

Original Article

Cardiorespiratory fitness and physical activity in youth with type 2 diabetes

Shaibi GQ, Faulkner MS, Weigensberg MJ, Fritschi C, Goran MI.
Cardiorespiratory fitness and physical activity in youth with type 2 diabetes.
Pediatric Diabetes 2008; 9: 460–463.

Objective: The increased incidence of type 2 diabetes (T2D) among youth is hypothesized to be due, in part, to low levels of fitness and activity. Therefore, the purpose of this investigation was to examine whether cardiorespiratory fitness and physical activity are reduced in youth with T2D compared with overweight controls.

Participants: Thirteen adolescent boys with previously diagnosed T2D (mean duration 2.4 ± 1.8 yr) were matched for age and body mass index to 13 overweight, non-diabetic controls.

Methods: Cardiorespiratory fitness was assessed during a progressive exercise test to volitional fatigue and physical activity was estimated from a 7-d physical activity recall.

Results: Youth with T2D reported performing ~60% less moderate to vigorous physical activity compared with their non-diabetic counterparts (0.6 ± 0.2 vs. 1.4 ± 0.3 h/d, $p = 0.04$). Furthermore, diabetic youth exhibited significantly lower cardiorespiratory fitness levels compared with controls (28.7 ± 1.6 vs. 34.6 ± 2.2 mL/kg/min, $p < 0.05$).

Conclusions: These findings support the hypothesis that cardiorespiratory fitness and physical activity are reduced in youth with T2D. Whether reduced fitness and activity contributed to the pathophysiology of the disorder cannot be determined from the cross-sectional analysis.

Longitudinal studies are warranted to examine whether improvements in fitness and increased physical activity can prevent the development of T2D in high-risk youth.

**Gabriel Q Shaibi^a,
Melissa S Faulkner^b,
Marc J Weigensberg^c,
Cynthia Fritschi^d and
Michael I Goran^{e,f}**

^aCollege of Nursing & Healthcare Innovation, Arizona State University, Phoenix, AZ, USA; ^bCollege of Nursing, University of Arizona, Tucson, AZ, USA; ^cDepartment of Pediatrics, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA; ^dDepartment of Medical-Surgical Nursing, University of Illinois at Chicago, Chicago, IL, USA; ^eDepartment of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA; and ^fDepartment of Physiology and Biophysics, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

Key words: exercise – fitness – pediatrics – type 2 diabetes – youth

Corresponding author:
Gabriel Q. Shaibi, PhD, PT
College of Nursing & Healthcare Innovation
Arizona State University
500 N 3rd Street
Phoenix, AZ 85004
USA.
Tel: 602-496-0909;
fax: 602-496-0921;
e-mail: gabriel.shaibi@asu.edu

Submitted 18 February 2008. Accepted for publication 13 March 2008

Pediatric obesity has reached epidemic proportions in many industrialized nations (1). In parallel with the increase in obesity among youth, type 2 diabetes (T2D) has emerged as a critical health condition in this population (2). As in adults, peripheral insulin resistance is thought to be a critical underlying component of T2D in youth (3, 4). Although several factors (i.e., puberty, obesity, and ethnicity) are associated with insulin

resistance in youth, reduced levels of physical activity and cardiorespiratory fitness may be contributory (5, 6).

Low cardiorespiratory fitness is associated with the development of impaired fasting glucose and T2D in adults (7). Whether the same predictive relationship holds true for younger populations has not been established. To date, very limited empirical data are

available describing cardiorespiratory fitness and/or physical activity in children with T2D and no studies have directly evaluated fitness or activity in children with and without T2D. Therefore, the objective of the current report was to compare cardiorespiratory fitness and physical activity between youth with T2D and non-diabetic controls matched for age, gender, and adiposity.

Research design and methods

Subjects

Thirteen adolescent males (aged 13–18 yr) with previously diagnosed T2D (mean duration 2.4 ± 1.8 yr, mean hemoglobin A1c = $8.4 \pm 0.8\%$) were examined in the General Clinical Research Center (GCRC) at the University of Illinois at Chicago (UIC). These youth were pair matched for age and body mass index (BMI) to 13 non-diabetic overweight males examined in the GCRC at the University of Southern California (USC). Written informed consent and assent were given by parents and children, and the studies were approved by the respective institutional review boards.

Procedures

Specifics regarding the study procedures have been presented elsewhere (8, 9). Briefly, height and weight were recorded to the nearest 0.1 cm and 0.1 kg, respectively, and BMI was subsequently calculated. An oral glucose tolerance test was administered in the non-diabetic subjects to confirm both fasting and 2-h glucose values were not in the diabetic range (10). Cardiorespiratory fitness (VO_{2peak}) was assessed during a progressive exercise test to volitional fatigue on an electronically braked cycle ergometer. Breath-by-breath respiratory gases were collected and measured through open-circuit spirometry and analyzed on either a SensorMedics® (Yorba Linda, CA) VMAX29 (UIC) or a MedGraphics® (St. Paul, MN) CardiO2 combined exercise system (USC). The exercise protocols were designed to elicit test termination between 8 and 12 min. Tests were terminated when the participant was unable to continue pedaling despite verbal encouragement from research staff. Heart rate was measured continuously throughout the test using an integrated electrocardiogram. VO_{2peak} was determined from the highest 20-s average achieved with the respiratory exchange ratio (RER) >1.0 . Physical activity was determined by the 7-d physical activity recall and is expressed as hours per day of moderate to vigorous activity (11).

Statistics

Descriptive characteristics between youth with and without T2D were examined by independent sample *t*

test. Analysis was performed using SPSS VERSION 15.0 (SPSS Inc., Chicago, IL, USA) with a type I error set at $p < 0.05$. Data presented are means \pm SE.

Results

Descriptive characteristics of the participants are presented in Table 1. No significant differences were noted in age, height, weight, or BMI. Furthermore, no significant differences were found in either peak heart rate or peak RER. However, T2D youth exhibited significantly lower fitness levels compared with their overweight non-diabetic counterparts (Fig. 1) despite similar peak workloads (200.8 ± 10.6 vs. 192.5 ± 11.7 W, $p = 0.680$). Furthermore, diabetic youth performed significantly less moderate to vigorous physical activity than non-diabetic youth (0.6 ± 0.2 vs. 1.4 ± 0.3 h/d, $p = 0.04$).

Discussion

T2D is an emerging epidemic in young people. It is hypothesized that increases in adiposity secondary to a sedentary lifestyle may be a contributory factor (12). To date, an absolute paucity of data exist comparing cardiorespiratory fitness in youth with and without T2D. To this end, we found that fitness was $\sim 18\%$ lower in adolescent males with T2D compared with age and BMI-matched controls. We also found that diabetic youth spent $\sim 60\%$ less time per day in moderate to vigorous activities compared with their non-diabetic counterparts. This is especially troubling given the fact that youth diagnosed with diabetes often receive education on the importance of increased physical activity for diabetes management.

Our data extend previous findings in adults that have shown that T2D is associated with reduced cardiorespiratory fitness and a sedentary lifestyle (7). Others have shown that fitness tends to be lower in overweight, severely insulin resistant, adolescents compared with adiposity-matched moderately insulin-resistant controls (13). It is thought that lower fitness levels are

Table 1. Characteristics of boys with and without T2D

	T2D	Controls	p Value
Age (yr)	16.4 ± 0.6	15.2 ± 0.5	0.13
Tanner stage	4.2 ± 0.3	4.7 ± 0.1	0.16
Ethnicity	8 AA/5 Hisp	13 Hisp	—
Height (cm)	176.0 ± 2.4	168.4 ± 2.8	0.06
Weight (kg)	99.2 ± 7.9	91.9 ± 5.3	0.45
BMI (kg/m^2)	31.7 ± 1.8	32.3 ± 1.6	0.80
Peak HR (bpm)	180.7 ± 3.6	190.3 ± 4.0	0.09
Peak RER	1.2 ± 0.1	1.2 ± 0.3	0.35
VO_{2peak} (L/min)	3.1 ± 0.2	2.7 ± 0.1	0.14

AA, African American; bpm, beats per minute; Hisp, Hispanic; HR, heart rate; RER, respiratory exchange ratio. Data presented are means \pm SE.

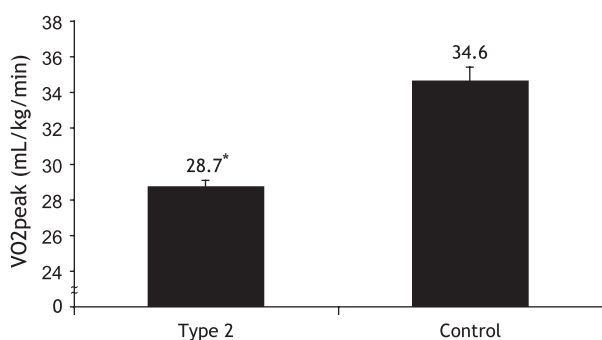


Fig. 1. Cardiorespiratory fitness and type 2 diabetes. Means \pm SE of VO₂peak in youth with (type 2) and without (control) type 2 diabetes. * $p < 0.05$.

indicative of, among other things, impairments in oxidative capacity of skeletal muscle (14). Skeletal muscle oxidative capacity is a significant predictor of insulin resistance in adults with T2D and therefore may be important in the pathogenesis of the disorder (15). The lower fitness observed in youth with diabetes may be an indication of early defects in the metabolic capacity of skeletal muscle, that is, mitochondrial dysfunction.

A secondary mechanism that may explain the reduced fitness levels in the diabetic youth relates to circulatory defects that limit oxygen delivery to exercising muscle. Studies in adults have found that diabetics have an inadequate oxygen uptake response relative to increases in exercise workload (16). As a result, oxygen consumption does not meet the demands at increasing exercise intensities. Therefore, lower VO₂ peak may be an indication of a compromised oxygen delivery system in conjunction with mitochondrial dysfunction in youth with T2D (17). While it would be expected that peak workloads would concomitantly be reduced with lower a VO₂peak, we found that diabetics attained similar peak work levels to controls. This finding is consistent with adult patients with cardiovascular disease who exhibit a greater reliance on anaerobic energy pathways to maintain high-power outputs when oxygen delivery is limited (18). While no studies to date have examined these issues in younger populations, adults with cardiovascular disease exhibit a similar pattern of endothelial dysfunction as T2D, which may indicate a common pathophysiologic link related to reduced fitness and vascular disease (19).

Beyond fitness, physical activity (especially moderate to vigorous) is a potent stimulator of glucose uptake in skeletal muscle (20). As such, the less active lifestyle observed in the diabetic youth may have contributed to their eventual diabetes diagnosis through a mechanism that is independent of aerobic capacity. In adolescents, fitness and activity are only moderately associated (21) which suggests that activity may mediate diabetes risk through a secondary pathway, for example, adiposity.

In the end, it is more than likely that the pathogenesis of T2D in youth is an end result of a complex interplay between genetic and environmental factors rather than exclusively because of lower fitness and/or a sedentary lifestyle (22). However, we did attempt to minimize some of the potentially confounding variables by matching participants by gender, age, and BMI in addition to including an insulin-resistant population of youth as controls.

Despite the above-mentioned attempts, there are limitations to our study that warrant mention. We cannot exclude the possibility that differences in body composition, fat distribution, or other intrinsic factors contributed to the observed findings. Youth with diabetes were a mixed ethnic group of African American and Hispanics, whereas the controls were exclusively Hispanics. We have previously observed that fitness is not significantly different between these minority groups, but we did not assess physical activity in that study (23). Therefore, it is possible that the ethnic makeup of our groups may have contributed to some of the observed differences in fitness and activity. Youth were recruited from different geographic sites with distinct seasonal and built environments, which may also impact physical activity patterns. Last, despite similar exercise protocols and calibration procedures, differences in the study site personnel and equipment may have influenced the overall results. The sum of the aforementioned limitations suggest that these data be interpreted as preliminary in nature with the impetus for future studies to build upon our results through incorporating more diverse samples of youth in terms of ethnicity and gender and better control for potential confounding effects of the environment. These limitations notwithstanding, there remains a dearth of available information in youth with T2D. The current report builds upon previous work and extends the scientific knowledgebase regarding this growing population of youth.

In conclusion, we found that both cardiorespiratory fitness and physical activity are lower in T2D adolescent males compared with their non-diabetic counterparts matched for age and BMI. Whether reduced fitness and activity contributed to the pathophysiology of the disorder cannot be determined from the cross-sectional analysis. Longitudinal studies are warranted to examine whether improvements in fitness and increased physical activity can prevent the development of T2D in high-risk youth.

Acknowledgements

We are grateful the participants and their families for their involvement as well as the GCRC staff at UIC and USC. This work was supported by the Thrasher Research Fund (02817-1), the USC Center for Interdisciplinary Research, USC GCRC (M01 RR 00043), National Institute of Nursing Research, (R01 NR07719), and UIC GCRC (M01-RR-13987).

References

1. LOBSTEIN T, BAUR L, UAUY R. Obesity in children and young people: a crisis in public health. *Obes Rev* 2004; 5: 4–85.
2. GROUP SfDiYS. The burden of diabetes mellitus among US youth: prevalence estimates from the SEARCH for Diabetes in Youth Study. *Pediatrics* 2006; 118: 1510–1518.
3. ELDER DA, PRIGEON RL, WADWA RP, DOLAN LM, D'ALESSIO DA. β -cell function, insulin sensitivity, and glucose tolerance in obese diabetic and nondiabetic adolescents and young adults. *J Clin Endocrinol Metab* 2006; 91: 185–191.
4. DRUET C, TUBIANA-RUFI N, CHEVENNE D, RIGAL O, POLAK M, LEVY-MARCHAL C. Characterization of insulin secretion and resistance in type 2 diabetes of adolescents. *J Clin Endocrinol Metab* 2006; 91: 401–404.
5. KU CY, GOWER BA, HUNTER GR, GORAN MI. Racial differences in insulin secretion and sensitivity in prepubertal children: role of physical fitness and physical activity. *Obes Res* 2000; 8: 506–515.
6. KASA-VUBU JZ, LEE CC, ROSENTHAL A, SINGER K, HALTER JB. Cardiovascular fitness and exercise as determinants of insulin resistance in postpubertal adolescent females. *J Clin Endocrinol Metab* 2005; 90: 849–854.
7. WEI M, GIBBONS LW, MITCHELL TL, KAMPERT JB, LEE CD, BLAIR SN. The association between cardiorespiratory fitness and impaired fasting glucose and type 2 diabetes mellitus in men. [erratum appears in *Ann Intern Med* 1999 Sep 7;131(5):394]. *Ann Intern Med* 1999; 130: 89–96.
8. FAULKNER MS, QUINN L, RIMMER JH, RICH BH. Cardiovascular endurance and heart rate variability in adolescents with type 1 or type 2 diabetes. *Biol Res Nurs* 2005; 7: 16–29.
9. SHAIPI GQ, CRUZ ML, BALL GD et al. Effects of resistance training on insulin sensitivity in overweight Latino adolescent males. *Med Sci Sports Exerc* 2006; 38: 1208–1215.
10. AMERICAN DIABETES ASSOCIATION. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2005; 28: S37–S42.
11. SALLIS JF, BUONO MJ, ROBY JJ, MICALE FG, NELSON JA. Seven-day recall and other physical activity self-reports in children and adolescents. *Med Sci Sports Exerc* 1993; 25: 99–108.
12. ALBERTI G, ZIMMET P, SHAW J et al. Type 2 diabetes in the young: the evolving epidemic: the international diabetes federation consensus workshop. *Diabetes Care* 2004; 27: 1798–1811.
13. BACHA F, SAAD R, GUNGOR N, ARSLANIAN SA. Are obesity-related metabolic risk factors modulated by the degree of insulin resistance in adolescents? *Diabetes Care* 2006; 29: 1599–1604.
14. LARSON-MEYER DE, NEWCOMER BR, HUNTER GR, HETHERINGTON HP, WEINSIER RL. ³¹P MRS measurement of mitochondrial function in skeletal muscle: reliability, force-level sensitivity and relation to whole body maximal oxygen uptake. *NMR Biomed* 2000; 13: 14–27.
15. BRUCE CR, ANDERSON MJ, CAREY AL et al. Muscle oxidative capacity is a better predictor of insulin sensitivity than lipid status. *J Clin Endocrinol Metab* 2003; 88: 5444–5451.
16. REGENSTEINER JG, SIPPEL J, McFARLING ET, WOLFEL EE, HIATT WR. Effects of non-insulin-dependent diabetes on oxygen consumption during treadmill exercise. *Med Sci Sports Exerc* 1995; 27: 661–667.
17. REGENSTEINER JG. Type 2 diabetes mellitus and cardiovascular exercise performance. *Rev Endocr Metab Disord* 2004; 5: 269–276.
18. HANSEN JE, SUE DY, OREN A, WASSERMAN K. Relation of oxygen uptake to work rate in normal men and men with circulatory disorders. *Am J Cardiol* 1987; 59: 669–674.
19. YU HI, SHEU WH, LAI CJ, LEE WJ, CHEN YT. Endothelial dysfunction in type 2 diabetes mellitus subjects with peripheral artery disease. *Int J Cardiol* 2001; 78: 19–25.
20. HOUMARD JA, TANNER CJ, SLENTZ CA, DUSCHA BD, MCCARTNEY JS, KRAUS WE. Effect of the volume and intensity of exercise training on insulin sensitivity. *J Appl Physiol* 2004; 96: 101–106.
21. MORROW JR, FREEDSON PS. Relationship between habitual physical activity and aerobic fitness in adolescents. *Pediatr Exerc Sci* 1994; 6: 315–329.
22. HUANG TTK, GORAN MI. Prevention of type 2 diabetes in young people: a theoretical perspective. *Pediatr Diabetes* 2003; 4: 38–56.
23. SHAIPI GQ, BALL GD, GORAN MI. Aerobic fitness among Caucasian African-American, and Latino youth. *Ethn Dis* 2006; 16: 120–125.