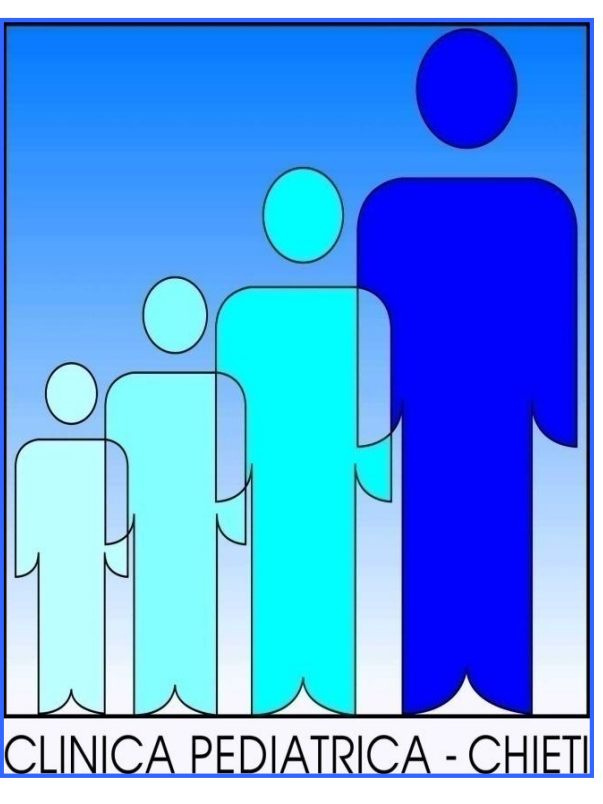


Iron metabolism disorder in prepubertal obese children with and without NAFLD

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Background

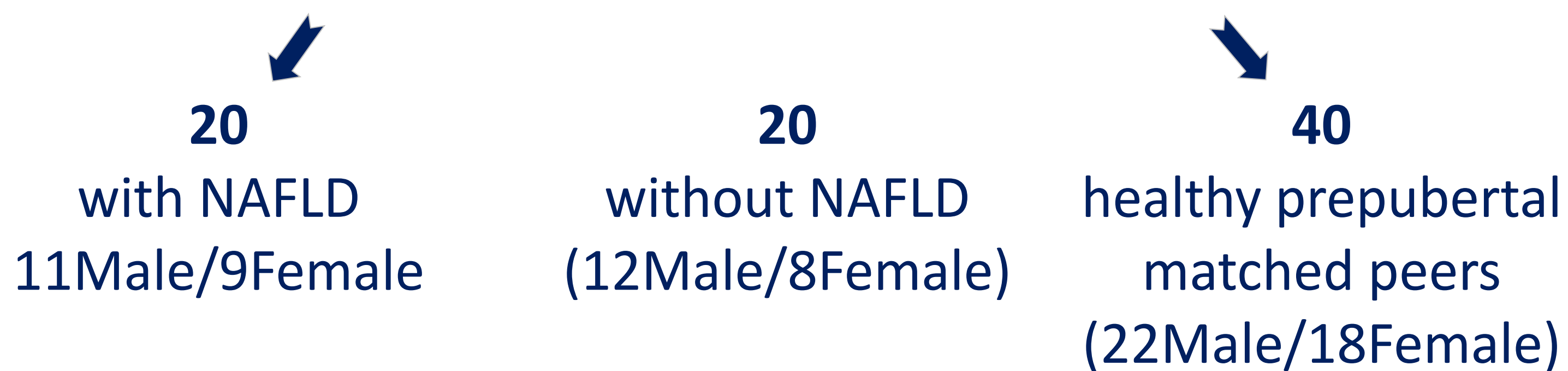
Childhood obesity is associated with non-alcoholic fatty liver disease (NAFLD). Previous studies in obese adult and pubertal children with NAFLD have shown that chronic inflammation/oxidative stress and insulin resistance might induce iron metabolism disorders, characterized by increased Hepsidin and Ferritin levels and decreased serum Iron levels. However, data evaluating these findings in a well selected population of obese prepubertal children are still missing.

Aims of the study

- ❖ we aimed to characterize iron metabolism in a group of 40 obese prepubertal children with and without NAFLD defined by ultrasonography, compared to 40 healthy prepubertal age- and gender matched peers
- ❖ we also investigated correlations between iron metabolism and both oxidative stress and metabolic markers

Materials and methods

STUDY POPULATION 80 prepubertal children



- ❖ Anthropometric measurements were determined
- ❖ Fasting blood samples were collected for measurement of insulin, glucose, lipid profile
- ❖ HOMA-IR was calculated as Insulin Resistance Index
- ❖ ALT, AST and iron profile including iron concentration, ferritin and hepcidin
- ❖ Lag-phase and MDA were evaluated as markers of oxidative stress

Statistical Analysis

- ❖ All values were expressed as means and SD
- ❖ Differences across the three groups were evaluated by One-way Anova test
- ❖ Post-hoc assessment was calculated by Bonferroni test
- ❖ In obese subjects a Pearson's correlation was used for searching correlations between Hepsidin and other parameters
- ❖ Significant values $P < 0.05$

Results¹

	Lean Healthy Controls	Obese without NAFLD	Obese with NAFLD	p*
Number	40	20	20	
Age (years)	8.8±1.9	8.9±1.6	9.1±1.9	NS
Sex (M/F)	22/18	12/8	11/9	NS
Weight (Kg)	30.7±18.8	48.5±13.1	56.4±15.2	<0.01; †‡
BMI (Kg/m ²)	17.8±3.6	25.1±3.4	27.6±4.3	<0.01; †‡
WC (cm)	57±5	78±12	86±11	<0.01; †‡§
SBP (mmHg)	103±9	110±15	112±13	0.03; ‡
DBP (mmHg)	59±5	63±8	63±8	NS

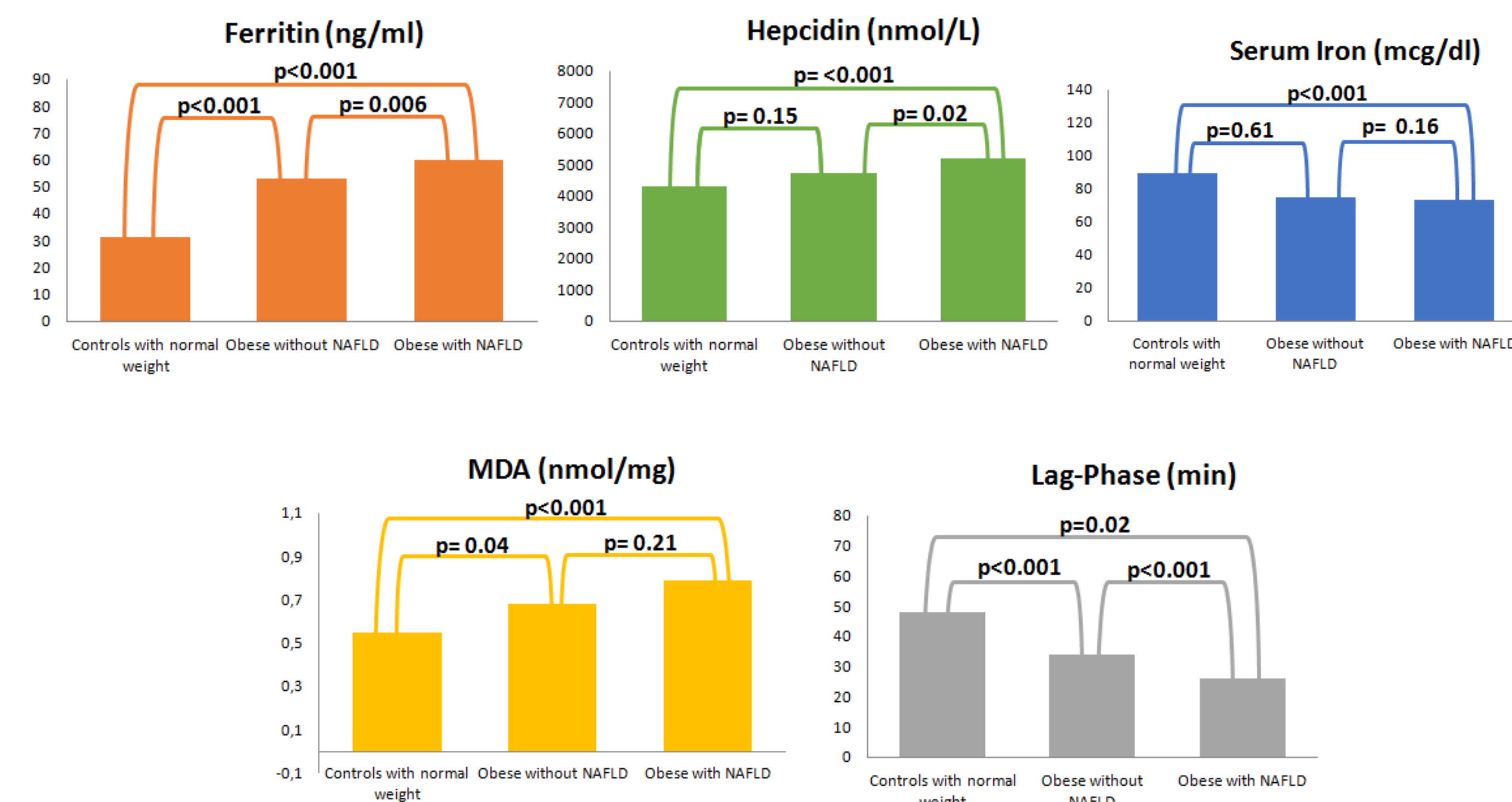
Obese without NAFLD versus Lean Healthy Controls[†]
Obese with NAFLD versus Lean Healthy Controls[‡]
Obese without NAFLD versus Obese with NAFLD[§]

Results²

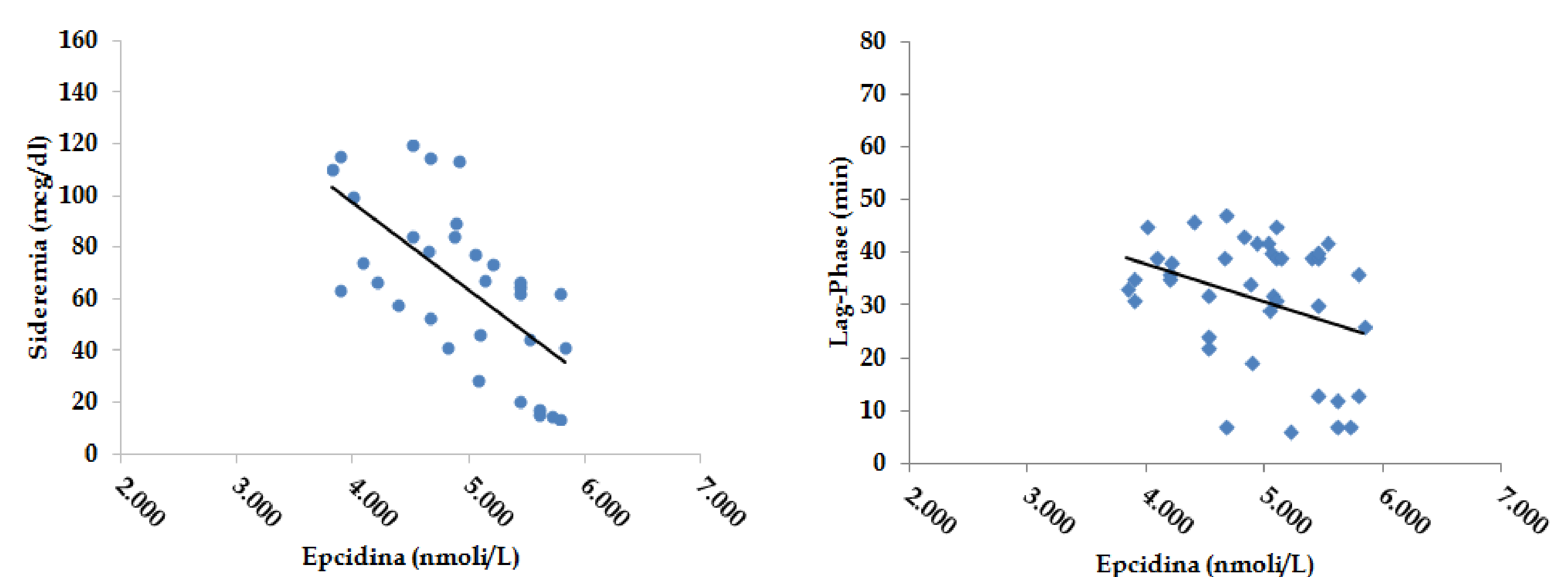
	Lean Healthy Controls	Obese without NAFLD	Obese with NAFLD	p*
LIPID PROFILE:				
Cholesterol total(mg/dl)	110±10	151±26	156±19	<0.01; †‡
TG (mg/dl)	80±8	87±65	87±45	<0.01; †‡
HDL (mg/dl)	51±8	45±10	45±6	0.02; ‡
INSULIN-RESISTANCE:				
Insulin (μU/mL)	5.8±1.5	12.1±7.0	13.7±8.7	0.03; †‡
Glycaemia (mg/dl)	83±12	84±9	86±8	NS
HOMA-IR	1.26±0.50	2.75±2.40	3.01±1.58	0.03; †‡
HEPATIC FUNCTION:				
ALT (U/L)	24±7	27±5	36±9	<0.01; †‡
AST (U/L)	22±5	27±8	37±26	0.01; †‡

Obese without NAFLD versus Lean Healthy Controls[†]
Obese with NAFLD versus Lean Healthy Controls[‡]
Obese without NAFLD versus Obese with NAFLD[§]

Results³



Results⁴



Conclusions

Obese prepubertal children show impaired iron metabolism disorders, especially in those subjects with NAFLD. The correlation between Hepsidin levels and increased oxidative stress activity in obese prepubertal children suggest a role of these components in the early pathogenesis of NAFLD in prepubertal children.