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Effects of diabetes family history and exercise training on the expression of adiponectin and leptin and their receptors

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The daughters of patients with diabetes have reduced insulin sensitivity index (ISI) scores compared with women with no family history of diabetes, but their ISI increase more in response to exercise training⁽¹⁾. The present study aimed to determine whether differences between these groups in exercise-induced changes in circulating adiponectin and leptin concentrations and expression of their genes and receptors in subcutaneous adipose tissue (SAT), could explain differences in the exercise-induced changes in ISI between women with and without a family history of diabetes.

The participants were female offspring of patients with type 2 diabetes (*n* 34, age 35.6 (95% CI 33.2, 38.0) years, BMI 28.1 (95% CI 26.4, 29.8) kg/m²) and matched controls with no family history of diabetes (*n* 36, age 33.6 (95% CI 31.6, 35.6) years, BMI 27.3 (95% CI 25.8, 28.8) kg/m²). Blood and abdominal SAT samples were obtained at baseline and after 7 weeks of endurance exercise.

At baseline no significant differences were observed between groups in circulating leptin or adiponectin concentrations or SAT expression of their genes or receptors. In response to exercise plasma leptin decreased more in offspring than controls and leptin receptor (LEPR) long mRNA increased significantly only in the offspring (Table). Leptin mRNA decreased similarly in both groups. Furthermore, changes in plasma leptin (*r* -0.432, *P*<0.001) and leptin mRNA (*r* -0.298, *P* = 0.019) correlated significantly with ISI changes. Plasma adiponectin decreased similarly in both groups, but no significant changes were observed in adiponectin-related gene expression (ADIPOQ, ADIPOR1 and 2). In multiple regression analyses changes in the adiponectin and leptin 'systems' (i.e. changes in circulating concentrations, SAT expression of the genes and their receptors and fat mass) explained 22.8% and 47.4% of the variance in the exercise-induced ISI change respectively.

	All subjects (<i>n</i> 62)			Controls (<i>n</i> 34)			Offspring (<i>n</i> 28)			<i>P</i>
	Mean	95% CI	<i>P</i> _‡	Mean	95% CI	<i>P</i> _§	Mean	95% CI	<i>P</i> _§	
ISI*	16.7	7.1, 27.2	<0.001	7.3	-3.0, 18.7	0.613	29.2	12.4, 48.5	0.001	0.035
Fat mass*	-15.2	-20.5, -9.6	<0.001	-12.1	-18.7, -4.8	0.022	-13.8	-18, -9.4	0.004	0.556
Adiponectin†	-13.8	-18.0, -9.4	<0.001	-15.2	-20.5, -9.6	<0.001	-12.1	-18.7, -4.8	0.007	0.477
Leptin*†	-19.5	-28.0, -10.0	<0.001	-7.3	-19.1, 6.2	0.725	-32.2	-42.5, -20.1	<0.001	0.005
ADIPOQ	-4.6	-12.3, 3.9	0.314	-6.8	-16.1, 3.6	0.633	-1.8	-14.6, 12.8	0.992	0.558
ADIPOR1	7.6	-1.0, 17.0	0.083	5.4	-3.9, 15.5	0.802	10.4	-4.7, 28.0	0.408	0.585
ADIPOR2	0.6	-5.8, 7.5	0.853	0.7	-8.1, 10.2	0.999	0.6	-8.8, 11.0	0.999	0.998
Leptin	24.9	-33.7, -14.9	<0.001	-25.0	-37.7, -9.7	0.008	-24.7	-36.1, -11.3	0.022	0.975
LEPRlong	21.0	3.8, 41.0	0.011	7.7	-13.0, 33.3	0.892	39.4	12.9, 72.1	0.026	0.101
LEPRshort	9.1	-2.5, 22.1	0.102	1.7	-9.3, 14.1	0.996	18.8	-3.1, 45.6	0.186	0.179

*Values reported previously⁽¹⁾, but included for comparison. †Plasma levels. Results were analysed by repeated measures ANOVA: ‡exercise; §*post-hoc* exercise; ||group × exercise interaction.

The findings show that exercise training has differing effects on leptin-related variables between women with and without a family history of diabetes and indicates that the effects of exercise training may differ at the molecular level between different population groups.

1. Barwell ND, Malkova D, Moran CN *et al.* (2008) *Diabetologia* **51**, 1912–1919.