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An inpatient lifestyle-change programme improves heart rate recovery in overweight and obese children and adolescents (LOGIC Trial)

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Abstract

Background: Impaired heart rate recovery (HRR) is a strong predictor of overall mortality and cardio-metabolic risk. This study aimed at investigating (1) the effect of participation in a lifestyle-change programme for weight loss on HRR in overweight and obese children and (2) potential associations between the changes in one minute HRR (HRR_1) and fitness, weight loss and cardio-metabolic risk.

Methods: The analysis included 429 individuals (169 boys) aged 13.9 ± 2.3 years who participated in an inpatient weight loss programme for four to six weeks. At baseline and the end of the programme clinical investigations were performed, including blood analyses, blood pressure, anthropometry and maximal cycle ergometer exercise testing with continuous heart rate (HR) monitoring. HRR was calculated as the difference between the highest exercising HR and HR at one, three and five minutes post-exercise.

Results: Average body weight decreased from 90.7 ± 22.5 kg to 81.9 ± 20.0 kg and peak exercise capacity increased from 1.66 ± 0.38 W/kg to 2.05 ± 0.45 W/kg ($p < 0.001$). Cardio-metabolic risk factors improved (waist circumference, LDL-cholesterol, HOMA insulin ratio, blood pressure; $p < 0.05$). HDL-cholesterol and triglyceride levels remained unchanged. Compared with baseline, at follow-up the decline in HR was more pronounced (+32%, +18% and +11% for HRR_1, HRR_3 and HRR_5; $p < 0.001$). Improvements in HRR_1 were weakly correlated with changes in exercise capacity ($p < 0.05$; $r < 0.13$), but not with changes in body weight and cardio-metabolic risk factors.

Conclusions: HRR considerably improved after an inpatient weight loss programme in overweight and obese children. This was not associated with improvements in body weight and cardio-metabolic risk; hence HRR would be a valuable addition to cardiovascular risk assessment in this group.

Keywords

Cardiovascular diseases, exercise testing, inpatients, overweight, weight loss

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Introduction

Heart rate recovery (HRR) refers to the decrease in heart rate (HR) after a specific time (usually one minute) after cessation of peak exercise. In adults, delayed one minute HRR (HRR_1), an indicator of decreased vagal activity and increased renin–angiotensin-sympathetic system activity, has been shown to be a strong predictor of cardiovascular disease and overall mortality.[1,2] Therefore, increasing rate of HRR_1 seems to have an effect on reducing cardiovascular disease risk.

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In addition, HRR has been observed to be inversely associated with cardiovascular and metabolic risk factors.[3,4] These relationships are likely present from an early age onwards. For instance, inverse cross-sectional correlations have been discovered with systolic blood pressure, serum triglycerides and serum C-reactive protein in healthy children and adolescents.[5] Furthermore, heart rate recovery may be related to adiposity. Several cross-sectional studies have reported a negative relationship between HRR and body weight, waist circumference and body mass index (BMI) in both adults[3,4] and children.[5,6] Accordingly, in overweight adults, HRR has been shown to improve as a result of participation in a three-month outpatient weight loss programme.[7,8] Prado and co-workers carried out a comparable study in children.[9] They investigated the effect of a four-month outpatient exercise and diet versus diet only treatment involving 30 obese children and found an improvement in HRR after weight loss, which could be contributed to exercise therapy.[9]

To our knowledge, this is the first study to evaluate the effect of a short-term (four to six weeks) inpatient lifestyle-change programme for weight loss focusing on a calorie restricted diet, adequate physical activity and behavioural therapy on HRR in a large sample of overweight and obese children and adolescents, and to investigate longitudinal associations between HRR and cardio-metabolic risk parameters. The primary hypothesis was that HRR improves from baseline to follow-up. In addition, we hypothesized that changes in one minute HRR are related to changes in exercise capacity, body weight and cardio-metabolic risk factors.

Methods

Participants

Six- to 19-year-old overweight and obese children and adolescents (hereafter referred to as children) who attended an inpatient lifestyle-change programme for weight loss and participated in the LOGIC trial between 2006 and 2008 (Long-term effects of lifestyle intervention in Obesity and Genetic Influence in Children; clinicaltrials.gov NCT01067157[10]) were asked to take part in exercise testing with heart rate monitoring. Participants of the LOGIC-trial were recruited consecutively once a month upon their arrival at the clinic. All children with a BMI $\geq$90th percentile[11] who arrived at the centre while the organizers were on-site were eligible unless they suffered from secondary obesity and syndromal disorders or monogenetic diseases such as the Prader–Willi syndrome. Children with complete HR datasets both at the start and the end of treatment were included into the presented analysis. All children and at least one legal guardian gave informed consent to participate in the study. The study has been conducted according to the declaration of Helsinki (Seoul, 2008) and was approved by the medical ethics committee of the Technical University Munich, Germany.

Weight loss programme

The standardized non-pharmacological weight loss programme has been described in detail before.[10] It focused on a calorie-restricted balanced diet, an increase in physical activity and behavioural counselling and has been conducted by an interdisciplinary team of paediatricians, exercise physiologists, dieticians, psychologists and pedagogues according to German guidelines for inpatient weight loss programmes (AGA, Arbeitsgemeinschaft Adipositas im Kindes- und Jugendalter).[11]

The children were required to participate in theoretical and practical lessons on healthy eating, physical activity and behaviour change skills based on the cognitive-behavioural theory. In addition, they were offered an optimized balanced diet prepared according to current guidelines (30%, 15% and 55% of the total energy content from fat, proteins and carbohydrates, respectively), with an allowed energy intake of 1200–1800 kcal/day, depending on body height and sex. The exercise therapy consisted of approximately 11 hours of structured and supervised moderate-intensity physical activity per week, that is therapeutic sports, swimming, hiking, group sports as well as strength and posture training, where children were encouraged to increase intensity over time, while the duration of the exercise programme did not change. Moreover, children had six hours of recreational non-structured exercise such as walking to town, individual playing and excursions.

Measurements

All measurements were conducted at the start (baseline) and at the end of inpatient treatment (follow-up) by trained medical staff according to standardized procedures. Symptom-limited peak exercise testing was performed by an incremental exercise test on a cycle ergometer (Jaeger ERGOSTEST ER 900) to volitional exhaustion. The first stage of exercise testing was set at a load (Watt) of half of the participants' sex, age and height related reference body weight,[12] and the load was increased by the starting load every second minute until volitional exhaustion. The participants pedalled at a cadence of 60 to 70 revolutions per minute. In the first three minutes post-test, the participants were instructed to continue pedalling with a resistance of 20 Watts to promote recovery, and for

the remainder of recovery they sat motionless. Heart rate was measured with an HR monitor and chest transmitter (Polar Favor, Polar Heart Rate Monitor, Polar Electro Oy, Kempele, Finland) and recorded pre-exercise while sitting on the bike without pedalling. It was also recorded at peak exercise and at one, three and five minutes post-exercise. Blood pressure was measured at the same time points at the right brachial artery using an appropriate size cuff and the Riva-Rocci method. Children were asked to refrain from exhausting exercise on the day prior to and the day of the exercise test. Heart rate recovery was calculated as the difference between peak HR and HR at one, three and five minutes post-exercise.

Body height and weight were measured in underwear to the nearest 0.5 cm with a rigid stadiometer and to the nearest 0.1 kg with a digital scale (Tanita BC-420 P MA Profi, Tanita Europe BV). Body mass index was calculated and transformed into a BMI standard deviation score (BMI-SDS) according to formulae developed by Cole and co-workers and reference values of German children were used.[12,13] Waist circumference was assessed during exhalation with a tape measure to the nearest 0.1 cm, midway between the lower rib margin and the iliac crest.

Blood sampling and metabolic risk factors

Blood samples were taken after an overnight fast. After centrifugation (Hettich EBA 3 S Type 2007) samples were stored at –80°C. Triglycerides were measured by the enzymatic endpoint method (*COBAS INTEGRA 6000 C*, Roche Diagnostics GmbH, Mannheim, Germany), total cholesterol by the cholesterol oxidase esterase and peroxidase method, low- and high-density lipoprotein (LDL and HDL) cholesterol were measured with the direct polyethylene glycol method. Plasma glucose was measured by the hexokinase method (*COBAS INTEGRA 800,* Roche Diagnostics GmbH, Mannheim, Germany) and serum insulin by enzyme-linked immune sorbent assay (*Mercodia*, Uppsala, Sweden). HOMA insulin ratio (HOMA-IR) was calculated as the product of insulin (fasting, µl/ml) and glucose (fasting, mmol/l) divided by 22.5.

Statistical analysis

Descriptive summary statistics were calculated using means and standard deviations. For normally distributed data, paired sample *t*-tests were performed to examine changes from pre- to post-measurement and independent *t*-tests were carried out to analyse sex differences. For skewed distributed data Wilcoxon signed rank tests and Mann–Whitney U-tests were used. Partial correlation analyses were carried out to determine the relationship between HRR_1 and the variables of interest (cross-section, i.e. at baseline and at follow-up, and change from baseline to follow-up). Cross-sectional correlations were adjusted for age and sex. Correlations of changes were adjusted for age, sex, HRR_1 at baseline and time to follow-up. Significance was assumed if $p < 0.05$. Statistical analysis was performed using SPSS 11 software for Mac OS X (SPSS Inc.®, Chicago, IL).

Results

Participants

As shown in Figure 1, 510 of 1478 children who started weight loss treatment at the clinic between 2006 and 2008 participated in the LOGIC-trial. The children who did not participate either arrived when the staff of the Centre for Prevention and Sports Medicine was not on site (due to monthly recruitment into the study but children arriving every second week to start treatment; $n = 772$), were not interested in study participation ($n = 192$) or they did not meet the inclusion criteria ($n = 4$).

In total, 429 children (169 boys) had complete HR datasets and were included into this analysis. Their mean age was 13.9 ± 2.3 years and they were mostly of Caucasian ethnicity (98%). Eleven children took medication for either hypertension ($n = 9$) or type 2 diabetes mellitus ($n = 2$). Of those, 338 children had complete blood sample datasets. Children with complete HR datasets did not differ from the whole LOGIC-collective ($n = 510$) in age, anthropometry, blood pressure, relative exercise capacity, pre-exercise HR and HRR at both baseline and follow-up and there were no meaningful differences in the mean values and correlations of sub-group analyses including the 338 children with complete blood sample datasets and the intent-to-treat analyses (back to baseline) performed with the whole group ($n = 429$).

Mean differences

Over the intervention period, which had an average follow-up time of 5.4 ± 0.9 weeks, mean body weight decreased from 90.7 ± 22.5 kg to 81.9 ± 20.0 kg ($p < 0.001$). Accordingly, waist circumference and BMI-SDS declined ($p < 0.001$; Table 1). Relative exercise capacity increased by 23% (1.66 ± 0.39 W/kg to 2.05 ± 0.45 W/kg; $p < 0.001$).

Compared with baseline, at follow-up HR was lower at every point of measurement, that is pre-exercise, throughout exercise testing and post-exercise ($p < 0.001$; Table 1 and Figure 2). Pre-exercise HR decreased from baseline to follow-up by on average

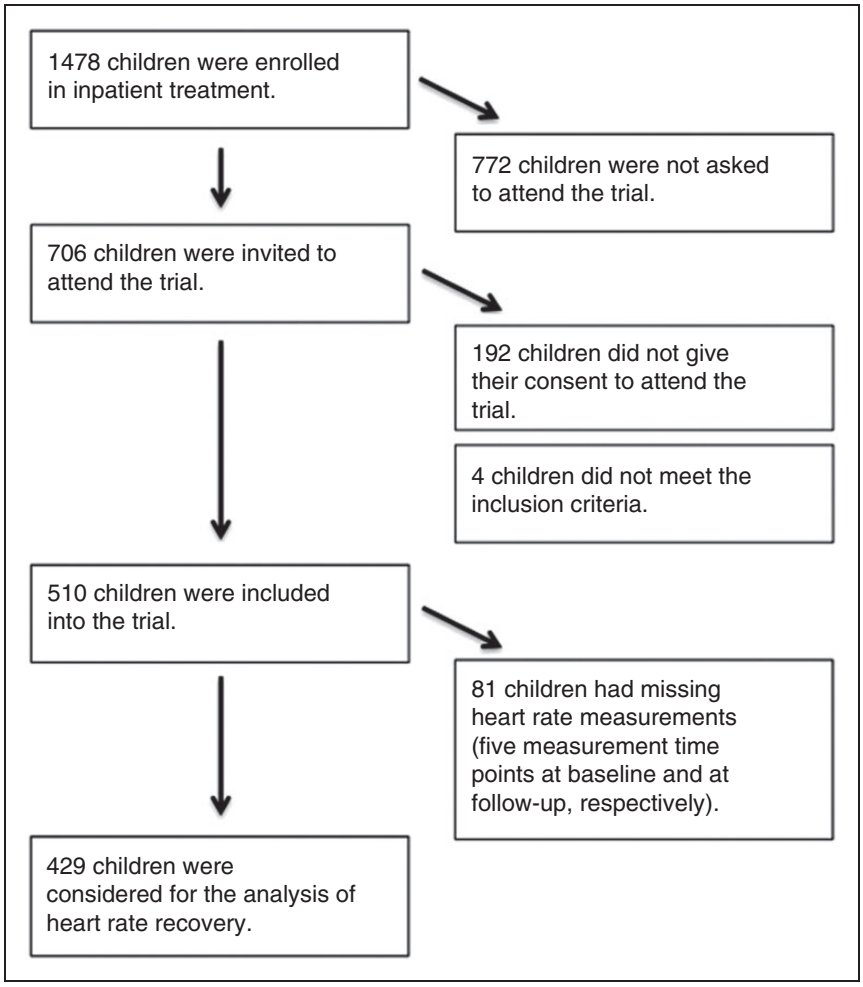

Figure 1. Flow-chart of study recruitment.

13 bpm (from 97 ± 14 bpm to 84 ± 13 bpm) and peak heart rate decreased by 7 bpm (from 183 ± 13 bpm to 176 ± 13 bpm; both $p < 0.001$). Heart rate recovery improved by 32% (from 28 ± 10 bpm to 37 ± 11 bpm) at minute one, by 18% (from 49 ± 12 bpm to 58 ± 13 bpm) at minute three and by 11% (from 70 ± 12 bpm to 78 ± 14 bpm) at minute five post-exercise ($p < 0.001$). In total, 82% of the children improved their HRR. Heart rate reserve increased by 7%; from 86 ± 16 bpm at baseline to 92 ± 16 bpm at follow-up ($p < 0.001$).

As also presented in Table 1, systolic blood pressure was between 2% and 8% lower at every measurement time point at follow-up compared with baseline ($p < 0.01$).

Mean blood values of the whole study group were in a normal range at both baseline and follow-up, except for HOMA-IR, which was above 2 in girls, a value which is considered to be a first indication for insulin resistance.[14] Nevertheless, levels of total cholesterol, LDL-cholesterol and HOMA-IR improved during the intervention ($p < 0.05$).

Correlation analyses

Both at baseline and at follow-up, HRR_1 was weakly correlated with relative exercise capacity, pre-exercise HR and peak HR (follow-up only) ($p < 0.01$, r always ≤ 0.3) as well as with body weight and BMI-SDS (baseline only) ($p < 0.05$, r always ≤ 0.2). There were no significant (total cholesterol and LDL) or weak correlations (systolic blood pressure, diastolic blood pressure at baseline, HDL and HOMA-IR ($p < 0.05$, r always ≤ 0.2)) with metabolic risk factors.

The changes in HRR_1 from baseline to follow-up were weakly correlated with the changes in relative and absolute exercise capacity ($p = 0.007$ and 0.016; $r = 0.133$ and 0.119; 95% confidence interval (CI) 0.04–0.22 and 0.03–0.21) but not significantly with body weight and BMI-SDS ($p = 0.224$ and 0.179;

Table 1. Basic characteristics of the study sample ($n = 429$)

	Total			Boys			Girls		
	Baseline	Follow-up	$p_{(change)}$	Baseline	Follow-up	$p_{(change)}$	Baseline	Follow-up	$p_{(change)}$
Age, years	13.9 (2.3)			13.6 (2.3)			14.1 (2.2)		
Body weight, kg	90.7 (22.5)	81.9 (20.0)	*<0.001*	92.1 (26.1)	82.0 (23.3)	*<0.001*	90.0 (19.5)***	81.9 (17.6)***	*<0.001*
Waist circumference, cm	97.0 (12.6)	88.2 (10.7)	*<0.001*	100.5 (13.4)	90.0 (11.6)	*<0.001*	94.6 (11.3)	87.0 (9.9)	*<0.001*
BMI-SDS	2.77 (0.52)	2.39 (0.52)	*<0.001*	2.67 (0.44)	2.26 (0.51)	*<0.001*	2.84 (0.56)*	2.48 (0.61)*	*<0.001*
Peak exercise capacity, W	148 (42)	163 (42)	*<0.001*	159 (50)	173 (51)	*<0.001*	141 (34)***	157 (33)***	*<0.001*
Peak exercise capacity, W/kg	1.66 (0.39)	2.05 (0.45)	*<0.001*	1.76 (0.44)	2.16 (0.51)	*<0.001*	1.60 (0.34)**	1.97 (0.39)**	*<0.001*
HR pre-exercise, bpm	97 (14)	84 (13)	*<0.001*	98 (14)	83 (12)	*<0.001*	97 (13)	84 (13)	*<0.001*
HR peak, bpm	183 (13)	176 (13)	*<0.001*	183 (14)	176 (14)	*<0.001*	183 (12)	176 (13)	*<0.001*
HR at 1' recovery, bpm	155 (16)	139 (16)	*<0.001*	153 (17)	137 (16)	*<0.001*	155 (16)	141 (16)*	*<0.001*
HR at 3' recovery, bpm	134 (14)	118 (14)	*<0.001*	132 (15)	117 (13)	*<0.001*	135 (14)*	118 (14)	*<0.001*
HR at 5' recovery, bpm	113 (13)	98 (14)	*<0.001*	112 (14)	97 (14)	*<0.001*	114 (12)	99 (14)	*<0.001*
RR_{sys} pre-exercise, mmHg	122 (14)	116 (12)	*<0.001*	126 (15)	117 (13)	*<0.001*	120 (12)**	114 (11)**	*<0.001*
RR_{dia} pre-exercise, mmHg	79 (9)*	75 (9)*	*<0.001*	81 (10)	74 (9)	*<0.001*	78 (9)	75 (9)	*<0.001*
RR_{sys} peak, mmHg	171 (23)	168 (23)	*<0.01*	172 (24)	170 (25)	*<0.01*	170 (22)	167 (20)*	*<0.01*
RR_{dia} peak, mmHg	82 (10)	79 (9)	*<0.001*	83 (12)	80 (10)	*<0.001*	81 (10)	79 (9)	*<0.001*
Triglycerides, mg/dl	64.3 (25.9)	69.4 (29.9)	*<0.001*	62.1 (31.7)	59.6 (25.8)	*>0.05*	67.6 (25.6)**	77.1 (30.6)***	*<0.001*
Cholesterol, mg/dl	155.6 (32.0)	134.0 (26.5)	*<0.001*	157.7 (34.5)	128.9 (25.4)	*<0.001*	154.1 (30.0)	137.9 (25.2)***	*<0.001*
LDL, mg/dl	101.0 (32.9)	78.3 (24.0)	*<0.001*	103.1 (34.6)	74.2 (24.2)	*<0.001*	98.8 (31.1)	81.2 (22.5)***	*<0.001*
HDL, mg/dl	50.5 (12.6)	50.0 (12.2)	*>0.05*	50.0 (12.7)	50.5 (12.6)	*>0.05*	50.4 (12.3)	49.2 (11.8)	*<0.05*
HOMA-IR	2.08 (1.26)	1.91 (1.05)	*<0.05*	1.83 (1.03)	1.71 (2.09)	*<0.05*	2.25 (1.36)**	2.09 (1.06)**	*<0.05*

Data are presented as means and standard deviations. Sex differences are indicated by: $^{*}p < 0.05$, $^{**}p < 0.01$ and $^{***}p < 0.001$. BMI-SDS: body mass index standard deviation; HR: heart rate; RR_{sys}: systolic blood pressure; RR_{dia}: diastolic blood pressure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; HOMA-IR: HOMA insulin ratio.

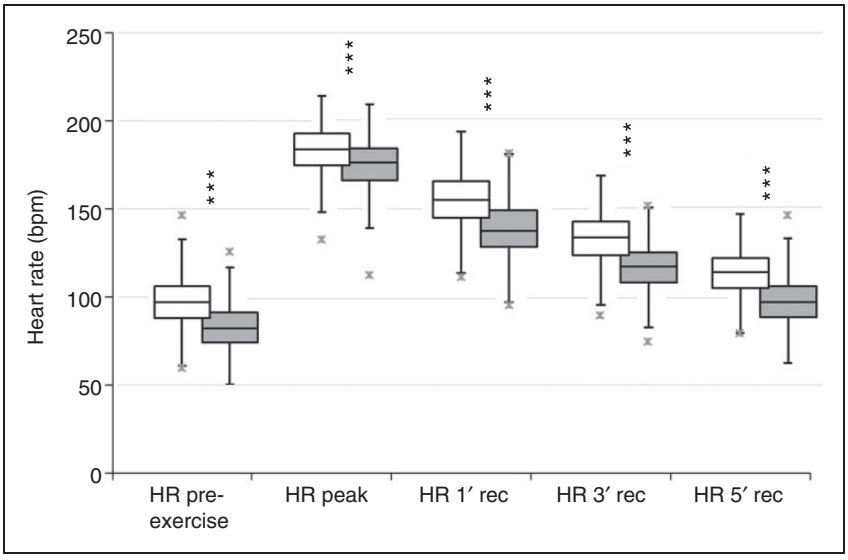

Figure 2. Box plots demonstrating the heart rate time trend before (white) and after the weight-loss programme (grey). Group differences are indicated by $^{***}p < 0.001$; HR: heart rate; 1', 3' and 5' rec: 1 min, 3 min and 5 min recovery.

Table 2. Partial correlations between the change in one minute heart rate recovery and the change in presented cardio-metabolic risk factors

	All		
	Mean change	*p*-value	95% CI
Waist circumference, cm	−8.5 (5.7)	0.182	−0.033; 0.175
RR_{sys}, mmHg	−6.6 (11.2)	0.596	0.068; 0.12
RR_{dia}, mmHg	−4.6 (9.7)	0.213	−0.033; 0.155
Cholesterol, mg/dl	−21.6 (22.7)	0.816	−0.093; 0.119
LDL, mg/dl	−22.8 (22.2)	0.715	−0.086; 0.126
HDL, mg/dl	−0.61 (7.2)	0.001*	0.074; 0.28
HOMA-IR	−0.17 (1.0)	0.663	−0.082; 0.13

The improvement in HRR_1 was 8.3 (12.2) bpm. The correlation models are adjusted for age, sex, HRR_1 at baseline and time to follow-up; $^*r = -0.18$; CI: confidence interval; RR_{sys}: systolic blood pressure; RR_{dia}: diastolic blood pressure; LDL: low-density lipoprotein; HDL: high-density lipoprotein; HOMA-IR: HOMA insulin ratio.

$r = 0.060$ and 0.067; 95% CI −0.03 to 0.15 and −0.03 to 0.16). Correlation analyses between the changes in HRR_1 and in cardio-metabolic risk factors are presented in Table 2. There were no significant correlations between HRR_1 and any of the cardio-metabolic risk parameters ($p > 0.05$), except for a weak inverse association with HDL-cholesterol ($p = 0.001$; $r = -0.18$; 95% CI 0.07 to 0.28).

Discussion

This is the first study to evaluate the effect of an inpatient lifestyle-change programme for weight loss on HRR in a large group of children and adolescents and to investigate the associations between change in HRR and changes in fitness, body weight and cardio-metabolic risk parameters. We found that HRR significantly improved after only one to 1.5 months of treatment, which is of great clinical relevance as an enhanced HRR has been associated with lower cardio-metabolic risk in both adults and children and reduced mortality in adults.[1,3–5,15–18] Average HRR during the entire five minutes post-exercise improved by about 10 beats per minute from baseline to follow-up, which is an enhancement of 11 to 32%. Interestingly, HRR was faster in our participants at both baseline and follow-up than in an investigation involving children of a similar age group by Lin and co-workers.[5] This is likely due to the higher peak HRs reached during exercise testing in the current study (approximately 30 bpm higher). Larger deviations from peak HR due to exercise lead to larger HRR,

independent of other potential physiological mechanisms.[19] Importantly, the greatest improvement in HRR was observed at minute one in recovery. HRR_1 has been suggested to have a strong prognostic power in predicting mortality and closely reflects vagal activity post-exercise,[1,20] which has been suggested to drive the reduction in HR immediately after peak exercise, whereas later in recovery sympathetic withdrawal becomes increasingly important in controlling HR.[21] Hence, the increased HRR_1 achieved in our study might reflect an increase of parasympathetic activity and a potential reduction in cardiovascular risk.

Noteworthy, relative exercise capacity improved during the programme in 91% of the children by an average of 23%. Somewhat surprisingly, there was only a weak longitudinal association between HRR and exercise capacity, which would indicate that this considerable enhancement in fitness was not a main determinant of the improvement in HRR. This observation is in line with findings by Brinkworth et al.,[8] who reported a significant improvement in HRR but no changes in either physical activity levels or peak aerobic capacity during their three-month weight loss intervention based on calorie restriction and involving healthy overweight and obese men.[8] The authors also found that the change in HRR was significantly correlated with the change in adiposity, which is in agreement with another study involving adults,[22] but not confirmed by our data and a further study involving children and adolescents.[9] In adults, weak correlations were also observed between the changes in HRR and cardiovascular risk factors such as the diastolic blood pressure and triglyceride levels from pre- to post-intervention.[8] We did not observe such correlations, although almost all of the measured cardio-metabolic parameters improved considerably from baseline to follow-up. For example, systolic blood pressure was elevated in 50% of the participants at baseline. After the relatively short intervention it was between 2% and 8% lower at every measurement time point of testing so that the prevalence of elevated blood pressure was only 16% at the end of the programme. No changes were observed in HDL levels, although fitness did improve substantially. This might potentially be explained by the effect of a decreased fat intake counteracting the effects of increased PA volume. Importantly, HDL levels were normal at baseline and remained normal throughout the investigation.

The lack in longitudinal correlations may be explained by differences in inter-individual responses to this lifestyle programme regarding weight loss, improvement of metabolic risk factors and change in cardiopulmonary regulation as reflected by our large standard deviations (see Tables 1 and 2) and also observed in a previous lifestyle intervention.[23–25] This may be explained by differing baseline characteristics and to some extent by genetic determination.[24,26,27] In addition, it might be that relationships are stronger in adults who have had risk factors for a metabolic syndrome for a longer period of time and in whom the symptoms would likely also be more present.[4]

A limitation of the study is that many of the participants seem not to have achieved maximal HR levels (see Table 1), which is likely to be due to the children not being used to this exercise testing protocol and for many exercising in general. This might explain the slightly lower peak HR at follow-up compared with baseline.[28] The study did not include a control group. Nevertheless, it is unlikely that in these children factors other than the changes in lifestyle, for instance growth or maturation, would have provoked comparable physiological changes in this short period of time. Furthermore, up to now it is unknown to what extent improvements in HRR_1 in children may reduce future risk of cardiovascular disease or all-cause mortality. However, there is strong evidence from the NHANES study that autonomic nervous function is linked to metabolic risks, even at ages 12–19.[5] It is very likely that the 16 bpm (32%) improvement in HRR_1 in the current study represents a positive effect on autonomic regulation and therefore on risk factors for cardiovascular disease.

To summarize, HRR significantly improved in children and adolescents after participation in a short-term inpatient lifestyle-change programme for weight loss. This was accompanied by considerable improvements in body weight, exercise capacity and cardio-metabolic risk factors. Improved HRR is related to improved parasympathetic function, whereas attenuated HRR is an independent predictor of all-cause mortality. Therefore, the findings of the current study add further support for a great benefit of lifestyle-change programmes for overweight and obese children and adolescents. Moreover, the improvement in HRR was weakly or not correlated with changes in body weight, fitness and cardio-metabolic risk factors and HRR would be a valuable addition to cardiovascular risk assessment in obese children.

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Conflict of interest

None declared.

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