats. Interesting echocardiographic changes were found in the rats treated with RES having RV function values similar to untreated controls and lower to MC-treated rats, values like TAPSE ($p<0.01$), RV output ($p<0.05$), RV output tract ($p<0.05$), RV free wall (p=ns), septum free wall (p=ns), RV chamber measurements (p=ns). However the values associated with lung pressure like PAAT ($p<0.001$), and PADT ($p<0.05$), were similar to the MC-treated rats. There were no differences among systemic blood pressure, weight gain or heart rate between the groups. Finally, we observed MC-induced changes in cell shortening in isolated cardiomyocytes, noted as a lower cell shortening in the MC group during adrenergic response compared with control animals ($p<0.001$).

These findings show that early changes in cardiomyocytes dysfunction secondary to pulmonary hypertension might be due to an inability to manage calcium dynamics and adverse remodeling in the cardiac muscle. The MC-treated group developed changes associated with PAH, but RES induced a cardioprotective effect in the hearts but not the lungs in the treated rats. Our results, suggested that RES might reduce fibrosis and hypertrophy, maintaining RV cell shortening, thereby preventing development of PAH.