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Circulating MOTS-c levels are decreased in obese male children and adolescents and associated with insulin resistance

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Background and Aims

A novel bioactive peptide, mitochondrial-derived peptide (MOTS-c), has recently attracted attention as a potential prevention or therapeutic option for obesity and type 2 diabetes mellitus (T2DM). MOTS-c profiles have not yet been reported in human obesity and T2DM. We aimed to determine circulating MOTS-c levels in obesity and explore the association between MOTS-c levels and various metabolic parameters.

Methods

In this case-control study, 40 obese children and adolescents (27 males) and 57 controls (40 males) were recruited in the Hubei Province of China in 2017. Circulating MOTS-c levels were measured using ELISA, clinical data (e.g., glucose, insulin and lipid profile) were recorded, and anthropometric measurements were performed. Finally, we investigated correlations between MOTS-c levels and related variables.

Results

- MOTS-c levels were significantly decreased in the obese group compared with the control group (472.61 ± 22.83 ng/mL vs. 561.64 ± 19.19 ng/mL, $p < 0.01$).
- After classification by sex, MOTS-c levels were significantly decreased in obese male children and adolescents compared to their counterparts (465.26 ± 24.53 ng/mL vs. 584.07 ± 21.18 ng/mL, $p < 0.001$), while they were comparable between the obese and healthy female subjects (487.89 ± 49.77 ng/mL vs. 508.85 ± 38.76 ng/mL, $p > 0.05$).
- Further, MOTS-c levels were negatively correlated with body mass index (BMI), BMI standard deviation score, waist circumference, waist-to-hip ratio, fasting insulin level, HOMA-IR, and HbA1c in the male cohort.

Conclusions

Circulating MOTS-c levels were decreased in obese male children and adolescents and correlated with markers of insulin resistance and obesity. Although the role of MOTS-c as a treatment for obesity and diabetes in humans will requires further investigation, it is possible that a decline in MOTS-c might be a biomarker of insulin resistance during childhood obesity.

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The authors confirm that there is no conflict of interests associated with this manuscript.

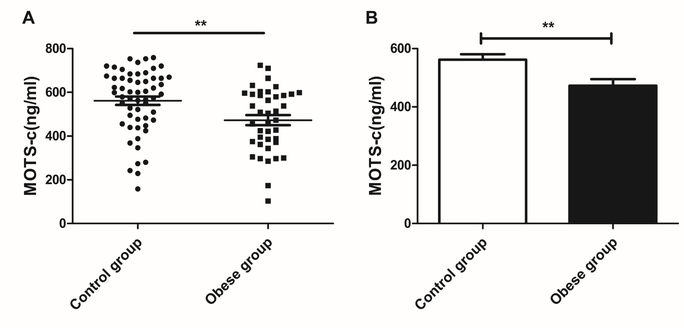

Fig. 1. Circulating MOTS-c levels were decreased in obese children or adolescents (A, B). Plasma concentrations of MOTS-c in the control group (n= 57) and obese group (n=40). Data are shown as mean ± SEM. $^{**}p < 0.01$.

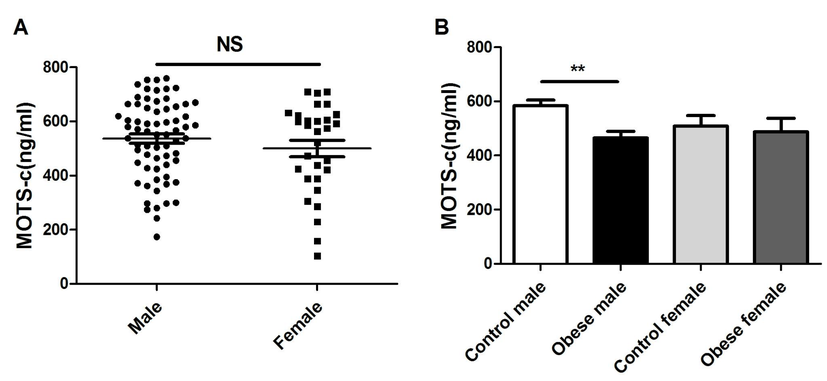

Fig. 2. Comparison of plasma MOTS-c concentration between sexes: (A) Plasma concentrations of MOTS-c in the all-male cohort (n= 67) and all-female cohort (n=30); (B) Comparison of MOTS-c concentration among male control (n= 40), obese male (n= 27), control female (n = 17), and obese female (n= 13) groups. Data are shown as mean ± SEM. $^{**}p < 0.01$; NS, no significance.

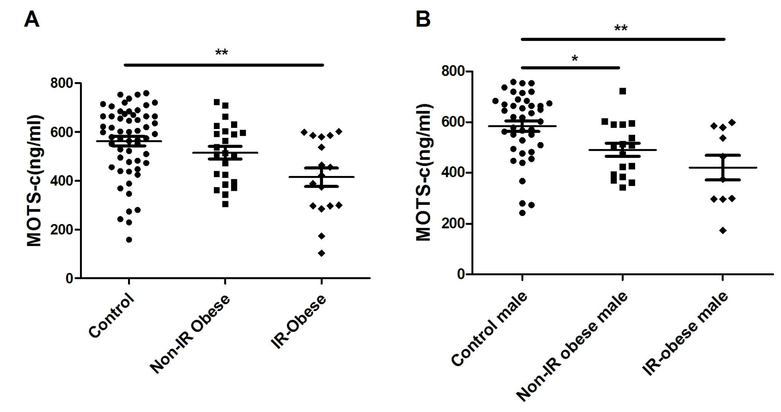

Fig. 3. Circulating MOTS-c levels were significantly decreased in the insulin-resistant obese male group. (A) The comparison of plasma MOTS-c concentrations among the control group (n= 67), the non- IR obese group (n = 23), and the IR-obese group (n = 17). (B) The comparison of plasma MOTS-c concentrations among the control male group (n= 40), the non- IR obese male group (n = 17), and IR-obese male group (n = 10) are shown as mean ± SEM. $^{*}p < 0.05$, $^{**}p < 0.01$.

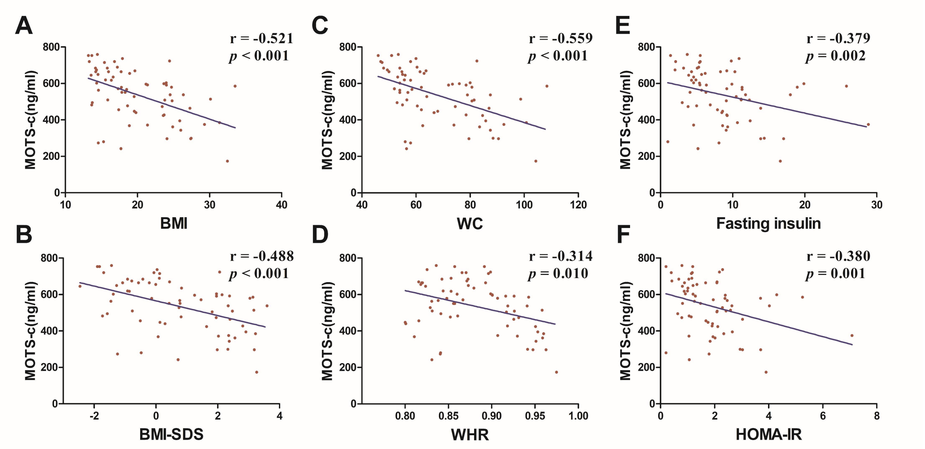

Fig. 4. MOTS-c concentrations were negatively correlated with body mass index (BMI), BMI-SDS, waist circumference (WC), waist-to-hip ratio (WHR), fasting insulin levels, and HOMA-IR of male children and adolescents. (A) BMI, r = -0.521, $p < 0.001$; (B) BMI-SDS, r = -0.467, $p < 0.001$; (C) WC, r = -0.559, $p < 0.001$; (D) WHR, r = -0.314, $p = 0.010$; (E) fasting insulin levels, r = -0.331, $p = 0.006$; and (F) HOMA-IR, r = -0.344, $p = 0.004$.


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