

## Aims

Leptin has recently been related to myocardial remodeling in animal experimentation studies on heart failure (HF). Furthermore, leptin has been reported to be related to diastolic dysfunction, however only in healthy population. With the emergence of new medical therapies targeting cardiac remodeling, there needs to be a better understanding of the metabolic pathways involving leptin. Our study aims to investigate leptin's correlation to parameters of systolic and diastolic heart function, as well as epicardial and pericardial adipose tissue (EAT and PAT) in heart failure (HF) patients.

## Methods

The study included 51 patients with chronic heart failure with reduced ejection fraction (HFrEF) of ischemic (n=22) and non-ischemic (n=29) origin (NYHA II-III, mean EF 29.56 %, SD 8.1; mean BMI 28.08, SD 5.8). Serum concentrations of leptin, NT-proBNP, HbA1c, LDL, and total cholesterol were also measured. Global longitudinal strain (GLS) and other LV function parameters were assessed in transthoracic echocardiography, as well as EAT and PAT in parasternal long and short-axis views.



Figure 1 and 2 : EAT and PAT measurement in parasternal long and short-axis views

## Results

We found a significant correlation between leptin serum concentration and epicardial, as well as pericardial adipose tissue (EAT  $r=0.336$ ,  $p=0.030$ ; PAT  $r=0.565$ ,  $p<0.001$ ). There was a significant negative correlation between leptin and GLS ( $r=-0.332$ ;  $p=0.045$ ), and a positive correlation between Leptin and E/E' ration ( $r=0.373$ ;  $p=0.039$ ). There was no significant difference between ischemic and non-ischemic HF patients.

Patients, N=51

|   |               |
|---|---------------|
| Age   | 61 ± 12       |
| Male/Female                                 | 33/18         |
| BMI   | 28.08 ± 5.8   |
| NYHA II/III                                 | 53/47 %       |
| Etiology – ischemic vs. non-ischemic origin | 43/57 %       |
| <b>Echo</b>                                 |               |
| LVEF (%)                                    | 29.56 ± 8.1   |
| LVEDd (mm)                                  | 59.68 ± 8     |
| Left atrial volume (ml/m <sup>2</sup> )     | 45 ± 19       |
| E/E'  | 15.14 ± 7.9   |
| Global longitudinal strain (%)              | -7.79 ± 3.1   |
| EAT (mm)                                    | 3.9 ± 1.8     |
| PAT (mm)                                    | 6.5 ± 3.1     |
| <b>Serum measurements</b>                   |               |
| NT-pro BNP (pg/ml)                          | 2183 ± 2380   |
| Creatinine (mg/dl)                          | 1.07 ± 0.33   |
| LDL (mg/dl)                                 | 91.86 ± 34.69 |
| Total cholesterol (mg/dl)                   | 162 ± 46      |
| HbA1C (%)                                   | 5.7 ± 0.7     |
| Leptin (ng/ml)                              | 16.6 ± 19.7   |
| <b>Risk factors</b>                         |               |
| Smoking                                     | 39 %          |
| Diabetes                                    | 20 %          |
| Family history of cardiovascular disease    | 27 %          |
| Hypertension                                | 54 %          |
| Dyslipidemia                                | 58 %          |
| Atrial fibrillation                         | 29 %          |
| <b>Medication</b>                           |               |
| Betablockers                                | 86 %          |
| ACE inhibitors/ARBs                         | 82 %          |
| Diuretics                                   | 53 %          |
| Aldosterone antagonists                     | 65 %          |
| Statins                                     | 59 %          |

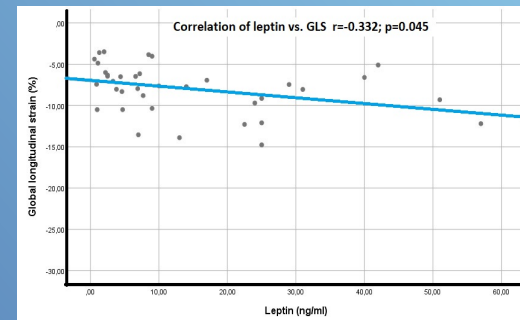
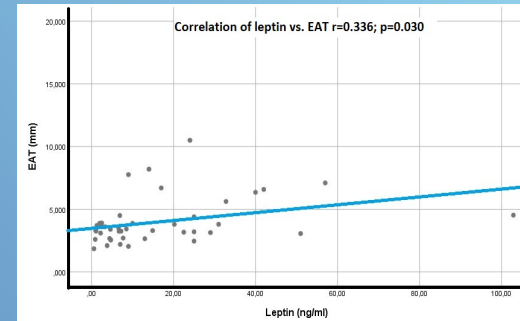


Table 1: Clinical characteristics  
 Data in mean ± SD or percentages

Figures 3 and 4: Correlation between EAT and leptin and between GLS and leptin

## Conclusion

We provide evidence of serum leptin correlation to remodeling parameters, as well as epicardial and pericardial fat tissue in HF patients. Whether leptin has positive effects on reversing or preventing remodeling in heart failure, needs further investigation.

\* The authors declare no conflicts of interest