**INTRODUCTION**

Preeclampsia (PE) occurs in 3.4% of all pregnancies. It is one of the leading causes of maternal and fetal morbidity and mortality, which account for ~50,000-75,000 of maternal deaths/year worldwide. It is related with maternal and fetal endothelial dysfunction. Preeclampsia may affect fetal growth and development, which may lead to programming of adult onset diseases later in life. This is associated with increased risk of cardiovascular disorders in the adult offspring. Pregnancies with male fetuses have higher incidence of later-onset PE.

**METHODS**

Freshly isolated, highly pure (CD31 dynabeads), unpassaged (P0) fetal endothelial cells (PE-HUVECs) from normal term (NT) and PE pregnancies (NT-F, NT-M, PE-F, PE-M) were treated with ECM-b (control) or TGF β (10ng/ml), the electrical resistance was constantly recorded for 25h. (n=5). *Differ from corresponding negative control (NegCtrl). (P<0.05, Kruskal-Wallis test).

**RESULTS**

- Fig. 2. Preeclampsia differentially dysregulates transcriptomic profiles and miR29a/c-3p target genes of PE-HUVECs. (A) Circos plots illustrate the chromosomal position of differentially expressed genes between NT-F vs. NT-G (grey dots, 1561 genes), PE-F vs. NE-G (red dots, 927 genes), and PE-M vs. NT-M (blue dots, 172 genes). Each dot represents one gene. Numbers and letters in the outer ring indicate the chromosome. For each scatter plot track, dots outside and inside of the centerline are up- and down-regulated genes, respectively (Zhou C et al., Hypertension 2019 ). (B) Multidimensional scaling (MDS) plot showing the similarities/disparities among samples. Each dot representing the gene expression pattern of miR29a/c-3p target genes in a sample.

- Fig. 5. Knockdown/overexpression of miR29-a/c-3p differentially dysregulates female and male fetal endothelial proliferation in responses to TGFβ1. Female and male P0-HUVECs from NT and PE were treated with vehicle, negative control, miR29a/c-3p inhibitor (-), or miR29a/c-3p mimic (+) for 24hrs. After serum starvation, sub-confluent cells were treated with ECM-b (control) or TGFβ1 (10ng/ml), the electrical resistance was constantly recorded for 25h. *Differ from corresponding negative control (NegCtrl). (P<0.05, Kruskal-Wallis test).

**CONCLUSIONS**

- PE differentially dysregulates miR29a/c and its targets genes in female and male fetal endothelial cells in association with differential cellular responses to TGFβ1.

- MiR29a/c-3p may play a role in the PE differentially dysregulated female and male fetal endothelial cells to TGFβ1.

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