

## MEETING ABSTRACT

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# 228 Functional cardiac phenotyping of vasoactive intestinal peptide (VIP) deficient mice by MR microscopy

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## Introduction

Mice with deletion of the gene for Vasoactive Intestinal Peptide (VIP) exhibit pulmonary arterial hypertension (PAH) with pulmonary vascular remodeling and RV hypertrophy in the absence of hypoxemia<sup>1</sup>. We hypothesized that these mice would also develop a decreased functional capacity of the right ventricle. We tested this hypothesis using MR microscopy to evaluate right and left heart ventricular volumes and corresponding ejection fractions in VIP-deficient and wildtype (WT) control mice.

## Methods

A total of twelve male mice (10–12 months) were used for the studies; Group 1 = Control; C57BL6/J male mice (n = 6) and Group 2 = VIP<sup>-/-</sup> male mice (n = 6). All animals were anesthetized and breathing spontaneously during the study. The electrocardiogram (ECG), respiratory rate, heart rate and body temperature were continuously monitored (SA Instruments). MR images were acquired on a 9.4 T horizontal bore Bruker magnet. Short axis bright blood views of the heart were obtained with an ECG gated multi-slice 2D-FLASH gradient echo sequence: TR = 7.5 ms, TE = 2.9 ms, Flip angle = 10°, in-plane-resolution: 0.133 × 0.133 mm<sup>2</sup>, slice thickness: 1 mm; interslice gap = 0.5 mm. The heart rate and

consequent R-R interval allowed for acquisition of 12 cine frames within each cardiac cycle with the given temporal resolution of 7.2 ms. Image analysis was performed using *SEGMENT* <http://segment.heiberg.se>. For all studies, end-systole was referred to as the frame with minimal ventricular cavity volume.

## Results

As can be seen from Table 1, in the WT control mice left ventricular (LV) and right ventricular (RV) ejection fractions (EF) were similar. However, in the VIP<sup>-/-</sup> mice the RV EF was significantly lower than the RV EF of the WT control group. Further, end-systolic volumes were approximately 50% larger in the VIP<sup>-/-</sup> mice compared to controls. Figure 1 demonstrates the enlarged end-systolic RV volume in a VIP<sup>-/-</sup> deficient mouse. The figure displays two series of MR microscopy short axis cardiac images at the time of end-diastole from a WT control mouse (upper panel) and a VIP<sup>-/-</sup> mouse (lower panel) and there is clear visual evidence of a dilated RV in the VIP<sup>-/-</sup> mouse.

## Conclusion

In conclusion, our preliminary data demonstrate that VIP<sup>-/-</sup> mice have RV disease phenotypically expressed as increases in RV end-systolic volume and a reduced

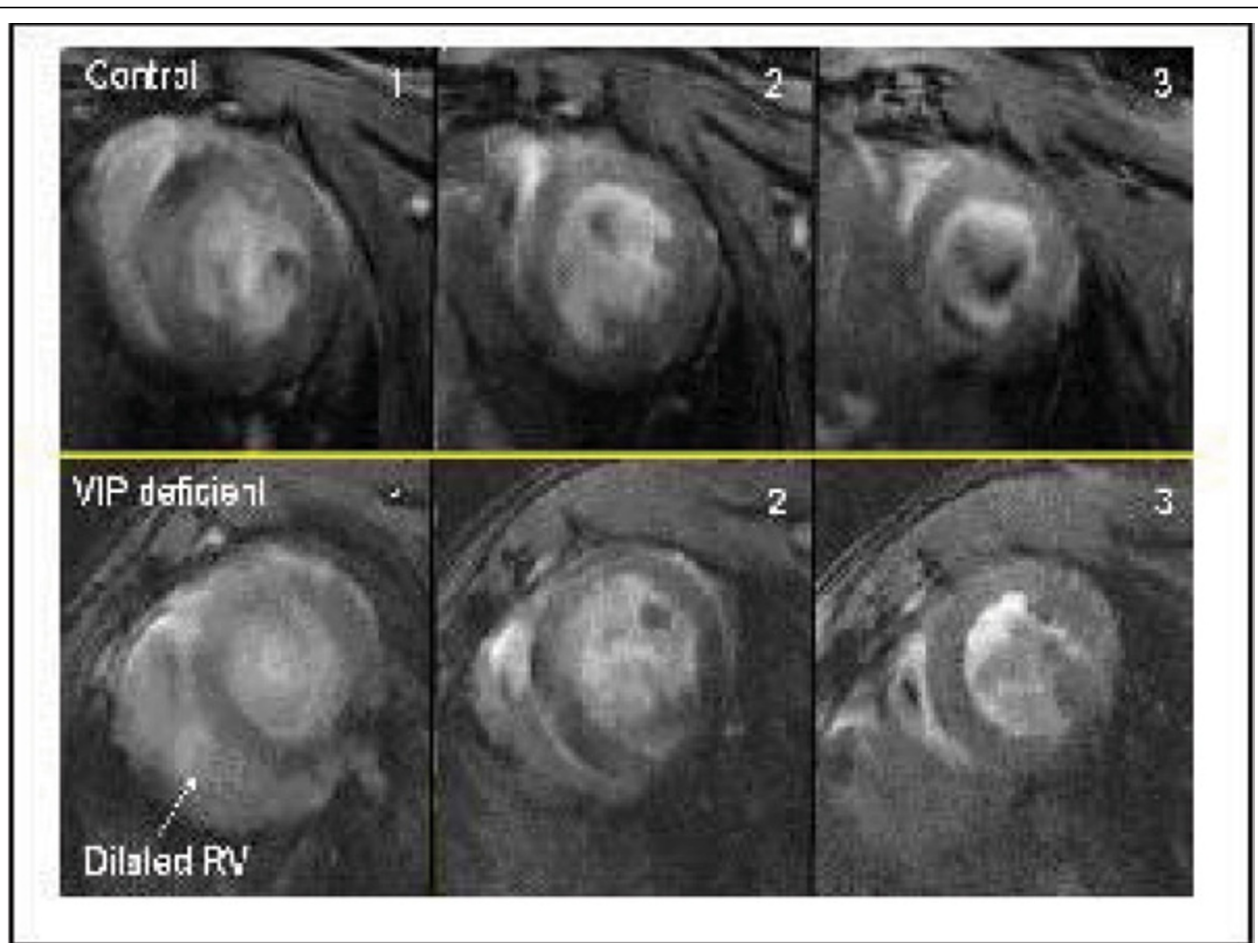
**Table 1**

Mouse Genotype	EDV (μl) LV	EDV (μl)RV	ESV (μl) LV	ESV (μl) RV	EF (%) LV	EF (%) RV
WT (n = 6)	47.0 ± 7.0	23.6 ± 2.6	15.9 ± 3.7	8.8 ± 3.0	65.7 ± 8.7	63.1 ± 12.4
VIP <sup>-/-</sup> (n = 6)	43.9 ± 7.4	32.0 ± 11.2	16.6 ± 4.4	19.3 ± 10.8**	62.5 ± 4.7	41.9 ± 12.4*

EDV = end-diastolic volume; ESV = end-systolic volume.

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**Figure 1** We quantified ventricular volumes from Cardiac CINE MR images to phenotype the cardiac functional capacity in Vasoactive Intestinal Peptide deficient (VIP<sup>-/-</sup>) mice known to develop pulmonary artery hypertension. Pronounced RV dilation was observed in VIP<sup>-/-</sup> mice when compared to controls.

RV EF where as the LV is unaffected when compared to WT controls. Studies are underway to characterize the development of RV pathology in VIP<sup>-/-</sup> mice over time and to examine the effect of VIP therapy on the RV functional capacity in these mice.

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