INTRODUCTION
Cortisol homeostasis dysregulation has been associated with hypertension in adults. Higher levels of cortisol have been described in preterm-born individuals, who have a higher risk of hypertension.

The 11β-hydroxysteroid dehydrogenase (11β-HSD) type 2 metabolizes cortisol into cortisone, preventing activation of mineralocorticoid receptors. The inverse process is mediated by 11β-HSD type 1. The A- ring reductases inactivate cortisol and cortisone into tetrahydro-metabolites. Defects in 11β-HSD1 and 11β-HSD2 produce cortisol-dependent mineralocorticoid excess. Additionally, 11β-HSD1 overexpression has been associated with hypertension and metabolic disorders.

AIM
To compare the serum concentrations of cortisol and cortisone, and urinary concentrations of cortisol (F), cortisone (E), tetrahydrocortisol (THF) and tetrahydrocortisone (THE) between very preterm (VPT) born and at term school-aged children, all adequate for gestational age (AGA).

METHOD

- In this cross-sectional study, 69 very preterm (<32 gestational weeks) and 42 full-term (>37 gestational weeks) school-aged children (4.9 to 8.9 years old) were included.
- Urine and serum samples were collected in the early morning after an overnight fasting period.
- All the metabolites were measured by mass spectrometry.

RESULTS
Serum cortisol and cortisone had similar profiles in both groups. Therefore, the serum F/E ratios are alike.

In the urinary samples analysis, cortisone had a higher concentration in VPT (p-value=0.01), but the groups maintain an equivalent urinary F/E ratios. In the case of urinary tetrahydro-metabolites, THE was higher in children born VPT, and the THF/THE ratio was lower in the same group.

CONCLUSIONS
School-aged children born very preterm adequate for gestational age had similar profiles of serum cortisol and cortisone compared to children born at term. However, at the renal level, the VPT children showed higher concentrations of cortisone and THE, an inactive metabolite, that could be caused by a disbalance between 11β-HSD1 and 11β-HSD2 function, with a predominance of 11β-HSD2. The disbalance could reflect an initial protective mechanism in preterm-born children to prevent blood hypertension in the first years of life.

REFERENCES

CONTACT INFORMATION
1. Alejandro Martínez-Aguayo alemarti@med.puc.cl
2. Gonzalo Domínguez-Menéndez gadoming@uc.cl